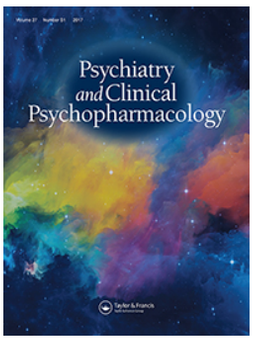

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Mine Elagoz Yuksel MD, Mahmut Cem Tarakçıoğlu MD, Ali Güven Kılıçoğlu MD, Ümit Işık MD, Bulent Bahçeci MD, Rümeysa Yeni Elbay MD, Kader Semra Karataş MD, Şahin Bodur MD, Özge Şahmelikoğlu Onur, Alişan Burak Yasar MD, Merve Çıkılı Uytun MD, Lale Gönenir Erbay MD, Mustafa Yıldız MD, Özlem Özcan MD, Erol Göka MD, Özlem Özcan MD, Rukiye Ay MD, Sevda Bağ MD, Ömer Yanartaş MD, Ömer Faruk Demirel MD, Musa Tosun MD, Ayse Sakallı Kani MD, Hatice Altun MD, Kemal Utku, Yazici MD, H. Tuna Çak MD, Ipek Percinel, Yazici MD, Birim Günay Kılıç MD, Necati Uzun MD, Halil Kara MD, Gamze Ergil Altın MD, Fatma Hülya Çakmak MD, Gamze Ergil Altın MD, Rabia Yılmaz MD, Miray Çetinkaya MD, Sencan Sertçelik MD, Özhan Yalçın MD, Özhan Yalçın MD, Esra Porgalı Zayman MD, Nilfer Şahin MD, Funda Gümüştaş MD, Ebru Çengel Kültür MD, Serhat Ergün MD, Serhat Nasıroğlu MD, Sadettin Burak Açıkel MD, Özden Şükran Üneri MD, Hesna Gül MD, Özden Şükran Üneri MD, Murat Altın MD, Ferda Korkmaz Özkanoğlu MD, Zeynep Esenkaya Usta MD, Gözde Narin Coşkun MD, Aynur Görmez MD, MRCPsych, Kadir Coşkun MD, Hatice Doğan MD, Yasemin Yulaf MD, Hasan Turan Karatepe MD, Arzu Çalişkan Demir MD, Hasan Turan Karatepe MD, Neşe Yorguner Küpeli MD, Hasan Turan Karatepe MD, Arzu Çalişkan Demir MD, Hatice Güneş MD, Özge Kılıç MD, Ahmet Gül MD, Sevinc Ulusoy MD, Ali Evren Tufan MD, Sümer Öztanrıöver MD, Mesut Yildiz MD, Mesut Yildiz MD, Didem Öztop MD, Bengi Semerci MD, Vahdet Gormez MD, MRCPsych, Filiz Şükrü Gürbüz MD, Gülşen Teksin Ünal MD, Ömer Faruk Akça MD, M. Kayhan Bahalı MD, Dilek Sarıkaya Varlık MD, Oğuz Tan MD, Sarper Taşkıran MD, Sarper Taşkıran MD, Şükrü Kartalcı MD, Sevgi Özmen MD, Esra Demirci MD, Erol Göka MD, Özgür Çağla Cenker MD, Mehmet Fatih Ceylan MD, Cagdas Oyku Memis MD, Selim Polat MD, Selma Tural Hesapçıoğlu MD, Hatice Doğan MD, A Cenk Ercan MD, Cenk Varlik MD, A Cenk Ercan MD, Hatice Aksu MD, Kadir Coşkun MD, Bilge Doğan MD, Tuba Mutluer MD, Mustafa Solmaz MD, Can Tuncer MD, Veysi Çeri MD, Veysi Çeri MD, Didem Öztop MD, Tuğba Uyar MD, Tuğba Uyar MD, Serkut Bulut MD, Murat Kuloğlu MD, İpek Güngör MD & Rayna Mandova MD, PhD

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SYMPOSIA ABSTRACTS

9th International Congress on Psychopharmacology & 5th International Symposium on Child and Adolescent Psychopharmacology

SYMPOSIA ABSTRACT: 345

Clinical Uses of Melatonin in Children and Adolescents

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ABSTRACT
Melatonin is an indoleamine often used in children and adolescents. Melatonin is considered to be an effective clinical management for dyssomnias, sleep disorders present in children with attention-deficit hyperactivity, autism spectrum disorders, developmental delays. Quick-acting capsules, controlled-release (CR) capsules, sublingual tablets and liquid forms are available. Melatonin is generally very well-tolerated in children and adolescents. The pharmacology, therapeutic applications, and side effects of melatonin are discussed.

SYMPOSIA ABSTRACT: 371

Unmet Needs in Attention Deficit Hyperactivity Disorder: Long Term Effects of ADHD Treatment in Children and Adolescents

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ABSTRACT
ADHD has been seen widely seen and leads significant functional impairment in childhood[1]. Although stimulants have been used most frequently in ADHD treatment their long term effects did not investigate well. Methylphenidate which is the most frequently prescribed stimulant's long term side effects on growth-development, cardiovascular, psychiatric and neurological systems are very important[2]. Stimulant diversion or misuse in long term has growing up in recent years and lead important consequences on community health[3]. Attention Deficit Hyperactivity Disorder Drugs Use Chronic Effects (ADDUCE) consortium was established in 2012 and experts on ADHD, drug safety, neuropsychopharmacology and cardiology developed a programme which investigates the long term possible side effects of stimulants[2]. In this presentation, long term side effects of stimulants (methylphenidate) will be reviewed through the perspective of ADDUCE work group study results.

KEYWORDS
ADHD; long term treatment; stimulants

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SYMPOSIA ABSTRACT: 374

Clinical Presentation, Comorbidity and Differential Diagnosis in Pediatric Bipolar Disorder

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ABSTRACT

Mood fluctuations are common in daily life, but when these fluctuations is severe, persistent and has a negative result or impairment clinicians must be aware of an affective disorder[1]. Affective disorders classified as Major Depressive Disorder, Cyclothymic Disorder, Other specified bipolar and related disorder, Unspecified bipolar and related disorder, Substance or drug-induced bipolar and related disorder, Bipolar and related disorder due to another medical condition, Bipolar I and Bipolar II in The Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5) according to its extent and severity of mood elevation[2]. Manic or hypomanic episodes are states of elevated mood and increased motor drive that are finite in time and differ in severity and length[1]. A hypomanic episode is defined in DSM-5 as persisting for at least 4 consecutive days, whereas a manic episode lasts for at least 1 week. Pediatric bipolar disorder has been recognizing increasingly as having roots in early life. Despite the fact that The DSM-5 does not distinguish the adult onset bipolar disorder from the childhood or adolescent onset bipolar disorder, there are important differences in clinical presentation and prognosis. Pediatric Bipolar Disorder (BD) shares characteristics of the severe adult BD (e.g. elevated mood, grandiosity, decrease of need for sleep, flight of ideas/racing thoughts, distractibility, talkativeness than usual, Increase of intentional activity or psychomotor agitation, excessive involvement in pleasant activities) but it has differences like shorter euthymic periods between episodes, rapid changes in mood, higher psychiatric comorbidity, more impairment and manic episodes is characterized more mix or dysphoric feature along with irritability. The most common comorbid disorder in pediatric BD is Attention Deficit and Hyperactivity Disorder (ADHD) with a rate of 11–90%. The other important comorbid situations are Oppositional Defiant Disorder (ODD) (9-88), Conduct Disorder (5–69%), anxiety disorders (12–60%), Obsessive Compulsive Disorder (7–47%), suicide attempts and Substance Use Disorder (4–32%). Anxiety disorders are also important in prognosis of Pediatric BD. The comorbidity of anxiety disorders with BD is a poor prognosis predictor and treating or preventing anxiety disorders is critically important in the prognosis of pediatric BD. The other important thing that clinicians must be careful about the comorbidity of BD and ODD. If we diagnose BD, we cannot diagnose ODD as comorbidity with BD[3]. The main difficulty in diagnosing Pediatric BD is to distinguish the normal developmental period from the psychopathology. For example hyperactivity, egocentric behaviors, imaginary plays/friends must be differentiated from mania[4]. The primary differential diagnosis of pre-adolescent BD is ADHD, because of the extent overlapping symptoms. The primary distinguishing method of BD from ADHD is the episodes of BD (severe fluctuations of affect) and the symptoms of mania. The other disorder that is hard to differentiate is Disruptive Mood Dysregulation Disorder. In DMDD the irritability is chronic and non-episodic which is helpful for the differential diagnosis. Conduct disorder and BD is the diagnosis that must be differentiated as well. In BD, behavior changes starts suddenly. Pediatric BD may also be confused with Schizophrenia if the psychotic symptoms are prominent. An abrupt onset, without affective flattening or abulia, and affective familial backgrounds, would be more characteristics of Pediatric BD. We should also consider the possibility of Pediatric BD in the cases of early onset Major Depression, with psychotic symptoms, elevated psychomotor inhibition and treatment resistant[5]. Finally, we should rule out other medical or drug diseases that could mimic manic symptoms

KEYWORDS

Pediatric Bipolar; Clinical Presentation; Comorbidity; Differential Diagnosis

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SYMPOSIA ABSTRACT: 397

Oxytocin and Vasopressin in Attention-Deficit/Hyperactivity Disorder and Conduct Disorder

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ABSTRACT

The etiology of attention-deficit/hyperactivity disorder (ADHD) and conduct disorder (CD) involves complex interactions of neuroanatomical and neurochemical systems, but the exact neurobiological mechanisms underlying of these disorders are currently unknown[1,2]. In recent years, researchers have raised concerns regarding the potential roles of neuroendocrine hormones in the etiopathogenesis of ADHD and CD. Oxytocin (OT) is classically known for its role as a hormone involved in parturition and lactation. Recent studies have shown that it also has crucial effects on attachment, trust, stress management, social cognition, and memory[3]. To date, few studies have investigated the role of OT in ADHD and CD. These studies suggest a possible role for OT as a biomarker for the ADHD and CD. The vasopressin system in brain has important roles in social communication and behavior. Social communication and behavior deficits are shown in patients with ADHD and CD, but only a few studies has so far investigated the association between the vasopressinergic system and ADHD and CD[4]. This panel will present current research on the relationship between OT and vasopressin and ADHD and CD.

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SYMPOSIA ABSTRACT: 461

Use of Herbal Treatment in Sleep Disorders

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ABSTRACT

Sleep disorders are common among adults, especially in older ages and has associated daytime consequences which impair job performance and quality of life[1]. It is also associated with increased risk of comorbidities such as chronic pain, diabetes and depression[2]. Chronic lack of sleep increases the risk of getting infections and illnesses related to cardiovascular, respiratory, gastrointestinal, urinary, neurologic, endocrine-metabolic and immune systems, and may cause psychological disorders[3]. With respect to growing public interest in complementary and alternative medicine (CAM), sleep disorders are also treated with different CAM modalities. CAM encompasses a variety of disciplines that include everything from diet and exercise to mental conditioning, lifestyle changes and psychological and behavioral interventions. Natural products are the most common products used for sleep disturbances[4]. Herbal products are one of the most preferred forms of CAM and the use of herbs as sleep aids is a common practice[4]. Unfortunately there is a common misperception that herbs are natural products without any risk for health[5]. It should be kept in mind that herbs may also have side effect if used unnecessarily in high dose[5].

KEYWORDS

Sleep; herbal treatment; psychiatry

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SYMPOSIA ABSTRACT: 464

Female Patients with Sexual Dysfunction: An Experience In A General Psychiatry Outpatient Clinic

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ABSTRACT

Sexuality is an important part of human life and plays a vital role in maintaining wellbeing and health. The World Health Organization defines overall sexual health as “a state of physical, emotional, mental and social well-being in relationship to sexuality; it is not merely the absence of disease, dysfunction or infirmary. It has been emphasised that there is a relationship between sexual health problems and quality of life and that problems in relation to sexual health affect individuals and/ or society’s well-being and quality of life, causing them to be perceived as "part of life" over time. Sexual health requires a positive and respectful approach to sexuality and sexual relationships, as well as the possibility of having pleasurable and safe sexual experiences, free of coercion, discrimination and violence.” The focus here is not just on physical sexual function – are the genitals “working” – but whether the individual can be fulfilled and satisfied in their physical, emotional, and social sides of sex[1] . A satisfying sex life is also an important component of a marriage. It has been shown that spouses with sexual dysfunction have a higher rate of deteriorated marital life in comparison to the healthy control groups and[1] the Masters-Johnson model was one of the first sexual response models developed in the 1960s which applies to both men and women. According to this model, sexual response progresses predictably and linearly from excitement to plateau, orgasm, and resolution. The main focus of this model is on physical response of the genitals. Helen Singer Kaplan, a psychologist and sex therapist, noted that many individuals had problems with sexual desire, denoting the importance of desire to sexual response. In the 1970s she modified the Masters-Johnson model to a three-phase model of desire, excitement, and orgasm. From 1968 to the present day, sexual dysfunctions were included in the DSM and ICD diagnostic classifications with minor changes. However, recent researches suggest that classification was insufficient to account for sexual behavior because there is no clear distinction between arousal and the phase of sexual response in women. For this reason, changes were made in the diagnostic criteria. The most recent edition of the Diagnostic and Statistical Manual (DSM-5), states that sexual dysfunctions “are a heterogeneous group of disorders that are typically characterized by a clinically significant disturbance in a person’s ability to respond sexually or to experience sexual pleasure.” As such, “female sexual dysfunction” is an umbrella term for four distinct disorders recognized in the DSM-5: Female Orgasmic Disorder, Female Sexual Interest/Arousal Disorder (FSIAD, which encompasses what were previously termed Hypoactive Sexual Desire Disorder and Female Sexual Arousal Disorder in the DSM-IV), Genito-Pelvic Pain/Penetration Disorder (which encompasses what were previously termed vaginismus and dyspareunia), and Substance/Medication-Induced Sexual Dysfunction. To diagnose any one of these disorders, the symptoms must be (a) present at least 6 months, (b) cause clinically significant distress in the individual [not solely in the individual’s sexual partner(s)], and (c) not be better explained by another issue, such as relationship distress or other stressors. From the studies carried out in our country, , it seems that there is significant proportion of people, both men and women , in the community having sexual dysfunction. In a survey conducted by the Sexual Training and Therapeutic Research Association (CETAD) in 2006, it was found out that 65% of the individuals did not receive any health care and counselling services, 15% had no sexual problems, 5% had no active sexual life, 11% had at least one health counselling, 4% of the respondents did not answer the question. It has been reported that about 31% of men and 43% of women in our country have sexual dysfunction[3]. There is an increasing demand on

sexual treatments / therapies in the country in recent years. There could be several reasons for this increased need in this area. One could be increased awareness of sexual problems and widespread media coverage, the role it plays within the community, the significant increase in sexual treatment facilities in recent years can also count. Despite the rapid increase in the number of sexual treatment centres, outpatient clinics and specialists in our country in the recent years, the increase in number of patients has overcome this rate. All these developments require medicine and all physicians to be better equipped and prepared within this area[4]. Women's involvement in education, social life, more workforce and increasing self-awareness is continual universally and in this country with each passing day. Being able to respond to women's relevant applications in this area gains importance when considering the benefits that both they and their families will see. Unfortunately initial referral to sexual treatment centers may take 3-6 years later in Turkey. The right referrals of patients by health professionals at the first consultation have a key role in preventing unnecessary delays and accessing the appropriate services at early stage[4]. The prevalence of vaginismus in Western contexts varies from 5 to 17% among those who sought treatment for sexual dysfunctions. This prevalence is much lower than the 43–73% found in Turkey[5]. In Turkey, the most frequently encountered primary sexual dysfunction found in women seeking treatment is vaginismus, and the second one is hypoactive sexual desire disorder. Vaginismus is a condition which cannot be treated by any medication or an operation. With sexual therapy, this problem can be treated by a standard therapy of 6-10 sessions over 2-4 month with a success rate of more than 90%. It is actually the most easily treated sexual dysfunction[4]. The treatment of sexual desire disorder takes very much of an individualized approach. In most cases it requires a multifaceted management. This could sometimes be a very basic intervention such as providing the patient with information about sexuality or correcting the sexual myth that individual and his/ her partner might have or switching to another medication which is less likely to cause sexual side effects. Through these simple yet significant interventions, patients can realize that 'there is no sexual problem as such, or in some cases it is sufficient to solve the problem easily[4]. Although with appropriate interventions vaginismus and sexual desire disorders cases can achieve high success rates, they can lead to several problems in people's lives if not recognized or treated. There are still a limited number of specialists serviced for sexual dysfunction disorders in the country which makes the role of general psychiatrist even more crucial in managing these cases. Within the last 1 year , 8 cases of vaginismus , one woman with a partner with premature ejaculation , 1 man and 3 women cases with sexual desire disorder, 3 men with erectile dysfunctions presented to my outpatient clinic. The vaginismus cases were couples married from 3 weeks to 1.5 years. When they presented to the clinic, they had not had sexual intercourse yet, and this had led to a significant strain on their relation. I managed vaginismus cases and some of the sexual desire disorder cases through therapy. During the assessment process, two of the cases with desire dysfunction were diagnosed with depressive disorder and their sexual problems resolved when the depressive illness was treated. I referred the erectile dysfunction cases and one case sexual desire disorder of three Erenköy Mental Health Research and Training Hospital for specialist services. In conclusion, sexuality is an important part of human life. Sexual disorders can lead to physical, mental, and social problems if not managed appropriately. It is crucial to train psychiatrists during their specialty training on this subject so that they are skilled enough to manage some of the most common and uncomplicated cases of sexual dysfunction disorders at the early stages. Considering that more women than men are affected by sexual disorders and the higher rates of vaginismus cases in Turkey, developing policies and providing culturally sensitive/ appropriate services for woman would be a significant step on the way of solution of this major issue.

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SYMPOSIA ABSTRACT: 465

Sleep Disorders and Law

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ABSTRACT

Scientists investigate the effects of sleep disorders to social and human life that has been one of the areas of concern. Researchers revealed individual and social harms of sleep disorders with many scientific data. They have raised the question of how to prevent these losses. While individual losses of sleep disorders may be prevented with person clarification and early detection in these areas, social losses can be minimized with legal regulations. Social consequences can result of individual impacts. Sleep disorders can be caused excessive daytime sleepiness, fatigue, cognitive slowing that impairs performance, can result social harms as traffic and industrial injury. Recent years legislative amendments have been done to limit the social causes of sleep disorders. These arrangements can clarify the presence of sleep disorder with crime or caused damage that persons have criminal responsibility or not, application of driver license, industrial injury and review the person's disability rate and military service. A crime, as defined by law, is the wrongful act, which is clearly cited in the laws, where a causality between the cause of the action and its effect is established, and which is committed by an individual by being completely aware of the meaning and the outcome of his/her act. Turkish Criminal Law (TCL) states that an individual committing a crime, who is not aware of the lawful meaning and the outcome of his/her or his/her ability to control his/her act was reduced or substantially reduced due to a mental disease or a temporary reason or being under the influence of alcohol or substance taken unwillingly, will not be punished or he/she will be given abatement in punishment[2]. Turkish Civil Law, emphasizes that an individual will be regarded having legal capacity and having power of discernment unless such individual does not have capacity to act plausibly due to his/her being juvenile, being mentally disabled, having mental weakness, being under the influence of alcohol or a similar other reason. Turkish Civil Law stipulates that the acts of an individual without having power of discernment will not be legally effective. According to the article covering the cases of individuals with disability, a disabled person is: who has difficulties in adapting to social life and in managing his/her daily requirements due to lack of physical, mental, psychological, sensorial or social abilities at various degrees, whether congenial or acquired in life; and therefore is need of protection, care, treatment, counseling and support services; and who is evidenced with a medical report that he/she is at least 40% disabled to work[3]. Published general principles for individuals applying to get driving license in Turkey require now passing screening for sleep disorders during their medical examination. There may be a relation between the accident and the disability in the work accidents; its relation with the sleep-state of being awake cycle may be questioned. It is another process to assess for people with sleep disorders before being enlisted for military service whether such person is eligible for the service [1,3].

Sleep-Related Violence

Sleep disorders from legal perspective are those involve a relation with sleep disorders with sleep-related violence. Sleep-related violence is defined as a person's damaging himself or his/her environment during sleep. Sleep-related violence may be in the form of verbal, physical and sexual actions and may lead to disability from injuries or even to death. Among the sleep-related violence which mostly resulted in criminal cases are reported as NREM parasomnia, various sexomania, parasomnia occurring with alcohol drug use, REM sleep disorder (RDB), nocturnal dissociative disorder, obstructive sleep apnea syndrome (OSAS) and Münchausen syndrome[1,2,5]. The criteria for mischievous discretion of a person in case of a criminal act are cited in the TCL No.5237 as the presence of freedom of conscious and action. For a person to be kept liable for a crime he/she committed, he/she must have full capacity to understand and wish (immutability). The article 32 of the TCL requires that a person must have freedom of conscious and wish. In terms of this provision, sleepwalking may be regarded as a mental disorder and under that, the perpetrator will not be considered without mischievous discretion but rather, his/her behaviors will not be regarded actions under the legal perspective. Some legal experts emphasize that sleepwalking as a mental disorder and therefore the mischievous discretion of a person committing crime under sleepwalking is influenced temporarily, which is covered by the article 34 of the TCL [1–3]. Whether sleep disorders affect the power of discernment of a person and whether his/her actions would have legal consequences is another matter. People with hypersomnia may cause accidents in traffic and workplace which may result in injuries and death. In researches, narcoleptic patients are reported to be in high risk group and their driver performance was found insufficient compared to the control group. There may be liability both for the employee and the employer in injuries where narcoleptic person was involved and although such person's situation was known and necessary measures were not at place.[1]. The article 22/3 of the TCL No.5237 defines the negligence, which is another form of liability ex delicto. Per that, "In such as case, although the person does not deliberately, if the result occurs, there is culpable negligence. The punishment in this case shall be increased by from one third to half of the punishment envisaged for negligent crime. People may face legal consequences such as if he/she has negligent offense or if he/she can be given driver license, depending on the gravity of his/her situation[1,3]. Sleep breathing disorders may vary from simple snoring to severe obstructive sleep apnea (OSAS). OSAS, repeated partial or complete obstructive airway block occurs and that situation may have some symptoms. These symptoms are nightlong arousals, bruxism, sleepwalking, sleep-disordered movement,

hypersomnia during the day, loss of attention irritability, amnesia, loss of reflex skills , neurocognitive disorders, and these may affect the life quality of the person and may cause personal and social damages[3,5]. There are medical condition rules during the medical examination of persons willing to get driver license and people with OSAS condition should not be allowed to drive motor vehicles before fully treated. Since the accidents caused by OSAS are foreseeable and preventable, doctors have specific responsibilities for these drivers. After the diagnosis, the patient should be advised about his/her condition and the risks associated with this situation and his/her acknowledgment of them should be documented. Injuries due to foreseeable and preventable accidents may have penal consequences[1]. Insomnia is a symptom which is seen sleep disorders, in medical conditions and psychiatric diseases[1–3]. Accidents caused by insomnia are foreseeable and preventable accidents. Penal consequences may arise if a person does not take necessary measures and does not pay due care beforehand[1,2].

Sleep Disorders and Disability

Sleep disorders where disability is involved are problems with initiating, continuing the sleep, insomnia, hypersomnia, sleep-wake cycle disorder, sleep staging disorders and parasomnia. According to the international sleep disorder classification, disability rate in such disorders are: 10% for insomnia; 35% for sleep breathing disorders; 10% for circadian sleep-wake cycle disorders; 10% for parasomnia; 35% for RDB; and 10% for sleep-disordered movement. There is not sufficient data in Turkey for the number of disabled people, types and causes of disabilities[1,3].

Sleep Disorders and Military Services

Transferring the military hospitals under the administration of state hospitals introduced a responsibility for the state hospitals to decide on fitness for military service. There is not a medical condition list for sleep disorders affecting the military service. However, chronic insomnia, narcolepsy and OSAS were listed in the diseases and conditions limiting the military service. It was emphasized that these disorders should be identified with polysomnography[3].

Conclusion

Sleep experts have responsibilities for expert examinations before and during the legal processes which the people with sleep disorders were involved. People with sleep disorders should be advised about their condition and its treatment and probable situations should be identified and foreseen. Increasing our knowledge about sleep disorders and educating legal experts on the legal consequences of these disorders will help solving the legal problems where the sleep disorders are involved[1,2].

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SYMPOSIA ABSTRACT: 466

Neurobiology of Sleep

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ABSTRACT

Sleep is a fundamental biological process observed widely in the animal kingdom, but the neural circuits generating sleep remain poorly understood. Understanding the brain mechanisms controlling sleep requires the identification of key neurons in the control circuits and mapping of their synaptic connections. Technical innovations over the past decade have greatly facilitated dissection of the sleep circuits. This has set the stage for understanding how a variety of environmental and physiological factors influence sleep. The ability to initiate and terminate sleep on command will also help us to elucidate its functions within

and beyond the brain. A series of findings over the past decade has begun to identify the brain circuitry and neurotransmitters that regulate our daily cycles of sleep and wakefulness. The latter depends on a network of cell groups that activate the thalamus and the cerebral cortex. A key switch in the hypothalamus shuts off this arousal system during sleep. Other hypothalamic neurons stabilize the switch, and their absence results in inappropriate switching of behavioral states, such as occurs in narcolepsy. These findings explain how various drugs affect sleep and wakefulness, and provide the basis for a wide range of environmental influences to shape wake–sleep cycles into the optimal pattern for survival. Sleep disturbances are also common to a number of psychiatric disorders. The neural substrates of sleep and wakefulness form a highly distributed and, to some extent, redundant network, with hypocretin, monoaminergic and cholinergic systems largely promoting wakefulness and GABAergic systems in the preoptic area, hypothalamus and brainstem promoting sleep. The hypocretin/orexin system seems to play a special role in the promotion of wakefulness and suppression of REM sleep by providing excitatory input to the monoaminergic and cholinergic systems. Sleep is not a unitary state but involves a cyclic alternation between NREM and REM sleep; the pons is critical for generating the multiple components (i.e., EEG synchronization, eye movements, muscle atonia, and so forth) that characterize REM sleep. A corticothalamocortical loop plays a major role in generating SWA measured in the EEG; cortical nNOS/ NK1 neurons may be important in coordinating and/or propagating SWA within the cortex. Because the control of sleep and wakefulness involves a complex orchestration of the activity of many neural systems, it is readily apparent that many nodes for dysfunction exist that can have implications for both physical and mental health. There has been significant progress in our knowledge of the mechanisms underlying the regulation of sleep and wakefulness. Novel neurotransmitters, pathways, and receptors have been discovered to refine prior theories and define new hypotheses. For example, recent studies have pointed to mutually inhibitory pathways that regulate the switch between wakefulness and sleep, and between NREM and REM sleep, much like flip-flop switches. Similarly, the importance of the wake-promoting orexin pathway has been demonstrated in animal models, as well as in humans with narcolepsy. These new findings have provided novel targets for the development of agents to manage clinical sleep disorders such as insomnia, hypersomnia, and narcolepsy. The concept of interacting circadian and homeostatic systems in regulating sleep and wakefulness has facilitated our understanding of sleep onset and maintenance. This two-process model will undoubtedly be modified as the molecular basis of each system is further elucidated. As we improve our understanding of sleep and wake regulation, novel behavioral and pharmacologic targets for the treatment of sleep disorders will emerge to improve sleep and wake functions and mitigate the negative effects of poor sleep on health.

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SYMPOSIA ABSTRACT: 467

Mood Stabilizator Use During Pregnancy and Breastfeeding

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ABSTRACT
Despite the claim that pregnancy may improve bipolar disorder in the past years, recent studies have not supported this and have shown that postpartum period bipolar disorder development and recurrence risk significantly increases. Many studies have shown that women with bipolar disorder have more depressive/ mixed episodes during their pregnancy than episodes of hypomania and mania. Determining the course of bipolar disorder in pregnancy, symptom proSymposia Abstract, risk of suicide, and possible treatments will prevent both occurrence of these episodes and the negativities that can develop. It is important to document the effects

of discontinuing mood stabilizers. A study showed that pregnant women who discontinued mood stabilizers at this time had higher recurrence rates than those who continued to use mood stabilizers. In the same study, it was stated that cutting the drug by decreasing it gradually leads to a more positive effect in increasing risk compared than sudden interruption. In another study, it is claimed that in the case of abrupt withdrawal of mood stabilizers in pregnancy, a new attack is shown at a rate as high as 100% while this rate is 30% in the cases of continuing to use lamotrigine. The risk of teratogenicity in the use of psychotropic drugs in the first trimester and the risk of developing negativities in pregnant women with bipolar disorder and babies should be considered together at the basis of the "profit-loss" account. Breastfeeding period which is a physiological consequence of the postnatal period, is necessary for survival of the newborn. Breastfeeding reduces the risk of developing diseases such as severe lower respiratory tract infections, atopic dermatitis, asthma, acute otitis media, gastroenteritis, obesity, type 1 diabetes mellitus, childhood leukemia, sudden infant death syndrome and necrotizing enterocolitis in infants while leads to a reduction in the risk of type 2 diabetes mellitus, breast cancer, over cancer and postpartum depression in the mother. It is essential that the use of postpartum drug should be decided by considering the loss profit rate of both mother and baby. The psychiatric anamnesis of the mother should be evaluated to record the risk of recurrence of the ongoing illness, the psychiatric charts encountered in previous periods and the medicines that benefit in previous pregnancies. On the other hand, whether or not the baby is born on time, accompanying development defect should be investigated. It is obvious that any developmental/metabolic problem that may delay the excretion of the intended drug is likely to lead to unpredictable results in the baby. Institutional medical communities of different countries have informative explanations and recommendations especially for clinicians regarding drug use during pregnancy and breastfeeding. Existing classification systems lead to limitations in practical use because of not mentioning personality-specific identifiers like drug exposure, exposure periods. Classification of “L” for medicinal use in breastfeeding mothers is based on how much of the drug has gone through the mother and the possible effects on the baby. In terms of mood stabilizers, Lithium is classified as L4, while valproate and carbamazepine are classified as L2 and lamotrigine L3. Four distinct pharmacokinetic parameters are used in evaluating the effects of the drug used during breastfeeding: The half-life (T1/2), maternal plasma/protein binding, milk-plasma ratio (M / P) and oral bioavailability. However, when maternal plasma protein binding is taken into account, the fact that the drug has a high maternal plasma protein binding indicates that the mother will also undergo less at that time. M/P higher than 1.0 suggests that the drug is above the acceptable level of adverse effect in the baby. The dose to which the baby is exposed varies considerably depending on factors such as the distribution of the drug in fat, the dose used, the metabolism of the mother, the peak concentration of the drug in the milk and the bioavailability of the baby. It can be assumed that the value of the relative infant dose (RID), which is the proportion of the daily dose of medicines exposed to the body weight of the baby, is less than 10%, is considered to be relatively safe. In a study in which long-term effects of interrelated abilities of children with anti-epileptic drugs were assessed by breast-feeding, there was no adverse cognitive effect in these children who were exposed to antiepileptic drugs during pregnancy and lactation and the children who received breast milk had higher IQ and verbal capacities[1].

Lithium

The ‘benefit / safety ratio’ of lithium use in the perinatal period is still favorable to lithium compared to other mood stabilizers. Lithium can be used in the mother who is in attack or in the high risk group as soon as the illness starts before the treatment plan. In one study, women with bipolar disorder who stopped using lithium due to pregnancy had a 2.9 fold higher risk of postpartum episode than nonpregnant women. Lithium use in pregnancy has been associated with Ebstein’s anomaly. The prevalance of Ebstein’s anomaly is 1:20000. With lithium use in pregnancy, this risk increases by 20-40 times, but the absolute risk remains low. Polyuria and polydipsia have also been reported among the changes that can be seen in mothers using lithium during pregnancy. It has been reported that as the pregnancy progresses lithium excretion is accelerated in proportion to the increased glomerular filtration rate and from the beginning of the pregnancy higher doses are needed. However, it should not be forgotten that the reduction in fluid compartments, after delivery, may result in toxicity of lithium levels. Newborn lithium exposure is associated with ECG anomalies lethargy cyanosis and hypotonia[2].

Valproate

The risk of neural tube defects that may be caused by the use of valproate in pregnancy, particularly in the first trimester, varies between 5-9%. It is recommended to take oral folic acid preparations in pregnancy to reduce the risk. However, this is theoretical information and it is not known how much it is actually beneficial. Again the use of valproate in gestation has been associated with a lower level of intelligence in children. In a controlled trial, IQ levels in children with valproate during pregnancy were found to be lower by an average of 9 points compared to the control group. Thrombocytopenic purpura and anemia were reported in an infant whose mother used valproate during pregnancy and continued to use 600 mg of valproate daily during breastfeeding and this clinical table improved with the abortion of the breastfeeding[3].

Lamotrigine

It is stated that the therapeutic dose should be increased due to the increase of excretion in the later stages of pregnancy. The lack of a standard therapeutic dose of lamotrigine in clinical practice makes it difficult to follow in this way. However, due to use of lamotrigine during

pregnancy has lower risk for the fetus compared to older anti-epileptic drugs, the current drug has an advantage for this period of use. The NICE guide does not recommend prescribing lamotrigine because of the possible risk of Stevens-Johnson Syndrome in infants of women[4].

Carbamazepine

Many authors agree that carbamazepine should be preferred during pregnancy only if other possibilities are not appropriate for the patient. The women who started carbamazepine after the pregnancy, have been shown to be more susceptible to severe side effects such as agranulocytosis, liver failure, and Steven-Johnson Syndrome compared with those who are already taking carbamazepine when pregnant. It is stated that the exposure of carbamazepine in the first 8 weeks after conception is the highest risky period. Intrauterine exposure of carbamazepine may cause midline anomalies on the face of the fetus. The risk of bleeding which may develop in the newborn due to the lack of fetal vitamin K, may be reduced by 20 mg / day given to the mother in the last month of pregnancy. Cholestatic hepatitis has been reported to develop in the mother of infant using carbamazepine during pregnancy and for postpartum 5 weeks[5].

Oxcarbazepine

It was claimed that when oxcarbazepine was compared with carbamazepine, it was safer to use during pregnancy because there is no epoxy metabolism.

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SYMPOSIA ABSTRACT: 468

Fast Efficacy in the Eye Movement Desensitization and Reprocessing Treatment of Complicated Mourning Disorders

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ABSTRACT

Grief is a process that after the result of losing someone However, prolonged (>6 months) grief process could prepare the ground for pathological situation. According to DSM-5, Persistent Complex Bereavement Disorder (PCBD) is diagnosed when grief continue for the deceased over twelve months. If the experience accompanies with a traumatic event, DSM-5 Appendix recommends including Traumatic Death Specifier. In the process of PCBD, individuals shows some responses as decreasing functionality, sleeping disorders, depressed mood, guilt feelings, somatic disorders and rejection in response to the death. Several treatment ways have been used in the treatment of PCBD and one of them is Eye Movement Desensitization and Reprocessing (EMDR). In the course some cases effectiveness of time-limited EMDR treatment on PCBD diagnosed patients will be explained.

SYMPOSIA ABSTRACT: 469

Possible biomarkers in children and adolescents with conduct disorder and psychopathic traits

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ABSTRACT
Conduct disorder, antisocial personality disorder and psychopathy are often seen as developmental disorders that span the life course and the terms are sometimes used interchangeably. There are, however, significant differences between them and their associated correlates. Whereas conduct disorder and antisocial personality disorder primarily focus on behavioral problems, psychopathy, as described by Hare, emphasizes deficits in affective and interpersonal functioning[1]. However, people with these traits exhibit a more severe, violent, and chronic pattern of antisocial behavior[2]. In the past two decades, a significant body of research has emerged refining how the key features associated with psychopathy may be expressed in children and adolescents. These conceptualizations have focused largely on the presence of callous-unemotional (CU) traits (e.g., lack of empathy and guilt, failure to put for the effort on important tasks, shallow and deficient emotions), which correspond closely to the affective dimension of psychopathy—core to the construct in adult samples[3]. As a result of these studies, the most recent revision of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013) has integrated these changes into the diagnostic criteria for conduct disorder. There is evidence that the presence of CU traits prospectively predicts later incidence of aggression and violence over and above impulsive conduct-disordered (I/CP) tendencies[4]. Recognizing the CU traits in the children is important to start to intervening early of childhood-onset conduct problems were should be an important aim for preventing later serious aggression and antisocial behavior. Additionally, in older children with severe antisocial behaviors, the most successful interventions are comprehensive interventions that are tailored to the unique needs of the individual child and so, research on the different developmental pathways to conduct problems could help to guide these individualized interventions. Several studies examined the etiology of CU traits and these studies were tried to find bio markers for these traits. Specifically, genetic and neuroimaging studies are important on this issue. Catechol O-methyltransferase (COMT), monoamine oxidase-a receptor (MAO-A), serotonin transporter (5-HTT), oxytocin receptor (OSTR) gene and prolactin receptor gene (PRLR) were investigated in children and adolescents with CU traits. Majority of neuroimaging studies were focused on amygdala and frontal cortex. With some exceptions, the most of studies to date have demonstrated reduced function, volume, and connectivity in the frontal cortex and the amygdala in psychopathic adults and adolescents, two brain areas strongly implicated in prosocial behavior and decision making[5]. In this present, these studies will be detailed and the probable biomarkers will be summarized.

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SYMPOSIA ABSTRACT: 470

Microbiota and Other Psychiatric Disorders

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ABSTRACT
Gastrointestinal system (GIS) and immune system work together. GIS products can display pathogenic effect on nervous system. The relation between GIS and central nervous system remains debatable. There are some theorems suggested for their relationship. One of these is that microbiota influences nervous system via immune stimulation. In another theorem, bacterial metabolites[1] and some bacterial products joining into tryptophan metabolism[2] has been introduced as an example for the interaction between microbiota and nervous system.

Neural pathways (via vagal nerve) and intestinal hormonal response are the other pathways demonstrated concerning this topic[3]. The effect of microbiota on anxiety and depression has been studied more in animals. In one of these studies, it was demonstrated that the depression due to enhanced hypothalamo-pituitary adrenal (HPA) axis response, which was experimentally created in rats, can be reversed by administering bifidobacterium infantis. Bifidobacterium infantis is found in substantial amount both in the intestine of newborns and probiotic medications. It is called as "psychobiotic" because it shows antidepressant efficacy[4]. In a study conducted in 2010, baby rats were separated from their mothers and divided into two groups. Depressive behaviors were measured by forced swimming test. Separation from mother led to decreased swimming in forced swimming test, decreased concentration of cerebral norepinephrine, increased secretion of peripheral IL-6, and increased concentration of corticotrophin releasing factor mRNA in the amygdale in baby rats. One of the groups received bifidobacterium infantis and the other group received citalopram. Probiotic therapy resulted in improved behavioral problems and normalization of the immune response and cerebral norepinephrine concentration. This study supported antidepressant effect of bifidobacterium infantis[5]. The relation between microbiota and anxious behaviors has been demonstrated in many studies performed in rats. Clinical studies found the bacterioides group bacteria to be associated with depression. It seems that the effects of intestinal microbiota and bacteria on human health will be the focus of interest in neuroscience for the next decade. There are authors introducing the microorganisms as a novel group of medications called as "psychomicrobiotic" in the treatment of psychiatric disorders. Intestine-brain axis may be a missing chain that would enable complete understanding and treatment of neuropsychiatric disorders.

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SYMPOSIA ABSTRACT: 472

How can we improve treatment adherence in patients with schizophrenia? The role of psychotherapies in the improving of medication adherence

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ABSTRACT

Schizophrenia is characterized by positive, negative, and cognitive symptoms and a reduced insight that contributes to functional outcomes through not accepting the disease and a low involvement in the treatment and care. Functional outcomes of schizophrenia mainly depend on the acceptance of the disease related troubles, adherence to pharmacological treatment and involving in psychosocial therapies. Although pharmacotherapy effectively improves some symptoms of schizophrenia, some others can remain affecting the patients' daily life activities nearly during their whole life. Psychosocial therapies have been used in success for many years as adjuncts to pharmacotherapy to help alleviate residual symptoms, to improve social functioning and quality of life, to improve medication adherence, to prevent relapses, to reduce re-hospitalization rates, and to decrease the cost of illness.

As in any other illness in which a long-term intake of medication is necessary, compliance is of high clinical relevance in patients suffering from schizophrenia and other psychotic disorders having problems of insight. It is a fact that around 40-60% of the schizophrenic patients in long-term treatment does not take their medication according to the physician's recommendations. Non-adherence can have serious consequences, including poor symptom control and an increased risk of relapse and hospitalization. Medication compliance in patients with schizophrenia is influenced by some factors. These are:

(1) Patient-related factors include demographic characteristics such as age, sex and social status, illness-associated characteristics such as type of disorder and psychopathological

KEYWORDS

Schizophrenia; medication adherence; psychosocial interventions

symptoms, poor insight, substance abuse, hostility, negative attitude or subjective response toward medication, previous nonadherence, etc.

(2) Clinician-related factors include poorer therapeutic alliance, clinician's attitudes toward psychotherapeutic approaches, barriers for meeting to clinician or treatment team, lack of informative materials about the disease and its treatment for patients and relatives, etc.

(3) Treatment-related factors include EPS, particularly akathisia, sedation, weight gain, drooling, sexual dysfunctions, complexities in medication regimen, obstacles for getting to affordable medication, etc.

(4) Environment-related factors include inadequate discharge planning or aftercare environment, lack of enough support from family and relatives, stigma and discrimination, unemployment, poverty, difficulties for accession to psychiatric, psychotherapeutic, and rehabilitative treatment facilities, lack of social supports, etc.

Medication noncompliance is a big challenge for the treatment of schizophrenia, which has high remission rates as much as 73% especially at the phase of first episode, but low recovery rates as less as 8–20%. Because of the deteriorating course of the illness, it is most important task that has to be done is to maintain medication treatment in patients with schizophrenia, in spite of the medication side effects or other obstacles. The factors related to medication adherence seem to be modifiable by using not only pharmacologic strategies, but also some psychotherapeutic and social interventions. Dealing with the medication noncompliance or treatment nonadherence in schizophrenia, many schizophrenia guidelines (APA, NICE, PORT, etc.) recommend the use of following psychotherapies as an adjuvant in the treatment of schizophrenia and related disorders: Cognitive Behavioral Therapy, Social Skills Training, Cognitive Rehabilitation, Family Therapy, Assertive Community Treatment, Supported Employment, Dual Diagnosis Treatment, Illness Self-management, Personal Therapy, Compliance Therapy, Adherence Therapy, and Integrated Therapies. These psychotherapeutic approaches have common elements which mediate the therapeutic efficacy, including psychoeducation, motivational techniques, and interpersonal relations as therapeutic tools. Some main psychotherapeutic approaches' effective domains by and large are shown in Table 1. Improved medication adherence is an important goal for psychosocial therapies along with psychosocial functioning because of the link between nonadherence and the risk of relapse and hospitalization. In this regard, compliance/adherence therapy and family therapies based upon psychoeducation and cognitive behavioral techniques beside other psychotherapeutic interventions should be considered as an adjunctive therapy in the treatment of schizophrenia. Medication non-adherence in patients with schizophrenia could be solved not only by using any individual psychotherapeutic approach, but also using some other therapeutic techniques or approaches when necessary upon the individual needs. It should be noted that the use of psychosocial interventions to improve treatment adherence don't add extra cost to the treatment, because its benefits are superior to expenditures.

Table 1. Domains of improvement with psychosocial interventions

Interventions	Domains mostly improved	Domains less improved
Cognitive Behavior Therapy	Psychopathology Residual symptoms	Adherence Social functioning
Social Skills Training	Social functioning Activities of daily life	Adherence Residual symptoms
Family Interventions	Adherence Relapse & Hospitalization Disease burden	Residual symptoms Social functioning
Cognitive Remediation	Cognitive functioning	Residual symptoms Social functioning
Integrated Therapies	Residual symptoms Social functioning	Adherence Relapse
Compliance/Adherence Therapy	Adherence Relapse	Residual symptoms

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SYMPOSIA ABSTRACT: 473

Attention Deficit Hyperactivity Disorder, Attachment Disorder, Posttraumatic Stress Disorder Co-occurrence and Differentiation and Their Reflections on Treatment in Preschool Period

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ABSTRACT

Attention Deficit Hyperactivity Disorder (ADHD) is a disorder , not a disease, and increasingly suggested to be etiologically heterogeneous. Current theoretical models of ADHD suggest multiple pathways to the disorder like emotion regulation deficit, extreme emotionality and insecure attachment patterns (1). On the other hand , some controversy exists regarding the diagnosis of ADHD in preschool children dependent on developmental characteristics of this period. These problems make it difficult for children to be diagnosed with ADHD, to make differential diagnosis and to choose appropriate treatment approaches. John Bowlby and Mary Ainsworth developed attachment theory based on observations of mother–child interactions. Forms of attachment include secure, insecure dismissing, insecure preoccupied, and insecure disorganized. Many psychological theories have claimed an association between attachment insecurity and ADHD. In a normal population, 60% of children have secure attachment competencies, whereas among children with ADHD lower than % 10 have secure attachment competencies (2). Attachment insecurity is thought to cause ADHD symptoms through negative expectancies on the self and others, as well as through negative effects on other domains of functioning important for goal-directed behavior, such as emotional functioning and cognitive control (1). İn addition, ADHD shares some etiological factors and symptoms with reactive attachment disorder (RAD). Studies found that emotional dysregulation is an important feature of both reactive attachment disorder and ADHD and perinatal period seems to be pivotal in both disorder(3). Posttraumatic stress disorder (PTSD), which is under the heading of stress and trauma related disorders in DSM 5 like RAD, similar with ADHD in preschool period. Complex trauma refers to the occurrence of chronic and prolonged adverse events in a child's life that has an early onset and is generally interpersonal and share a constellation of symptomatology found in children diagnosed with ADHD. In this presentation, ADHD, PTSD and attachment disorder relationship will be discussed through the presented the case. Psychotherapeutic approaches to emotional regulation based on these three disorders will be emphasized.

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SYMPOSIA ABSTRACT: 474

Terrorist and The Meaning of Life

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ABSTRACT

Before getting into the particulars of the subject of psychology of terrorists, the question that who is a terrorist should be answered. Unfortunately, no social or psychological proSymposia Abstract exists solely with regards to terrorists. Research indicates that terrorists have (1) a consistent line of thought; (2) absolute faith in their purpose; (3) a tendency to take risks;

and (4) a will to act, and these consist the common characteristics of terrorists. However, these researches failed to shed light onto the issue as to whether people also have such characteristics before becoming a terrorist. What is nevertheless known is that the terrorists responsible for 9/11 attacks have intolerant and narcissistic reactions as well as extreme anger against those that are not "one of them". So how do these people that may not have such characteristics before getting into a terrorist organization develop these characteristics after becoming a terrorist? One potential factor for this emerges from the structure and activities of terrorist organizations. To elaborate, terrorist organizations have a harsh self-criticism mechanism as well as merciless punishments for not acting in accordance with the organization's rules. These cruel experiences and the mechanisms in the terrorist organization can turn once angry persons to soldiers of ferocity in time. Another potential cause may derive from the cults and ideologies of terrorist organizations. As a result of such cults and ideologies, terrorists may tend to attribute nearly divine meanings to their purpose, leader or even to themselves, and disdain any other person that is not among them. So terrorists may actually enjoy from making such "inferior" people suffer. But, we still do not know for sure. Therefore, researchers should not include terrorists and terrorism to already known psychological illness categories, as terrorist acts require a complex functioning of mind and organization. We all should refrain from psychological reductionism and simplification. Only by taking all the above into account, it can be said that minds of terrorists acknowledge a narrowed set of values. In this context, it should be noted that person is a social being as much as it is an individual being, and the concept of "group" is different than a mere crowd or mass. People live within different groups, and the "groups with close / emotional relations" affect people's individual psychology the most. Since terror is eventually a group act, evaluating the formation of close relationship groups is crucial. The principle of "similarity", in other words the principle that people having similar personalities and understanding tend to get close and together (e.g. people related to mafia or certain spiritual groups), seems to be the most reasonable principle for explaining how people become close. This principle also sheds some light to persons' process of joining terrorist organizations. It should also be noted that, at the basis of terrorism's psychology and people's affiliation with terrorist groups lies a very strict religious and political attitude or in other words, an exceptional fanaticism. Terrorists tend to take refuge in the delusion of a utopia, as their understanding of life does not include any meanings that will enable them to maintain their daily life. Only persons who know the value of life for himself/herself can understand such value in respect of other people's lives. Terrorists lack the understanding of such value or in other words, "the meaning of life".

Prominent French political scientist Oliver Roy also supports the above position by stating that ISIS does not represent the "radicalization of Islam", but the "Islamization of radicalism" through a nihilist riot of a certain generation. According to Roy "terrorists do not derive from the radicalization of a certain group of Muslim population, but they reflect the uprising that affected a particular category of young generation. […] They are "more Muslim than Muslims", especially more than their mothers and fathers. […] They have made significant efforts (albeit in vain) to make their parents faithful again. […] The ones that become Muslim subsequently have chosen Islam, as it is the only religion on the market of radical uprising, and it is certain that you will create terror when you join ISIS." Indeed, violence and terror are "social and historical" phenomena that occurred in all periods of the history and in every system of faith. As a corollary, these cannot be attributed to Islam. Concepts such as "jihad", "martyrdom" or "pledges for heaven to martyrs" do exist to an extent in every system of ideology or belief. On a separate note, we should also remember the underlying reasons for emergence of Political Islam. Political Islam is an anomaly in the history of Islam that harbors the vengeance of a hopeless and humiliated society. Notwithstanding the above, nihilist approaches are not common in Turkey, and it is again Islam and Muslims that form the basis of social values and create meaning against such nihilism. Turkish government makes great efforts to spread and convey these values both in schools and mosques. Having said that, there were and still are involvements to ISIS from Turkey, but we are still trying to come up with vast theories as to the reasons of terrorism. Maybe, instead of creating such theories, we should focus on the connection between terror and nihilism, which arises from the state's inability to convey values to the society, to see the bigger picture.

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SYMPOSIA ABSTRACT: 475

Autism Spectrum Disorder and the Gut Microbiome

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ABSTRACT

Studies about gut microbiota contributed to understanding of the "brain-gut-microbiota axis". Gastrointestinal microbes influence brain function and behavior through several sets of complex pathways. The gut and nervous systems communicate via neural mechanisms such as parasympathetic innervation by the vagus nerve and sympathetic innervation from multiple levels ,endocrine pathways such as the hypothalamic-pituitary-adrenal (HPA) axis. On the other hand, immune-mediated mechanisms play important roles in the way the gut and brain influence each other's function and development. All this information suggests that there may be a relationship between microbiota and psychiatric diseases especially in neurodevelopmental disorders such as an autism spectrum disorder. Autism spectrum disorder (ASD) is a heterogeneous group of complex neurodevelopmental disorders with evidence of genetic predisposition. In recent years, there are increasing opinions about potential the role of microbiota in the pathophysiology of ASD. Researches and clinical observations are frequently reported gastrointestinal disturbances in infants with ASD, which may correlate with the severity of the disorder. Also intestinal permeability is increased and microbial composition is different from normal controls in ASD patients. In addition to different microbial composition, treatment with vancomycin provided transient improvement in gastrointestinal symptoms and cognitive skills in patients with regressive-onset autism. In this presentation, the relationship between microbiota and ASD will be discussed. In addition, the clinical implications of this relationship will be addressed.

SYMPOSIA ABSTRACT: 476

Suicide in Obsessive Compulsive Disorder

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ABSTRACT

Suicide is an aggression and destruction act towards an individual's personality and is the ending of own life intentionally. Suicide and suicide attempts are important causes of morbidity and mortality all around the world. There is a psychiatric disorder in most of the individuals who attempt to suicide. The psychiatric problems that are associated with suicide and serious suicide attempts are mood disorders. Although obsessive-compulsive disorder (OCD) is a psychiatric problem characterized by chronicity, treatment resistance, and frequent comorbidities, risk factors for suicide in OCD have been studies more rarely compared to other diseases. Early studies reported that although it was thought that suicide is rare in this patient group because these patients are on alert always to avoid aggressive impulses and potential threats, suicidal ideations and attempts are increased if affective disorders and anxiety disorders coexist. A recent study identified current and lifetime rates of having suicidal ideations in OCD patients as 28% and 59%, respectively. The suicide rate in the past was found as 27%[1]. Religious and aggressiveness obsessions, repetition and checking compulsions has been found more common in those with suicide attempts history than those without suicide attempts history. In sociodemographic risk factors for suicide in OCD, a significant relationship was found between suicide attempt and being single, and having a family history of suicide attempt; and between suicidal ideations and low socioeconomic level and not having religious activities[2]. In another recent study, the following was identified as risk factors for suicide: Axis 1 diagnosis, comorbid depression and high anxiety scores, severe obsession, concomitant hopelessness and previous history of suicide attempt[3]. In another study published concurrently, while personality disorders and substance abuse were identified as risk factors for suicide; female sex, high education level, comorbid anxiety were proposed as protective factors against suicide[4]. The information on types of suicide in OCD patients is limited. One study reported that 60% of study subjects with OCD attempted suicide with poisoning[5]. Beck Hopelessness Scale, Beck Suicidal Ideation Scale, Suicidal Behaviors Scale, Suicidal Intent Scale, Suicide Probability Scale, Suicidal Ideation Inventory, Suicidal Risk Evaluation Scale are frequently used to evaluate the suicide

KEYWORDS

Obsessive-compulsive disorder; suicide; risk factors

risk. Interventions to prevent suicide can be classified into primary, secondary and tertiary interventions. The aim of the primary prevention is to determine and to eliminate the factors that increase the suicidality in the society and to reduce suicide incidence. The purpose of secondary prevention is identifying risk groups and risk factors and developing preventive measures against them. The aim of tertiary prevention includes the treatment of patients who attempted suicide and prevention of re-attempts. Although the relationship between OCD and suicidal behavior has been put forward with cross-sectional studies, longitudinal research of the relationship of OCD and suicide seems intriguing. To determine which patients will attempt suicide, OCD patients with suicidal ideations should be evaluated prospectively in a longitudinal study. Determination of risk factors in OCD patients and development of interventions to prevent suicide in patients at risk are required.

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SYMPOSIA ABSTRACT: 479

Antidepressant Use During Pregnancy

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ABSTRACT

Considering the fact that most psychiatric disorders occur in childbearing ages and that the pregnancy is not protective in this direction, some women are expected to use antidepressant medications because they are not aware of the beginning of their pregnancy[1]. In our country, there is no data yet. Patients often discontinue medication when they notice that they are pregnant. The prevalence of use falls sharply to one-third in the second and third trimester of pregnancy compared to the time when the gestation is realized. According to a study, 37% of patients take antidepressants only in the first trimester, 7% in the third trimester and 22% in all trimesters. For example, in a large cohort study conducted in the Scandinavian countries published in 2015, the SSRIs most commonly used were reported as citalopram and sertraline, respectively. ın the USA it is reported that the most common used antidepressants after SSRIs is bupropion and followed by TCAs and trazodone. All antidepressants pass to the fetus via transplacental and amniotic fluid. It is expected the concentration in the cord blood to increase as the dose increases. However, although this hypothesis is shown in sertraline and fluoxetine, negative results have been reported about citalopram and paroxetine. Studies show that venlafaxine has a higher level of amniotic fluid and placenta transfer compared to SSRIs. In one study, the lowest cord/plasma ratio was found to be in sertraline and followed by paroxetine, fluoxetine and citalopram, respectively. The rate of nortriptyline in TCA is reported as 68% and in clomipramine it is reported as 60%.The role of the fetal effect of the drug on the degree of exposure of the fetus in through a transplacental or amniotic fluid is not clear. Major malformations leading to death, severe loss of function or cosmetic problems are seen in 2–4% of women under 35 years old, most commonly cardiac malformations. In fact, the overall malformation rate was reported as 3.7% in a large cohort study of newly published and containing 36722 infants exposed to SSRI or venlafaxine and covering 5 countries[2].

SSRIs

In the FDA categorization, paroxetine is classified as 'D', others as 'C'. SSRIs as a group are not associated with increased malformations according to most studies and systemic reviews. The reported risks by Furu et al., in a large-scale cohort study, including 11193 using citalopram and 7245 using sertraline 1.19 and 1.06 for general malformation, 1.15 and 1.13 for cardiac defects,

1.45 and 2.47 for anal atresia and 0.99–1.30 for craniosynostosis respectively. In infants of 366 mothers who used sertraline, malformations 1.11 cardiac malformation risk 1.16 cardiac septal defect risks 1.34 and craniosynostosis risk 2.03 were reported according to controls. There are no meta-analytical results against these two drugs and are suggested as the first choice SSRIs by some authors. Escitalopram, which resembles citalopram and is frequently used clinically, does not appear to be associated with congenital anomaly according to a few studies. In a cohort study in 3950 infants exposed to escitalopram, the overall and cardiac defect risk was 0.83 and 0.89, respectively; in other words, there is no risk increase. Fluvoxamine is the SSRI, the least we know about[3].

TCAs

A study reported a 1.63 times increased risk for malformations in general, but no relationship was found in most studies. The most commonly used imipramine, amitriptyline and clomipramine in the general clinical practice in our country are categorized in the C class by the FDA.

Other Antidepressants

Among the commonly used antidepressants, venlafaxine, mirtazapine, fluoxetine, trazodone and bupropion are categorized in the C group in the FDA classification and there is little information on their use in pregnancy. There is no clinical data on agomelatin, vortexetine and milnacipran. TCAs do not seem to be associated with SGA in restricted data. There is also evidence that there is no significant difference between antidepressants.

Persistent pulmonary hypertension is a rare condition that develops with diminished pulmonary vesical resistance following birth and can result with death in 10–20%. The association with antidepressants was noted in a 2006 case-control study of infants with PPH, reporting that the rate of SSRI use was 6 times higher in these cases. The fetal central nervous system development lasts throughout pregnancy and serotonin plays an important role from cell migration to synaptic connections. In some animal studies increased serotonergic activity has been reported to negatively affect neuronal migration and cortical development. However, it has been shown that in most cases fluoxetine administration is not effective in the development of brain regions, particularly in the cortex hippocampus, striatum, or in the way of reducing depression.

Post natal adaptation syndrome

In the literature also referred to by names neonatal behavioral syndrome, weak neonatal adaptation syndrome, cut-off syndrome. A number of central nervous system, gastrointestinal autonomic and respiratory system related respiratory stresses ranging from hypoxemia to convulsions occur. It's more like adults' withdrawal. It is not only unique to antidepressant users but can also be seen in antipsychotic and benzodiazepine users. It is more appropriate for the baby to be discharged after monitorization in the newborn unit for 48–72 hours. There is evidence that breastfeeding at birth reduces the risk of PNAS. The risk of PNAS in TCAs and SNRIs is similar to SSRIs in terms of symptoms and prognosis. Venlafaxine is one of the most commonly reported antidepressants which cause PNAS. PNAS ratio is reported similar to SSRIs in mirtazapine. The most frequent and most severe symptoms in TCAs are seen in clomipramine.

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SYMPOSIA ABSTRACT: 481

Cardiovascular Aging in Depressive and Anxiety Disorders

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ABSTRACT

Cardiovascular disease, anxiety and depression are among the most prevalent health problems. Previous studies suggest that; although vascular health is mainly determined by age, acute and chronic mental stress may also cause cardiovascular disease. Epidemiologic studies have shown that patients with anxiety and/or depressive disorders have higher

risk of fatal myocardial infarction and sudden cardiac death compared to normal population and also the risk of a fatal event increases in higher levels of anxiety and depression. Arterial stiffness is one of the major signs of vascular aging, which has been documented as a prominent independent prognostic factor of cardiovascular mortality. Progressive loss of arterial elasticity may cause complications such as stroke, left ventricular hypertrophy, and hypertension. Therefore, arterial stiffness has increasingly drawn attention for its role in the cardiovascular disease. Increased arterial stiffness was linked with systemic inflammation in patients with psoriasis. In a study anxiety was associated with cardiovascular diseases and it was hypothesized that inflammation has a role in that association. Thus, inflammation may have critical role between arterial stiffness and anxiety and depression. Previous studies have demonstrated that depression and anxiety may play an important role in vascular aging. It has found that increased pulse wave velocity is related to panic disorder. The comorbidity of anxiety and depressive disorders has been shown to be a distinct risk factor for increased arterial stiffness parameters, respectively. The usage of antidepressant medication may have effects on cardiovascular physiology but its relation to arterial stiffness is unclear. In three studies both decreasing symptom severity and using of antidepressant medication have been found to be related to arterial stiffness. The aim of this study is to evaluate the association between arterial stiffness parameters and depression/ anxiety symptom severity in patients with depressive and anxiety disorders.

SYMPOSIA ABSTRACT: 482

Electroconvulsive Therapy in Schizophrenia

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ABSTRACT

Electroconvulsive therapy (ECT) is a medical treatment that is effective for many psychopathological conditions. In 1938, Ugo Cerletti and Lucio Bini, two Italian psychiatrists, were the first to use anticonvulsant therapy in the field of psychiatry, and the first patient given the treatment had a psychotic disorder. Several observations of ECT response in psychotic states inform our mechanistic inquiry. It is suggested that the benefit of ECT in schizophrenia is not a specific "antischizophrenia" effect per se, but rather is a more general antipsychotic effect. This suggestion is illustrated by the fact that ECT has shown effectiveness in treating psychosis associated with a host of causes, despite their presumed divergent pathophysiologic mechanisms. In this general sense, it may be thought that ECT mirrors the effectiveness of antipsychotic drugs, but ECT's effectiveness in patients who are antipsychotic nonresponders suggests a different or more potent mechanism of action for some patients. In schizophrenia, ECT is frequently considered as a treatment option, particularly in drug-resistant cases with persistent psychotic symptoms, when rapid symptom relief is required, for adolescents and young adults with first-episode schizophrenia, and for prevention of suicidal behavior. Recent studies have shown that combination of ECT and antipsychotics has a significant advantage in terms of rapidity and quality of response for schizophrenia patients. Besides this, augmenting ECT on clozapine resistant schizophrenia found to be clinically effective and safe in many studies. This presentation aims to discuss the place of ECT for treatment of schizophrenia.

KEYWORDS

Schizophrenia;
Electroconvulsive therapy;
Clozapine

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SYMPOSIA ABSTRACT: 489

What is Electroconvulsive Therapy, how it works in the brain?

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ABSTRACT

Electroconvulsive therapy (ECT) is a biological treatment modality in which epileptic seizure is done in the brain by the help of electrical stimulation. ECT was first used to treat mental disorders in 1938 as a substitute for the chemical induction of seizures. Almost 80 years have passed since its first use, and ECT is still widely administered worldwide. It is largely considered to be a treatment for affective disorders in most western countries, while in many eastern countries ECT is mainly applied as a first-line treatment for schizophrenia. Increased receptor sensitivity, hypothalamic stimulation, increased turnover of serotonin and noradrenalin, increased activation of monoaminergic pathways, increased permeability of blood-brain barrier, regulation of circadian rhythms, increased syncronization between both hemispheres and therapeutic effect of convulsion theories have been studied to explain mechanism of action of ECT. For explaining the mechanism of ECT, connectivity, neurotransmitter change, neurotrophic factors, inflammatory response, genetic polymorphism have been studied for recent years. Pinna et al. (2016) suggested that ECT appears to exert pleiotropic effects on the CNS. Its actions target different areas of the signaling system of the brain probably in a top-down fashion: from the top level of the connectivity between multiple cerebral areas, where it appears to modulate and dampen the hyperconnectivity observable in MDD patients, to the intermediate neurochemical signaling level (neurotransmitters), and the bottom level of cellular and intracellular regulatory systems, including neurotrophic factors modulating neurogenesis and neuroplasticity, such as the brain-derived neurotrophic factor (BDNF), as well as gene expression, and genetic polymorphisms. Aim of this presentation is talking about mechanism of action of ECT in the light of new research and to discuss its effect on the brain.

KEYWORDS

Electroconvulsive therapy; mechanism; neurobiology

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SYMPOSIA ABSTRACT: 490

Differences and Similarities in Neuroimaging Findings of Schizophrenia and Bipolar Disorder

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ABSTRACT

The conceptual distinction of schizophrenia and bipolar disorder dates back to 100 years ago, when Kraepelin created the first dichotomous approach based on the clinical course and outcome. However these two mental disorders remain to be classified in two different categories in current diagnostic manuals, similarity in clinical appearance such as delusions, disorganized thought and mood symptoms is a well-known fact in our practice[1]. Furthermore, familial clustering, efficacy of new antipsychotics in both disorders, diagnostic shifts on the course of the illnesses and frequency of schizoaffective cases make us think that these disorders might not be two apart categories but might be subcategories along the

entire psychosis spectrum[2]. Converging evidence from researches in genetics, neuropathology, neuroimmunology, neuropsychology and finally neuroimaging, also leads to a discrepancy with the dichotomous model and support the evidence in terms of shared neurobiological features[3]. In order to make accurate diagnosis and provide appropriate treatment and more importantly to develop more targeted and effective therapeutic interventions it is crucial to find reliable and valid biomarkers identifying pathologic processes underlying the clinical symptoms. Neuroimaging is a beneficial scientific tool to examine the overlap as well as pertaining neural patterns of schizophrenia and bipolar disorder. In this presentation we aim to provide information regarding differentiating and overlapping neuroimaging findings in schizophrenia and bipolar disorder. Brain structural, functional, and connectivity alterations and their implication for the clinical and cognitive manifestations of these disorders will be presented based on the current evidence from the relevant neuroimaging studies.

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SYMPOSIA ABSTRACT: 492

Vitamin D Levels In Children And Adolescents With Obsessive Compulsive Disorder

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ABSTRACT

Obsessive compulsive disorder (OCD) is a common, chronic, and treatment-resistant neuropsychiatric illness in which the person suffers from recurrent obsessions and/or compulsions that cause severe anxiety or distress. Obsessions are recurrent and persistent thoughts, impulses or images that are experienced in an intrusive and inappropriate way, cause marked anxiety and distress, and persist despite all attempts to try to ignore or suppress them. Compulsions are repetitive behaviors or mental acts that a subject feels driven to perform in response to obsessions and are aimed at preventing or reducing anxiety. OCD frequently begins during childhood and adolescence. The prevalence of OCD is 1%-3% [1]. The current Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition classifies OCD under the "obsessive-compulsive and related disorders." The standard neurobiological model of OCD is especially focused on the cortico-striato-thalamic-cortical pathway, and most OCD-related research has been related to the serotonergic and dopaminergic systems. In addition, glutamatergic system has been emphasized in recent years. However, the etiology of OCD is still unclear. But it is known that genetic, environmental, psychological, social and biochemical factors are involved in the etiology.

Vitamin D is an essential molecule mainly involved in the regulation of calcium and phosphorus in the body. It is structurally part of a group of sterols. Biosynthesis of vitamin D begins with the sterol provitamin D3 molecule 7-dehydrocholesterol. The main source of vitamin D comes from the conversion of 7-dehydrocholesterol to provitamin D3 in the skin by solar ultraviolet B radiation, while a lesser amount of vitamin D comes from food intake. The active form of vitamin D is a steroid with potent endocrine, paracrine, and autocrine effects, induced by binding to its specific ligand-the vitamin D receptor (VDR). Vitamin D is produced in the skin during sun exposure or obtained from the diet. It is converted sequentially in the liver to 25-hydroxyvitamin D [25(OH)D] (the major circulating form which is used to determine vitamin D status) and then in the kidneys by the 25-hydroxyvitamin D-1 alpha-hydroxylase (also known as CYP27B1) to its active form 1,25-dihydroxyvitamin D [1,25 (OH)2D]. After formation, 1,25(OH)2D enters the circulation and interacts with its nuclear VDR to regulate serum calcium levels. CYP27B1 is also expressed in other organs such as brain. Circulating 25(OH)D passes the blood–brain barrier and enters neuronal and glial cells and is converted to 1,25(OH)2D. Then the active form of vitamin D interacts with the VDR to regulate several thousand genes responsible for up to 80 different metabolic processes

KEYWORDS
Adolescents; Children; Obsessive compulsive disorder; Vitamin D

including cellular proliferation, differentiation, apoptosis, DNA repair and modulation of both cellular and innate immunity [2,3]. In this way, recent studies have indicated that vitamin D plays a significant role in the development of the nervous system and the regulation of its functions. Investigators have reported that vitamin D is very important for normal brain development and its functions; plays a role in various tasks in the nervous system such as cell proliferation, differentiation and neurotransmission, and shows neurotrophic, neuroprotective and anti-inflammatory effects. Various preclinical and clinical studies have drawn attention to the fact that vitamin D deficiency causes dysfunctional changes in the brain, and may increase the risk of incidence of neuropsychiatric diseases and cognitive disorders such as major depression, autism, and schizophrenia [4]. In a preclinical study reporting a specific impairment of perseverative response inhibition in the case of vitamin D deficiency, it was suggested that this situation may be associated with stereotyped/repetitive behaviors observed in disorders such as OCD and autism [5]. Both the VDR and 1α-hydroxylase enzyme (CYP27B1) are prominently distributed in the dopamine–rich region of the human brain, especially in the hypothalamus and substantia nigra, which considered to play an important role in OCD development. Vitamin D which is expressed to have a regularity role in the nigrostriatal pathway has been reported to have protective effects on dopaminergic neurons against toxins. It is reported that, in the case of vitamin D deficiency, a weakening may occur in the antioxidant defense system like glutathione and therefore, dopaminergic neurons are more susceptible to damage by oxygen free radicals. In the literature, also there are some studies which indicate a possible relationship between OCD and oxygen free radicals. In addition, the active form of vitamin D, 1,25(OH)2D, has been associated with the tyrosine hydroxylase enzyme [dopamine and other catecholamines rate-limiting enzyme in the biosynthetic pathway] [2]. All of these mechanisms supports the hypothesis that vitamin D deficiency may play a role in the pathophysiology of OCD.

In a study conducted in our clinic, vitamin D and related parameters of calcium, phosphorus and alkaline phosphatase levels were evaluated in children and adolescents with OCD. To the best of our knowledge, this is the first study to evaluate the levels of vitamin D and related parameters in children and adolescents with OCD. In this symposium, the possible association of OCD and vitamin D deficiency will be discussed in the light of the literature. In doing so, our study will be taken into consideration.

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SYMPOSIA ABSTRACT: 493

Definition and Etiology of Tic Disorders

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ABSTRACT

Tic disorder is a common neurodevelopmental disorder of childhood. Tics is described as "sudden, rapid, recurrent, nonrhythmic motor movements or vocalizations, generally preceded by urge" in DSM-5. It should be noted that young children, less than ten years of age, most often do not report urge, and that could be either because there is no urge or it is difficult to describe. DSM-5's tic disorders include Tourette's disorder (TD), persistent motor or vocal tic disorder, provisional tic disorder, other specified tic disorder, and unspecified tic disorder. Tic disorders were included as a sub-category of neurodevelopmental disorder chapter in the DSM-5 and the term chronic tic disorder was changed to persistent tic disorder, transient tic disorder was changed to provisional tic disorder and Tourette syndrome was changed to TD. Tics can affect any part of the body. Tics may be further

KEYWORDS

Children; etiology; genetic; neurobiology; tic disorder; Tourette's disorder

classified as simple motor tics such as eye blinking, nose twitching, shoulder shrugging and simple vocal tics such as throat clearing, coughing, sniffing or complex motor tics such as repetitive touching objects, jumping, back arching and complex vocal tics such as repetitive touching objects, jumping, back arching [1,2]. TD is the subtype of the most studied tic disorder in the literature. TD is a severe form of tic disorder, characterized by multiple motor and vocal tics that persist for at least 1 year. It is a disorder that typically starts in childhood and often improves in adulthood. Recent studies have shown that the incidence rate of TD in childhood is 1% and 3-5 times higher in males. Persistent tic disorder occurs 2-4 times more often than TD and about 20% to 30% of school children experience at least one transient tic [2,3]. The pathophysiology and etiology of tic disorders is still unclear. TD has long been known to be a complex disorder etiologically, with both genetic and non-genetic factors. However, clear specific risk factors for TD, either genetic or non-genetic factors, have been difficult to identify and/or replicate. There are many evidences from twin and family studies that point to inheritability of TD and persistent tic disorder. However, TD has been associated with several environmental factors as well, with Group-A Streptococcal infection and psychosocial stress being the most prominent among them. The malfunction of the cortico-striato-talamo-cortical pathway is thought to be responsible for the etiology of tic disorder. Disturbances of diverse neurotransmitter systems such as the dopamine, gamma-aminobutyric acid, glutamate, histamine and serotonin have been considered to play an important role in the pathogenesis of TD. Various changes in brain regional volume have also been reported in neuroimaging studies of TD, although the results have been inconsistent [4,5].

Despite numerous of studies conducted to elucidate the genetic and nongenetic basis of tic disorders, the field are still in the developmental stage.

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SYMPOSIA ABSTRACT: 494

Behaviorally Challenging Preschoolers: A Case of Chronic Irritability and Multiple Comorbidities

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ABSTRACT

Irritability is a ubiquitous symptom of many childhood psychiatric disorders and is a DSM-V criterion for bipolar disorder (BD), major depressive disorder (MDD), generalized anxiety disorder (GAD), and oppositional defiant disorder (ODD).[1] A new diagnosis, Disruptive Mood Dysregulation Disorder (DMDD) was recently included in DSM-5 based largely on the research on severe mood dysregulation (SMD).[1] Criteria for DMDD are similar to SMD, with the exception that the SMD hyperarousal symptoms were removed from the criteria for DMDD, the upper boundary of the age of onset is set earlier at age 10, and an ODD diagnosis is excluded with a DMDD diagnosis. The hallmark symptoms of DMDD are recurrent severe temper outbursts in context of a chronically irritable mood. DMDD shares many characteristics of ODD including irritable mood and temper outbursts.

Case: "A" was initially seen at age 6 years, 2 months for persistent irritability, oppositional behavior and tantrums lasting hours on end. Her mother was exhausted and reported trying almost every approach recommended by child psychiatrists with no improvement.

"A" presented with angry and destructive outbursts that appear uncontrollable and result in emotional and physical upheaval at home. The outbursts occurred almost 3-4 times a day at home or at school. Triggers included, but were not limited to, even minor frustrations; redirection and attempts to calm or distract her; any transition or changes in routine; and any perceived criticism. Her mood would rapidly change from expansive to rage and tearfulness. At times she shared feelings of self-loathing and guilt, but for the most part she

KEYWORDS

Disruptive mood dysregulation disorder; severe mood dysregulation; preschooler; irritability; temper loss; comorbidity

blamed others or circumstances for setting her off. Her mother also reported frequent inappropriate wandering and leaving and running away from the classroom, interrupting others and impatience. About three weeks ago "A" threw her friend's backpack through a glass window at school because her teacher told her to sit down in her desk. When her teacher tried to intervene, "A" grabbed her hair, pulled it violently and punched her in the arm. She ran out of the class to the schoolyard, tried to open the gates and run away. This event was representative of the outbursts "A" has engaged in for more than a year. She could not attend school from then on. Her mother said that she couldn't identify any severe mood swings that appear to be abnormal. Rather, "A" is just cranky and irritable all the time. She can be compliant around the house, but those times are rare and can dissipate without warning. From toddlerhood through about six "A" was seen by different child psychiatrists for significant sleep difficulties; hyperactivity and temper outbursts. She was diagnosed as ADHD and ODD. She was put on several medications including risperidone, aripiprazole, haloperidol, OROS methylphenidate and zuclopenthixol with no improvement. Her medical history was noncontributory. Wechsler Intelligence Scale for Children (WISC-R) revealed a verbal intelligence quotient (IQ) of 89 and performance IQ of 111. Her mother suffered from chronic depression and her father has an obvious history of ADHD and still had chronic sleep problems, reactive aggression, and alcohol misuse and tended to categorize her behavior as childhood willfulness. On the mother's side there is a family history of depression, anxiety and a grandfather with completed suicide with significant psychiatric issues likely Bipolar in nature. On the father's side there is a family history of ADHD and autism. "A" was fidgety and entered the office willingly. She was appropriately dressed and appeared her stated age. She was smiling and started talking right away about the dresses and shoes she wants her mother to buy, her speech was circumstantial and pressured along with racing thoughts. She kept wandering in the room while she was talking, seated when asked but got up very quickly and kept wandering in the room. Her mood was happy, and her affect was mood congruent and intense, shifting quickly. Her thought content was age appropriate, and she talked about frustrations when her mother does not buy what she wants. There was no suggestion of hallucinations or delusions. Her cognitive ability seemed normal and her vocabulary was average for her age. Her thought process was linear, with frequent derailments. When prompted, she could be redirected, and could remember her trail of thought. She showed no deficits in recalling events and was oriented in time and place. She denied the fact that she had frequent age inappropriate outbursts. The diagnosis and management of young children with recurrent temper loss and persistent irritability is an important concern in child and adolescent psychiatry. Further research is needed to determine whether such difficulties are best characterized as a unique disorder or as a trait contributing to more severe functional impairment across multiple disorders. However regardless of our approach to the diagnostic classification of problems of temper loss and persistent irritability, therapeutic approaches to these problems in early childhood are critical.

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SYMPOSIA ABSTRACT: 495

Emotion Recognition and Regulation in Children and Adolescents with Exogenous Obesity

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ABSTRACT

Obesity has been defined by the World Health Organization as abnormal or excessive fat accumulation that may impair health. Exogenous obesity refers to cases of obesity occurring in the absence of underlying disease and accounts for the majority of patients. Obesity is classified under "Other Conditions That May Be a Focus of Clinical Attention" in the Diagnostic and Statistical Manual of Mental Disorders - 5 (DSM-5). However, obesity may be seen in individuals with "Binge Eating Disorder", which is classified under Eating Disorders in DSM-IV and DSM-5. Obesity in children increases susceptibility to many medical diseases and may indicate psychiatric problems as well. It is difficult to identify a direct relationship between obesity and psychosocial problems. However, several recent reports indicate that psychological problems may lead to abnormal eating attitudes and behaviors and thus may

KEYWORDS

Adolescent; children; emotion recognition; emotion regulation; obesity

influence both the formation and exacerbation of obesity [1]. In addition, psychiatric problems arise in children with obesity, suggesting that obesity leads to psychiatric disorders. Altogether, the current literature indicates that overweight children or children with obesity experience difficulties in social communication and interaction and often have fewer friends than their peers [2]. Healthy social interaction depends upon the ability to recognize and interpret the feelings of others and to use these social cues to develop compatible social behavior. The interpretation of emotional information obtained from facial expressions, voice intonation, or from a combination of each allows the individual to make inferences regarding the mental states underlying the behavior of others (Theory of mind) [3]. The behavior is collectively known as social cognition. Another skill that is critical for social communication is emotional regulation, which can be defined as managing the sensation, experience, and expression of existing feelings [4]. The difficulties in social communication and interaction reported in children with obesity may be related to the perception, identification, or interpretation difficulties of their own feelings or the feelings of others. Difficulties that these individuals face in social relationships may be associated with abnormal emotional regulation. Improved understanding of emotional recognition and emotion regulation in young people with obesity may improve their social adaptation and helps by treatment of their disorder. The emotional recognition skills of obese children were found to be inhibited in comparison to non-obese children [5]. Moreover, neither emotion recognition nor emotion regulation difficulties have been evaluated together in a population containing obese children and obese adolescents. In a study that conducted in our clinic, emotion recognition and emotion regulation functions were assessed in obesity patients aged 11-18 years relative to matched control subjects. In this symposium, the possible association of emotion recognition/emotion regulation and obesity will be discussed in the light of the literature. In doing so, our study will be taken into consideration.

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SYMPOSIA ABSTRACT: 496

Development of Evidence Based Treatment Models in the Scope of Translational Developmental Neurobiology in Preschool Depression

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ABSTRACT

Brain development is not a preprogrammed linear and deterministic anatomical maturation process, but a transactional process in which genes, behavior, and environment each play an important role. In this process, the establishment of new links between brain regions or the beginning of an experiential situation (e.g. adolescence) prompts the change of pre-established interactions and the formation of more extensive reorganizations. For this reason, psychopathological processes within the brain can be better understood within a developmental conceptual framework. Many of the psychiatric disorders begin early in the developmental period. In neurodevelopmental disorders, early psychological and physiological experiences and disorder manifestations may increase dysfunctional configuration in important neural systems, leading to more permanent behavior-related problems. During this period where neuronal plasticity is more prominent, interventions may provide long lasting benefits. Therefore, the investigation of the psychopathology dimensions based on the neurobiological basis rather than the categorical diagnostic

approach is also important for the development of evidence based treatment methods. Understanding the brain-behavior relationships in a developmental perspective and the development of evidence-based treatment modalities in a translational neuroscience scope in preschool psychopathologies is a relatively new subject. In this presentation, treatment approaches in preschool depression will be discussed in this context. The normative developmental negative affect pattern detected in early childhood has a peak around age 3 and then declines. However, there are considerable individual differences in this course. In some children there is an increase in negative affects with age when compared to their peers, and functionality may gradually deteriorate. Negative affect in early stages of life is a predictor for later depression. The increase in amygdala activity in preschool children with high levels of negative affect can be considered as an important biomarker. The increased amygdala response to negative emotional stimuli is thought to reflect an increased bias in this type of information processing. Negative attention biases (NAB) may be an important behavioral link between early onset depression and altered brain function. NAB has been a major cause of concern in the past decade. Basic research findings have suggested that NAB in depression is likely the product of a failure to disengage attention from negative information once engaged rather than a tendency to more rapidly orient to it. There is supportive evidence that "attention bias modification" (ABM) method is therapeutic in depression. Selective attention and cognitive models of learning provide a framework for how ABM provides a therapeutic change in attention bias and depression. On the other hand, when the affective, cognitive and behavioral components of depression are evaluated altogether; early detection of developmental deviations in generalized negativity, emotion regulation and reward and threat systems are important in terms of identifying children at risk. In this context, pilot studies conducted in preschool depression are promising but extensive research is needed.

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SYMPOSIA ABSTRACT: 500

Anxiety Disorders and Oxytocin-Vasopressin

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ABSTRACT

Anxiety disorders are common disorder during childhood and adolescence and are a group of diseases that negatively affect the social life, school success and quality of life significantly. The prevalance of anxiety disorders in childhood and adolescence is 6–20%[1]. The most common subtypes are Generalized Anxiety Disorder, Separation Anxiety Disorder, Social Phobia and Specific Phobia. There are roles of biological and environmental factors in the etiology of anxiety disorders. Among biological factors, neuropeptide substances oxytocin and vasopressin are pointed out in the literature in recent years. As well as physiological regulation features of these two neuropeptides in some disorders such as depression, social anxiety disorders and posttraumatic stress disorder was found variability in serum levels. Oxytocin synthesized in the posterior hypophysis extending neurons of the supraoptic and paraventricular nuclei of hypothalamus. Studies have shown the effect of oxytocin on lactation, maintenance and maternal behavior. Vasopressin is reported to be a hormone that increases the susceptibility to anxiety and depression, in contrast to oxytocin[2]. In an animal study increased anxious response to psychogenic stressors was observed in female rats which doesn't have oxytocin gene, and the hypothalamus-pituitary-adrenal axis was suppressed when mice were given oxytocin during stress[3]. In a clinical study, serum oxytocin

KEYWORDS

Anxiety disoders; oxytocin; vasopressin

levels were measured in individuals with social anxiety disorder, and anxiety symptom severity was positively correlated with high levels of oxytocin. However, there was a negative relationship between social dissatisfaction and high levels of oxytocin[4]. In another study, increased oxytocin levels were reported to reduce anxiety and increase the desire to establish social relationships[5]. Data obtained from studies in animals and clinical samples about anxiety disorders and social behavior is remarkable but clinical researches shows that the results are inconsistent in this regard.

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SYMPOSIA ABSTRACT: 501

Neurobiology of Sleep as Related to Autism Spectrum Disorder

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ABSTRACT

Sleep is a critical component of health. Although we do not understand all its functions, sleep is essential to grow, to restore our body and immune system and to enhance and solidify memory and learning. Sleep disorders are a common coexisting condition in autism spectrum disorders (ASDs)[1]. Between 44% and 83% of children ASDs, have sleep problems, with insomnia being the most common sleep concern[2]. Sleep may be affected in individuals with ASDs for a number of reasons, including psychiatric and medical comorbid conditions, coexisting sleep disorders, behavioral and developmental causes, and potentially neurotransmitter abnormalities related to ASDs. For children with ASD, insufficient sleep appears to impact daytime behaviors, making challenging behaviors worse. In addition, sleeping difficulties for the children lead to sleeping difficulties for parents and sometimes siblings, adding to the stress and challenges of parenting a child who has autism. Gamma-aminobutyric acid (GABA) and melatonin play critical roles in promoting sleep and establishing a regular sleep-wake cycle; both are affected in ASDs. GABA is an important neurotransmitter in preoptic area of the hypothalamus, a major sleep-promoting system. Brainstem regions, including the pedunculopontine and laterodorsal tegmental nuclei, the locus cereleus and the dorsal raphe, form a neuronal network regulating arousal from sleep[3]. Neurons projectıng from the preoptic area of the hypothalamus inhibit these brainstem regions, thus promoting sleep. In ASDs, GABAergic interneurons appear disrupted, and an ASD-related genetic susceptibility region has been identified on chromosome 15q that contains GABA-related genes[4]. The expression of these autism susceptibility genes may affect sleep by interfering with the normal inhibitory function of GABA via the preoptic area neurons. The circadian system is also involved in the regulation of the sleep-wake cycle, with suprachiasmatic nucleus receiving information regarding the environmental light/dark cycle from the retinohypothalamic tract. Melatonin is released by the pineal gland in response to signals from the suprachiasmatic nucleus and inhibited by light[5]. Abnormalities in melatonin in blood, or its metabolites in urine, have been documented in individuals with ASDs. Neurotransmitters which are acetylcholine, glutamate, histamine, GABA, Orexin, Galanin, noradrenalin, melatonin, serotonin, adenosine, dopamine, melatonin concentrating hormone (MDGs) associated with sleep and wakefulness. Neural systems that regulate sleep and wakefulness, ascendant reticular activating system, tuberomammillary nucleus, ventral tegmental area, orexin (hypocretin) neurons, ventrolateral preoptic area. Serotoninergic, GABAergic and glutamate-related systems have been associated with autism. In brain imaging studies, anatomical changes were also found in many studies.

KEYWORDS

Sleep; neurobiology; autism spectrum disorder

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SYMPOSIA ABSTRACT: 502

Interpersonal Psychotherapy in Perinatal Period

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ABSTRACT

Interpersonal psychotherapy (IPT) is one of the evidence-based formal psychotherapies for perinatal mood disorders. IPT is an effective, time-limited psychosocial treatment that focuses on the relationship between interpersonal stressors and depressive symptoms and aims to decrease depressive symptoms by improving interpersonal functioning. The original development of IPT was initiated by Gerald L. Klerman and Myrna M. Weisman in the late 1960s. Its theoretical roots can be traced in works of authors such as Harry Stack Sullivan, John Bowlby, and Adolf Meyer. The basic assumption of IPT is that the interpersonal relationships between the depressed patient and significant others have a major influence on the onset, course and outcome of depression, as the development of the disorder occurs in a social and interpersonal context. Social support and stressful life events have also been found to play a role in the development of depressive episodes. Within IPT, interpersonal relationships are the focus of therapeutic attention as the means to bring about change, with the aim of helping patients to improve their interpersonal relationships or change their expectations about them. The treatment also aims to assist patients to improve their social support network so that they can better manage their current interpersonal distress. IPT is typically offered as a short-term treatment, generally lasting 16 to 20 sessions. The IPT model conceptualizes three general areas in which a person may be having relationship difficulties: loss and grief, interpersonal disputes, and role transitions. Interpersonal disputes should be selected as a problem area when overt or covert disputes are contributing to the patient's problems or illness. IPT would aim to identify the dispute and determine its stage, detect sources of misunderstanding via ineffective communication, explore how nonreciprocal expectations contribute to the dispute, examine how the quarrel is perpetuated, develop alternative solutions for the dispute, and promote the development of skills needed to negotiate future interpersonal conflict. Role transitions are conceptualized as situations where the patient has to adapt to a major change in life circumstances. In IPT, the patient with role transition difficulties is helped by the therapist to reappraise the old and new roles, to identify sources of difficulty in the new role, and to generate and implement solutions for these difficulties. The grief problem area aims to facilitate the delayed mourning process and later, to help the client reestablish interests and relationships that can substitute for what has been lost. The therapist, while maintaining an empathic listening stance to help facilitate the mourning process, seeks to reconstruct the patient's relationship with the deceased, helps to address unresolved issues in this relationship, and guides the patient to recognize the link between depression and the feelings for the deceased. A primary aim of the grief work is to help the patient build new relationships and increase his or her social support system. Perinatal depression is a common complication of pregnancy and postpartum period with potentially enduring consequences for the mother, infant, and family. The availability of IPT as an effective treatment for perinatal periods especially important as pregnant women and postpartum women may have concerns about exposing their infants to antidepressant medication. Both pregnancy and the postpartum periods are the times of significant physiological and emotional changes which also influence interpersonal relationships. Interpersonal risk factors like insufficient social support and increased social conflict can have an important impact on the women's mental and physiological health during this period. Such distressing factors which can occur during pregnancy and delivery are compatible with the main problem areas that interpersonal psychotherapy addresses so that the therapist can easily use interpersonal psychotherapy in order to solve such difficulties.

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SYMPOSIA ABSTRACT: 503

Autism Spectrum Disorder in Adolescence: Characteristics and Behavioral Problems

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ABSTRACT

Autism spectrum disorder (ASD) is a heterogeneous neurodevelopmental disorder defined in the Diagnostic and Statistical Manual of Mental Disorders 5th Edition (DSM-5) as occurring across a spectrum of severity of pervasive deficits that appear in early childhood and endure across the lifespan. Core symptoms include persistent impairments in social communication (e.g., limited verbal and non-verbal communication, poor eye contact, difficulty understanding others' body language), persistent deficits in reciprocal social interaction (e.g., difficulties in developing and maintaining relationships) and at least two patterns of restricted, repetitive behavior (e.g., unusual repetitive speech, unusual object use, ritualized behavior, excessive rigidity, fixated interests, heightened or diminished reactivity to sensory input). Adolescence is a time of dramatic physical, cognitive, emotional, behavioral, and social changes, which may have unique consequences for individuals with autism. This is the developmental period surrounding the transition from late childhood to early adulthood, which includes the period between sexual maturation and the attainment of adult roles and responsibilities. With adolescence comes pubertal maturation as well as the challenge of new developmental tasks (e.g., formation of high-quality friendships, acquiring autonomy from parents, forming romantic and sexual relationships). Adolescence is a period during which we see the emergence of many social-emotional problems, including depression, anxiety disorders as well as a broad range of domains of problem behaviors that include risk taking, alcohol and substance use, aggression, and violence. Adolescents with autism spectrum disorders not only share these same risks with their non–autism spectrum disorder peers but also may face additional vulnerabilities during this developmental period.

Among specific symptoms, communication skills are the most widely improved area of functioning, with as many as 93% of adolescents and adults showing improved language and communication skills over their lifetime. Most outcome studies report improvements in social skills over the course of development, though social functioning remains impaired relative to typical peers. The most commonly reported areas of social improvement are in social responsiveness and conversational skills, whereas the quality of friendships and other relationships remains impaired. In contrast to the lifetime improvement observed in communication and social functioning, the severity of restricted/repetitive behaviors and interests appears to be relatively stable over time. Psychiatric and medical comorbidities are quite high in this population, occurring in up to 65% of adolescents and adults with ASD. Epilepsy is the most commonly reported medical comorbidity, occurring in up to 38% of individuals on the spectrum. The most commonly reported psychiatric disorders are affective disorders, particularly social anxiety and depression. Increased rates of obsessive-compulsive disorder, Tourette's syndrome, and attention-deficit/hyperactivity disorder have also been found in individuals on the spectrum. Adolescents with ASD, especially boys, may develop severe problems associated with sexual growth. Exposure of their own body, masturbation in public and touching other people can be embarrassing to those confronted by the behavior, and to parents and siblings. Although the transition from childhood through late adolescence is generally marked by improvements in core symptoms and some social and cognitive skills an estimated 30% of children with autism experience deterioration in functioning for several years or more with the onset of puberty. There are particularly vulnerable neural regions in which reduced volume and increased neuron loss emerge in adolescence and extend into adulthood among individuals with autism. In particular, the amygdala exhibits early overgrowth in childhood followed by a distinct decline in volume

during adolescence for individuals with autism. In addition, the caudate nucleus of the striatum reveals an atypical growth pattern; it shows an increase in volume during adolescent years for individuals with autism. The risk that children ASD will have an increase in neuropsychiatric problems around the time of adolescence is substantial, so services for them (schools, residential care, family support, medical care) should be planned so as to cause minimal stress during that period.

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SYMPOSIA ABSTRACT: 504

Teenager or Mother: Adolescent Pregnancy/ Risk Factors and Negative Outcomes

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ABSTRACT

The World Health Organization (WHO) defines teenage pregnancy as pregnancy in which the mother is under the age of 20 at the time the pregnancy ends. Adolescent pregnancies are now a global concern and an important issue for any society. They are recognized as a risk for both adolescent mothers and their newborns. About 16 million girls aged 15 to 19 and some 1 million girls under 15 give birth every year- most in low- and middle-income countries. The 2014 World Health Statistics indicate that the average global birth rate among 15 to 19 year olds is 49 per 1000 girls. Lack of knowledge about sex and family planning and the lack of skills to put that knowledge into practice put adolescents at risk for pregnancy. Effective sexuality education is lacking in many countries. Education itself is a major protective factor for early pregnancy; birth rates among women with low education are higher than for those with secondary or tertiary education. In low-income countries, sexual activity for girls is often initiated within the context of marriage, or as a result of coercion, frequently with older men. Latin America, the Caribbean, and high-income countries have higher rates of adolescent pregnancy outside marriage than southern Asia, and rates vary across sub-Saharan Africa. In addition, uses of contraceptives are not common among adolescents and they have difficulties to reach access to sexual and reproductive health services. Complications occurring during pregnancy and labor represent the second most frequent cause of death in teenagers 15–19 years of age. The babies born to this age group are at a higher risk of dying than those born to women 20 to 24 years of age. Early, unwanted pregnancies are associated with increased levels of induced abortion, which when carried out in unsafe conditions carries severe health risks, including death. Findings confirm that infants born by teens are at higher risk of preterm delivery, low birth weight, FGR (fetal growth restriction), and fetal distress. Some studies found a high incidence of postpartum hemorrhage and of manual or instrumental uterine revision in the teenage group compared with the young adult group. In addition to physical and mental difficulties lived by both mother and child, childbearing teenagers are also often under the obligation to face decisions, such as abandoning school and, having a long-term impact on their personal life, their family and their community. The social consequences for the adolescent motherhood include attainment of fewer years of schooling and socioeconomic difficulties like potential dependence on social welfare and unemployment. Early marriage puts girls and women at risk of psychological violence including emotional pressure from their husband and in-laws. Developmental immaturity,

low self-esteem, poor negotiation skills and limited financial resources make these girls more vulnerable to these psychosocial challenges. Prevalence of depressive disorder in pregnant adolescents is over twice that of 10–12 % found among pregnant adults. Perinatal depression poses risk for poor maternal-infant bonding, infant attachment style, and behavior problems in offspring.

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SYMPOSIA ABSTRACT: 505

Potential Biomarkers In Autism Spectrum Disorder

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ABSTRACT

Over the past 50 years much effort has been expended in the search for biomarkers for autism. The term "biomarker" has usually been practically defined as a biological measure that differs across groups or is associated with some aspect of a condition. The hope has been that biomarkers would be informative regarding mechanisms of the atypicalities associated with autism, might be useful in early identification, predicting risk and course, might define subgroups, and might index or predict treatment response. Genetic, biochemical, neuropsychological, neurophysiological and neuroimaging measures have been investigated and, at times, proposed as biomarkers for autism. In order to examine the biochemical markers of autism, publications related to the subject were searched after 2010 with the help of databases such as PubMed, google academic. Oxytocin is a neurohormone with a role in social behavior. The relationship between the oxytocin receptor gene (OXTR) and autism has been established. Plasma oxytocin level was found to be positively correlated with social functions in autism. Chronic inflammation and microglial cell activation were detected in the postmortem brain. Calcium binding protein S100B, produced in astrocytes and indicative of brain damage, was found to be high in autistic individuals. Irregular cytokine proSymposia Abstract was associated with autism and a positive relationship was found with the severity of regressive onset and behavioral symptoms. Changes in complement proteins, chemokines, adhesion molecules, growth factors, proinflammatory cytokines have been identified. Maternal antibodies are able to cross the blood brain barrier. Maternal infectious or immunocompromised fetal neurogenesis, synaptic plasticity, changes in cytokine levels affect neuronal migration. Children with higher levels of serum IFN-g, IL-4, and IL-5 were found to have a higher prevalence of autism in their children. It has been determined that fetal Il-6 exposure may cause morphological changes in the hippocampus and learning difficulties in the following years in the late periods of pregnancy. The prevalence of gastrointestinal dysfunction in autistic individuals is 9 to 70%. GIS symptoms may increase behavioral symptoms in autistic individuals. In many studies, changes in GIS flora have been detected in autistic individuals. Between the brain and the intestine there is a gut-brainstem, which is defined as a complex network of two-way communication, which is regulated by the production of neurotransmitters such as vagal innervation, serotonin and GABA, and gastrointestinal peptides.

Serotonin has been found at high rates in many studies on autistic individuals. Elevation of serotonin was detected in autism and not detected in intelligence. Excessive serotonin exposure of platelets is thought to be associated with alterations in the platelet serotonin binding capacity. Melatonin is known for its role in circadian rhythm, immune response, and neuronal plasticity. Melatonin is synthesized by serotonin with acetylserotonin (ASMT) enzyme.

Significantly lower levels of plasma melatonin in autism have been detected in a number of studies and it is thought that this low level of ASMT enzyme level and altered synthesis of circadian melatonin. Vitamin D has been shown in many studies that have decreased in autistic individuals. In many studies it has been determined that the redox mechanism has changed or is inadequate and at the same time oxidative stress has increased autism. Reduced glutathione, glutathione peroxidase, methionine and cysteine have been shown to have increased reduced glutathione in autism. Mitochondrial diseases are more common in autistic children than in the normal population. Mitochondrial function markers also change in autistics without mitochondrial diseases. Therefore, mitochondrial dysfunction changes are thought to be different in autism subtypes. The variety of symptoms in autism, including monozygotic twins, emphasizes the strong role of epigenetics. Epigenetic changes can be caused by factors such as oxidative stress, mitochondrial functions, methylation, maternally induced infections, environmental toxins, and diet. Structural imaging studies showed increase in total brain volume, increase in frontal lobe volume, increase in cortical thickness in temporal and parietal lobes, decrease in adolescents and adult cortical thickness in children. Structural changes were also observed in the corpus callosum, amygdala, cerebellum and basal ganglion. Activation changes in the fusiform gyrus in the face recognition and decreased fusiform gyrus and amygdala activity in the emotional face recognition task were found in FMRI studies given to the social task. Abnormal right hemisphere lateralization of the temporal cortex was detected in tasks assigned to evaluate language skills. FMRI studies have also shown that in the resting state, the number of nearby links increases whereas the number of remote links decreases

Although many avenues have been tried for identifying biological markers for ASD, a clinically valuable ASD biomarker is not yet in sight. For a biomarker to become clinically valuable, it would need to be highly sensitive and specific (even if limited to a well-defined subgroup of ASD patients or to a developmental window), be feasible for use in the clinic, and not be cost prohibitive. The majority of studies currently available suffer from small cohort sizes and lack of replication in independent datasets, which make the estimation of biomarker reliability hard to evaluate. The difficulty of putting together a large ASD research cohort may be balanced out in the near future by more open data sharing, allowing investigators to replicate their results using published data.

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SYMPOSIA ABSTRACT: 506

Neurobiology of Sleep in Anxiety Disorders

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ABSTRACT

Sleep is an essential process that is associated with neural restoration and physiological maintenance of multiple systems. Healthy sleep leads to clarification of metabolic waste from the brain. Sleep is vital for cognitive functions; yet lack of it is linked to many adverse effects including dysregulation of circadian processes such as cortisol secretion, emotion regulation disabilities. The tight regulation of sleep/wake cycles causes the individual to many other disorders like obesity and psychiatric problems (1). Many neurochemical systems are related to generating wakefulness and sleep. It's now known that neurons producing ACh and monoamines such as NE, 5-HT, DA, and HA promote various aspects of wakefulness(2). In addition, orexins/hypocretins help sustain long periods of wakefulness while suppressing REM sleep. For example orexin neurons are associated with the development of narcolepsy. NREM sleep is mainly regulated by neural pathways originating in the VLPO and other preoptic regions, yet it is also influenced by diffusible somnogens such as adenosine. REM

KEYWORDS

Sleep; anxiety; orexin; melatonin; norepinephrine; stress

sleep is driven by neurons in the pons that make ACh and GABA(3). Recent data highlights the clinical potential of single and dual orexin receptor antagonists (SORA and DORA's) for neuropsychiatric conditions including addiction, anxiety and depression. Anxiety is able to cause enhanced excitatory drive to dopaminergic and noradrenergic neurons in the locus ceruleus –an important projection target of orexin neurons. Patients exhibiting panic anxiety displayed increased CSF orexin levels compared to patients exhibiting panic anxiety with comorbid major depressive disorder (4). Norepinephrine (NE) regulates wakefulness and sleep, as many studies provide evidence. The NE system may be important in promoting arousal under circumstances that require responding to a stimulus, challenge or particularly a stressful condition. In addition, the locus ceruleus and NE neurons in the ventral medulla are active during stress. Animal studies have revealed that lack of NE and lesions of locus ceruleus after stress exposure lead to less behavioral arousal and cortical activation. Also excessive NE tone with anxiety likely contributes to insomnia (2). Melatonin (MLT) is a pleiotropic neurohormone controlling many physiological processes and whose dysfunction may contribute to insomnia. Melatonin and the nonselective MT1/MT2 receptor agonist agomelatine have displayed anxiolytic-like properties in classical animal paradigms of anxiety. MT2 receptors have great potential for pioneer drug discovery in the treatment of anxiety disorders and related sleep disturbances (5).

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SYMPOSIA ABSTRACT: 508

Imagery Rescripting

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ABSTRACT

Some of the patients with psychiatric problems can report severe traumatic experiences like physical or sexual abuse in childhood. For others, they didn't have a strong relationship with parents to fulfil basic child needs such as warmth, empathy, unconditional acceptance, guidance, stable and predictable emotional attachment[1].They can have dissociative symptoms and mental disorders. Severity of dissociative phenomena impacts emotional processing of stressful events. These patients are resistant to pharmacy and also to therapies that don't address these stressful memories. These memories serve as an important maintaining factor for a range of mental health conditions. Imagery rescripting techniques have long been used as a part of cognitive behavioral therapy. This approach seems to shorten the patient's treatment and lead to faster recovery than traditional exposure therapy[2].

The process may be divided into several steps[3]:

(1) Creating a therapeutic atmosphere: The first step is to create feelings of security and control for the patient. The therapist shows emotional support, empathetic interest, acceptance and appreciation.

(2) Imaginal exposure to painful experiences: The patient recalls one of important events experienced during the childhood. The patient is asked to close the eyes and describe in the present tense, what is happening.

(3) Expressing negative emotions towards aggressors or persons who could not protect: This step helps to express emotions that have not been expressed and could have been hold in for years.

(4) Expressing the child's needs towards the person who should have protected him or her: In imagination, the patient tells a close person what he or she would need or asks for help, defense, protection or punishment of the aggressor.

(5) Experiencing a better end-imagery rescripting of the story: If it is possible to imagine that the patient solves the situation in a different way and by himself or herself, he or she is asked to do that in imagination. If it is unimaginable, the patient may imagine himself or herself as an adult protecting a small child in the traumatic situation. Another possibility is to imagine that another person or the therapist enters the situation[4,5]. The patient is asked whether in the past, there was a person he or she trusted and loved and who could have protected him or her in the traumatic situation.

(6) General calming: The imaginary experience is verbally discussed and put in the context of the patient's entire story. It is important to praise the patient both for having the courage to enter the experience and for how he or she managed to rescript it.

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SYMPOSIA ABSTRACT: 509

Prodromal Symptoms in Children and Adolescents with Bipolar Affective DIsorder-Prodromal Mania and Comorbidity with Substance Use Disorder

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ABSTRACT

There is a general consensus for premorbid and prodromal stages of schizophrenia. In DSM 5 some criteria were proposed for prodromal schizophrenia under the title of "attenuated psychosis syndrome". There is an also prodromal phase for bipolar disorder. In patients diagnosed for bipolar affective disorder with a loaded genetic risk, the first premorbid symptoms usually start before the age of 10. The premorbid symptoms for bipolar affective disorder in children may consist of high levels of anxiety, sleep disorders, mood symptoms and affective lability, behavioral disinhibition, hyperactivity, impulsivity, cyclothymic-reactive-hypersensitive temperamental features, neuroticism, ruminative thoughts, depressive symptoms, conduct disorder and ADHD symptoms, cognitive inflexibility, social cognition deficits, memory deficits. The appearance of mania and psychosis may be too similar in adolescence. There is often significant diagnostic controversy and challenge resulting in diagnostic shifts. The decision for final diagnosis should be in the follow up. In 16.7% of the adolescents with schizophrenic disorder, final diagnosis is bipolar disorder. In 9.5% of the adolescents who thought to be experienced psychotic mania the final diagnosis is schizophrenia. In adolescents the first stage of the bipolar episodes could be bipolar depression. Adolescent depression with psychomotor retardation, catatonic features, confusion-stupor, psychotic symptoms, atypical depressive, sometimes melancholic depressive symptoms, excessive sleepiness may convert to bipolar affective disorder in the follow-up. Also depressions with fast respond to antidepressants or on the contrary treatment resistance to antidepressants may be the first episode of bipolar affective disorder in children and adolescents. Family history of bipolar disorder is also so significant for suspecting prodromal stage of bipolar affective disorder. Attenuated thought abnormalities and ideas, judgment difficulties are more predictive for prodromal schizophrenia. OCD findings, suicidality, thought and communication-interaction difficulties, depressive findings, concentration-memory deficits, anergia, physical agitation or retardation, affective instability, depression with mix features are more predictive for prodromal mania. Also panic disorder, separation anxiety disorder, generalized anxiety disorder, conduct disorder symptoms, ADHD, impulsivity, criminal behaviors in children could be a risk factor for developing bipolar affective disorder. In spite of prodromal schizophrenia, prodromal mania may be associated with better functionality before the disorder, shorter period of untreated illness (DUP), mild elevated mood, labile mood and affect, hyperactivity, mild flight of ideas, orientation difficulties, subthreshold delusions, metaphysical-religious-grandiose obsessions, being female. In patients with these symptoms initiation of the disease can be with psychotic episode but these people are risky for the conversion of diagnosis to bipolar affective disorder or schizoaffective disorder. Also mild autistic symptoms in childhood may be a risk factor for schizoaffective disorder. Quetiapine, lurasidone, lamotrigine, fluoxetine + olanzapine combination might be more appropriate for

KEYWORDS

Bipolar affective disorder; prodromal mania; substance use disorder

the symptoms of suspected prodromal mania and depression with mix features. Synthetic cannabinoids and cocain-crack could induce manic symptomatology and LSD may lead to symptoms similar to schizoaffective disorder. In bipolar children and adolescents substance use disorder comorbidity rates are extremely high. Also substance use may complicate the diagnosis of bipolar affective disorder in adolescents. Substance use may mimic rapid cycling bipolar affective disorder but patients with bipolar affective disorder with rapid cycling pattern also have greater tendency for substance use.

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SYMPOSIA ABSTRACT: 510

The Association Between Substance Use Disorders and Childhood Negative Life Experiences and Psychosocial Traumas

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ABSTRACT
There is a very complicated and bidirectional psychosocial, genetic, environmental, epigenetic, familial interactions between traumas in childhood-adolescent period and developing substance use disorder. Traumas also may transmit between the generations for families with addiction problems. Parental substance use disorder, makes children vulnerable to traumas because of that not only genetic but also epigenetic factors may lead to a substance use disorder diagnosis. Also ADHD is a risk factor for substance use disorder, but ADHD may also make children vulnerable to psychosocial traumas which are an also separate risk factor for developing substance use disorder. Traumatic experiences may lead to substance use for self-medication but substance use also heightens the risk for a traumatic experience. Especially in young girls sexual abuse may induce substance use disorders and as a result, this condition may lead polyvictimization and revictimization. Especially sexual abuse leads to substance use other than different traumas. In this presentation it is going to be endeavored to enlighten the complicated and bidirectional association between "traumatic spectrum disorders" (ADHD, PTSD, complex trauma disorder, dissociation and dissociative disorders, borderline personality features, criminal and antisocial behaviors, attachment problems, aggression) and substance use disorders.

KEYWORDS
Trauma; substance use disorder; early negative life experiences

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SYMPOSIA ABSTRACT: 512

What is Microbiota? Yesterday, Today, and Tomorrow…

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ABSTRACT

Gut microbiota is a complex structure formed by collecting bacteria, viruses and some single-cell eukaryotes in the intestine and functioning like an organ system.

The gut microbiota is not stable. The intestinal mucosa, which varies depending on the person, is sensitive to endogenous and exogenous factors[1]. The content and density of gut microbiosis vary from proximal to distal, from superficial to luminal, due to differences in physiological and anatomical structures. Babies receive the initial microbiome from their mothers but it change with aging. Gut microbiota can change under the influence of diet, medicine and stress. 1 year old the infants form a complex gut microbiome like adults[2,3]. In healthy humans, intestinal microbiology can be divided into 6 bacterial classes as Firmicutes, Bacteroidetes, Proteobacteria, Actinobacteria, Fusobacteria and Verrucomicrobia[4]. Bacteroidetes and Firmicute constitute 90% of gut microbiota. Intestinal microbiota has various benefits such as host homeostasis, nutrient synthesis, detoxification, epithelial development and immune system[5]. Microbioms can also affect the health of a person in the negative direction and cause certain diseases. These diseases are pre-defined as excessive intestinal bacterial growth, dyspepsia, rosacea, irritable bowel disease[6]. Recently it is thought that microbiomes are involved in the pathophysiology of systemic diseases such as inflammatory bowel diseases, infectious diarrhea, colon cancer, type 2 diabetes and obesity[7]. The latest researches show that changes in gut microbiota could affect the brain's physiological, behavioral, and cognitive functions, but its precise mechanism has not been fully understood yet[8,9]. Gut microbiota affects the brain not only through the nervous system (gut-brain's neuroanatomical pathway) but also through the endocrine system, immune system, and metabolic system. Furthermore there is significant evidence linking gut microbiota and neuropsychiatric disorders such as schizophrenia, autism spectrum disorder, anxiety, depression[10,11,12]. As a result, the effects of gut microbiota on human health are likely to be important in neuroscience in the coming years. The gut brain axis might perhaps provide a complete understanding and treatment of neuropsychiatric disorders.

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SYMPOSIA ABSTRACT: 513

Differential Diagnosis in Tic Disorders

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ABSTRACT

In order to made the diagnosis of tic disorder, one should exclude tics seen in other clinical conditions, tic related to medical condition or substance use, and tic-like movements. Tics are generally characterized by sudden, repetitive movements or sounds with preceding premonitory sensation associated with an impulse, distress, irritability or other sensorial phenomenon. The differential diagnosis of tic disorders includes chorea, athetosis, dystonia, myoclonus, tremor, stereotypies and abnormalities related to obsessive compulsive disorders. Careful observation and detailed history are important in distinguishing tic disorders from other movement disorders. Absence of current or previous facial tics, tics without wax/wane in severity or flow from one body part to another, delayed onset of tics without history of tics and presence of complex tics in the absence of simple tics may indicate other movement disorders, although there are no clear borders for distinguishing tics from other movement disorders[1].

Treatment of Tic Disorders

In the children with tic disorders, the goal of treatment is to resolve tics and to enhance social functionality and potential of the child in the school environment. At the initial phase of therapy, psychoeducation of child and parents has a remarkable role. During psychoeducation, information about nature and course of disorder should be provided and concerns should be addressed. Symptoms, associated risks, potential comorbidities, course of tic disorders and treatment modalities should be explained to parents and child clearly. Severity, effectiveness of treatment modalities, tolerability and patient's preference should be taken into account when planning individualized therapy. If behavioral therapy is failed in the treatment of tic disorders or if tics are excessively severe, pharmacological therapy should be added to behavioral therapy or behavioral therapy should be switched to pharmacological therapy. Conditions that may accompany to tics should be questioned before making decision about therapy. Whether or not comorbid psychiatric disease is present, current social functionality and presence or absence of developmental delay are of important when planning treatment. After diagnostic assessment, the condition should be formulated, treatment plan should be established and therapeutic hierarchy should be created. When making decision about therapy in the presence of comorbid conditions, treatment should also direct comorbid condition.

Behavioral Techniques Used in Treatment of Tic Disorders

Main behavioral psychotherapy approaches used in the treatment of tic disorders include relaxation training, massed negative practice, biofeedback, exposure and response prevention, contingency management, and habit reversal training[2].

Exposure and response prevention technique is based on the assumption that tics are voluntarily performed to relieve himself/herself from unpleasant/negative sensations. In this technique, habituation to premonitory sensations is important. If the patient is aware of premonitory sensations, he/she will orientate to these sensations by response prevention. As a result, desire to perform sound or movement will decrease; thus, motor and vocal tics will be reduced over time[3].

The primary elements of habit reversal training include awareness training, self-assessment, relaxation training, competing response training and motivational techniques. Awareness training aims to identify patient his/her sensation and behaviors before and during tic. Competing response training is an important component of HRT and suggests that competing response should be fully reciprocal to tic movement which must be applied at least a few minutes and can be performed during normal daily activities. The motivational techniques involve encouraging child to cope with tics and ensuring social support through positive reinforcement by family members.

Pharmacotherapy in Tic Disorders

Alpha-2 receptor agonists and antipsychotic agents are most commonly used agents in the pharmacotherapy of Tourette disorder. The alpha-2 receptor agonists are moderately effective in the control of tics. Both clonidine and guanfacine stimulate alpha-2 receptors in the central nervous system. Although clonidine was most commonly used alpha-2 receptor agonist in the past, guanfacine is preferred due to lesser sedation and advantage of daily dosing today. Clonidine, either alone or in combination with methylphenidate, can be used particularly in cases with ADHD and tic disorders together[4]. Typical antipsychotic agents have higher affinity to D2 receptors than D1 receptors. In addition, they interact with serotonin, acetylcholine, histamine and norepinephrine receptors, which can lead several side effects. Thus, there are concerns about using typical antipsychotic agents as first-line therapy. Haloperidol, pimozide and fluphenazine have also been addressed in the treatment of tic disorders. Adverse effect profile of typical antipsychotic agents make their use challenging; thus, atypical antipsychotic agents are preferred[5]. When compared to typical antipsychotic agents, atypical antipsychotics exert more selective D2 receptor blockade; in addition, they also interact with serotonin receptors. They have minimal EPS side effects. Risperidone has greater affinity to D2 and 5HT receptors. It was suggested that atypical antipsychotic agents are effective in the treatment of Tourette disorder and that best evidence are associated to risperidone[6]. Aripiprazole is another antipsychotic agent used in the treatment of tic disorder. It has D2 antagonist activity; in addition, it is partial agonist for D2, agonist for 5HT1A receptor and antagonist for 5HT2A receptor. It has become a promising alternative in

the treatment of tic disorders due to its marked effectiveness on tic symptomatology and comorbid conditions with mild side effect profile[7]. There are studies suggesting that other atypical antipsychotic agents including olanzapine, ziprasidone and quetiapine improve tics. In addition, there are reports indicating effectiveness of sulpiride and amisulpride on tic disorders. Moreover, agents with low level of evidence could be used in cases not responding or in cases with intolerance to alpha-2 receptor agonist or antipsychotic agents although it is controversial. In this presentation, we will discuss differential diagnosis and treatment of tic disorders.

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SYMPOSIA ABSTRACT: 514

Autonomic Dysfunction/ Autonomic Nerve System and Emotions

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ABSTRACT

When it comes to emotion, all roads lead to the autonomic nervous system (ANS). Whether it is the generation, expression, experience, or recognition of emotion, the role of the ANS is critical. In many theories, emotions organize the activity of ANS and other physiological systems. Two kinds of patterned activity are discussed: (1) coherence; emotions organize and coordinate activity within the ANS, and between the ANS and other response systems such as facial expression and subjective experience, and (2) Specificity; emotions activate different patterns of ANS response for different emotions[1]. There is some empirical evidence for the coupling of individual basic emotions to discrete patterns of autonomic response, e.g., changes in heart rate, skin conductance and skin temperature or distinguishable patterns of cardiorespiratory activity. No single autonomic dimension (e.g., sympathetic arousal) appears to be sufficient to characterize different emotions. Physiological responding in anger-eliciting contexts of harassment or personalized recall describes a modal response pattern of reciprocal sympathetic activation and increased respiratory activity, particularly faster breathing[2]. Anxiety has been almost unanimously characterized by sympathetic activation and vagal deactivation, a pattern of reciprocal inhibition, together with faster and shallower breathing[3]. Disgust-related autonomic responding falls into two partially overlapping patterns: (a) disgust elicited in relation to contamination and pollution (e.g., pictures of dirty toilets, cockroaches, maggots on food, foul smells, facial expressions of expelling food), characterized by sympathetic–parasympathetic co-activation and faster breathing, particularly decreased inspiration (Physiological response associated with vomiting); (b) disgust elicited in relation to mutilation, injury, and blood (e.g., injections, mutilation scenes, bloody injuries), characterized by a pattern of sympathetic cardiac deactivation, increased electrodermal activity, unchanged vagal activation, and faster breathing[2]. Inducing embarrassment by experimenter humiliation, watching a video of oneself singing, or imagery, studies consistently indicate broad sympathetic activation and vagal withdrawal, a pattern of reciprocal inhibition. Studies on fear point to broad sympathetic activation, including cardiac acceleration, increased myocardial contractility, vasoconstriction, and increased electrodermal

KEYWORDS

Autonomic nerve system dysfunctions; emotions; psychiatric disorders

activity. In distinction to the physiological response to anger, peripheral resistance typically decreased in fear, whereas it increased in anger. This response is accompanied by decreased cardiac vagal influence and increased respiratory activity, particularly faster breathing based on decreased expiratory time, resulting in decreased carbon dioxide blood levels. The activating sadness response, which partially overlaps with the physiological response of crying sadness, is characterized by increased cardiovascular sympathetic control and changed respiratory activity, predominantly reported in studies using personalized recall, and some studies using film material. On the other hand, the deactivating sadness response, which partially overlaps with the physiological response of non-crying sadness, is characterized by sympathetic withdrawal, reported in the majority of studies using film material, as well as music excerpts, and standardized imagery. The autonomic response pattern of happiness is characterized by increased cardiac activity due to vagal withdrawal, vasodilation, increased electrodermal activity, and increased respiratory activity. This response pattern points to a differentiated sympathetic activation state of decreased α and β adrenergically mediated influences, while at the same time cholinergically-mediated effects are increased. Happiness shares with various negative emotions a central cardiac activation component due to vagal withdrawal, whereas it is distinguished from these by peripheral vasodilation[4]. Psychiatric disorders may cause autonomic nervous system dysfunctions at various levels. Autonomic regulation of the heart plays a key role in cardiovascular functioning. Heart rate variability, the assessment of beat-to-beat variation in the heart over time, provides a reliable index of cardiac autonomic function. Reductions in HRV have therefore been considered a marker for various disease states, most notably a greater risk for cardiovascular disease and all-cause mortality. Psychotic, anxiety and depressive disorders are associated with reduced HRV at various levels[5]. Depressive disorders that onset in the juvenile years have been linked to far-reaching adverse consequences, making it imperative to elucidate key mechanisms and contributory factors. Excessive use of regulatory responses that exacerbate sadness (maladaptive mood repair) or insufficient use of regulatory responses that reduce adaptive mood repair may reflect behavioral mechanisms of depression risk. Cardiac vagal control, indexed by patterns of respiratory sinus arrhythmia has received attention as a putative physiological risk factor for depression[6].

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SYMPOSIA ABSTRACT: 515

Complicated Preschool Attention Deficit/Hyperactivity Disorder and Pharmacologic Treatment Approaches

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ABSTRACT

Attention deficit/hyperactivity disorder (ADHD) is a highly comorbid diagnosis and conditions like dysphoric affect, low self-esteem, anxiety, and obsessional traits are often found in patients with a primary ADHD diagnosis, but they can also occur as separate conditions. Comorbidity should be evaluated for all cases with ADHD symptoms. After gathering all the relevant data from clinical interviews, rating scales, and other sources, the clinician must weigh and integrate this information to determine whether the patient meets the diagnostic criteria for ADHD and/or another learning or psychiatric disorder. On the other hand, in many cases it can be impossible to make an official Axis I diagnosis. Some symptoms like irritability can be trans-diagnostic and it can be difficult to make differential diagnosis.

KEYWORDS

Disruptive mood dysregulation disorder; preschool ADHD; irritability; temper loss; comorbidity

Another issue in diagnostic accuracy is the absence of official definitions of symptoms that are developmentally sensitive. Emotional lability is a highly prevalent condition in child psychiatry cases, it can be observed in nearly half of the child psychiatry admissions, and it can be difficult to differentiate an ordinary developmental feature from pathological phenomenology of a spectrum. Clinicians should consider the developmental features during diagnostic evaluations. Official diagnosis is important to plan an evidence based treatment, however symptoms associated to both depression and ADHD can be viewed as occurring along a dimension from sub-syndrome to disorder. Even when these two set of symptoms are viewed as categories, the point at which depression and ADHD criteria reached diagnostic levels hinges on whether enough symptoms are co-occurring to be recognized as a syndrome and whether these symptoms are severe enough and persistent to cause functional impairment. On the other hand, considerable evidence indicates that, subthreshold depressive or ADHD symptoms have found to be associated impaired functioning[1,2]. The rates of internalizing comorbidity also not change between threshold and subthreshold ADHD[1]. When planning treatment, highly comorbid symptoms can be viewed as a dimension and "treat what is there" approach can be main strategy to choose pharmacologic agent. In our case of preschool ADHD, chronic irritability and temper outbursts are the main associating symptoms to ADHD symptoms. These symptoms are frequent feature of many disorders like mood disorders, anxiety disorders, disruptive, impulse control disorders and substance use disorders. Irritable children are described as grumpy, annoyed easily and in negative mood in their background. The other feature they have is outbursts with flashes of anger or explosiveness, verbal and/ or physical, that are excessive responses to requests or events, and they are usually disruptive to their environment. It can be difficult to differentiate excessively irritable cases from bipolar cases, DSM-5 described a manic episode with A criterion as distinct period of elevated, expansive or irritable mood and increased goal directed activity or energy, lasting at least 1 week and present most of the day nearly every day; B criterion described many associating symptoms to A criteria which should be noticeably different from baseline usual behavior. Other than distinct episodes, family history of bipolar disorder can help to differentiate irritable-ADHD cases from bipolar disorder. Irritable-ADHD cases were described as SMD at first, and in DSM-5 hyperarousal symptoms which may overlap with B criteria of bipolar disorder are excluded from severe mood dysregulation criteria and described as disruptive mood dysregulation disorder. Dimension of angry- irritable mood is highly associated with externalizing disorders and they predict future anxiety and depressive disorders, high comorbidity and functional impairment[3,4]. So, disruptive mood dysregulation disorder is placed under depressive disorders. Disruptive mood dysregulation disorder is a highly possible diagnosis for our case since irritability is not episodic and stable in between episodes of temper outbursts. Accurate diagnosis can be very difficult, it is very important to make separate family and child evaluations and to follow-up the case in order to observe episodes, change from baseline usual behaviors, presence of symptoms in more than one situation, co-occurrence of symptoms as bundles, routine pattern of outbursts, mood between outbursts, and consider cultural features and developmental level, think about impairment that irritability produces. Treating mood problems in behavioral disorders could be critical to helping these individuals and may be important for their overall outcome. Treatment of these cases is suggested mainly similar to treatment of ADHD with anxiety or depression. So, stimulants and treatments for anxiety depression such as selective serotonin reuptake inhibitors, cognitive behavioral psychotherapies, or a combination of these could be considered. Tourian et al.[5] examined empirical evidence supporting the pharmacological treatment for severe angry-irritable mood and found that pharmacotherapeutic treatment for both aggression and chronic irritability includes various options, such as antidepressants, especially selective norepinephrine reuptake inhibitors, mood stabilizers, psychostimulants, antipsychotics, and alpha-2 agonists. Environmental intervention can also be helpful, and non-pharmacological treatments should also be considered in treatment of severe irritability with ADHD. The available evidence suggests potential efficacy of psychotherapies which have previously been developed for internalizing and externalizing disorders.

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SYMPOSIA ABSTRACT: 516

Similarities and Differences in Neurocognitive Impairments in Patients with Schizophrenia and Bipolar Disorder

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ABSTRACT

Similarities and differences in neurocognitive impairments in patients with schizophrenia and bipolar disorder is a hot topic in researches. Cognitive impairment is a common and persistent feature of schizophrenia. It is one of the most important determinants of functional impairment observed in schizophrenia[1]. Generalized impairment in multiple cognitive domains including verbal and visual memory, episodic memory, working memory, processing speed, attention, executive functions that persists in every clinical state[2]. Persisting cognitive deficits and functional impairment had been traditionally considered as specific features of schizophrenia in comparison to bipolar disorder. Neither cognitive impairment nor functional deficits are specific to schizophrenia. Recent researches showed that bipolar disorder is also associated with cognitive impairment and functional deficits which are also evident in euthymic patients[3]. Thus, neurocognitive deficits have been considered trait-related markers of bipolar disorder. A recent meta-analytic review which compared neurocognitive deficits in first-episode bipolar disorder (FEBP) with healthy controls and first-episode schizophrenia (FES) reported that FEBP patients were significantly impaired in all cognitive domains. FES patients significantly underperformed FEBP patients in most cognitive domains. Similar to chronic patients, cognitive functions in FEBP lie intermediate between FES and healthy controls. So they comment that neurodevelopmental factors are likely to play a significant role not only in schizophrenia but also in bipolar disorder[4]. The existence of premorbid cognitive deficits in people with schizophrenia is well established. On the other hand, many patients with bipolar disorder have normal or supranormal cognitive abilities. The fact that relatively large proportion of patients with BP has good cognitive skills is important for explaining observations suggesting a relationship between BP and creativity and premorbid scholastic achievement. It is likely that a neurobiologically distinct subtype of BP might be characterized by normal neurodevelopment and cognitive functioning. It is important to note that there have been some conflicting results in studies investigating the relationship between above average scholar achievement/intellectual abilities and risk for BP. According to results of the studies about premorbid cognitive functioning in BP, the risk for BP is not only associated with good scholastic achievement but also with low premorbid cognitive functioning and scholastic underachievement. These findings suggest a U-shaped relationship between premorbid cognitive impairment and risk for BP in which both poor and good premorbid cognitive/scholar functioning predict BP in adulthood and risk for BP[3]. In a recent quantitative analysis individuals with clinical risk to develop psychosis exhibited deficits with an intermediate degree of severity between healthy control and schizophrenia patients and comparable to the level of impairment in individuals with a "genetic" familiar risk. Moreover, the degree of cognitive dysfunction was more pronounced among clinical high risk that was also at familiar risk. Transition to psychosis was associated with additional cognitive impairment, characterized by deficits of medium/large effect size (ES) across several domains[5]. While evidence for the relationship between premorbid cognitive deficits and schizophrenia is stronger compared to BP, emerging evidence suggests that some patients with BP have also premorbid cognitive deficits. It is important to note that cognitive heterogeneity within and between schizophrenia and BP can have a significant impact on findings of these studies[3]. The presence of substantial cognitive impairment also in first degree relatives (FDRs) of individuals with schizophrenia has provided evidence supporting a role of certain cognitive deficits as candidate endophenotypes for schizophrenia. Meta-analyses of studies on nonaffected adult relatives of schizophrenia patients indicated the presence of medium ES differences as compared to controls in the domains of declarative and verbal memory and executive functions, while smaller ES differences were observed for attention. Consistently, subsequent analyses of cognitive performance in adult relatives of schizophrenia patients confirmed prior findings of moderate ES deficits in tasks of working memory, sustained attention, as well as set-shifting and response inhibition[6].

The presence of cognitive impairment not only in BD patients but also in FDRs led to the hypothesis that specific aspects of cognitive dysfunction may be trait markers of the disorder and potential candidate endophenotypes. For instance, while large ES were observed for euthymic individuals with BD for deficits in verbal memory and in executive functions, FDRs exhibited deficits of smaller magnitude but significantly different compared to healthy controls in verbal memory and executive functions. A subsequent meta-analysis suggested that deficits in response inhibition could be a potential endophenotype for BD and a potential marker of ventral prefrontal dysfunction in BD. In this quantitative study, deficits of

small-to-medium ES were reported for set shifting and verbal memory as well, and have been postulated to represent potential generic markers of psychosis, being candidate endophenotypes for both BD and schizophrenia. Moreover, separate components of sustained attention have been postulated to have a role as potential endophenotypes for BD and schizophrenia[6]. Data from the available meta-analytic literature suggest a significant overlap of impairment between the neuropsychological profiles in established BD and schizophrenia, with differences being more quantitative than qualitative. Finally, it should be noted that both schizophrenia and BD are heterogeneous phenotypes. Maybe, we have to try to understand the disorders in a continuum model instead of dichotomic classification.

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SYMPOSIA ABSTRACT: 517

Mental Health of Iraq Refugee Children in Turkey's Refugee Camp

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ABSTRACT

It is well known that refugee problems have a history that dates back to the period before the invention of writing and that all civilizations have been displaced because of war[1]. Human Rights by the United Nations General Assembly on December 10, 1948. This declaration remains one of the most important documents in the international human rights law[2]. It is reported that people who live in refugee camps, in particular, are at a risk of developing mental health problems stemming from injury, separation from homelands, torture, killing of family members, lack of adequate social support, age- and gender-related factors, and conditions and demographics of the host country[3]. A study on the high rate of PTSD (20%) among Sudanese refugees in the United States attributes this finding to their feelings of discomfort and experience of marginalization and isolation[4]. In Turkey, refugees who live in camps are divided into two groups: municipalities accommodate one, and the other lives in the camps of the Disaster and Emergency Management Presidency. According to data from the latter, in 2015, 258,537 refugees were living in Turkish camps: 36,460 in Kilis, 15,087 in Hatay, 49,956 in four tent camps and one container camp in Gaziantep, 14,000 in Şanlıurfa, and 17,295 in a central tent city in Kahramanmaraş. Most of the Yazidi population resided in the Sinjar district of Iraq. The Uçkuyular, Oğuz, Onbaşı, and Uğurca villages of Batman, Turkey are located close to one another and accommodate people of similar religious beliefs and economic and social conditions. The Yazidi people living in these villages were regularly visited for 10 days in the ninth month after their immigration by two child and adolescent psychiatrists who could speak their native language. The Turkish version of the Kiddie Schedule for Affective Disorders and Schizophrenia–Present and Lifetime (K-SADS-PL) was used in this study. Posttraumatic stress disorder (PTSD) was detected in 20 children (36.4%), depression in 18 (32.7%), nocturnal enuresis in six (10.9%), and anxiety in four (7.3%). The following factors were found to be associated with depression: witnessing violence and/or death, being a girl, having older parents, being the elder child, and having multiple siblings ($p<0.05$)[5].

KEYWORDS

Mental health; refugee; posttraumatic stress disorder; child

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SYMPOSIA ABSTRACT: 522

Mood Disorders and Oxytocin

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ABSTRACT

Major depressive disorder in children and adolescents is characterized by either a depressed or irritable mood or a loss of interest or pleasure and at least four additional symptoms of depression. Depression in children has been reported to be a recurrent and associated with increased psychosocial and medical morbidity and mortality[1]. Oxytocin is a neuropeptide that is synthesized in the supraoptic (SON) and paraventricular (PVN) nuclei of the hypothalamus. Oxytocin is released into the brain, distributed oxytocinergic pathways and receptors are located in various brain regions. This neuropeptide can improve maternal-infant attachment, decrease anxiety and stress. Oxytocin is also released in response to acute psychogenic stressors. Oxytocin inhibits activity in the HPA axis in rats which induced by stress and plays an important role in the response to stress because it has an association with corticotrophin-releasing factor[2]. Many studies have examined oxytocin levels in both major depressive disorder (MDD) and bipolar disorder (BD). These studies have conflicting results. There is a complex relationship between oxytocin levels and mood disorders[2]. Frasch et. al. reported that plasma oxytocin level is found different between patients with MD and healthy controls[3]. Differences are more significant in older patients. In another study, plasma oxytocin levels reported lower in female patients with both MDD and fibromyalgia than in patients with fibromyalgia without MDD or HC. Oxytocin levels and the scores for depression and anxiety were negatively correlate, and oxytocin levels and the scores for happiness were positively correlate. Scantamburlo et al. found a negative correlation in patients with MDD between oxytocin plasma levels both Hamilton Depression Rating Scales scores (HAM-D) and anxiety scores on the State-Trait Anxiety. This finding indicates that comorbid anxiety may be a moderating factor of the effects in depression[4]. Thompson et al. studied on the rs2254298 polymorphism. They studied the association between early adverse parental environment and the polymorphism in predicting poor psychosocial outcomes in 9–14 year-old girls and their mothers. They measured depressive and anxiety symptoms in the girls, and performed genotyping. Heterozygous ('AG') girls with maternal history of recurrent MDD reported higher symptoms of depression, physical anxiety, and social anxiety than did girls without maternal history of MDD and/or with homozygous ('GG') genotype[5]. As a result, oxytocin plays a role in the stress response, and the evidence for a complex interrelationship of the oxytocin system and mood, there is need for larger clinical studies of oxytocin in mood disorder patients. Currently, the lack of available data prevents any supposition about its potential role in the treatment of mood disorders. There are limited data about oxytocin and mood disorders in adolescence.

KEYWORDS

Oxytocin; depression; mood; disorders; adolescent

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SYMPOSIA ABSTRACT: 524

Use of Antipsychotics in Childhood

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ABSTRACT

Antipsychotic medications can be divided according to chemical structure, type of receptor binding, and clinical profile into two main groups: first generation or typical antipsychotics and second generation or atypical antipsychotics. The first antipsychotic drug, chlorpromazine, was introduced in 1952, and then many other antipsychotics were synthesized. A new group of antipsychotics, called second generation or atypical antipsychotics, emerged in the 1980s. This second generation of antipsychotics showed similar effectiveness but fewer extrapyramidal effects compared to first generation antipsychotics[1]. Atypical antipsychotic use in pediatric patients has increased in the 1990s[2]. Antipsychotic drugs have received United States (US) Food and Drug Administration (FDA) approval for only schizophrenia, bipolar mania, irritability associated with autism, and Tourette syndrome in children. Literature shows us most pediatric use of antipsychotics are off label (for conditions which had not been approved by the FDA) [3]. Many hypotheses have been generated to explain the increased use of antipsychotic medications in children and adolescents. Some of these are described below[2].

Greater acceptability of psychotropic medication use in children
Increased knowledge and awareness
Limited access to non-pharmacologic treatments
Demand for quick and affordable treatments
Inadequate provider time and reimbursement for managing behavioral problems
Limited treatment options for vulnerable populations

Olfson et al. (2015) reported for USA National Data that;

- For younger children those receiving antipsychotic medications most often carried a diagnosis of ADHD with aggression and/or disruptive behavior disorders.
- For adolescents, most carried a diagnosis of depression.

Another result of this study was that less than 25% of the children being treated with antipsychotics were receiving any type of talk therapy or family instructions on behavioral control, and many children who were prescribed antipsychotic medications were concurrently prescribed other classes of medication in addition to their antipsychotics.

While the extant scientific evidence about the atypical antipsychotics in youths is growing, much is still not known about the efficacy, tolerability, and long-term safety of these drugs in young people. The AACAP Practice Parameter on the Use of Psychotropic Medication in Children and Adolescents presents a series of principles to guide the clinician when using psychotropic medications in children and adolescents. Those principles are below[5];

- A careful diagnostic assessment,
- Attention to comorbid medical conditions,
- A review of other drugs the patient is being prescribed,
- The creation of a multi-disciplinary plan, including education and psychotherapeutic interventions for the treatment and monitoring of improvement,
- A thorough discussion of the risks and benefits of psychotropic treatment with both the youth and their guardians.

KEYWORDS

Children; adolescent; antipsychotic; typical; atypical

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SYMPOSIA ABSTRACT: 525

Autism in a Developmental Perspective

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ABSTRACT

Autism spectrum disorder (ASD) is a complex neurodevelopmental disorder (NDD) characterized by motor, social and cognitive deficits that develop early during childhood. (Association, 2013). Although ASD is also one of the most heritable brain disorders, as shown by family and twin it is also highly polygenic. For example, 500 genes identified to date can explain nearly 20% studies, of ASD cases (Geschwind, 2011). In addition to genetic factors, various environmental factors also present ASD risks, including exposure to drugs and toxins, viral infections, and pre- and post-natal immune dysfunctions (Chaste & Leboyer, 2012) .

According the studies, underlying mechanisms of autism could be regulated during time dependent windows and critical periods during normal brain development.

Here, we will discuss the developing utility of ASD, as well as current genetical susceptibility and environmental models in a neurodevelopmental approach.

Genetic Factors of Autism

To date hundreds of diverse ASD susceptibility genes have been identified, yet none of the mutations found account for more than a small subset of autism cases. Recent autism gene expression studies highlight genes that are expressed in the brain, immune system, and processes such as cell metabolism and embryology. Various biological processes have been shown to be implicated with ASD individuals as well as differences in gene expression levels between different types of biological tissues (Ansel, Rosenzweig, Zisman, Melamed, & Gesundheit, 2016). Most of the genes surveyed were shown to be consistently down or up-regulated across different source types in different studies. This strongly suggests that, in fact, these genes are not coincidentally higher or lower in ASD but might actually be active players in the underlying pathogenesis of the disorder. In this part of the model, gene expression studies in ASD by tissue types and comorbidities will be addressed .

Environmental Factors (Non-Genetic) of Autism

Strikingly, at least 200 industrially applied or produced chemicals have been associated with neurotoxicity in humans. Through a combination of human epidemiological and animal experimental studies, many environmental toxicants have distinct sensitive time-windows during which exposure may disrupt critical developmental events, thereby increasing the risk of developing autism. The majority of these time-windows occur prenatally rather than postnatally (Heyer & Meredith, 2017). In this part of the panel, environmental toxicants including Polychlorinated Biphenyls (PCBs), lead, arsenic, pesticides, bisphenol A; maternal use of medication during pregnancy including thalidomide, valproic acid, misoprostol, SSRIs; and other environmental factors including maternal infection, ionizing radiation and maternal malnutrition will be addressed.

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SYMPOSIA ABSTRACT: 526

Psychodrama with Children

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ABSTRACT

Psychodrama is a psychotherapy technique developed by Jacob Levi Moreno, whose key concepts include creativity, spontaneity, and action. In order for this technique of psychotherapy to be comprehended, however, the terms of stage, protagonist, director, and *auxiliary egos* must first be understood[1].

- The stage is the area in which psychodrama takes place. No matter the subject of the therapy, it is the stage which allows us to conceptualize that subject.
- The protagonist is the main character of the drama and is the person whose life is brought up onto the stage.
- The director is the person who is responsible for each session of the therapy. The director can also be called the leader or the psychotherapist. There is only one director in each session, however, there can be more co-directors.
- The *auxiliary egos* are those from the group who act out certain people, concepts, and emotions et cetera of the protagonist who is chosen by the protagonist.

During a psychodrama session, emotions, thoughts, and behaviors are expressed through games, thus, a psychodrama session typically consists of a warm-up, action, and a sharing. The warm-up activities build group trust and coherence[2]. For example the group leader may introduce the purpose of the role-plays and then interview each group member about potential scenarios that they may wish to explore through a dramatic experience. The goal is to foster spontaneity and a willingness to try new behaviors and a sense of playfulness. The action is the part where the protagonist and/ or the other participants get to face their lives. And in the sharing part, group members should discuss how the enactment affected them and avoid analyzing the protagonist or offering advice. Sharing with the group leads to bonding and a sense that one is not alone[3]. Children are by their very nature spontaneous. And their tendency to learn through actions and creativity makes them suitable for psychodrama practices. Psychodrama therapies can either be conducted individually (known as monodrama) or as a group. Children are known to benefit from monodrama therapies. However, it is more common to see group therapies in clinic applications of psychodrama. The lower age cap of psychodrama therapies is generally 5-6 years old. For those children who are younger, the application of monotherapy techniques is more appropriate. For those kids who are thought to be capable of handling group therapies, that method may also be used[4]. Being in psychodrama groups presents the children with the opportunity to learn from their own peers as they get the chance to be with and observe children their age. Moreover, they get people such as the director or the therapists whom they can see as role-models. In short, psychodrama groups allow the child to observe different behavioral patterns and to try out different ways of acting without fear of judgement, thus allowing for them to adapt different behavioral patterns. During psychodrama, kids are encouraged to display their emotions related to the difficulties they face during their lives through games. This way, there starts a process where change occurs through actions. Children experience changing roles and trying to understand other people in psychodrama. They can find new solutions to problems[2–5].

KEYWORDS

Children; group; therapy; psychodrama

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SYMPOSIA ABSTRACT: 528

The Neurobiology of Personality Disorders and Implications for Pharmacotherapy

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ABSTRACT

Personality disorder (PD) is the primary psychiatric illness encompasses individuals with interpersonal or psychosocial dysfunction that impairs their social or professional function. The

prevalence of PD among Turkish community in various studies has been reported to be between 3% and 20% but it is important to remember that these statistics are affected by culture and socioeconomic variables. While biomedical and psychosocial aspects of personality disorders are quite complex, advances in neuroscience have furthered our understanding of the role of the neuromodulators and brain circuitry in the pathogenesis of psychopathology. Personality is seen as organized around basic psychological dimensions and this alternative approach to the diagnosis of personality disorders was included in American Psychiatric Association (APA) Diagnostic and Statistical Manual of Mental Disorders (DSM-5). As there are many dimensions of variation in personality, it has been identified four broad psychological dimensions based on disturbances in the domains of mood/affect, impulse/action, attention/cognition, and anxiety, each of which is grounded in biology and genetics. These dimensions can be formulated as affective dysregulation or instability, impulsivity, cognitive disorganization, and anxiety. Understand the neurobiological mechanisms of these dimensions may help clinician's in daily clinical practice. The prefrontal cortex controls planning, emotions and selection of appropriate behaviors. Excessive aggressive behavior represents a failure of cortical "top-down" control of limbic emotional systems hyperresponsive to events or situations that can trigger frustration, anger, or fear. According to previous studies, a low threshold for impulsive aggression, as observed in borderline and antisocial personality disorders has been related to excessive amygdala reactivity, reduced prefrontal inhibition, and diminished serotonergic facilitation of prefrontal controls. Affect instability is characterized by marked, brief shifts from baseline mood to depression, irritability, anxiety or by a marked reactivity of mood. Affective instability mediated by excessive limbic reactivity in gabaminergic/ glutamatergic/ cholinergic circuits, resulting in an increased sensitivity or reactivity to environmental emotional stimuli as in borderline personality disorder and other cluster B personality disorders. The modulation of limbic structures, including the amygdala, the entorhinal cortex, and the insula, is likely dependent on glutamatergic/gabaminergic balance in interaction with the cholinergic system Studies with bipolar affective disorder (BPD) showed that valproate, which enhances gabaminergic activity, has stabilized affect-driven impulsivity and topiramate and lamotrogine, which reduce glutamatergic activity, as well as enhancing gabaminergic activity, have been reported to ameliorate BPD symptoms (Nickel et al. 2005). While affect instability is a part of personality disorders, especially B cluster disorders, same treatment approaches has been used and showed efficacy in patients with personality disorders. Cognitive dysfunction seen in many personality disorders. In addition, serious cognitive disorganization, manifest in odd speech, disturbed thinking patterns, eccentric appearance is a hallmark of cluster C personality disorders. Disturbances in cognitive organization and information processing contribute to the detachment, desynchrony with the environment, and cognitive/perceptional distortions of cluster A or schizophrenia spectrum personality disorders. Several studies findings suggest that dopaminergic activity can be relatively increased or decreased, depending on the predominance of psychosis-like or deficit-like symptoms (Siever and Davis 2004). Increases in dopamine activity are associated with hypervigilance and stereotypic cognitions/behaviors that are precursors of psychosis, while decreases in dopamine activity are associated with deficits in working memory, cognitive processing, and hedonic tone (Siever and Davis 2004). Reduced dopaminergic and noradrenergic activity in the prefrontal cortex may contribute to the cognitive impairment in SPD. Pharmacological intervention studies with amphetamine, guanfacine and pergolide suggested improvements in working memory, executive function, and, to a lesser degree, sustained attention and verbal learning, as well as improving the deficit-like symptoms. A low threshold for anxiety may contribute to the avoidant, dependent, and compulsive behaviors observed in cluster C personality disorders. These alterations in critical regulatory domains will influence how representations of self and others are internalized.

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SYMPOSIA ABSTRACT: 529

Evaluation of Frontal Lobe Function

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ABSTRACT

In its modern form, cognitive neuropsychology is relatively young. In cognitive psychology, frontal lobe functions are immensely important. Neurophysiologists has been seen the frontal lobes mainly as structures for regulating voluntary movement. Just 64 years ago, in 1953 H.G.M had been operated for temporal lobe epilepsy. He had bilateral medial temporal lobe resection along with the hippocampal formation and adjacent structures, including most of the amygdaloid complex and entorhinal cortex. He lost his memory on an operating table. His acquired memory deficit played a very important role for the development and understanding of cognitive neuropsychology. And as for today, "do we really know so much about cognition?". Prefrontal cortex, constitutes almost one-third of the neocortex, is a late developing region and has its own, almost separate individual maturation and evolution. Recent neuroanatomical, neuropsychological, and functional imaging literature, clearly shows that prefrontal cortex and subcortical structures are also closely involved in regulating higher cerebral processes that control cognition. Five parallel circuits have been described between the frontal lobe and basal ganglia. Two of these circuits are related to motor function. The remaining three loops are connected with non-motor areas in the frontal lobe. A dorsolateral prefrontal circuit, which mediates "executive" functions; an anterior cingulate circuit, which is involved in motivational mechanisms and an orbitofrontal circuit, which has lateral and medial divisions. The medial portion of the orbitofrontal circuit allows integration of visceral-amygdala functions with the internal state of the organism, while the lateral portion is involved with integration of limbic and emotional information into contextually appropriate behavioral responses. Because of this, assigning parts of cognitive function to separate lobes is not a useful approach. Furthermore, some cognitive tests such as Wisconsin Card Sorting and Stoop Tests are sensitive but not specific to frontal lobe lesions. "Frontal Lobe Syndromes", "Frontal Network Syndromes", "Executive Dysfunction" and "Metacognition" are all different terms which have been used to describe disorders involving the frontal lobes. The appropriate term seems to be "Frontal Network Syndromes" as it emphasizes the extensive network between the frontal lobes and other parts of the brain.

Many neuropsychiatric disorders such as Tourette's Syndrome, Huntington's Disease, Obsessive-Compulsive Disorder, Attention Deficit/Hyperactivity Disorder, Schizophrenia, and Mood Disorders may result from disturbances that have a direct or an indirect impact on the integrity or functioning of the prefrontal cortex and its circuits. For the last 60-70 years, neurologists have been examining their patients' cognitive functions to facilitate differential diagnosis (e.g., Alzheimer's Type Dementia or Frontotemporal Dementia). Psychiatrists have been checking their patients' cognitive functions to decide which condition better explains a clinic presentation (e.g., Depression or Dementia). Neurosurgeons had used cognitive function tests in the past for localization of lesions. Improvements in imaging technology solved most of the diagnostic problems. Now, needs are changing. Clinicians and scientists wish and expect to pinpoint underlying pathology and discover mechanisms of disease.

People with cognitive dysfunction have problems in daily life. Researchers and clinicians observe some of them well. But documenting these observations in a scientific manner for diagnosis, for follow-up and evaluation of the efficacy of treatment as well as disease progression and/or remission. To evaluate the Frontal Network Syndrome, many tests can be used. Some of them can be performed bedside, some of them take a long time and some of them require special equipment. In day to day clinical practice, time is of essence. Aspects of cognitive function such as attention and working memory, must be practically evaluated in Frontal Network Syndromes, in as short time as possible. Wisconsin Card Sorting Test (WCST), Stroop Neuropsychological Screening Test, Luria Test, Letter and Category Fluency List Generation and Boston Diagnostic Aphasia Evaluation Test are well known, widely used cognitive function tests. In recent years several computerized screening test have become available. Mind Streams, CANTAB, CNS Vital Signs and MOXO d-CPT (distracted-Continuous Performance Test). As computers are becoming increasingly involved in our daily lives, their undeniable and enormous effect in scientific advancement is becoming apparent as well. Clinicians' and scientists' making use of computerized tests is expected to increase in the near future. These computerized tests are continuing to be developed. TOVA and Conner's Test, which are continuous performance tests for frontal network systems to detect ADHD, have been used for many years. A new continuous performance test, MOXO d-CPT has been shown to have high sensitivity for frontal network dysfunction. Our day to day lives include so many distractors that paying attention to a specific task is in itself a challenge in daily life. Until recently, all tests used to evaluate attention were performed in pretty much idealized laboratory conditions, different from real life. With MOXO d-CPT mimicking real life conditions with distractors, it is possible to at least outline the effects of visual and auditory distractions on other aspects of cognitive function.

SYMPOSIA ABSTRACT: 530

Autism Spectrum Disorder and Normal Sexual Development

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ABSTRACT

Dreams… Plans… Chromosomes… Hormones… Organs… Feelings… Attitudes… Imposed…

After "Is the baby healthy?", the property we wonder most is " What is the baby's gender?" Although there are many stages of life and development periods, gender and sexuality and related processes arouse excitement, horror, and interest the most. Such as circumcision, military service, and marriage…There may be demands and expectations regarding the gender when we plan to have a baby. 'We want a girl.' Whatever we want or imagine, there is a gender that accompanies the baby existentially and decided by fertilization. XX or XY. The most significant factor in the sexual development of humans biologically is chromosomes and hormones expressed by the effects of chromosomes; organs shaped by the hormones. However; starting from birth, families and other social environment codes to children gender-specific responsibilities, rights, expectations, and obligations, which can be totally called as social roles. If expectations are compatible with the realities, there is not any problem most of the time but not always. Such as the choice and orientation of the child do not match expectations of the families and biological facts…The first condition of noticing abnormal situations is knowing normal properties and noticing. Children development goes by physical growth, cognitive and mental maturation. All these areas are individually participating in and support the process of sexual development of the child. What are the features of a healthy sexual development and how does it happen? There are theoretical approaches that address human development with different views: psychosexual, psychosocial, social learning, cognitive learning, etc. A common property of theories is that the developmental process is divided into stages. Projected course of the development is universal. Although there are not sharp boundaries between period features, none of the steps is skipped. Physical development of the body and the spiritual and mental development may not occur concurrently; each can take place in a different stage. In Freud's theory of the psychosexual development, the most prominent of these theories, attributed great worth to gender and sexuality concepts not only in the sexual identity development but also in spiritual development and personality development of the individual. However, although the main factors that are proposed to have roles in the human development and highlighted in other theories are different, if they are considered holistically, they can serve as a guide to understanding all the aspects of sexual development similar to other development areas. If we briefly discuss, in the light of the available information, children's developing gender and their interest in sexuality specific to children's age and development period. The discovery of the body and in the world up to the age of three. The discoveries of the regions of pleasure such as mouth, anus and genitals, which changes with age consecutively, and a number of accompanying emotional changes. After the age of three, awareness about girl and boy identity and determination of own sexual identity, identifications, pReferences. During this period, awareness of tangible situations related to differences between names, clothes, hair lengths, hair pins, played games and toy pReferences. Curiosity for opposite sex is satisfied through playing games such as family and doctor. At the same time, families give, involuntarily and spontaneously in most cases, messages that transmit their expectations regarding sexual identity. Boys don't cry, girls should act like a lady… Children's behaviors and attitudes compatible with their sex are approved and stiffened with encouragement. Asynchronous behavior according to the family are ignored….For girls; my diligent girl, my skillful girl; for boys; my strong son, my lion son. Children start to make an effort to learn, interiorize, and live these social roles and expectations related to sexual identity. And on the other hand, questions such as 'where did the baby come from?', 'How did the baby get out? ', 'How did the baby enter into mom's belly', etc. occupy children's mind; and their knowledge and beliefs on sexuality are formed by the answers to these questions. The most intense sexual interest in children is in the pre-school period. Interest would fall during school period and pushing to subconsciousness and suppression by superego with avoiding sexual topics, shame and prohibitions follow. After this latent phase, genital period also known as puberty begins[2,3]. Autism spectrum disorder (ASD) is a common neurodevelopmental disorder which symptoms present in the early developmental period and characterized by persistent deficits in social communication and social interaction, restricted, repetitive patterns of behavior, interests or activities[1]. The etiology of this disorder, however, is largely unknown, although emphasized that the significance of interaction between non-genetic risk factors and the known and unknown genetic predispositions. Whether children with Autism Spectrum Disorder (ASD), who may have problems with overall identity development, could develop sexual identity is debated

KEYWORDS

Autism spectrum disorder; sexual development; childhood; adolescence

for a long time. However, In a study of Abelson on children with ASD, it was demonstrated that children with ASD have the potential to develop sexual identity although this mostly depends on cognitive skills and intellectual age[4]. In this presentation, I am going to attempt to answer the following questions: How sexual development does happen in children with ASD, who has impairments in social skills, limitations in verbal and nonverbal communication, repeated interests and behaviors, and deteriorations in executive functions? Are these theories and hypothesis proposed for healthy children also suitable and sufficient to understand and evaluate sexual development in these children? What are the potential effects on the normal sexual development of the theories suggested to explain the etiology of ASD, such as the extreme male brain theory[5] and the neuroendocrine hypothesis based on the oxytocin/cortisol balance[6]? How social sexual identity development in children with ASD could be affected according to mind theory[7]? Sexual development in children with ASD will be discussed in the light of current research by mentioning sexual roles, sexual orientation and sexual identity development and problems in children with ASD.

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SYMPOSIA ABSTRACT: 531

Autonomic Dysregulation in Anxiety Disorders

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ABSTRACT

The autonomic nervous system is the part of the peripheral nervous system that controls involuntary movements and organ functions. It is inadvertently affected in situations such as heart rate (HR), digestion, respiration, salivation, sweating, menses, defecation, sexual arousal. It is divided into parasympathetic nervous system (PNS) and sympathetic nervous system (SNS). The sympathetic system sends signals to tissues and organs, generally acting in a way that increases the activity of the body, energy consumption. The parasympathetic nervous system slows down our movements. The parasympathetic system sends signals to tissues and organs and generally acts in the body to maintain energy. SNS and PNS are interdependent regulatory systems that act on different time scales. Normally balanced sympathetic and parasympathetic branches of the autonomic nervous system (ANS) was historically described as "autonomic tuning," in contrast to the disorders of arousal which are characterized by ANS dysfunction, affective lability, anxiety, stress, and emotional disorder. Recent studies of the relevance of the ANS to stress and mental disorders are becoming increasing in number 1. ANS dysregulation impacts on both physical (increasing cardiovascular risk) and mental (compromising psychological well-being) health at multiple levels. Loss of regulation of normal autonomic control of cardiac adjustment to environmental stressors thus leads to negative impacts on physiological function affecting arterial blood pressure, heart rate and rhythm, and vagal afference. Tonic PNS activity under basal conditions keeps the heart rate lower in comparison to HR of the denervated heart, e.g., after heart transplantation. Consequently, fast HR increases are mediated by parasympathetic withdrawal, before sympathetic activation will mediate a slower and larger HR increase. Most studies suggest an association between psychopathologies and prolonged ANS imbalance with sympathetic hyperactivation. However, research in anxiety disorders have recently focused beyond sympathetic activation, examining also the role of the parasympathetic branch in ANS dysregulation, although it is well established that the PNS does not influence HR independently of the SNS. Dysregulation of the autonomic system has been associated with the physical description of anxiety. The nature and severity of somatic responses to strase can vary widely. The most common symptoms are muscle tension,

palpitations, frequent breathing, dizziness, and feelings of incontinence, urinary frequency and defecation. In addition, pupil dilatation, percent fever, piloerection and tremor can be observed. Muscle tightness increases. These are due to the parasympathetic and sympathetic stimulation of stress hormones such as epinephrine, norepinephrine, cortisol, growth hormone and prolactin. Patients with anxiety show higher muscle tension and decreased autonomic variability than those who are not anxious even at rest.

Increased fatigue, hypertension, hyperlipidemia, obesity, myocardial infarctions, cardiovascular risk, and low-grade inflammatory excess state have been repeatedly reported in anxiety disorder patients. Although the precise brain processes and mechanisms preceding and following the state of hyperarousal in anxiety disorder remain unclear, a central regulation by the CNS is presumed with the prefrontal cortex (PFC) and amygdala playing a major role in influencing allostatic systems through the vagal nerve. PFC hypofunction, as observed in PTSD, underlies the deficient rational control of emotional responses resulting in exaggerated amygdala activity. Amigdala is the region that has the most important role in feeling anxiety and fear. Dysregulation of the central autonomic network through different autonomic core centers, from PFC via the amygdala and hypothalamus to the brain stem, is expected to alter peripheral ANS activity and eventually the dynamics of heartbeat intervals. Retrograde anatomical labeling studies identified the complete autonomic network innervating the heart in rats. The role of the amygdala as trigger for arrhythmogenesis in the malfunctioning heart has been demonstrated in the pig. Transient amygdala inactivation by local cooling abolished the arrhythmias elicited by restraint stress in pigs previously subjected to transient occlusion of coronary arteries.

Differences in anxiety and heart rate and heart rate variability were found in different studies. In a study conducted by Agorastos et al. in 2015, an increase in heart rate was reported in anxiety patients. In some studies, anxiety has also been shown to reduce heart rate variability. Autonomic dysregulation increase heart rate and reduce HRV.

In the study of Alkozei et al. in 2015, no group differences at rest or in response to stress were found. The findings suggest that childhood anxiety disorders may not be characterized by inflexible autonomic responding, and that previous findings to the contrary may have been the result of differences in subjective anxiety between anxious and nonanxious groups during the tasks, rather than a function of chronic autonomic dysregulation.

In a study of Dennis et al. in 2014; for individuals with posttraumatic stress disorder (PTSD), a disorder characterized by hyperarousal and frequent physiological symptoms related to anxiety and stress, dysregulation of the autonomic nervous system has been identified as an important precursor to cardiovascular disease, diabetes, and other health risks. Indeed, a key indicator of autonomic functioning and cardiovascular health, heart-rate variability (HRV), is often depressed amongst individuals with PTSD. Although the link between PTSD and HRV is generally discussed as a purely psychosomatic phenomenon, a number of behavioral risk factors- namely smoking, alcohol misuse, obesity, and sleep disturbance- may account for this link. In this study, HRV was assessed amongst younger adults (18- to 39-years-old) with and without PTSD to determine whether autonomic dysfunction in individuals with PTSD is in part attributable to the higher rates of smoking, drinking, obesity, and sleep disturbance that often coincide with PTSD. The SNS stimulates excitation (e.g., increased heart rate and blood pressure) in response to unexpected changes in the body and/or environment through the release of catecholamines. The PNS restores cardiovascular activity to baseline levels via vagal innervation. When these two systems are in disequilibrium- either because the SNS is hyperactive or the PNS is hypoactive-HRV attenuates. Low HRV is both an indicator and a precursor of disease. It may signal some underlying irregularity, such as immune dysfunction resulting from diabetes, osteoporosis, arthritis, Alzheimer's disease, and some cancers. Reduced HRV may also stimulate deleterious effects on cardiovascular health. Lower HRV is a risk factor for arrhythmia and in turn is predictive of heart disease and cardiac arrest. Reduced HRV may also accelerate atherosclerosis and result in increased variability in blood pressure, which is itself an independent risk factor for coronary artery disease. Exposure to psychological trauma increases the risk of developing PTSD, a disorder characterized by persistent re-experiencing of the traumatic event, avoidance of stimuli associated with the event, and increased arousal. These symptoms have long been known to convey autonomic dysregulation, such as elevated heart rate and increased blood pressure both at baseline and in response to stressors. Even though the SNS is instrumental in the etiology of anxiety disorders, experimental evidence suggests that the PNS may be responsible for the maintenance of elevated physiology in psychopathology. For instance, the administration of catecholamine (SNS) antagonists prior to induced panic attacks does little to reduce heart rate, suggesting that the SNS plays a minor role in such attacks. However, lactate, which is a known suppressor of vagal (PNS) activity, accumulates during panic attacks, and is even administered in laboratory studies to induce panic attacks and stimulate PTSD symptoms. Thus, suppressed PNS activity and consequently reduced HRV are implied in individuals with anxiety or trauma related disorders such as PTSD. Indeed, individuals with PTSD exhibit reduced HRV in both short-term laboratory-based measurements of HRV and 24-hour ambulatory measures. In turn, individuals with PTSD are more likely than those without PTSD to develop cardiovascular disease and face an increased risk of cardiac death.

Although reduced HRV in PTSD is primarily attributed to the direct impact of psychological hyperarousal and anxiety on the autonomic nervous system, behavioral risk factors could partially account for that association. Individuals with PTSD are more likely than individuals

without PTSD to smoke and do so heavily, abuse alcohol, be obese, and suffer from sleep disturbance stemming from flashbacks and nightmares. Each of these risk factors are independently associated with reduced HRV, suggesting that the relationship between PTSD and HRV may in part be due to the behavioral health risks that frequently accompany PTSD.

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SYMPOSIA ABSTRACT: 534

Women with Autism: Are There Any Differences?

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ABSTRACT

Gender has been recognized as a risk factor for mental ill health and gender differences in psychopathology are well recognized. Women are more likely to be diagnosed with internalizing problems, such as affective and anxiety disorders, whereas men are more likely to meet diagnostic criteria for externalizing problems such as substance use. The male predominance of Autism Spectrum Disorders (ASD) is one of the best-known, and at the same time, one of the least understood characteristics of these disorders. The ratio of boys to girls with ASD is commonly reported as 4:1 overall and 10:1 for ASD without intellectual disability[1]. How this male dominance relates to etiologies and liability of autism and how it shapes the research, clinical practice and the service development have been widely discussed with little consensus. Gender issues in assessment: Standardization samples for ASD instruments consist of predominantly males with a ratio of approximately 3:1 and this raises the issue of validity of diagnostic criteria by gender. It has been increasingly discussed in the literature whether separate criteria content/cut-off scores based on gender are required. Gender differences in prevalence: Due to higher ratio males, some researchers suggested female protective model suggesting that girls may develop ASD only if they experience a greater etiological burden which also relates to lower average IQ in female ASD population. Some other studies described ASD as an extreme expression of male behavior even in females[2]. Representation in research: Girls with ASD are poorly represented in relevant studies because of the difficulty in recruiting large enough sample to make statistically significant comparisons. Gender differences in IQ: The male to female ratio gets larger in people with high functioning autism (10:1). This might suggest that females need extra/more pathology to develop autism. Although there is enough evidence to suggest overlapping genetic factors related to ASD and ID, the causal relationship between ID, ASD and gender differences remain unclear and warrants further investigations. Gender differences in ASD symptoms: Small evidence suggests that there may be female/ male phenotypes of autism. The phenotypic gender differences between boys and girls with ASD seem somewhat similar to the typically developing children.

Age of diagnosis:

Girls are more likely to develop strategies to mask their ASD symptoms and get diagnosed late or even not diagnosed at all. They are less likely to present with problem behaviors to get the attention of parents or professionals. Some blamed the culture for this such that girls are brought up to be more considerate and socially accommodating. What happens after diagnosis? Girls with ASD can face different or harder challenges than boys. Girls have less chance to develop social skills at special education and other service environments as there will be far less females using these services which are usually designed for male prototypes. Limited number of outcome studies report that girls are likely to be more socially isolated

KEYWORDS

Autism; ASD; gender; female; woman

and less likely to have gone to college or have paid employment[4]. It has been consistently documented that girls are more likely to be underdiagnosed or diagnosed late. They have poorer outcome than their male counterparts. It can be argues that women with ASD face double disadvantages; one from the neurotypical female population and the other is from the male with ASD[5]. Hence it is of paramount importance to do more research to investigate the different presentations and needs of female population in order to better understand the differences and develop gender-sensitive services and give women a chance of better outcome. This will obviously require involving more women in research samples and being more attentive to them and their carers' needs and wishes.

Table 1. Anecdotal Descriptions About Behavioral Sex/Gender Differences in Autism[3]

Domain	Characteristics More Often Present in Females Than in Males
Social interaction	Greater awareness of the need for social interaction Desire to interact with others Passivity (a "loner"), often perceived as "just being shy" Tendency to imitate others (copy, mimic, or mask) in social interactions, which may be exhausting Tendency to "camouflage" difficulties by masking and/or developing compensatory strategies One or few close friendships Tendency to be "mothered" in a peer group in primary school but often bullied in secondary school
Communication	Better linguistic abilities developmentally Better imagination (fantasizes and escapes into fiction and pretend play, but is prone to being nonreciprocal, scripted, and overly controlled)
Restrictive repetitive patterns of behavior, interests or activities	Restricted interests tend to involve people/animals rather than objects/things (e.g., animals, soap operas, celebrities, pop music, fashion, horses, pets, and literature), which may be less recognized as related to autism
Other	Tendency to be perfectionistic, very determined Tendency to be controlling (in play with peers)

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SYMPOSIA ABSTRACT: 535

Ways to Rediscover the Past: Prolonged Exposure Therapy

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ABSTRACT

Posttraumatic stress disorder is a psychiatric disorder that affects the quality of life and social functioning of a person who develops after a life-threatening, unpleasant or desperate life threatening person's life and body integrity. Most of the symptoms of post-traumatic stress disorder, which can be transmitted to the traumatized person, fall short of being diagnosed within a year but in a significant part the indications will be continuous. Treatment of the disorder is done by using pharmacological and psychosocial therapies together. When we focus on psychosocial treatments, we see that the most work in this area is related to cognitive behavioral therapies. It is known that cognitive behavioral therapies improve symptoms of post-traumatic stress disorder by 70% in combination and single use. Therefore, many treatment guidelines have included cognitive-behavioral therapy as a first-line treatment option. Exposure is an application to reduce "pathological fear" and its related

feelings that are common in post-traumatic stress disorder and other anxiety disorders as a commonly used behavioral treatment technique. During exposure application, patients are faced with exaggerated objects, situations, thoughts and similar stimuli to reduce anxiety levels under control. Exposure practice can be done in two different ways: "in vivo exposure" or "imaginal exposure". Prolonged exposure therapy, which began in the 1980s and has been shown to be effective with various empirical studies, is a special treatment program designed for post-traumatic stress disorder treatment and is based on "emotion processing theory". It has been reported that prolonged exposure therapy has a high efficacy in improving post-traumatic stress disorder, depression and anxiety symptoms in the results of all day-to-day studies. This treatment program has been developed with the addition of new cognitive and behavioral techniques to the basic structure of behavioral techniques over time. This treatment program consists of four main parts: 1. Psycho-education about trauma and post-traumatic disorders, 2. Breathing exercise education, 3. In vivo exposure, 4. Imaginal exposure. The program consists of 1 to 2 times a week, 60-90 minute interviews and takes 9–15 weeks in total. According to the theory of emotional processing, which is the theoretical basis of prolonged exposure therapy, fear is represented as a cognitive structure that allows danger to escape from memory, and this structure against danger is called "fear structure". This "structure of fear" consists of objects or situations (stimuli) that reveal fear, physiological and psychological responses to stimuli, and implications related to these stimuli and reactions. The traumatic moment of the traumatized person repeatedly activates with emotional participation, recalling the thoughts and feelings associated with the event, sharing with others, correcting information about the world and the self, confronting the objects and situations reminding trauma in real life activates the trauma memory again and again. In this way the memories are organized appropriately and the natural healing process takes place. This condition, called emotion processing, reduces the belief in the belief that the world after trauma is completely dangerous and that the person is totally inadequate. According to emotion processing theory; the most important cause of the natural healing process in the person experiencing trauma is the cognitive and behavioral avoidance of traumatic memories and all the situations that cause the responses to be resurrected. Avoidance prevents trauma memories from being processed, causing the structure of fear in the memory of a person to become pathological (pathological fear), and therefore the disorder becomes processive.

The purpose of prolonged exposure therapy is to assimilate the knowledge of fear that has become pathologic after trauma in the previous memory system and to improve the process of the feeling about trauma as it is healing from the natural path. In order for the pathological structure of fear to be rearranged into the previous memory system, two conditions must be met. The first thing to do before these conditions is to activate the "fear structure" because if not activated, it will not be available for editing. Secondly, it is possible to "reverse" other information to the teaching/ conditioning of the traumatic event (e.g., all men are dangerous, etc.) simultaneously with the activation of the fear structure. If these two conditions are met, information about the incident can be absorbed in the existing memory. Through systematic implementation of this method the patient begins to see the invalidity of his (negative) thoughts, learns that the anxiety is diminishing over time and can conquer it, learns that the trauma distinguishes itself from trauma-like situations, thinking of trauma is a worrying situation, which it is different, and that thinking is not a danger on its own. All these awareness provide cognitive restructuring and allow the patient to reassess the traumatic event in a new way of thinking and in a safe environment during therapy. All of these changes help to reduce the number and severity of symptoms, and to provide a feeling of superiority and competence over the event, leading to major depression, generalized anxiety, anger, and guilt symptoms that develop after trauma. The Prolonged Exposure therapy program consists of 60–90 minutes of talks that last an average of 9–12 weeks. Following the first interview after the evaluation interview (interview 1), a review of the treatment program for the patient is presented, and the rationality of the treatment is explained. Methods used in treatment are introduced. Information about the patient's index trauma, posttraumatic state, and life before the trauma or trauma-like events are collected. In the last part of the first interview, breathing exercises are done with the patient for 2–3 minutes by giving information about breathing exercises. In the second interview of the therapist, psychoeducation is given about the common responses to trauma and symptoms associated with PTSD. Common symptoms in the disorder are explained. Cognitive avoidance, behavioral avoidance, and nonfunctional cognitions about the world and the self, which are the three important factors that play a role in the extension of posttraumatic problems, are emphasized. First, the rationality of in vivo exposure is discussed after the avoidance is addressed and the results are evaluated. Avoidance behaviors (person, object or situation, etc.) are determined. The patient is introduced with SUDS (Subjective Unit of Distress Scale) scores and is listed in a hierarchical order according to the level of distress experienced by the avoidant behaviors (1% distress to 100% distress). The initial in vivo exposure task is chosen to include a level of distress that the patient can make. This is usually an exercise that causes 40–50% subjective difficulties. In the last part of the interview, talk about the concerns that may arise during in vivo exposure, emphasizing the importance of breathing exercises. During the first in vivo exposure, information about the expected change in anxiety level (habituation) is given and the patient is alerted to possible safety seeking behavior or avoidance. The first in vivo exposure experience must be successful, the

fear structure must be activated, and the corrective emotional experience must be experienced. The third interview begins with the control of the first homework. The patient should always be asked what they learned from this first homework. Following the disclosure of the rationality of imaged exposure, it is started to be applied on index trauma which is previously determined. The patient is asked to sit comfortably, to close his eyes and to describe the moment of trauma, as if it were the case at the moment, with the clues established at the present time. This process takes about 45–60 minutes and is recorded on the one hand. This patient is eventually encouraged to talk about patient trauma, so the information processing process is ensured and imaginary exposure is processed. It is expected that the level of anxiety will be reduced and the patient will be able to talk about emotional thoughts on trauma. At the end of the interview, the in vivo exposure application is selected from the list. Voice recordings are delivered to the patient. The first imaginary exposure homework is given together with the in vivo exposure homework. The patient is asked to listen to the sound recording with the headphones closed for at least 45 minutes a day, in a focused state. The main structure of the interviews after the 3rd interview (4th to 11th interviews) is roughly similar. The sessions begin with homework control and the difficulties that arise during the practice of homework are addressed. Again, the trauma is reported within each time period (imaginal exposure), and then the processing section of the talk about trauma consisting Again, the trauma is reported within each time period (imaginal exposure), and then the processing section of the talk about trauma is passed. As negotiations progress, the patient is asked to enter details in the trauma story. There may be some parts of the narcissistic anxiety that the patient has difficulty telling during the trauma story. These sections are called "hot spots". On further interviews, the patient is asked to detail "hot spot" moments and to tell them many times until anxiety is reduced (habituation). This process is detailed in repetitive sessions and processing continues.

Therapy ends on average twelfth session. However, according to the situation of the patient, it can be extended until the twentieth interview. Last interview begins with homework control. Imagine exposure is performed for 20–30 minutes and the entire traumatic moment is explained once again. After the relapse prevention talks, the therapy is terminated by asking the patient about the changing factors of the trauma until now and the thoughts about the treatment effect.

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SYMPOSIA ABSTRACT: 536

Tic Disorders and Attention Deficit Hyperactivity Disorder

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ABSTRACT

Attention deficit hyperactivity disorder (ADHD) is the most common comorbidity seen in tic disorders (TDs). It has been reported that combined type ADHD is more common in tic disorders. Presence of TDs in association with ADHD doesn't affect the symptoms ADHD; however, it aggravates tic-related symptoms. Even in such condition, it should be taken into account that ADHD diagnosis is more important for long-term dysfunction. There are other comorbidities in majority of cases having these two diagnoses (70%). In addition, comorbid ADHD increases risk for development other comorbidities in TDs[1]. In population-based studies, comorbid ADHD was observed in 38% of cases with TDs. In clinical samples, ADHD was observed in 60% of children and 40% of adults with TDs. It is particularly observed in male patients. Mostly, ADHD symptoms are firstly recognized at 3–5 years of age, appearing

2-3 years before onset of tics[2]. Thus, debates whether drugs used for treatment of ADHD trigger tics in children and adolescents could be affected by onset of complaints. In general, it is accepted that disruptive behavior disorders and aggression could be increased in the presence of comorbid ADHD diagnosis with TDs and that self-perception and anger control are associated with ability to focus in these children. Comorbid ADHD diagnosis increases risk for additional psychopathologies in TDs and worsens their course. In fact, in a study on 5247 cases, it was reported that obsessive-compulsive disorder (OCD) was increased by 1.4 folds whereas specific learning disorder by 3.7 folds, mood disorders by 1.9 folds, anxiety disorders by 1.2 folds, conduct disorder and oppositional-defiant disorder by 6.1 folds, sleep disorders by 1.7 folds and anger control problems by 3.2 folds in children with association of TDs and ADHD when compared to those with TD alone. Again, in the same sample, both self-harming behavior and coprolalia/copropraxia were increased by 1.6 fold in the association of TDs and ADHD. It was also reported that problems related to social skills were observed 3.3 folds more frequently in these cases[1]. In adult patients with association of TDs and ADHD (n=1628), it was seen that the most common comorbidities were obsessive-compulsive disorders and mood disorder (Odds Ratio, OR: 1.5 and 1.3, respectively); in addition, anger control problems (OR: 2.8) and problems related to social skills (OR: 2.2) were maintained. In adult tic disorders, association with ADHD can be missed and assessment regarding ADHD is recommended[1].

Tic Disorders and Obsessive Compulsive Disorder

Obsessive compulsive disorder and sub-threshold symptomatology may be added to TDs as well as tics may be present in cases with OCD. In fact, prevalence of TDs and tics has been reported as 7% and 20% in cases with OCB, respectively[3]. OCD is second most common comorbidity in TDs and it is generally accepted that it has a hereditary association with tic disorders. Threat perception related to obsessions is less clear in the association of TDs/chronic motor and vocal tic disorder (CMVTD) and OCD[1]. Again, compulsions and "exact localization rituals" are prominent in such cases. Thus, it may be difficult to distinguish compulsions and rituals from complex tics. In addition, sexual, religious and somatic obsessions involving violence, counting rituals, tic-like compulsions and saving are frequent in these patients. It has been reported that OCD accompanied by tics has an earlier onset and fluctuating but persistent course and that it is more prevalent among male patients[2]. This association increases risk for other psychopathologies and worsens prognosis of TD.

In children with TDs, trichotillomania, scratching skin, nose picking, finger cracking, teeth grinding while awake, self-harming, sniffing, splitting and opposite impulse phenomenon are more frequently seen in the presence of comorbid OCD. Among comorbidities in TDs, OCD is linked to perinatal problems, intrauterine alcohol/nicotine/caffeine exposure. In cases with TD/CMVTD, the diagnosis and symptoms of OCD may be more important for dysfunction. In these cases, OCB may not improve over time unlike tics. It is thought that cases without saving and obsessive compulsive personality traits which show early response to therapy have better prognosis[1].

Tic Disorders and Aggression

In an international study, it was found that point and lifetime prevalence of anger control problem/aggression were 25% and 37% among cases with TDs, respectively. Aggression seen in TDs is mostly reactive and associated to ADHD symptoms. In such cases, oppositional defiant disorder (ODD), affective disorder (AD) and OCD diagnoses may be increased; however, it is thought that ADHD has primary importance for aggression. It has been reported that executive function disorders (EFDs) may promote aggression in cases with TDs. Aggression worsens prognosis and increases risk for additional psychopathology in patients with TDs[1].

Tic Disorders and Other Anxiety Disorders

Anxiety disorders are one of the most commonly seen psychopathologies in children and adolescents. These disorders are most frequently associated to other anxiety disorders. Available data suggest that prevalence of anxiety disorders show no marked increase in children with TDs. For example, in the study by Specht et al., ADHD, social anxiety disorder, generalized anxiety disorder and OCD were found in 26%, 21%, 20% and 19% of children with CMVTD, respectively. In children with TD, anxiety disorders are also linked to sleep problems, acute alterations in mood and trichotillomania. Based on available data, it may be suggested that comorbid anxiety disorder is present in TB and should be evaluated systematically[1].

Tic Disorders and Affective Disorders

It has been reported that affective disorders, primarily major depressive disorder (MDD) and persistent depressive disorder (dysthymia in DSM-IV-TR), can be frequently associated to TDs; however, these diagnoses are commonly missed. In general, it is accepted that there is interplay between affective disorders and TDs, and both disorders may aggravate each other. Regardless of diagnosis of affective disorder, temporary sub-threshold depressive state and passive suicidal thoughts may be present in periods where tics are increased. It was reported that such complaints and findings can appear as a response to sensation of failure and social challenges caused by loss of control on tics; however, each case should be assessed in details for a potential underlying affective disorder. Lifetime anxiety disorder was reported by 75% whereas ADHD by 0-57% and ODD/AD by 0-79% in cases with association of TDs and affective disorder[1].

Tic Disorders and Other Comorbidities

Specific Learning Disorder

The prevalence of comorbid specific learning disorder (SLD) is found as 22% in TDS while its prevalence has been reported as 15-20% in general population. Thus, it may be suggested that TDs doesn't increase risk for SLD specifically. Especially, comorbid ADHD may mediate to SLD seen in association with TDs. However, SLD subtypes differ when they accompanied to TDs. Reading disorder is more common SLD in general population while mathematics and written expression disorders are primarily seen SLDs in association with TDs. In individuals with TDs, social responsiveness and other communication functions can be affected in the presence of comorbid SLD, worsening prognosis[4].

Self-Harming Behaviors

Prevalence of self-harming behavior can range from 17% to 53%. Some authors reported that self-harming behaviors are associated to motor tics while Kurlan advocated these behaviors are independent from tic severity. It has been reported that self-harming behaviors seen in TDs are independent from intelligence level; can be a marker for underlying depressive disorders; and obsessionality may mediate this symptom[2].

Autism Spectrum Disorders

TDs frequently accompany to autism spectrum disorders (ASD) and this association creates specific challenges in terms of diagnosis. It was suggested that tics such as hand clapping and sniffing make diagnosis in particular. In some cases, OCD and TDs can occur during adolescence[1,2,5].

Mental Retardation

There is a simplex relationship between TDs and mental retardation (MR). TD prevalence is increased in cases with MR but vice versa isn't true. Additional challenges may be present in the diagnosis as stereotypes are frequently seen in cases with MR. It has been reported that diagnosis is challenging in cases with moderate-to-severe MR[1].

Developmental Coordination Disorder, Trichotillomania, Substance Use Disorder

Developmental coordination disorder (DCD) accompanies to TDs especially in the presence of comorbid ADHD and academic problems. It may increase risk for development of psychopathologies over time and this impact may be mediated by social and academic problems. SLD and speech disorders may be seen commonly in these cases[1].

Trichotillomania is increased in TDs. In particular, it is more common among women with TDs and may increase risk for anxiety disorders. Substance use disorders alone don't increase risk for TDs. The group with association of TDs and ADHD are particularly at increased risk for development of substance use disorders. In these cases, it was reported substance use could aim self-medication and to control tics[1,2].

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SYMPOSIA ABSTRACT: 537

The Effect of Divorce on Family and Child

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ABSTRACT

Every year over 1 million children and adolescents are involved in a divorce[1].

Parental divorce is a major risk factor for internalizing and externalizing problems in children and adolescents. Cross-sectional and longitudinal studies show that children of divorced parents report more psychological maladjustment than children of married parents. A large body of research has revealed that children of divorced parents exhibited a heightened prevalence of conduct problems, anxiety and depression symptoms, and academic difficulties[2]. Findings suggest that divorced parents' communication skills are supportive help buffer stress and anxiety by changes in cortisol level[3,4]. On the other hand children of lower educated parents have worse life chances in the first place and when their parents divorce or separate, they are affected more negatively[5].

In this session, with the context of scientific researches the effects of divorce on children and family from different sights will be discussed.

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SYMPOSIA ABSTRACT: 539

ACT Interventions in Anxiety Spectrum Disorder

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ABSTRACT

Acceptance and Commitment Therapy also called third wave is a behavioral psychotherapy model that includes mindfulness and acceptance-based interventions. ACT focuses on planning people's behaviors in line with their long-term values, and suggests that efforts to reduce undesirable internal experiences hamper this behavior[1]. The efficacy of ACT has been shown in randomized controlled trials in many cases, including anxiety disorders such as generalized anxiety disorder, obsessive compulsive disorder, and posttraumatic stress disorder[2]. The aim of ACT applications in anxiety disorders is to eliminate the overactivity with anxiety and the experiential avoidance, which is defined as the attempt to change the frequency, intensity and shape of internal experiences such as thoughts, emotions, somatic signs and memories. The goal is to help people shape their behaviors in the direction of values they value by providing an anxiety and a more flexible and self-relationship. ACT approach to consultant; to show how to live with them by accepting their presence rather than repressing or abandoning the unpleasant symptoms of anxiety[3]. Approaches to ACT in anxiety disorders include:

(1) Inelastic behavior such as avoidance, abatement, and control of anxiety is a problem, not a solution!
(2) When faced with anxiety statements, 'acceptance' can be a functional alternative instead of struggling with them.
(3) To develop self-acceptance and volunteerism against aversive thoughts, emotions, somatic statements when the client feel anxiety.
(4) Clarify the values and act on values. The aim is to help the counselor for a meaningful life. ACT serves to determine the behaviors of the client in the direction of the values rather than try to reduce the symptoms.

At the end of this experiential course, participants will be able to formulate ACT-based case conceptualization and make the behavior analyses. Also the other aim of this course is to improve the using basic ACT processes in clients with anxiety disorders

KEYWORDS

Acceptance and commitment Therapy; Anxiety; behavior therapy

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SYMPOSIA ABSTRACT: 540

Autism and Nutrition

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ABSTRACT

Nourishment is utilization of nutrient needed by people for growing, building and living a healthy life. Healthy nutrition behavior occurs from establishing interrelation between child and parents. In the development of nutrition behavior, many variables like homeostatic mechanisms, reward system, motor, sensorial and socioemotional yeti capacity have a role since babyhood. On the other hand, child care and attitude skills of parents, social environment and cultural elements are closely related to the development of nutrition behavior. As to parents play an essential role in the configuration of nutrition behavior. They can help passing self-nutrition of children by providing consistent nutrition routine, presenting proper type and amount of food, allowing determination of how much she will eat and enforcing behavioral limits. Nutritional deficiency is the behavior of not eating sufficient type or amount of food needed for maintaining the weight, satisfying the nutritional needs and growing against the attempts of parents or child caretakers. In the occurrence of these defects, medical problems, individual features and interpersonal relations may have role. It is estimated that 25–35% of babies and children have nutritional deficiency. As nutritional deficiencies may be seen in the children growing exactly normally, it may be seen in the children having medical disorders, neurodevelopment problems in definite periods. Autism spectrum disorder (ASD) is a lifelong neurodevelopmental defect which manifests itself with deficiency in language development and social progress, and also difference and loss in cognitive enhancement. It is estimated that nutrition problems have a high rate like 90% in the children having ASD[1]. Nutrition problems related to ASD generally include eating rejection, eating anxiety, and excessive selectivity of food. It is asserted that the children having ASD, who are selective and precise about eating, show disfavor indications to food in certain temper, color, smell, heat, and belonging certain brand. Certain strict behaviors and routines seen in children having ASD determine when, where and how they will eat their meals and which kind of food they will consume. They are described as 'difficult eater' and 'selective eater' children on future dates. It is thought that nutrition problems cause big problems such as obesity, growth, and developmental delay. According to the research conducted on this basis, it is estimated that children having ASD have more obesity risk than normal developed children[2]. Also, according to many studies, it is asserted that people having ASD exhibit more complaints about gastro intestinal system like diarrhea, constipation, reflux, vomiting, gas pains, abdominal distention, and discomfort than healthy control and other siblings[3, 4]. It is reported that, in the cases like ASD in which nutrition problem is usually seen, mothers show less touching behavior during the interaction with children, and children are in tendency to keeping away from mothers, too. Parents may respond with different feeding attitude to different eating behavior seen in people having ASD. Nutrition problems developed in early age cause physical, cognitive, and mental problems in the child. Besides that, the child cannot develop healthy nutrition relation with parents and therefore feeding problems show up. Considering all of these issues, the evaluation of nutrition in ASD gains importance. In this presentation, 'Autism and Nutrition' relation will be discussed with the results of a study.

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SYMPOSIA ABSTRACT: 541

When Therapy is Going Nowhere: Journey from Barriers to Progress

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ABSTRACT

TUNING INTO THE OTHER

Acceptance and commitment therapy (ACT, typically pronounced as the word "act") is a form of psychotherapy commonly described as a form of cognitive-behavior therapy. It is an empirically-based psychological intervention that uses acceptance and mindfulness strategies mixed in different ways with commitment and behavior-change strategies, to increase psychological flexibility The objective of ACT is not elimination of difficult inner experiences (like thoughts, emotions, memories); rather, it is to be present with what life brings us and to "move toward valued behavior. ACT therapists try to develop *cognitive defusion* towards to the unwanted thoughts and *acceptance* towards to undesirable feelings and emotions on their clients. At the same time ACT therapists tries to keep clients now and here (*present to moment*), contact with her/his contextual self-perception (*self-as-context*) and notice and take action about the important aspects of her/his life (*values and committed action*). Throughout therapy, these six—interrelated—processes are studied in order to achieve psychological flexibility. But importance and power of ACT comes from the application of these six processes to the therapist through the therapy session with clients. Beyond the empathy, ACT wants therapist's to stay here and now and touch to her/his own self-perception with her/his values about the work, himself, life, etc. It's a hard work but more harder is to touch her/his pains (especially pain about the session and being a good therapist) and work with her/his unwished thoughts. ACT focuses on ways of relating, one human being to another, which fosters a powerful working alliance and makes valued living a shared creative act in the here and now. In this process two people come and stay at here and now with their pains. With the awareness of their sufferings, inadequacies, difficulties, etc., they are interested in each other and place and time where the process' ongoing

KEYWORDS

Acceptance and Commitment Therapy; mindfulness; psychological flexibility

SYMPOSIA ABSTRACT: 542

Endophenotypic similarities and differences in schizophrenia and bipolar disorder

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ABSTRACT

Schizophrenia and bipolar disorder have many similarities in terms of symptom pattern, course characteristics, familial cluster and treatment options, although they are categorized as two different disorders in the psychiatric diagnostic classification. As the heterogeneous structures of both disorders are considered, it is observed that there are transitions from one diagnosis to another in longitudinal follow-up. The fact that the boundaries of these two disorders are unclear also makes it difficult to understand the etiopathogenesis that has not been fully lightened yet (1). Genetics, neuro-imaging, endophenotyping and cognitive studies are in process to elucidate the etiopathogenesis of these two diseases. The expectation proposed by endophenotypes is that they will point toward specific correlated genes that provide insight into the underlying biology, thus aiding in classification (2).

Endophenotypes are defined as measurable, heritable, and reproducible biologic traits that vary continuously in the population at large which are correlated with an illness in the population in part due to shared underlying genetic influences. They are primarily state independent, unobservable by the naked eye, some of them revealed only through a provocative test, set apart with illness within families, and are found in nonaffected family members at a higher rate than in the general population (1,3). Classical endophenotypes for psychotic illnesses have included measures of brain structure and function and related cognitive and physiologic phenomena (4). Smooth pursuit oculomotor, saccadic eye movements, structural magnetic resonance imaging, gray matter volume, diffusion tensor imaging, task-based functional MRI, resting functional MRI, prepulse inhibition, P300 event-related potential, P50/paired stimulus processing, resting electroencephalogram and

cognition are candidate endophenotypes for psychotic illnesses (1). Many studies were conducted so far to discover endophenotypes in schizophrenia and bipolar disorder. Studies reveal that although some of these various measures differ between schizophrenia and bipolar disorder, most of them are similar between the two illnesses and co-exist on a continuum of severity (1). This course aims to discuss the boundaries of these two diseases by compiling the data about endophenotypes obtained from the previous studies.

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SYMPOSIA ABSTRACT: 544

New Horizons in Psychotherapies for Understanding the Internal Worlds of Our Patients

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ABSTRACT

A New Look at Psychopathology: Functional Contextualism and ACT

Functional Contextualism, a philosophy of science, is a pragmatically based epistemological interpretation of Skinner's work on behavior (Radical Behaviorism). Functional contextualism, which is rooted in pragmatism and contextualism and often used in the context of behavioral science and behavioral analysis, was conceptualized by S. Hayes at the end of the eighties. The form of contextualism from which functional contextualism emerged is the one described by the philosopher Stephen C. Pepper in his book *World Hypotheses: A Study in Evidence*. In this work, Pepper noted that philosophical systems tend to cluster around a few distinct "world hypotheses" or "world views". Each "world view" is characterized by two hypotheses, ontological (root metaphor) and epistemological (truth criterion). The ontological assumption of contextualism is based on "act in context" and the truth criterion of contextualism is often dubbed "successful working", whereby the truth and meaning of an idea lies in its function or utility. Steven Hayes has overcome the clinical application of functional contextualism by addressing contextualism in the field of psychology on the basis of "predicting and influencing behavior". It is necessary to clarify the contextual variables in order to predict and direct behavior according to functional contextualism. Thought may be related to a certain behavior, but only a certain historical and situational context provides the basis for the emergence of this thought and behavior. Functional contextualism has developed its own clinical practice through research on the concept of rule governed behavior, focusing on the thought's dominance over the behaviors. For that matter there is no need to change thoughts because they have no reason to determine the behaviors. What needs to be done is to make interventions that reduce the dominance of the thoughts over the behaviors. Acceptance and Commitment Therapy, developed on this philosophical basis, is a third generation behavioral therapy approach that centers the meaning for the client by setting the value fields of the individual as functional reference points[1]. ACT assess the psychopathology as an intervention with the environment of the organism rather than a structural problem and try to develop acceptance towards to the private experiences' of clients, without trying to change them. And also ACT focuses on the analysis of problem-centered behaviors by taking precedence functionality rather than diagnostic classification.

KEYWORDS
Functional contextualism; behaviorism; acceptance and commitment therapy

SYMPOSIA ABSTRACT: 545

Nutrition in Autism Spectrum Disorder

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ABSTRACT

Nourishment is utilization of nutrient needed by people for growing, building and living a healthy life. Healthy nutrition behavior occurs from establishing interrelation between child and parents. In the development of nutrition behavior, many variables like homeostatic mechanisms, reward system, motor, sensorial and socioemotional yeti capacity have a role since babyhood. On the other hand, child care and attitude skills of parents, social environment and cultural elements are closely related to the development of nutrition behavior. As to parents play an essential role in the configuration of nutrition behavior. They can help passing self-nutrition of children by providing consistent nutrition routine, presenting proper type and amount of food, allowing determination of how much s/he will eat and enforcing behavioral limits. Nutritional deficiency is the behavior of not eating sufficient type or amount of food needed for maintaining the weight, satisfying the nutritional needs and growing against the attempts of parents or child caretakers. In the occurrence of these defects, medical problems, individual features and interpersonal relations may have role. It is estimated that 25–35% of babies and children have nutritional deficiency. As nutritional deficiencies may be seen in the children growing exactly normally, it may be seen in the children having medical disorders, neurodevelopment problems in definite periods. Autism spectrum disorder (ASD) is a lifelong neurodevelopmental defect which manifests itself with deficiency in language development and social progress, and also difference and loss in cognitive enhancement. It is estimated that nutrition problems have a high rate like 90% in the children having ASD[1]. Nutrition problems related to ASD generally include eating rejection, eating anxiety, and excessive selectivity of food. It is asserted that the children having ASD, who are selective and precise about eating, show disfavor indications to food in certain temper, color, smell, heat, and belonging certain brand. Certain strict behaviors and routines seen in children having ASD determine when, where and how they will eat their meals and which kind of food they will consume. They are described as 'difficult eater' and 'selective eater' children on future dates. It is thought that nutrition problems cause big problems such as obesity, growth, and developmental delay. According to the research conducted on this basis, it is estimated that children having ASD have more obesity risk than normal developed children[2]. Also, according to many studies, it is asserted that people having ASD exhibit more complaints about gastro intestinal system like diarrhea, constipation, reflux, vomiting, gas pains, abdominal distention, and discomfort than healthy control and other siblings[3, 4]. It is reported that, in the cases like ASD in which nutrition problem is usually seen, mothers show less touching behavior during the interaction with children, and children are in tendency to keeping away from mothers, too. Parents may respond with different feeding attitude to different eating behavior seen in people having ASD. Nutrition problems developed in early age cause physical, cognitive, and mental problems in the child. Besides that, the child cannot develop healthy nutrition relation with parents and therefore feeding problems show up. Considering all of these issues, the evaluation of nutrition in ASD gains importance. In this presentation, 'Autism and Nutrition' relation will be discussed with the results of a study.

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SYMPOSIA ABSTRACT: 546

Pediatric Bipolar Disorder: Genetics and Neurobiology

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ABSTRACT

Pediatric bipolar disorder (PBD) is one of the most severe psychiatric disorders in childhood which is associated with higher rates of hospitalization, psychosis, suicidal ideation and suicide attempts[1]. Epidemiological studies indicate that the overall rate of PBD is 1.8%[2]. Although the pathophysiology of PBD is unknown, recent advances in genetic and magnetic resonance imaging (MRI) studies of affected children and adolescents are enhancing the understanding of the genetic and neurodevelopmental basis of the disorder[1,3]. Family and twin studies have shown that early onset BD is associated with increased familial risk[3]. Multiple genomic regions including regions on chromosomes 2, 4, 6, 8, 11, 12, 13, 16, 18, 21, 22, and X have been found to be possibly linked to BD. Moreover, several candidate genes catechol-Omethyltransferase (COMT), brain-derived neurotropic factor, tyrosine hydroxylase, D-amino acid oxidase activator, and neuregulin have been proposed to be possibly associated with BD[4]. Neuroimaging studies found that PBD is related to functional, anatomic, and biochemical abnormalities in structures that are hypothesized to be involved in the neuroanatomic circuits of emotion processing and regulation, including the limbicthalamic-prefrontal circuit and the limbic-striatal-pallidal thalamic circuit[1]. Studies have shown that children and adolescents with BD have smaller cerebral volumes (bilateral parietal and left temporal lobes), smaller cingulate volumes and larger right nucleus accumbens of the basal ganglia compared to children and adolescents with other psychiatric conditions and those without psychiatric diagnoses[4]. Diffused gray matter abnormalities have been identified in patients with PBD, specifically volumetric changes in the anterior limbic network (ALN), including the prefrontal regions, thalamus, striatum, amygdala, hippocampal complex, and the midline cerebellum. Reduced prefrontal cortex and increased globus pallidus volumes are among the most consistent findings in children and young adults with BD[5]. Out of scanner tests and functional magnetic resonance imaging (fMRI) studies have suggested that children and adolescents with BD have abnormal functioning in face processing, response inhibition, frustration, and cognitive flexibility[2]. Magnetic resonance spectroscopy (MRS) research indicates that PBD may be associated with specific neurochemical abnormalities. For instance, using MRS, unmedicated youth with bipolar disorder were demonstrated to have significantly lower glutamine levels in the anterior cingulate cortex compared to subjects with a BD who were currently receiving medications and subjects without a psychiatric diagnosis[4]. Diffusion tensor imaging (DTI) studies have found lower fractional anisotropy (FA) in the anterior corona radiata (ACR) and superior frontal white matter in PBD[5]. The aim of this presentation is to review recent genetic and neurobiological advances with respect to PBD.

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SYMPOSIA ABSTRACT: 547

Diagnosis and Treatment of ADHD in College Students

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ABSTRACT

Individuals with ADHD face substantial difficulties in academic performance, relationships, and self-esteem in college and across the life span. Estimated rates of ADHD in college are reported 2–8% in studies carried out in USA and these rates are based on studies that utilized self-reported symptoms or diagnostic status from convenience samples of students at individual campuses but not comprehensive assessment conducted with nationally representative samples[1]. College health care providers feel discomfort in diagnosing and treating ADHD and this could represent a barrier to care for college students[2]. The overarching goal of this presentation is to provide a clinically useful review of the available evidence that practicing clinicians can use to aid in the diagnostic assessment and treatment of ADHD in college students.

Diagnosis of ADHD in college students

Symptoms of ADHD in adulthood may not be as clear as in childhood. Despite recommendations regarding the diagnosis of emerging adults, there is not a strong consensus regarding the ideal method for diagnosing ADHD, additionally research on the diagnostics and follow up of ADHD in this population is limited. According to a recent study by Dyvorsky et al, rating scales can be used effectively to evaluate ADHD on college campuses if both parent and student rate childhood symptoms. The authors concluded collecting parent ratings helps against possible student malingering to obtain ADHD medications or accommodations and parent ratings of childhood ADHD symptoms of inattention were the strongest predictors of current diagnostic status of ADHD[3]. Another study in college students explored the association between level of impairment and the DSM-5 threshold of symptoms and suggested that DSM-V threshold of five symptoms for ages 17 years and older is not necessarily predictive of ADHD-related impairment in college students and may not be preferable to other thresholds[4]. Criteria to diagnose ADHD in adults were emphasized by other authors. These were:

(1) confirmation of at least 4 inattentive and/or hyperactive impulsive symptoms which contributed to current impairment,
(2) evidence of ADHD symptoms prior to 12 years of age that had an impact on impairment in multiple domains across the lifespan,
(3) third party corroboration of symptoms and impairment,
(4) the confirmation that impairment is not due to another disorder[5]. Outcome monitoring and assessment for comorbid conditions such as substance use, mood and anxiety disorders to properly treat these students.

Treatment of ADHD in college students

ADHD is one of the most referred disorders by college counselling centers for medication evaluation and treatment along with depressive and anxiety disorders[6]. Dopaminergic and noradrenergic deficits in the frontal cortex or regions projecting into that area have been implicated in the inattentiveness and/or hyperactivity associated with ADHD. Pharmacotherapy for adult ADHD often targets core symptom reduction, Current standard and first-line pharmacotherapy for adult ADHD is stimulant medication[7]. Psychostimulants relieve symptoms by increasing intra-synaptic dopamine, norepinephrine and serotonin. There are two classes of psychostimulants which are amphetamine-based and methylphenidate-based psychostimulants. Atomoxetine is a non-stimulant ADHD medication that is currently available for use in Turkey. Atomoxetine, inhibits norepinephrine transport and has also been approved for ADHD treatment as first-line pharmacotherapy in many countries[8]. Meta-analyses have provided evidence for the efficacy of stimulants[9] and beneficial effects of atomoxetine in ADHD in adults[10]. Among alternative compounds, amphetamines (mixed amphetamine salts and lisdexamfetamine) have the most robust evidence of efficacy, but they may be associated with serious side effects (e.g., psychotic symptoms or hypertension). They are not currently available in Turkey. Noradrenaline or dopamine enhancer antidepressants have an evidence of efficacy however not appropriate for patients with comorbid bipolar disorder[11]. Extended-release guanfacine and extended-release clonidine are two non-stimulant medications licensed for use in USA. Atypical antipsychotics are not indicated for treatment of core ADHD symptoms. According to a recent meta-analysis, stimulant drugs should be preffered over non-stimulant drugs due to superior efficacy. The efficacy of pharmacological treatment should be monitored over time because it may decrease progressively[12]. For adults with ADHD who continue to experience clinically significant symptoms following first-line medication treatment at the maximum tolerated dose, discontinuation of stimulant therapy and initiating non-stimulants like atomoxetine and bupropion is recommended. Although medication therapy has the most empirical support as treatments for ADHD in adults, many adults with ADHD continue to experience significant residual symptoms. Research evidence most strongly supports the use of cognitive-behavioral therapy (CBT) targeting deficits in executive function as well as the comorbid symptoms of anxiety and depression that tend to be present in these patients. Other psychological treatments consist

CBT-oriented coaching[13] and social skills training. Adapted Dialectical behavior therapy for adult ADHD addresses emotional awareness and regulation, mindfulness, organization, behavior analysis, stress management, interpersonal effectiveness, depression, and substance use disorders however there is little evidence from randomized trials to support the efficacy

of DBT in adults with ADHD[14,15]. Available data support the use of structured, skills-based psychosocial interventions as a viable treatment for adults with residual symptoms of ADHD. These treatments, however, require further study for replication, extension and refinement[16]. There are no trials comparing psychotherapies with adult ADHD, treatment selection could be guided by availability, the clinical features of the patient's presentation, and existing evidence. For patients with prominent symptom clusters or deficit areas (e.g., executive dysfunction, emotional dysregulation, or impulsivity), it is suggested that a psychotherapy targeting that cluster or area be used. The presence of a co-occurring disorder treatable by psychotherapy will also influence the treatment provided.

Adults with ADHD and with prominent deficits in executive function, augmentation of medication with cognitive behavioral therapy (CBT) that targets executive dysfunction seems more efficacious rather than other nonpharmacologic treatments or medication alone.

In conclusion, there is no single test that can reliably diagnose ADHD. The diagnosis of ADHD in college students is based on a comprehensive clinical assessment. This presentation will mention several structured diagnostic interviews (The Adult ADHD Clinical Diagnostic Scale (Adult ACDS), ADHD Lifespan Functioning Interview (ALFI), Conners' Adult ADHD Diagnostic Interview for DSM-IV (CAADID), Diagnostic Interview of ADHD in Adults (DIVA), Structured Clinical Interview for DSM-5 (SCID-5)- Adult ADHD Module, symptom measures to evaluate Adult ADHD in college students. The available Turkish versions of the interviews and measures and their practical application will be highlighted.

How ADHD is best managed across the lifespan and across key transition periods such as in college needs much more investigation[17] however the optimal medication and psychosocial treatment strategies will be discussed considering the current literature.

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SYMPOSIA ABSTRACT: 549

Effects of Synthetic Cannabinoids on ECG Parameters

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ABSTRACT

Synthetic cannabinoids (SCBs) are a growing class of highly potent, highly efficacious cannabinoid agonists that, until recently, have been falsely marketed as 'safe' and 'legal' alternatives to marijuana (Ford, Tai, Fantegrossi, & Prather, 2017). Also they are more attractive than cannabis owing to ease of purchase as well as increased odds of negative urine and blood testing. A variety of adverse clinical effects have been reported in the setting of synthetic cannabinoid use, including psychiatric, neurological, neuromuscular and cardiovasculer symptoms. Among cardiovascular symptoms, tachycardia (44%),with heart rates of 106-180 beats per minute, and hypertension (41%) [140–223 mmHg (systolic); 82-103 mmHg (diastolic)] were the most reported. Bradycardia (11%) and hypotension (11%) were also linked to these substances and 18% of patients complained of chest pain after smoking SCs. Exposure to SCs was found to cause serious health problems including myocardial ischemia and myocardial toxicity (Tournebize, Gibaja, & Kahn, 2016). Information about the effects of synthetic cannabinoids on the ECG parameters is limited because they are considerably new. According to case reports and recent researches on topic, P-wave dispersion (PD) (Aydin Sunbul et al., 2016) and ST elevation (McKeever et al., 2015) are the most common changes after acute use.

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SYMPOSIA ABSTRACT: 554

ACT Interventions In Anxiety Spectrum Disorders

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ABSTRACT

Acceptance and Commitment Therapy (ACT) is an innovative acceptance-based behavior therapy that has been applied broadly and successfully to treat a variety of clinical problems, including the anxiety disorders like generalized anxiety disorder, obsessive-compulsive disorder, and posttraumatic stress disorder[1]. Throughout treatment ACT balances acceptance and mindfulness processes with commitment and behavior change processes[2].

As applied to anxiety disorders, ACT seeks to undermine excessive struggle with anxiety and experiential avoidance- defined as a tendency to engage in behaviors to alter the frequency, duration, or form of unwanted private events (i.e., thoughts, feelings, physiological events, and memories). The goal is to foster more flexible and mindful ways of relating to anxiety so individuals can pursue life goals important to them[3].

Specifically, an ACT approach to anxiety disorders is designed to teach clients the following:

(1) Rigid and inflexible attempts to control anxiety are the problem, not a solution.

(2) Acceptance –opposed to struggle- is a viable alternative agenda when faced with anxiety responses.

(3) Practice mindful acceptance and willingness when experiencing aversive thoughts, feelings, and bodily sensations during anxiety.

(4) Identifying values- committed action/ ACT- style exposure.

KEYWORDS

Acceptance and commitment Therapy; anxiety; psychological flexibility

Unlike typical CBT approaches, the primary goal is not to reduce anxiety. The goal is to help clients live a full, rich, meaningful life. ACT- style exposure is done in the service of client values and life goals, not as a means to reduce symptoms. At the end of this experiential course, participants will be able to do ACT-based case conceptualization and make the behavior analyses of any anxiety related problems. Also, improving therapist skills by applying six core ACT processes while working with clients who anxiety related problems is one of the aim of this course.

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SYMPOSIA ABSTRACT: 555

How do we evaluate emotion regulation in clinical practice and research?

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ABSTRACT
"Emotions" are complex phenomena involving subjective experiences, cognitive processes, behaviors and psychophysiological changes[1]. "Emotional regulation"; in turn, involves initiation, inhibition and modulation of emotions to help personal functioning[1–3]. Various models attempted to elucidate emotion regulation in differing terms such as antecedent focused vs. consequence focused, automatic vs. controlled, top- down vs. bottom- up, intrinsic vs. extrinsic[1–4]. Emotional regulation and its problems are pervasive among clinical populations with internalizing and externalizing disorders[5]. They form both targets for treatment interventions and moderate the effects of interventions for other problems[5]. Those problems are also relatively less researched than other neuropsychological constructs, although newer attempts at classification such as DSM-5 and RDoC may have changed this view. The prevailing paradigm in both clinical practice and research on emotion regulation involves using subjective self-reports such as scales to assess both inter-individual variability and pathology[4]. However, this approach has its limitations in evaluating emotional phenomena which also involves psychophysiological changes and automatic/ bottom- up processes. This presentation will focus on various methodologies to evaluate emotion regulation such as monitoring electro-dermal activity, emotional Stroop paradigms, evoked potentials and attention blink paradigms and present results from studies conducted in Turkey.

KEYWORDS
Emotion; emotion regulation; psychopathology; psychophysiology; neuropsychology; Emotional Stroop

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SYMPOSIA ABSTRACT: 556

Lost Memories

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ABSTRACT

Traumatic experiences in early childhood, even during pregnancy are stored in the body and can affect our behavior. Awareness in mother's uterus is believed to start around week 28-32 of pregnancy. According to Verny, emotions and thoughts of the mother are reflected in the body of the child. It is found out that children who are accepted by their mother are much healthier to those who are not[1]. Early breakup in the mother-child connection and long-term absence can be devastating for the baby[2]. Hippocampus-Prefrontal Cortex communication in children has not fully developed until after 2 years. Therefore, early breakup-trauma is stored as memory fragments, physical feelings, images and emotions instead of clear memories[3]. Since there is not description of events during period before talking, these events are not stored in conscience[4]. These lost and undescribed memories that are stored in implicit memory become parts of sub conscience[5]. As a result, the person may not remember past experiences but games, behaviors and physical symptoms can reflect the trauma.[4] Unspoken traumatic memories are suppressed and just like a conditioning experiment it can effectively influence movements[2]. There is also grief where there is trauma[4]. Stress reaction system of the brain in traumatic evecnts can be over-reactive or tolerance can develop. Generally, when there is no control over the situation, sensitivity against stress develops. Even a small stimulus can lead to a strong reaction[5]. Sometimes terrorizing situations in early years lead to being more instinctive and aggressive, and less thoughtful and affectionate in later years.[5,4] If the traumatic event depends on a template and is re-occuring, tolerance improves against stress and this stops reaction of the patient against the experience for a long period[5]. In one study, chronic low levels of cortisol were observed in people with TSSB and their children[6,7]. Siegel recommends integrating logic on the left hemisphere with emotions on the right hemisphere during treatment of the trauma. "Letting children describe or describing details of the event in chronological order enables them to understand the experience they have with their emotions and body" he says[8]. Levine suggests a similar approach. The adult describes first from its own perspective. The child is asked if he/she wants to add anything to this story and to describe own version of the story[4]. Four cases presented have lost memories. There were memory fragments in an even involving a child but it was never talked about the event. Common properties of the cases were absence of crying due to sadness, second hand emotions (feeling own pain through other's) and weakness in controlling anger. Three cases were performed mother-child session, where the mother was asked to describe the events with EMDR therapy. The little child was observed about how he/she felt from his/her own eyes and the case was asked about how he/she felt about it. This way, lost memories were brought back to today and the feelings at right hemisphere were integrated with logic on the left hemisphere. In one case, picture of the patient during illness was used. Another patient was asked to imagine the mother uterus experience through feelings of her unborn child. It was evaluated as positive that crying due to sadness and living the grief of the event were observed after processing the traumatic memory. They were able to control anger much better after this.

To summarize, to act out emotions is necessary for the physical and mental health. Also, none of these cases that had trauma was able to fully live their childhood and was capture in child ego situation. They were still showing addictive behavior on parents or their partners. Because they were lacking adult skills. But they were also lacking child and adolescent skills. Because of this, homework on child and adolescent behavior (safe) were given with priority on child skills.

Case- 1 A. A. (17-yo, female, student)

Psychiatric History: Not accepted her boyfriend leaving her, excessive sadness, inability to cry, laughing in situations that she would normally cry.

She had intra-cranial bleeding due to falling when she was 5 at kindergarten. She stayed in coma for 3 days, her recovery was said to be "miracle" despite expectations of her dying or staying handicapped. When she was 15 years old she was unable to stay alone at home and was sleeping with her parents. Crying was unhealthy according to her. Head trauma and staying at hospital at 5 was taken as EMDR therapy. She did not have memory about the event. She was given homework for child skills. Mother-child session was made. Her mother was let speak in chronological order. When the emotional tone was increased, the child was asked about how she felt. The observation was that she was following her mother carefully and smiling when her mother was crying. What she felt was anger, she was feeling this in her stomach. This was evaluated as positive in terms of her living the grief from the trauma. Her extreme sadness recovered.

Case-2 D. A. (23-yo, male, student)

Psychiatric History: Substance abuse, school looks irrelevant, is sick of his friends

He stayed at hospital for 2.5 months at 5 years old due to vomiting and weight loss without any diagnosis. He had operation with diagnosis of choledochal cyst and had peritonitis and sepsis. He had vertigo after lhis sister was admitted to hospital due to head trauma, and nightmares where he and his brother died; he also could not sleep alone. He could not accept his girlfriend leaving her when he was 17, addiction to her. In the first session, homework was given on child skills. In the second meeting, mother-son session was made. There was no memory of the event in the conscious memory. When her mother was

KEYWORDS

Childhood; emdr; lost; memory; trauma

describing, she was halted and the emotions, surprise and anxiety of her parents in the child's eyes were observed with BLS. In the next session, memory fragments came about his sister staying at the hospital. He did not feel anything about these events. There was physical perception in terms of weight and sadness in his chest, other memories followed. He expressed sadness and happiness together afterwards.

Case-3 D. B. (16-yo, female, student)

Psychiatric History: Letting go when sick, nausea, fever. Afraid that his illness and nausea will never go away, fear of being bound to bed and lying all day. Stayed at hospital when she was 1-yo due to leukemia, recovered at 2-3 yo. Since primary school 2nd-3rd grade he is afraid that he will not recover when she gets sick. She was using Citalopram 20mg/d. Her fears were believed to be related to childhood trauma, there was no memory about the event. The picture of her at 2-yo with hair fallen off was taken as target event. There was "I am at danger" negative cognition, no sign of emotion however there was an unnamed feeling in her stomach. When visual stimulus light stopped, she said she felt like "dying". She felt like going to and coming from home with ambulance. She saw people running quickly. "Everyone should be next to me. I want it like that now" she said. Homework was given about child and adult skills. In the 3rd session tolerance of the illness increased. Self-confidence increase, case was able to defend herself. In the 4rd session she said her loneliness came from childhood (integration). Fears of the case disappeared completely.

Case 4 A. H: (7-yo, female, Primary School 2nd Grade, has sister who is 4.5 yo, living abroad)

Psychiatric History: Claustrophobia cannot close toilet door, does not let her mother leave the car for short time leaving her alone. Does not like sleeping (finds it boring), no crying due to sadness, cries when angry, aggressive towards her friends. Her mother stayed at hospital for 2 weeks when they came to Turkey and she was 2.5-yo due to premature birth. A total of 5 EMDRs were done. After first session, door was able to be shut half and after 2nd session fully. Mother-child session was made. She was able to close the toilet door fully and also wait her mother at car.

Case-5 E.O. (31 years old, male, married, physiotherapist)

Psychiatric History: Inability to control anger, dizziness, flashbacks to past. There were inner sounds that did not stop. He was fantasizing about killing with torture, doing extreme sports and had empathy about Hitler. He could not cry. Record of physical abuse and neglect from parents during childhood, had rickets as a child. His brother was decapitating his toys as "punishment" every 2-3 days. His wife was pregnant and one afternoon there was unhappiness and depression after calling by phone and saying "We are going to meal with the baby". While they were walking, the silhouette of the baby came before his eyes. The sympathetic image of baby in mother's uterus along with the baby's need of mother and that she could not help her baby, and sadness, sympathy came to her. EMDR was done with this image and "I am helpless" negative cognition. "The child is actually smiling, but looks like poor" idea and the fact that mother's own experience was reflect in the baby was observed.

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SYMPOSIA ABSTRACT: 557

Shared and Distinct Genetic Vulnerabilities Between Schizophrenia and Bipolar Disorder

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ABSTRACT
Schizophrenia and bipolar disorder are among the most debilitating psychiatric illnesses, and both are characterized by disturbances of thought, behavior, and mood. According to the Kraepelinian dichotomy, schizophrenia and bipolar disorder are two distinctly separate diseases each with its own pathogenesis and disease process[1]. But, recent studies have shown that these 2 disorders overlap significantly with regard to genetic susceptibility, clinical presentation and treatment. Genetic factors are known to play an important role in susceptibility schizophrenia and bipolar disorder. Genetic architecture of both disorders still remain largely unknown. But, studies have demonstrated that schizophrenia shows substantial genetic overlap with bipolar disorder. The results of numerous family, twin, and adoption studies have provided a genetic correlation between two disorders[2]. There is also strong evidence for genetic overlap between schizophrenia and bipolar disorder in terms of genome-wide association studies. GWAS studies involving high patient numbers suggest that single nucleotide polymorphisms (SNPs) related to immunity, transcriptional modification, synaptic plasticity and calcium channels are associated with increased risk for both disorders[3]. Instead of investigating genetic markers individually, polygenic risk scoring method was used by combining the associated markers. But, its sensitivity and specificity was low to be used as a predicitve test[4]. In recent years, studies exploring large chromosomal structural variants, in particular copy number variants (CNV) has increased. CNVs are rare and they a stronger influence on schizophrenia than bipolar disorder[5]. The establishment of large consortiums to understand genetic etiology has increased our knowledge about the etiology of both disorders. Studies including increased sample sizes, investigating distinct phenotypes of both disorders with new genetic techniques will provide better insight into the overlapping and diverging aspects of schizophrenia and bipolar disorder.

KEYWORDS
Schizophrenia; bipolar disorder; genetics

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SYMPOSIA ABSTRACT: 558

Antipsychotics and QTc Prolongation

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ABSTRACT
The QT interval is measured on the ECG from the beginning of the QRS complex to the end of the T wave. Because the QT-interval varies with the heart rate, the corrected QT-interval (QTc) should be used. QTc-prolongation can lead to ventricular arrhythmias like Torsade de Pointes (TdP) and sudden cardiac death in some cases[1]. Some factors are known to be associated with QTc prolongation: female gender, increasing age, structural heart disease, family history of cardiac death, hepatic or renal failure, hypokalemia or severe hypomagnesemia, starvation and obesity. Antipsychotic medications have long been known to have the potential to cause QTc interval prolongation and TdP. Antipsychotic medications are also associated with an increased risk of sudden cardiac death[2]. In general, low potency typical antipsychotics are thought to carry a greater risk than high-potency agents, and this risk is thought to be dose related. Phenothiazines were more associated with QTc prolongation than other antipsychotics in case control studies. Although haloperidol is a highly potent antipsychotic, haloperidol is associated with QTc prolongation especially in case reports[3]. Compared to oral and intramuscular forms, intravenous haloperidol is more risky in terms of QTc prolongation. Since intravenous haloperidol is frequently used in intensive care units, care must be taken in this regard. ECG monitoring is recommended if haloperidol is given intravenously. Within the atypical antipsychotics, ziprasidone, amisulpride and sertindole are more at risk for QTc prolongation than other atypical antipsychotics. Ziprasidone causes the greatest mean QTc

KEYWODS
Typical antipsychotic; atypical antipsychotic; QTc prolongation; risk factors

prolongation compared with olanzapine, risperidone, and quetiapine. Aripiprazole is safe in comorbid medical conditions in terms of QTc prolongation. Apart from antipsychotics; alcohol, cocaine, stimulants, sitalopram within SSRIs, methadone, lithium (if level > 1.2) and trazodone (in higher doses) are associated with QTc prolongation. Before prescribing any antipsychotic drugs, clinicians should be aware of the risk factors for QTc prolongation, sholud take the other prescribed medicines into account, should evaluate on a case-by-case basis, and if necessary, QTc should be assessed before and during antipsychotic use.

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SYMPOSIA ABSTRACT: 560

Is Emotion Regulation Treatable?

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ABSTRACT
Emotion regulation should be reserved for intentional manipulation of triggers of unwanted emotions and the course of the triggered emotion. Intention to stop and think, in turn requires finding reasons to do so under the current conditions. The emotion dysregulation model is based on the premise that "individuals with psychopathology have poor understanding of their emotions, negative attitudes about emotions and maladaptive emotional regulation and management strategies. Preliminary evidence indicates that emotion dysregulation (ED) is associated with impairments across the developmental spectrum, such as social impairment and risky behaviors, and that its relative absence/ presence is differentially associated with treatment response. The treatment methods that were developed based on the emotion dysregulation model seeks to improve emotional regulation as a means to improve the symptoms. The components include: relaxation exercises; reframing of beliefs; education about emotions; emotion skills training, experiential exposure exercises and DBT (Dialectical behavior therapy). DBT based on cognitive-behavioral intervention consists of multiple modalities of intervention that include individual therapy, skills training, telephone consultation, team consultation, and the structuring of ancillary treatments. Although empirical evidence has been shown that these interventions, such as DBT and emotion regulation group therapy, have a specific focus on improving emotion regulation and providing skills for coping with emotion dysregulation, a number of questions remain whether or not ED is malleable."

SYMPOSIA ABSTRACT: 561

ADHD and Crime

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ABSTRACT
Most of the literature tries to explore the relationship between mental illness and juvenile delinquency; however, there lies a gap in the literature on how mental illness affects severity of the committed crimes. Some psychiatric disorders, such as presence of conduct disorder

KEYWORDS
ADHD; juvenile; delinquency; conduct disorder; crime; mental illness

and childhood ADHD emerge as predictors of more violent crimes[1]. Adolescent ADHD is also more likely to get in trouble with the law. Adolescents with co-morbid ADHD and CD/ ODD appear to have higher levels of impulsivity and delinquency. The risk of committing crimes increases with age, and shows higher prevalence in children displaying co-morbidity with neuro-psychological deficits like low IQ scores and learning difficulties. Aggressive behavior, although not an adequate diagnostic criterion for ADHD, is displayed by more than 50% of the patients; and in childhood, it is found to be associated with ADHD, addiction to drugs and other antisocial behavior patterns and criminality[2,3]. In comparison to children with pure conduct disorder (CD), antisocial behavior and disposition to criminality are more prevalent in children with comorbid ADHD and CD[4]. Virtually all constructs of ADHD, such as impulsiveness, hyperactivity, restlessness, not considering consequences before acting, poor ability to plan ahead, low sense of control, risk taking and poor ability to delay gratification, measured in different ways, are persistently associated with offending[5]. It is crucial to spend more effort to diagnose and treat ADHD in order to prevent delinquency in children and adolescents.

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FILE 562

Cognitive Behavioral Therapy of Posttraumatic Stress Disorder in Youth

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ABSTRACT

Subsequent to exposure to a range of traumatic events including sexual abuse, accidents and natural disasters, children can develop posttraumatic stress disorder (PTSD). PTSD generally results in significant impairments in child functioning[1] and about a third of children may suffer chronic course[2]. Effective intervention is shown to be related with improved functioning after the therapy and two years follow up period[3]. Trauma-focused cognitive behavioral therapy (CBT) is shown to be effective in children with PTSD[4]. Treatment approaches based on the model of PTSD which suggest that disorder is maintained by disjointed and poorly elaborated trauma memories, idiosyncratic misappraisals of the trauma and trauma-related symptoms and dysfunctional coping strategies, parental reactions and coping strategies[5]. TF-CBT targets these perpetuating factors by developing a coherent narrative of the trauma, challenging unhelpful appraisals of the trauma and sequelae, changing maladaptive avoidant coping strategies, modifying parents` unhelpful trauma-related appraisals, and recruiting parents as co-therapists. Basic TF-CBT includes *cognitive formulation* that personalizes treatment intervention. *Psycho-education* and normalization of trauma experiences of the child, subsequently *treatment rationale* and *goal setting*. *Imaginal reliving* helps elucidating trauma memories and identifying hotspots at this stage and cognitive restructuring and *updating trauma memories* follows. After that stage *working with triggers* and stimulus discrimination work needed to accomplish. After that *other interventions* like behavioral experiment, sleep hygiene and image work are used to suit child's needs. *Relapse prevention* plan is the last step of the intervention. Trauma related therapy is a tough area and generally become complicated by such factors like grief reactions, ongoing threats, multiple traumas and high co-morbidity rates. Emphatic and flexible style of the therapist may improve results of the patients.

KEYWORDS

PTSD; trauma; children and adolescents; CBT

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SYMPOSIA ABSTRACT: 564

Cognitive Behavioral Approach to Personality Disorders

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ABSTRACT

One of third of people who require mental health services have at least one personality disorder (PD) characterized by maladaptive inner experience and behavior. Since people with PDs can possess very different personality disturbances, they have at least one thing in common: their mental illness will not remit without professional intervention. However, exactly what that intervention should consist of remains a subject for debate. People with personality disorders exhibit chronic, pervasive problems getting along with people in all kinds of different contexts. As a result, people with the disorders often don't seek treatment, and those who do often drop out. For example, people with borderline personality disorder (BPD)- the most commonly treated personality disorder--quit treatment programs about 70 percent of the time. The bulk of research has focused on BPD. While the challenges are numerous and the research is preliminary, two interventions in particular--dialectical behavior therapy (DBT) and cognitive therapy (CT)--show promise for BPD. Still, psychologists seeking to treat the other nine personality disorders face a paucity of existing research. The good news: New theories on the underlying emotional regulation, interpersonal styles and thought patterns characteristic of these less-studied PDs have laid the groundwork for developing interventions. Many people with BPD harm themselves to regulate their emotions. In an effort at self-stabilization, some use physical pain--which has been demonstrated to reduce emotional arousal. Practitioners encourage BPD patients to develop alternative ways to control their frequently overwhelming and confusing feelings. For instance, a therapist may teach mindfulness, a concept borrowed from Zen Buddhism. Practicing mindfulness allows clients to observe their emotions without reacting to them or seeking instant relief through self-harm. At the same time, the therapist needs to appreciate the reality of the client's emotions. BPD patients require emotional acceptance--a DBT staple--because they often lacked it as children. In an invalidating environment, for example, a child might express anger and be told by a parent that she is jealous. They never gain a sense that their needs, wants and desires are reasonable. Adding that such circumstances can lead to emotional difficulties and a problematic sense of self. DBT helps these people restore their sense of self, and legitimizes their emotional experience. Evidence seems to back DBT's efficacy. In one study published 58 women with BPD were either assigned to DBT or treatment as usual--generally a weekly session with a psychotherapist. In the study, a team of clinicians assessed the participants' self-harming and damaging impulsive behavior, such as gambling and substance abuse, using the Borderline Personality Disorder Severity Index. After seven months of therapy, DBT-treated participants more successfully reduced suicide attempts, self-mutilating and self-damaging behaviors than those who received treatment as usual. Additionally, DBT patients were nearly twice as likely to stay in therapy. This study, shows that DBT can be learned and applied effectively by teams other than her own, she explains. While DBT emphasizes emotional regulation, CT conceptualize all 10 personality disorders as dysfunctional core beliefs about the self, others and the world. The cognitive therapist helps people with these disorders learn to identify and change these core beliefs. This is most often accomplished by weekly sessions with a trained therapist. A person with BPD, for example, may believe "I'm defective, helpless, vulnerable and bad. Everything that they do, everything that happens, ends up maintaining these beliefs.nIf they don't give money to a homeless person, they think they are bad. If they do, they think they should have given more. To root out such dysfunctional beliefs, CT practitioners often must help patients revisit and reinterpret early-childhood experiences. For example, a person may have picked up the belief, "I'm inadequate," because his parents had assigned him responsibilities he was not developmentally ready for. "Perhaps he was asked to take care of his younger siblings, and, not unreasonably, he failed". CT practitioners ask clients to move beyond thinking of such events as proof of inadequacy and instead explore alternative meanings. Ideally, the patient comes to understand the underpinnings of dysfunctional core beliefs and works to change them. However, problems can emerge if a patient interrupts that process by

applying his or her dysfunctional beliefs to therapy itself. Axis I patients often come to therapy believing 'I can trust my therapist, this is going to work. Axis II patients may think things like 'I can't trust my therapist, she might hurt me,' or 'If I listen to my therapist it will show how weak I am and how strong she is. To counteract such dysfunctional thinking, therapists should be ready to help patients examine dysfunctional beliefs about the therapist or therapy. Preliminary trials of cognitive therapy for BPD lend support to Beck's theory. In one such study 32 people with BPD benefited from cognitive therapy sessions conducted weekly over one year. Their borderline symptoms came down significantly after a year of therapy. At follow-up, 55 percent of the participants no longer met diagnostic criteria for BPD. While pilot studies have been promising, cognitive therapy has not yet been shown as an effective therapy for personality disorders other than BPD. Despite the divergences of their approaches, many psychologists agree that while treating personality disorders is not easy, it isn't impossible. "That personality disorders are not treatable was a myth that occurred because there was very little empirical research [on treatments]. As more studies get published, we will see that start to change.

SYMPOSIA ABSTRACT: 565

Antipsychotic Use in Period

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ABSTRACT
The fertility rate among women suffering from schizophrenic and other severe and persistent psychiatric disorders has increased since deinstitutionalization. This may be as a direct result of availability of sexual partners or concurrent changing attitudes toward conception among those with serious *mental illness*. Unfortunately, however, unplanned and unwanted pregnancies occur more frequently in women with SPPDs than in the general population. This may result in delayed or poor antenatal care. Both first and second generation antipsychotics are known as indispensable effective medications for this groups. So we reviewed antipsychotic use in pregnancy period.

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SYMPOSIA ABSTRACT: 566

General Overview on Oxytocin and Vasopressin

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ABSTRACT
Oxytocin and vasopressin are neuropeptides synthesized in the paraventricular and supraoptic nuclei of hypothalamus, and are released in the systemic circulation through the posterior pituitary gland (Ludwig and Leng, 2006). These neuropeptides act as hormones regulating a range of physiological functions including breastfeeding, labor and sexual activity. They are also released in the central nervous system, acting as neuromodulators and influencing various neurophysiological and behavioral processes including feeding, anxiety, aggression, social recognition and the stress/fear response (Stoop, 2012, Hashimoto et al., 2012). Evidence from animal studies demonstrated oxytocin and vasopressin play a significant role in the regulation of social behavior and cognition. These neuropeptides are associated with

KEYWORDS
Oxytocin; vasopressin; psychiatric disorder; neuropeptide; behavior

complex social and emotional processing in healthy people which may result in some psychiatric disorder symptoms if impaired. Furthermore, there are also some reports indicating the use of these neuropeptides in the treatment of various psychiatric disorders including psychosis, autism spectrum disorders and affective and anxiety disorders (Gumley et al., 2014). Several reliable methods including intracerebral microdialysis, targeted delivery of neuropeptide agonists or antagonists, gene knockout and viral gene transfer are used in animal studies to determine the effects of central oxytocin and vasopressin levels on behaviors (Pagani et al., 2014). However, these are not available in humans, hence researchers have turned to peripheral assays as proxy measures including plasma/serum, saliva, urine or cerebrospinal fluid levels of these neuropeptides have been tested as putative biomarkers in ASD, psychosis, bipolar disorder, major depressive disorder, as well as in anxiety, personality and eating disorders (Grazia et al 2016). However, results obtained from the human studies indicate conflicting results. Future well designed studies on this topic will improve our knowledge about the effect of oxytocin and vasopressin on human behaviors.

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SYMPOSIA ABSTRACT: 568

Current Approaches for the Treatment of Bipolar Disorder in Children and Adolescents

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ABSTRACT

Bipolar disorder is a significant mental health problem for both children and their parents. We observe an increase in incident of bipolar affective disorder as child and adolescent psychiatry clinicians each passing day. In children which is the most important stage of biopsychosocial development emergence of bipolar disorder leads to significant developmental problems. For this reason diagnosis and treatment of bipolar disorder in children is extremely significant. As in adults the most important part of the treatment consists of pharmacological treatments. Additionally psychotherapeutic methods are in use as supportive treatment. In this presentation it is going to be discussed general features and treatment options for children and adolescents with bipolar affective disorder with current perspective.

KEYWORDS

Bipolar disorder; children; treatment

SYMPOSIA ABSTRACT: 569

The Effects of Thyroid Functions on Psychiatric Conditions in Geriatric Population

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ABSTRACT
The ratio of elderly population is increasing in the community; so the old age and associated medical, psychiatric and social problems we face in coming years will increase. As mental disorders take so important place among the old age, greater emphasis are being given to them. Depression and hypothyroidism are progressively more prevalent in the population over 60 years of age[1]. Studies addressing the correlation between TSH levels and mood disturbances have been scarce in the elderly. However, the relationship between subclinical hypothyroidism and depression remains controversial many studies have suggested that subclinical hypothyroidism and thyroid autoimmunity may be risk factors for depressive disorders[2,3]. In addition, negative association has been reported between TSH levels and memory performance on cognitive tests[4]. In this presentation we will talk about association between thyroid functions and psychiatric conditions in elder adults, diagnostic difficulties and treatment options.

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SYMPOSIA ABSTRACT: 570

How to Treat Refractory Obsessive-Compulsive Disorder: Biological Approaches

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ABSTRACT
Obsessive-compulsive disorder (OCD) is quite a common disorder, with a life-time prevalence of approximately three percent[1]. Long-term outcome studies show that nearly half of the patients still suffer from the illness at the end of five years[2]. We aimed to review biological approaches in treatment-resistant OCD. We conducted a search in PubMed (until February 2017) and Scopus (until February 2017) by using the following terms: "refractory OCD", "resistant OCD", "pharmacogenetics", therapeutic drug-monitoring TDM)", "transcranial magnetic stimulation (TMS)". It is well evidenced to taper clomipramine and selective serotonin reuptake inhibitors (SSRIs) to the highest possible doses[3]. A combination of clomipramine and selective serotonin reuptake inhibitors might be beneficial.[2] Augmentation with certain antipsychotics has an evidence-based place in refractory OCD. Some other pharmacological options such as adjunctive topiramate or memantine are promising. Meta-analyses have supported the efficacy of TMS[4]. Pharmacogenetic approaches and TDM have not been established in OCD treatment[5]. Some augmentation strategies and TMS can help in managing treatment-resistant OCD.

KEYWORDS
Treatment-resistant obsessive-compulsive disorder; augmentation; pharmacogenetics; therapeutic drug-monitoring; transcranial magnetic stimulation; brain stimulation

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SYMPOSIA ABSTRACT: 571

Mentalization Based Psychotherapy Principles in Treatment of Children and Adolescents

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ABSTRACT

Mentalization is the imaginative mental activity whereby we make sense of the subjective states and mental processes of others and ourselves. This process can be described as imagining what others might be thinking or feeling; and being aware of what is going on in our own mind and someone else's. Mental states (e.g., beliefs, wishes, feelings and thoughts) influence our behavior, and mentalization includes perceiving and interpreting these behaviors as conjoined with mental states (Bateman, & Fonagy, 2006).

Mentalization involves both cognitive and affective processes, and can be implicit or explicit (Bateman, & Fonagy, 2006). Explicit mentalization is automatic, requires careful attention and is slow. Implicit mentalization on the other hand, is an automatic, fast and non-conscious process (Fonagy, & Luyten, 2009). Interpreting one's behavior in terms of their mental states is prone to error and uncertainty because mental states are readily changeable. Our reactions to others' behaviors may be based on wrong assumptions about their mental states, which may lead to unforeseen negative circumstances (Bateman, & Fonagy, 2006). Our ability to mentalize is determined by our early life. Infants surrounded by attentive and caring adults, who sufficiently understand the infant's mental states, develop a better ability to understand others. As such, psychological trauma in childhood can disrupt mentalizing in adulthood, as it weakens the capacity to think about mental states or the ability to give narrative accounts of past relationships (Bateman, & Fonagy, 2010). In other words, mentalization capacity is a developmental achievement rather than a given; and is disrupted by disruptions in early attachment (Fonagy, & Luyten, 2009).

From a mentalization point of view, most mental disorders can be seen as 'the mind misinterpreting its own experience of itself' (Bateman, & Fonagy, 2006). For example, individuals with chronic depression may view negative self-appraisals as reality instead of seeing them as merely cognitions. Thus, depression may be defined as a failure to distinguish between mental states and reality; a disruption of mentalization. Similarly, anxiety disorder may be seen as experiencing a threat or fear with the full force of reality rather than seeing fear as a mental state (Bateman, & Fonagy, 2006). Bateman and Fonagy (2010) suggest that the relationship between disorganized attachment and problems with affect regulation, attention and self-control in borderline personality disorder (BPD) is meditated by an under-developed mentalizing capacity. Patients with BPD have deficits in regulating emotional responses and coming up with effective strategies to control their thoughts and feelings. Especially at emotionally salient times, their capacity to understand actions in terms of mental states deteriorates. When the mentalization capacity is lost, negative self-appraisals are intensified, potentially leading to depression. When this happens, poorly regulated impulsive behaviors and self-harm override (Rossouw, & Fonagy, 2012).

Mentalization Based Treatment

MBT is a psychodynamic psychotherapy approach. The primary aim of an MBT intervention is to restore or maintain the ability to understand the mental states of oneself and others, when it is lost in emotionally challenging circumstances. As MBT is a more permissive approach, it can be used by clinicians from various backgrounds (Bateman & Fonagy, 2016).

The initial goals of MBT include the reduction of impulsive self-damaging behaviors and the development of a psychodynamic formulation with the patient. The long-term goals of MBT are to work on the identification and expression of affect and their appropriate expression with others, and to work on personal integrity, personal responsibility and interpersonal function. In order to prevent impulsive behaviors and set the base to start considering internal representations, the first aim of treatment should be to work on the identification and expression of affect. Treatment should aim to identify affect (e.g., love, hurt, catastrophe) and focus on the patient's mind, not just behavior (Bateman & Fonagy, 2016).

Similarly, MBT for adolescents (MBT-A) aims to enhance patients' capacity to mentalize. The program involves weekly individual MBT-A sessions and monthly MBT-family (MBT-F), and focuses on impulsivity and affect regulation (Rossouw, & Fonagy, 2012). Treatment consists of four phases. After the assessment phase, patients receive a written formulation and a crisis plan and risk assessment. Unstructured sessions focus on interpersonal experiences and mental states, separation issues and managing challenges using mentalization skills.

Russouw and Fonagy (2012) in their randomized controlled trial examined whether an MBT-A intervention would reduce self-harm in adolescents. Their sample consisted of 80 adolescents (85% female) presenting with comorbid self-harm and depression, who were randomly allocated to either MBT-A or treatment as usual (TAU) groups. The primary outcome of this study was self-reported self-harm, and secondary outcomes were depression, risk-taking and

borderline features (emerging BPD). Participants were assessed for these measures at baseline and every 3 months until 12 months after randomization. Mentalization and attachment status were also measured at baseline and at 12 months. A reduction in self-harm and depression at 12 months was apparent in both treatment groups, however these improvements were significantly greater in the MBT-A group compared to the TAU group. Because risk-taking at baseline was greater in the MBT-A group, the findings for this construct were difficult to interpret. When controlled for baseline risk-taking, there was a marginally significant difference in the two treatment groups, favoring MBT-A ($p<0.073$). The effect of MBT-A on self-harm appeared to be mediated by positive changes in mentalization and reduced attachment avoidance. BPD diagnoses and traits also decreased at the end of MBT-A treatment. Although both treatment groups improved in terms of self and observer reports of self-harm, 69% of the sample was still self-harming at the end of 12 months, demonstrating that recovery was not complete. The MBT-A group had a slightly better recovery rate (44%) compared to the TUA group (17%). In conclusion; the focus of the talk will be on introduction of contents of mentalization, therapeutic application of mentalization based treatments in children and adolescents, and review of research studies in this area. The talk will further extend insights into adapting mentalization techniques to cultural norms (especially Turkish culture). Lastly, a discussion of further study designs will be discussed. The participants are expected to learn about basic mentalization concepts and treatment applications

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SYMPOSIA ABSTRACT: 572

How Does Emotional Dysregulation Translate into Psychopathology?

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ABSTRACT

Emotion regulation has been traditionally linked with and externalizing disorders in children and adolescents. Although externalizing disorders are understood as problems of behavior rather than affect, these conditions are almost always tied in with emotional processes, and emotion and emotion regulation would appear to be centrally involved in such conditions (Gross J., 2007). It is crucial to consider specific forms of the externalizing behavior patterns when examining emotional and emotion regulatory processes, as deficits in emotion regulation are relevant to some but not all forms of externalizing behavior. Emotional regulation may not always be present in an individual, and it can further be said that emotional regulation "accidents" are norm rather than exception. Emotional regulation is a developmental process that is acquired in infancy and childhood. However, a persistent pervasive pattern of emotional dysregulation is a pattern that may suggest future psychopathology and thus be carefully investigated. Chronic emotional dysregulation, although a much broader concept can be recognized by presence of chronic irritability in youth. The clinical importance of severe, impairing and chronic irritability among youth has been recognized since 1990s although its diagnostic relevance has been controversial. Some authors posited that pediatric Bipolar Disorder (BP) could be divided into "narrow" and "broad" phenotypes with the former displaying classical symptoms of mania/ hypomania (i.e.,

grandiosity/ euphoria) in an episodic course (Leibenluft et al. 2003). According to this position; patients with the "broad" phenotype (also called Severe Mood Dysregulation Disorder, SMDD) displayed chronic, non- episodic, impairing irritability and hyper- arousal without classical symptoms of mania. Probably as a consequence of this position, there had been a dramatic rise in rates of pediatric BP from the mid-1990s to the early 2000s along with debates on the "true" phenotype of pediatric BP. Further studies revealed that episodic and chronic irritability in youth had distinct consequences and etiologies. Accordingly, it was posited that severe, episodic irritability in childhood correlated with BP in adulthood while severe, chronic irritability in childhood correlated with unipolar depression and anxiety disorders. This distinct phenotype further evolved into a new diagnosis, recognized by the American Psychiatric Association, and was listed in DSM 5 as Disruptive Mood Dysregulation Disorder (DMDD). DMDD is a novel diagnosis that is included in the DSM-5 among Depressive Disorders partly to solve this quandary although it has its detractors (APA 2013). It is characterized by severe, pervasive, impairing, developmentally inappropriate and recurrent temper outbursts that are grossly out of proportion to the situation at hand. The outbursts may be manifested verbally and/ or behaviorally and should occur at least three times per week for one year or more with a symptom-free interval of less than 3 consecutive months. Between outbursts, children with DMDD display a persistently irritable or angry mood, most of the day and nearly every day. The onset of symptoms must be before age 10, and a DMDD diagnosis should not be made for the first time before age 6 or after age 18. Bipolar Disorder, Oppositional Defiant Disorder and Intermittent Explosive Disorder should be excluded for diagnosis. DSM-5 reports a prevalence that is probably between 2-5 % with a male preponderance both in the community and in clinical samples. Homotypical continuity in 1-year follow-up is reported to be approximately 50 % and Major Depressive Disorder, Attention Deficit/ Hyperactivity Disorder (ADHD) and Anxiety Disorders are reported to be the most common comorbid diagnoses.

DMDD is criticized due to its potential to pathologize normal behavior (i.e. temper tantrums) with a consequent elevation in use of drugs, paucity of empirical evidence supporting the validity of diagnosis, low test-retest reliability and supporting studies focusing at selected centers and a not entirely overlapping diagnosis (McGough 2014). However, there are also studies supporting its validity as a distinct diagnosis (Dougherty et al. 2014). Thus, DMDD should be further studied to understand the neuropsychological underpinnings and differentiations from other disorders. By definition, DMDD is primarily a mood disorder however its association with disruptive Behavioral Disorders is undeniable. Symptom clusters that are needed to establish the diagnosis have commonalities with non-mood disorders such as Attention Deficit Hyperactivity Disorder (e.g., impulsivity). Although not listed in diagnostic criteria; a large proportion of ADHD population is known to have irritability and low self-soothing skills. The literature review shows that measures of sustained attention such as continuous performance tests (CPT) have not been tested for DMDD population. These tests involve execution of a predetermined reaction to target and non-target stimuli. Failure to execute the response to target stimuli is called an "omission error" and reacting to non-target stimuli is called "commission error". Omission errors may be linked to problems with attention, and some subtypes of commission errors may be associated with "inadequate control", or impulsivity. Studies report that children and adolescents with ADHD demonstrated higher rates of omission errors than unaffected peers. Although the clinical utility of these tests has been controversial, a large meta-analysis of 26 studies revealed that children with ADHD made significantly more errors of omission and commission than normal children. These errors are significantly corrected by use of psychostimulant medications. Studies also highlight the importance and effectiveness of incorporating distractors (auditory, visual and combined) in CPT to better distinguish ADHD from non-ADHD children. To our knowledge, there are no studies that compare DSM-5 DMDD diagnosis with ADHD subjects in terms of neuropsychological functioning. Prior investigations of neuropsychological functioning in ADHD and severe mood dysregulation compared to healthy controls revealed that performances of children with ADHD were significantly poorer than the healthy control group's performances. As a result, it can be said that children with ADHD-Combined differed significantly from healthy controls in the neuropsychological tests used, however comparison with SMD did not yield any differences in neuropsychological tests. Those two disorders differed only at the behavioral level.

In conclusion; the current talk will review current findings on emotional dysregulation, discuss where emotional dysregulation and chronic irritability fit in the current diagnostic system. The presenter will also discuss his unpublished data from ongoing research on face emotion regulation measures, neuropsychological functioning of subjects with DMDD, and a meta-analyses on Correlates and Comorbidity of DMDD.

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SYMPOSIA ABSTRACT: 573

Microbiota and Schizophrenia

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ABSTRACT
In the last century, it was suggested that dementia praecox or other psychiatric disorders could be linked with certain microorganism. Several aspects of schizophrenia suggest a possible microbial origin. One is that individuals sometimes present with the clinical symptoms of schizophrenia, in the course of developing a known microbial disease. Another epidemiological aspect of schizophrenia is the fact that both have a modest seasonal birth predominance in the winter and spring months. Lastly, being born in an urban environment has been found to be related with an increase in the risk of schizophrenia. Toxoplasma gondii and CMV are the microorganisms which have been studied most commonly in this topic[1]. Thousands of commensal microorganisms living within our bodies constitute the human microbiota. Influences of the microbiota extend beyond the gastrointestinal tract, playing a major role in the functioning of the central nervous system[2]. In recent years, the connection of the microbiota and psychiatric diseases has been the focus of attention of psychiatry community. It is now increasingly recognized that bidirectional communication exists between the brain and the gut microbiota. The role of the gut microbiota in schizophrenia is also under investigation[3]. The microbiome is known to play a critical role in the modulation of a range of neurotrophins and proteins. It is proposed that an altered microbiome may contribute to abnormalities in metabolic function in schizophrenia. Evidence that indicate possible microbiota alteration in schizophrenia includes structural damage to the gastrointestinal tract, a heightened immune response to infectious pathogens and food antigens[4]. Microbiota model epidemiologically suits the course of schizophrenia. The neurotrophic properties of the microbes and the chance of being infected in early life are consistent with neurodevelopmental theories of schizophrenia. They are also capable of reactivation in early adulthood, the peak time for the onset of the symptoms of schizophrenia. However, the relationship with microbiota-schizophrenia has not yet been fully elucidated. Further investigation is required to accurately put forward the causality between microbiota and schizophrenia.

KEYWORDS
Microbiota; gut-brain axis; schizophrenia

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SYMPOSIA ABSTRACT: 574

Childhood Depression and Biomarkers

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ABSTRACT
The occurrence of depressive symptoms in children and young people constitutes a serious public health issue, addressed by the WHO. International population-based studies in childrenaged 7–13 years show that the occurrence of severe depressive symptoms ranges from 4% to as high as 26.1%. Numerous studies have documented that early depressive episodes persist or recur into adult life along with ongoing psychosocial difficulties. A better understanding of the etiology and pathophysiology of pediatric depression will be helpful in the development and implementation of more effective primary and secondary preventive strategies. There is a general consensus that depression results from complex interactions between multiple genetic and environmental factors. There is fairly consistent evidence that childhood onset depression has familial determinants. In a large twin study the hereditability for depression was 29% in males and 42% in females, respectively. Research to date has emphasized candidate biomarkers based on depression , most notably the role of monoamine neurotransmission, immune-inflammation, neuroplasticity and neuroendocrine function. Especially dysregulated neurotransmitter systems are contributors to the development of depression: individuals with depression show alterations in neurotransmitters such as serotonin, dopamine, γ-aminobutyric acid (GABA), and glutamate. The relationship between depression and inflammatory markers was observed in unaffected adolescents who were at high-risk for depression based on familial or cognitive vulnerability, particularly in those who experienced childhood adversity. Depression was associated with inflammatory and metabolic markers in youth with diabetes. Reduced neurotrophic factors (including BDNF) have been reported in pediatric depression.

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SYMPOSIA ABSTRACT: 575

Possible Biomarkers in ADHD

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ABSTRACT
Children diagnosed with ADHD represent a heterogeneous group with a high variation in their symptoms. The diagnostic process is still somewhat unstructured and can be relatively easily biased. A meta-analysis of genetic information showed several implicated genes, although

KEYWORDS
ADHD; biomarkers; etiopathology; diagnosis

none of them can be used as a true predictive marker. Several genetic studies identified the main contributing factors as prefrontal dopamine deficiency and central dopaminergic dysfunction, but changes in oxidative metabolism and immunity were also suggested. Polymorphisms in the dopamine transporter and D4 receptor were also suggested as biomarkers Environmental risk factors, including heavy metals and substance/chemical exposures and nutritional factors; hypothalamic–pituitary–adrenal axis (HPA) alterations; and markers involved in other aspects of brain functioning (growth hormone and thyroid function , other neurotransmission systems, neurotrophic factors, complement C4-B, pineal hormone melatonin, oxytocin were also assessed as biomarkers in ADHD. In addition, Analysis of miRNAs in peripheral whole blood of Attention-Deficit/Hyperactivity Disorder (ADHD) patients revealed reduced levels of miR-18a, miR-22-3p, miR-106b and miR-107 in recent studies. It was suggested that The assessment an electroencephalographic (EEG), specially theta/beta ratio (TBR), could be a biomarker with a clinician's ADHD evaluation. Along with findings of aberrant brain volumes,cortical thickness, tissue microstructure, neural activation and neurotransmitter levels, recent findings of atypical brain iron levels have added another promising biomarker to examine. Urinary serotonin levels have been studied in ADHD using proteomic studies involving LC–MS/MS. A case studies series spanning 40 years revealed significant heterogeneity of size effects across studies for several potential biomarkers and confirmed association between studies for neuropeptide Y (NPY), dehydroepiandrosterone (DHEA). Additionally, free cholesterol, HDL and ApoA1 were found to be higher in children with ADHD versus controls. While the search for biomarkers has improved knowledge about the molecular biology, etiopathology, diagnosis and drug treatment efficacy of ADHD, further information is needed.

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SYMPOSIA ABSTRACT: 576

The Psychology of July 15

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ABSTRACT

A *coup d'état* attempt had taken place in Turkey, on July 15, 2016. This coup attempt was prevented by the active resistance of people at the expense of 250 martyrs and more than 2200 injured. When we talk about "The Psychology of July 15", we actually mean 2 events and 2 sides. The first side is the dimension of betrayal, the side of the coup; whereas the other is the side of the resistance which turned the coup attempt and the experience of trauma into the victory, the side of the people. We have to take into account these 2 sides, when we are talking about "The Psychology of July 15." In this speech, firstly, we will try to make evaluations about the psychology of the coup and the soldiers of the coup which currently become a common issue in the context of the democracy struggle in Turkey. Of

course, we will evaluate the particular characteristics of the July 15 *coup d'état* attempt. In this respect, we will try to explain the similarities and the differences of the July 15 *coup d'état* attempt from the previous coups. It is seen more and more clearly every day that July 15 coup attempt was orchestrated by a militant esoteric organization, called FETO or Parallel State Organization (PDY), which spreads spiritual insanity and sneakily settled in the state, especially in the army to seize power. In our speech, we will discuss the psychodynamics, the aims and the mechanisms of FETO; we will particularly try to clarify how they can find pathological motivations to embark upon a mass murder of their own people. In this context, we will concentrate on the concept of "mystery", which is vital for such organizations as well as the phenomenon of "to be identified in an organization" which appears to be a unique case in that is particular to our country. Another significant matter that must be taken into consideration in evaluating the psychology of July 15 is the resistance that the people did not demonstrate in previous coups. The key question to explain this resistance is "why people kept silent for previous coups", and especially for the executions of Prime Minister Adnan Menderes and his cabinet members, who came into power by election with a high majority vote, following the 1960 *coup d'état* in Turkey. If we could explain such matter (i.e., the silence of people), it would be relatively easy to show why people resisted on the July 15 coup attempt. In this presentation, I will try to discuss my observations with a theoretical background regarding the July 15 failed *coup d'état* attempt in Turkey and the above-mentioned issues as a psychiatry professor.

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SYMPOSIA ABSTRACT: 580

Correlation Between Thyroid Dysfunction, Autoimmunity and Mental Disorder in Pregnancy/Postpartum Period

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ABSTRACT

The peripartum period is one of the most unique periods throughout a female's life. During pregnancy and postpartum period numerous changes on the physiological, psychological and cellular level occur, that prepare the female for the challenges of motherhood. Those changes in maternal physiology, plasticity of the maternal brain, and maternal behavior will not only be essential for the offspring, but also help to maintain the physiological and mental health of the mother (1). Perinatal mental illness is largely under diagnosed and undertreated (2). Postpartum mental disorders can be divided into three classes: maternal blues, postpartum psychosis and postpartum depression. The first two groups are accepted as typical for the postpartum period and it is hypothesized that they are related to hormonal disturbances. Postpartum depression is an episode of non-psychotic depression that onset within 1 year of childbirth regarded as not being any different from other depressions (3). Etiology of postpartum depression (PD) can be related to major life events such as stress, marital conflict, previous psychiatric illness and other socioeconomic problems or it may have some biological factors; genetic vulnerability and hormonal changes (4). Hormones reviewed include progesterone, estradiol, cortisol, corticotropin-releasing hormone, prolactin, thyroid-stimulating hormone and triiodothyronine/thyroxine (5). Thyroid hormones regulate metabolism and affect almost every organ in the body. The hypothalamic-pituitary-thyroid (HPT) axis have been studied by psycho-neuro-endocrine researches, especially focusing on mood disorders. Thyroid function abnormalities generally appear to be associated with an increased rate of psychiatric symptoms (6). Monitoring of thyroid markers is important when on lithium therapy, for the risk of developing hypothyroidism; and to identify subclinical thyroiditis as a causative factor in treatment resistant depression (7). During pregnancy, thyroid hormones play a critical role for the development of a healthy baby (8). Other than pregnancy, thyroid tests are also evaluated in the postpartum period. Postpartum thyroid dysfunction (PPTD) was diagnosed in patients with thyroid dysfunction presented in the postpartum period (9). The specific clinical and psychiatric morbidity associated with PPTD is

still uncertain (10). Thyroid dysfunction is mostly caused by thyroid autoimmunity. Around 10% of women over 20 years of age have elevated concentrations of thyroperoxidase antibodies (TPOAbs), an early sign of thyroid autoimmunity (11). There have been conflicting results about the possible relationship between positive thyroid antibody status, PPTD and PD. Some studies have shown that women who are positive for thyroid antibodies are susceptible to postpartum depression. Both the TPOAb and thyroglobulin antibody are associated with PPTD and some studies have shown an association of postpartum depression with PPTD (12-15). On the other hand; some others found no relationship between the onset and evolution of both disorders (9,10).

Moreover; according to the some researchers the relation between the onset and the clinical course of PPTD and PD has never been clearly defined and further researches are needed to clarify the relationships between PPT and PD (3,16). The aim of this presentation is to discuss the correlation between thyroid dysregulation, autoimmunity and psychiatric disorders in pregnancy/postpartum period.

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SYMPOSIA ABSTRACT: 581

Depot Antipsychotic Drug Applications in Children and Adolescents

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ABSTRACT

Although the use of oral antipsychotics in children and adolescents has been well studied, there is little information on the intramuscular use of long-acting injectable antipsychotics. To date,

KEYWORDS

Adolescent; adverse effects; children; depot antipsychotic;

effectiveness

risperidone is the best studied antipsychotic agent in pediatric populations. A previous study investigated the effectiveness of long-acting injectable risperidon (LAIR) in adolescents with bipolar disorder[1]. In a study conducted in our clinic (Child and Adolescent Psychiatry Department of Ankara Yildirim Beyazit University Yenimahalle Education and Research Hospital), totally 42 patients (12–18 age range) who were non adherent to oral antipsychotic drugs, received 25 mg/day of LAIR intra muscularly every two weeks. Totally 81% of the patients (conduct disorder, bipolar disorder, schizophrenia) markedly improved with LAIR in children and adolescents. Weight gain, daytime somnolence, muscle stiffness and spasms, impaired concentration, fatigue, and menstrual problems in girls were the most common side effects. The LAIR treatment was terminated in 26.2% of the patients due to weight gain, dystonia, and galactorrhea. In children and adolescents who show noncompliance with oral drugs, LAIR may improve treatment compliance. LAIR is a reliable treatment in terms of its effectiveness. Typical antipsychotic depot drugs (zuclopenthixol decanoate, flupenthixol decanoate) are rarely used in children and adolescents due to extrapyramidal system and anticholinergic system side effects. Typical antipsychotics should be used with caution. As a result, when treating children and adolescents with oral antipsychotic drugs, noncompliance is a common problem due to familial non collaboration, lack of insight of the patient, and obstinacy. Thus, the LAIR regimen, which is administered twice a month, is more practical than the twice a day oral regimen in adolescent patients.

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SYMPOSIA ABSTRACT: 582

Autistic and Schizotypic Markers on Psychosis and OCD Axis

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ABSTRACT

Obsessive Compulsive Disorder (OCD) and autism spectrum disorders (ASD) share similar clinical characteristics, most notably symptoms of repetitive or stereotyped behaviors. The Diagnostic and Statistical Manual (DSM-5) describes the core features of ASD as persistent deficits in social communication and social interaction across multiple contexts and restricted, repetitive patterns of behavior, interests, or activities. ASD also includes specific deficiencies in social-emotional reciprocity, nonverbal communicative behaviors, and the ability to cultivate and maintain meaningful interpersonal relationships. Individuals with ASD may exhibit stereotyped or repetitive motor movements or language usage, the need for sameness, inflexibility, intensely fixated interests, and/or hyper- or hypo-reactivity to sensory input. The DSM-5 core features of OCD include the presence of obsessions, compulsions, or both. Similar to fixed interests in ASD, obsessions are defined as thoughts, urges, or impulses that are recurrent and persistent. However, the distinction between a circumscribed interest and an obsession is that obsessions are intrusive and distressing. Individuals with OCD attempt to neutralize their obsessions with a compulsive, repetitive thought or action. Other topographically similar behaviors shared by OCD and ASD include inflexibility, the need for sameness, and the repetitive nature of characteristic behaviors (compulsions, stereotypies)[1]. Baron-Cohen et al. (1985) raised the question as to whether the autistic child had a theory of mind (ToM) impairment, that is, a problem with basic metarepresentational capacity that restricted or prevented the child from imputing thoughts to others or being able to take another's perspective. Subsequently, a number of studies have demonstrated theory of mind deficits in autism that are common to autism in comparison to typical controls or to persons with other developmental disabilities. The inability to take another's perspective could account for many common behaviors seen in autism including a lack of showing, sharing, pointing, and comforting behaviors. Many social conventions logically flow from a theory of mind, and it is easy to imagine how someone could drift into idiosyncratic and seemingly bizarre behavior in the absence of the ability to see one's own behavior from the vantage point of someone else. In recent years, theory of mind deficits have also been demonstrated in schizophrenia. Dozens of studies largely done in the past 5 years suggest a direct relationship between ToM deficits and the presence and severity of delusions and schizotypal symptoms. Investigators have also demonstrated that family members of subjects with schizophrenia also have more difficulty with theory of mind tasks than does the general population has gone so far as to raise the question as to whether schizophrenia is, at its core, a

disorder of social cognition. Virtually all of these findings replicate similar results from studies that have focused on a population with autism spectrum disorders. Although ToM deficits are neither specific to autism nor schizophrenia and have been demonstrated in a variety of other conditions including, for example, Tourette syndrome, methamphetamine abuse, and some children with deafness. On the other hand, the fact that ToM deficits extend to non-affected relatives of individuals with autism and schizophrenia is intriguing. Moreover, in an interesting study examining social cognition in a population with both schizophrenia and deafness, impairments in facial affect processing exerted a significant mediating effect on outcome. Advances in our understanding of the pathophysiology and neuropsychology of autism and of schizophrenia, coupled with genetic findings that invite a broader look at where diagnostic boundaries have been drawn around these disorders, have created an opportunity to revisit their relationship. Although certain symptoms have come to be pathognomic of specific disorders, and clinically, it is not difficult to discriminate autism from schizophrenia, there is growing evidence that the broader phenotypes of these spectra may overlap. Taking a more inclusive look at these areas of convergence may importantly inform and advance our understanding of the pathogenesis of these conditions. Thus, whether schizophrenia lives on the autism spectrum, or vice versa, or whether there is a sub-population at their intersection, reconnecting these phenotypes may provide new insights in terms of early identification and treatment[2]. Schizotypal personality disorder (SPD) shares common phenomenological, biological, genetic and treatment response characteristics with schizophrenia. Inter-correlation between schizotypal and obsessive-compulsive (OC) dimensions has been consistently found in non-clinical samples of students selected on the basis of self-report scales of schizotypy, as well as in the treatment-seeking OCD population. Nevertheless, the rate of occurrence of SPD in OCD is yet to be clarified. The observed rates vary substantially (0–50%). This may be accounted for by differences in the definition of schizotypal features (categorical vs. dimensional), method of evaluation (structured interview vs. chart review) and the patient population studied. Despite their methodological differences, studies are consistent in demonstrating that OCD patients with associated SPD exhibit a more deteriorative course and poorer prognosis than those with "pure" OCD. None of these studies, however, was prospective. The clinical validity of the OCD–schizotypy association has also been supported by showing differences in demographic and clinical characteristics of OCD patients with and without schizotypal features. Specifically, early age of onset, male gender, counting compulsions and a history of specific phobia substantially increased the odds of schizotypy in patients with lifetime OCD. Furthermore, neurocognitive findings lend some credence to the division of OCD into subgroups based on the presence of schizotypy. Whereas patients with "pure" OCD display impaired performance on measures sensitive to orbitofrontal cortex (alternation learning, response inhibition, delayed memory), OCD patients with SPD perform poorly on tests sensitive to both orbitofrontal and dorsolateral prefrontal cortex (e.g., executive function). Deficits in cognitive performance on tests sensitive to dorsolateral prefrontal cortex are consistently found in patients with schizophrenia and SPD. An additional validator of the OCD–SPD association is the differential treatment approach required in patients with both disorders as compared to those with OCD alone. Compelling evidence indicates that the presence of SPD predicts poor response to standard pharmacological (SSRIs) and behavioral intervention in OCD patients. Moreover, the presence of SPD is a good predictor of response to low-dose antipsychotic agents (pimozide, olanzapine) added to SSRIs. It is of note that therapeutic efficacy of low-dose antipsychotics has been demonstrated in non-OCD related SPD. Overall, a category of OCD with schizotypal features seems to have clinical and predictive validity and probably etiological specificity. However, a lack of well-designed controlled studies evaluating neurobiological and neurocognitive markers and predictors of treatment response in OCD patients with and without schizotypal features precludes comprehensive characterization of an OCD–SPD subgroup[3].

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SYMPOSIA ABSTRACT: 583

Psychiatric Conditions, Treatment Approaches and Rehabilitation After Traumatic Brain Injury (TBI)

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ABSTRACT
Traumatic brain injury is caused by an external mechanical force injuring the brain and leading to its dysfunction. The syndrome usually results from a violent blow or concussion to the head. The increase in the working population together with industrial and technological developments has raised the incidence of accidents, many leading to traumatic brain injury. In addition to the acute effects of head trauma; sensory, motor, cognitive and psychiatric disorders may present according to the area affected by the injury. Although motor and sensory deficits improve over time, emotional and behavioral changes last longer[1,2]. Delirium and amnesic disorder are common in the acute phase Of the injury. On the other hand, cognitive impairments such as attention deficit and loss of abstraction, mood changes such as depressive or manic episodes, psychotic symptoms and personality changes can be seen in long term after the traumatic injury[4,5]. In a recent study, attention deficit hyperactivity disorder has been shown to be a risk factor for early head trauma[3]. Long-term psychiatric effects of head trauma have been reported as loss of energy, dizziness, headache, irritability, memory complaints and depression respectively. Studies have shown the importance of psychiatric comorbidity in traumatic brain injury.

KEYWORDS
Head trauma; psychiatric approach; brain damage; posttraumatic situation

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SYMPOSIA ABSTRACT: 584

Adverse Effects of Antipsychotics in Children and Adolescents

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ABSTRACT
In recent years, antipsychotic use, especially atypical antipsychotics, in children and adolescents has dramatically increased[1]. Side effects that occur during the use of antipsychotics may have a detrimental effect on the patient's treatment compliance and quality of life. Children and adolescents are reported to be more susceptible to adverse effects of antipsychotic medications than adults. Neuromotor, cognitive, metabolic, cardiac, anticholinergic, urogenital untoward effects have been detected in children and adolescents treated with antipsychotic agents. Treatment factors such as dosing and exposure time may contribute to the emergence of these adverse events. Importantly, drowsiness, sedation, and impaired attention may affect learning and may disrupt the feedback between maturational brain changes and cognitive processes that empower to develop more effective intellectual and social tasks. Neuromotor side effects are side effects such as acute dystonic reaction, parkinsonism, akathisia, withdrawal dyskinesia, tardive dyskinesia, neuroleptic malignant syndrome. In a study carried out in our clinic, the acute dystonic reaction was observed in 30 (6.8%) of 441 patients with a mean age of 13.05±3.50 who received antipsychotic treatment. This ratio is 2.06% in 388 patients treated with single antipsychotic, 17.6% in 34 patients using dual antipsychotics, and 84.2% in 19 patients using three combined antipsychotics. Dystonia was most common with haloperidol (11.11%) followed by olanzapine (5.88%), aripiprazole (3.22%) and risperidone (0.86%) in the single antipsychotic users. The doses of risperidone, aripiprazole, olanzapine, quetiapine and haloperidol used in patients with dystonia were observed to be significantly higher than those who did not develop dystonia. In a meta-analysis study of antipsychotic use in early-onset schizophrenia, all antipsychotics except asenapine, quetiapine, and ziprasidone were reported to cause more akathisia than placebo[2]. Parkinsonism and dystonia are more common in children and adolescents, while akathisia and tardive dyskinesia are more common after adolescence. It is also known that antipsychotics lower the seizure threshold.

Endocrinologically, metabolic changes such as incompatible with age weight gain, glucose and lipid metabolism abnormalities can be observed in both genders. Hyperprolactinemia and associated amenorrhea, oligomenorrhea, galactorrhea and tenderness in the chest are frequently seen. Alters bone density, sexual maturation, or the risk of breast cancer or benign

KEYWORDS
Adolescent; adverse effects; akathisia; antipsychotic; children; dystonia

prolactinomas may emerge due to long term hyperprolactinemia[3,4]. Antipsychotics may differentially prolong the heart rate-corrected QT interval. In particular, ziprasidone should be used with attention because of higher Q-T prolongation risk. Attention should be required to anticholinergic side effects such as constipation, narrow-angle glaucoma, and a decrease in body secretions, which are more common with typical antipsychotics. In conclusion, antipsychotic use causes more sedation, acute EPS, withdrawal dyskinesia, hyperprolactinemia, age unconformity-inappropriate metabolic abnormalities in children and adolescents. Clinicians and researchers should use age-appropriate side effect measures to evaluate and manage antipsychotic risks and benefits in young patients.

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SYMPOSIA ABSTRACT: 586

Tic Disorders and Comorbidities

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ABSTRACT

Tic Disorders and Attention Deficit Hyperactivity Disorder

Attention deficit hyperactivity disorder (ADHD) is the most common comorbidity seen in tic disorders (TDs). It has been reported that combined type ADHD is more common in tic disorders. Presence of TDs in association with ADHD doesn't affect the symptoms ADHD; however, it aggravates tic-related symptoms. Even in such condition, it should be taken into account that ADHD diagnosis is more important for long-term dysfunction.

Tic Disorders and Obsessive Compulsive Disorder

Obsessive compulsive disorder and sub-threshold symptomatology may be added to TDs as well as tics may be present in cases with OCD. In fact, prevalence of TDs and tics has been reported as 7% and 20% in cases with OCB, respectively[3].

OCD is second most common comorbidity in TDs and it is generally accepted that it has a hereditary association with tic disorders.

Tic Disorders and Aggression

In an international study, it was found that point and lifetime prevalence of anger control problem/aggression were 25% and 37% among cases with TDs, respectively. Aggression seen in TDs is mostly reactive and associated to ADHD symptoms.

Tic Disorders and Other Anxiety Disorders

Anxiety disorders are one of the most commonly seen psychopathologies in children and adolescents. These disorders are most frequently associated to other anxiety disorders. Available data suggest that prevalence of anxiety disorders show no marked increase in children with TDs.

Tic Disorders and Affective Disorders

It has been reported that affective disorders, primarily major depressive disorder (MDD) and persistent depressive disorder (dysthymia in DSM-IV-TR), can be frequently associated to TDs; however, these diagnoses are commonly missed.

TIC DISORDERS and OTHER COMORBIDITIES

Specific Learning Disorder

The prevalence of comorbid specific learning disorder (SLD) is found as 22% in TDS while its prevalence has been reported as 15-20% in general population. Thus, it may be suggested that TDs doesn't increase risk for SLD specifically.

Self-Harming Behaviors

Prevalence of self-harming behavior can range from 17% to 53%. Some authors reported that self-harming behaviors are associated to motor tics while Kurlan advocated these behaviors are

KEYWORDS

Tic disorders; Tourette's syndrome; comorbidity

independent from tic severity.

Autism Spectrum Disorders

TDs frequently accompany to autism spectrum disorders (ASD) and this association creates specific challenges in terms of diagnosis. It was suggested that tics such as hand clapping and sniffing make diagnosis in particular. In some cases, OCD and TDs can occur during adolescence[1,2,5].

Mental Retardation

There is a simplex relationship between TDs and mental retardation (MR). TD prevalence is increased in cases with MR but vice versa isn't true.

Developmental Coordination Disorder, Trichotillomania, Substance Use Disorder

Developmental coordination disorder (DCD) accompanies to TDs especially in the presence of comorbid ADHD and academic problems.

Trichotillomania is increased in TDs. In particular, it is more common among women with TDs and may increase risk for anxiety disorders.

Substance use disorders alone don't increase risk for TDs. The group with association of TDs and ADHD are particularly at increased risk for development of substance use disorders.

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SYMPOSIA ABSTRACT: 589

Iodine Deficiency, Nuclear Disaster, and Autoimmunity: Psychiatric Disorders in an Endemic Goiter Area of the Eastern Black Sea Region

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ABSTRACT

It has been known that thyroid hormones have manifest effects on human brain and behavior. In many studies, clinical or subclinical thyroid dysfunction have been found to accompany neuropsychiatric outcomes such as bipolar disorder, unipolar depression, psychosis, anxiety disorders and dementia (1-3). Although Turkey is a country with moderate iodine deficiency, the Eastern Black Sea Region, where kale (Brassica oleracea Acephala) is widely consumed, is one of the locations where endemic iodine deficiency is profound. For this reason, Turkey's first and only goiter research hospital is in Fındıklı Province of Rize, serving the region (4,5). In spite of opposing studies in the literature, the nuclear explosion that broke out in Chernobyl in 1986 may be another reason for the frequent occurrence of thyroid dysfunction and psychiatric disorders in this region (6,7). Autoimmune thyroid diseases may bind with other pathologies such as neurological and mental disorders. Historically, this relationship was assumed that the concurrent non-thyroid disorders were consequences of exposure to elevated levels of thyroid hormones. Recent studies have shown that these psycho-immune-endocrine disorders frequently share common etiological and pathological mechanisms (8). In both unipolar and bipolar depression patients, autoimmune thyroid antibody levels were found to be higher than in healthy individuals (9).

There are many studies indicating the association between the presence of thyroid antibodies and treatment-resistant, atypical, gestational, postpartum and perimenopausal depression as well as rapid cycling and mixed bipolar disorder (10-12). Most of the untreated individuals who were diagnosed with Graves' disease, an autoimmune disease with hyperthyroidism, were found to have comorbid major depression, generalized anxiety disorder and hypomania (13). On the other hand, some studies have found that patients with schizophrenia-spectrum disorders are more likely to have autoimmune thyroid disease comorbidity than those with affective disorder (14,15). In addition to be an area deficient in iodine, the Eastern Black Sea Region is one of the lands most exposed to the Chernobyl disaster. Comorbidities of thyroid dysfunction and psychiatric disorders observed in the region will be discussed in context with the literature.

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SYMPOSIA ABSTRACT: 590

Bakırkoy Community Menthal Health Center Program as an Integrative and Individualized Treatment of Schizophrenia

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ABSTRACT

Our center, which opened as Day Hospital in 2006, has been serving as a Community Mental Health Center since 2011. 792 registered patients are treated and rehabilitated. Our team consists of psychiatry specialist, psychiatrist assistant, psychologist, nurse, ergotherapist and social worker. Apart from the treatment team, there are teachers of occupation. The entire team works as a case manager. All the patients who are followed in the center are the case managers, and all the patients are informed about the whole team. The training and supervision of the team is the task of the psychiatrist specialist who is in charge of the center. Case management is a process in which all services are coordinated and coordinated in order to meet the various needs of the individual (treatment, social, accommodation, financial, employment, rest, cultural needs). Assertive Community Treatment (ACT) is the model we have taken as an example. The ACT model is one of the most comprehensive assessed mental health interventions and there is good evidence of effectiveness. (1998 study). The mobile crisis resolution model aims to treat people at home during the acute mental crisis and to avoid hospital needs. It is a dense, limited-time approach. (A few weeks or month). This model is inadequate in solving the problems of heavy and ongoing people. This is why ACT was developed. In ACT functioning, the theme is passed in the environment where the individual lives, at home, at the park, at the café, at the work place. Multiple trials, flexible and diverse approaches, friendly approach, practical support offer, no retirement policy, secure connection to maintain long-term relationship, working with teamwork, frequent visits by patients and discussions, supervision meetings, team talents and cooperation with external agencies There are tasks such as setting up.

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SYMPOSIA ABSTRACT: 591

Social Sequelae, Functionality, and Legal Dimension Due to Traumatic Brain Injury

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ABSTRACT

Traumatic brain injury (TBI) is a result of a mechanical force to the head, causing widespread brain damage. The prefrontal cortex is affected so that the patients have difficulties on executive functions and communication skills in everyday life (1). Cognitive training has been found to be effective in improving cognitive and functional outcomes in patients suffering, therefore it should play role in post-acute TBI rehabilitation (2). Investigating and treating self-awareness is another key point to improve the concomitant neuropsychiatric disorders. Group therapies help to acquire self-awareness, focusing on impulsivity and unrestrained emotions and cognition (3). The most severe behavioral symptoms were found to occur in the context of severe emotional symptoms, even in the absence of cognitive impairment. Likewise, problematic behavioral symptoms were also present in the context of severe cognitive impairment, even in the absence of emotional symptoms. The emotional, cognitive, and behavioral changes due to TBI have personal aspects depending on the area affected and premorbid traits. Therefore, personalized therapies and rehabilitation programs should be managed for this population (4). TBI appear to affect overall quality of life and mental health negatively. Affected people show significantly more depressive symptoms than controls. However, perceived quality of life ratings had no significant difference between the TBI patients and controls. A high level of physical and social independence were found to be positive determinants of a perceived high quality of life (5).

Rehabilitation services should be improved and should be more accessible, reaching the families to provide them sufficient education in order to involve in the treatment and rehabilitation process (6).

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SYMPOSIA ABSTRACT: 592

Autistic and Schizotypal Traits in Childhood Mental Disorders

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ABSTRACT

Autism Spectrum Disorder (ASD) is characterized primarily by lack of persistence in interpersonal interaction and communication, and repetitive, restricted, stereotypical behavior and is classified under the heading neurodevelopmental disorders.

Although it is known that hyperactivity symptoms are frequently observed in individuals with autism, the frequency with which autistic symptoms occur in ADHD is not well researched.

Individuals with ADHD and ASD show significantly overlapping symptoms at high rate.

These are problems such as social-communication skill deficits, delays in language development, sensory hypersensitivity, attention problems, oppositional behavior and emotional regulation. The most frequently reported autistic symptoms in children with ADHD are deficits in social interaction. The symptoms of stereotypic and strict behaviors observed in ASD have similar patterns to obsessions and compulsions, which are the main features of obsessive compulsive disorder (OCD). In the literature, the interaction between repetitive behaviors and OCD are examined in a small number of studies. The findings of neuroimaging studies of symptoms seen in both disorders strengthen the idea that there are similarities between these two disorders. Some researchers have suggested that, despite these behavioral and anatomical similarities, there is a significant difference in autism in terms of repetitive behaviors when compared to OCD. Schizotypal personality disorder is characterized by cognitive or perceptual distortions and unconventional behaviors, such as discomfort in close relationships and reduced ability to build relationships. Schizotypal personality disorder overlaps with the social problems seen in autism spectrum disorders, such as inappropriate and limited emotion, lack of close friends, inadequacy in the social environment. Long-term follow-up studies suggest that schizotypal disturbances develop in individuals with ASD and that schizotypal symptoms can be observed in individuals with ASD during adolescence. It is important to notice that adolescents with schizotypal personality traits should also be considered in terms of ASD. Additionally, autistic features are shown to be higher in mood disorders and anxiety disorders than other psychiatric disorders in studies. In this presentation, the relationship between OSB and schizotypal features and other childhood psychiatric disorders will be discussed in lights of recent studies in various aspects.

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SYMPOSIA ABSTRACT: 593

Ways to Rediscover the Past: Prolonged Exposure Therapy

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ABSTRACT

Posttraumatic stress disorder is a psychiatric disorder that affects the quality of life and social functioning of a person who develops after a life-threatening, unpleasant or desperate life threatening person's life and body integrity. Treatment of the disorder is done by using pharmacological and psychosocial therapies together. It is known that cognitive behavioral therapies improve symptoms of post-traumatic stress disorder by 70% in combination and single use. Therefore, many treatment guidelines have included cognitive-behavioral therapy as a first-line treatment option. Exposure is an application to reduce "pathological fear" and its related feelings; in this way patients are faced with exaggerated objects, situations, thoughts and similar stimuli to reduce anxiety levels under control. Exposure practice can be done in two different ways: "in vivo exposure" or "imaginal exposure". Prolonged exposure

therapy is a special treatment program designed for post-traumatic stress disorder treatment and is based on "emotion processing theory".This treatment program consists of four main parts: 1. Psycho-education about trauma and post-traumatic disorders, 2. Breathing exercise education, 3. In vivo exposure, 4. Imaginal exposure. The program consists of 1 to 2 times a week, 60-90 minute interviews and takes 9-15 weeks in total.

According to the theory of emotional processing the "structure of fear" consists of objects or situations (stimuli) that reveal fear, physiological and psychological responses to stimuli, and implications related to these stimuli and reactions. According to emotion processing theory; the most important cause of the natural healing process in the person experiencing trauma is the cognitive and behavioral avoidance of traumatic memories and all the situations that cause the responses to be resurrected. Avoidance prevents trauma memories from being processed, causing the structure of fear in the memory of a person to become pathological (pathological fear), and therefore the disorder becomes processive. The purpose of prolonged exposure therapy is to assimilate the knowledge of fear that has become pathologic after trauma in the previous memory system and to improve the process of the feeling about trauma as it is healing from the natural path. In order for the pathological structure of fear to be rearranged into the previous memory system, two conditions must be met; 1.activation of fear structure, 2. provision of corrective feedback. If these two conditions are met, information about the incident can be absorbed in the existing memory. All these awareness provide cognitive restructuring and allow the patient to reassess the traumatic event in a new way of thinking and in a safe environment during therapy. All of these changes help to reduce the number and severity of symptoms, and to provide a feeling of superiority and competence over the event, leading to major depression, generalized anxiety, anger, and guilt symptoms that develop after trauma.

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SYMPOSIA ABSTRACT: 594

Autistic and Schizoid Features in Social Anxiety Disorder

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ABSTRACT

Social Anxiety Disorder (SAD) is one of the most common mental disorders, as shown by two recent general population surveys in the USA, which both found that almost one in 10 individuals suffers from SAD at some point in their lifetime[1–3]. Onset typically occurs in childhood or early adolescence and takes a chronic course. SAD may cause significant distress and impairment in educational attainment, employment opportunities, the development of professional, peer and intimate relationships, and financial independence.4 The early onset and chronic course with rare spontaneous remission frequently causes secondary comorbidities, such as depression and alcohol-related problems[4]. The conceptualization of SAD has rapidly altered and expanded during just the past few decades as progress has been made in SAD treatment strategies. The disorder was initially considered as just a type of phobic reaction to a specific social situation akin to a specific phobia in DSM-III.5 With the advent of DSMIII-R, six diagnostic criteria for the disorder underwent significant expansion. The specifier 'generalized' was introduced to the formal nosology in DSM-III-R6 to describe persons experiencing social fears in 'most or all' situations while allowing concurrent diagnosis of avoidant personality disorder. The label 'social anxiety disorder' was introduced in DSM-IV, 7 and replaced 'social phobia' in DSM-5[1]. Moreover, in the current version of DSM, typical fear and anxious situations are defined as: (1) social

KEYWORDS

Social; anxiety; disorder; autism; schizoid; spectrum

interaction fears; (2) observation fears; and (3) performance anxiety (in criterion A). Thus fear of performance in public was deprioritized compared to social interaction in DSM-5.

Autism spectrum disorders have been of heightened concern in recent years. Persistent impairment in reciprocal social communication and social interaction (Criterion A) and restricted, repetitive patterns of behavior, interests, or activities (Criterion B) are their essential features[1]. Initial interview with individuals who have generalized SAD frequently cannot correctly assess social communication capacity because of patients' anxiety in the appointment. However, careful observation in future appointments and additional assessment will reveal adequate reciprocal social communication and social interaction with familiar or intimate partners. Autism spectrum disorders have been of heightened concern in recent years. Persistent impairment in reciprocal social communication and social interaction (Criterion A) and restricted, repetitive patterns of behavior, interests, or activities (Criterion B) are their essential features[1]. Initial interview with individuals who have generalized SAD frequently cannot correctly assess social communication capacity because of patients' anxiety in the appointment. However, careful observation in future appointments and additional assessment will reveal adequate reciprocal social communication and social interaction with familiar or intimate partners.

Autism spectrum disorders have been of heightened concern in recent years. Persistent impairment in reciprocal social communication and social interaction (Criterion A) and restricted, repetitive patterns of behavior, interests, or activities (Criterion B) are their essential features[1]. Initial interview with individuals who have generalized SAD frequently cannot correctly assess social communication capacity because of patients' anxiety in the appointment. However, careful observation in future appointments and additional assessment will reveal adequate reciprocal social communication and social interaction with familiar or intimate partners. As with schizoid personality disorder, the social avoidance of schizophrenia should be distinguished from the social fear of SAD. However, due to the chronic course of generalized SAD, it can be associated with schizophrenia, though predictors have not been found. The differential diagnoses for these psychiatric diseases must be studied carefully. We aimed to review these common and concerning points about these three disorders.

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SYMPOSIA ABSTRACT: 595

Tic Disorders and Comorbidity: Approaches to Treatment Refractory Cases with Tourette's Syndrome

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ABSTRACT

Tourette's syndrome (TS) is a complex neurodevelopmental disorder marked by both motor and phonic tics over a period of at least 1 year with the onset in childhood or adolescence. Although symptoms usually decline by adulthood, a significant number of patients fail to respond conventional medical and behavior treatments. Most of the patients with TS have associated psychiatric comorbidities which may have a role of apparent refractoriness. First, concomitant psychiatric comorbidities, the current definitions and clinical characteristics of treatment refractory TS will be presented. Then, the strategies for the management of treatment refractory TS, potential new treatments such as transcranial magnetic stimulation (TMS), deep brain simulation (DBS) and novel pharmacological treatments will be discussed.

Finally, two cases of treatment refractory TS and their follow-up period will be presented.

Comorbidities

A recent cross-sectional study of over 1000 TS patients found that 86% of all patients had at least one psychiatric disorder, and 58% had 2 or more psychiatric disorders (Hirschtritt et al., 2015). Psychiatric comorbidities contribute essentially to functioning and to limit treatment response in TS, and might also increase the likelihood of receiving medical therapy for tics.

ADHD is the most common comorbidity in the TS patients, ranging between 60 and 80 %, is the leading factor affecting disruptive behavior in TS (Kumar, Trescher, & Byler, 2016). ADHD symptoms precede the onset of tics by 2–3 years and begin around the age of 3–5 years. The hyperactive subtype is predominant in the younger patients, whereas inattentive type is more common in adolescents. Untreated ADHD may also complicate therapeutic interventions such as habit reversal therapy (HRT), making them less effective. The prevalence of OCD ranges from 11 to 80 % of patients with TS. The clinical presentation of OCD symptoms in TS patients can be different than the typical presentation of patients with primary OCD. TS patients are found to have greater rates of symmetric obsession, obsessional counting, and "just right" perception, however primary OCD patients reported higher rates of cleaning rituals, compulsive washing, and fears of contaminations. Malignant TS, which has life threatening symptoms or requires hospitalization, is found strongly associated with the presence of OCD or obsessive-compulsive behaviors. In fact, OCD may contribute more to tic severity than other comorbid conditions (Kious, Jimenez-Shahed, & Shprecher, 2016). There has been a significantly increased prevalence of impulse control disorder (especially intermittent explosive disorder) in TS patients. Depressive symptoms also found to present thirteen to seventy-six percent in TS. Depression has been shown to be associated with social stigma such as bullying, teasing, and receiving derogatory nicknames in TS children. Twelve to sixty-two percent of patients with TS reported to have various sleep problems such as nightmares, night terrors, somnambulism, trouble initiating sleep, and restlessness (Kumar et al., 2016). There are no reliable estimates regarding treatment-refractory, and there is little known about the mechanisms that promote refractoriness. Greater tic severity in childhood and early fine motor deficits predicts tic persistence in adulthood. Reductions in total caudate volume in childhood found to be correlated with tic severity in early adulthood.

Definitions and clinical characteristics of treatment refractory TS

It is crucial to carefully assess the adequacy of treatment trials and the reasons they appear to have failed before declaring a patient to be treatment-refractory. This reasons could be incorrect diagnosis, insufficient doses, side effects, inaccessibility to available therapies (including expert psychiatric care and behavioral therapy). TS should be considered truly treatment-refractory if multiple, well-established interventions, including psychotherapy, fail to produce sufficient treatment response either alone or in combination, and there has been adequate control of comorbid psychiatric conditions. The most recent and detailed definitions of treatment-refractory TS have been suggested through guidelines for the selection of patients for DBS: a DSM-5 diagnosis of TS, involvement of ethics committee for cases younger than age 18 years, tics as the major source of disability, with YGTSS total tic severity upper than 35/50, video documentation of movements, failure of conventional therapies (medications from 3 pharmacologic classes), and a trial of CBIT if feasible. Additionally, appropriate documentation and treatment of psychiatric comorbidities, stable medical condition and psychosocial status, and the absence of suicidal or homicidal ideation for 6 months are also recommended.

Management of treatment refractory TS:

Repetitive transcranial magnetic stimulation (rTMS)

rTMS is non-invasive, safe and well-tolerated neuromodulation technique, has been utilized in a variety of psychiatric and neurologic disorder. An average reduction of YGTSS scores of 34% and improvement of comorbid conditions are demonstrated in two open-label trials of rTMS. A recent study in adults had less encouraging results which reported a trial of deep rTMS in 10 adult patients with TS with or without OCD. Subjects tolerated the intervention well, but there was also no significant reduction in tic severity, except in the subgroup of patients with comorbid OCD. Recently a randomized controlled study could not find significant difference in the reduction of YGTSS scores between the rTMS group and sham. Larger, well-controlled trials of rTMS are needed to identify the optimal stimulation parameters and paradigms. Additional clinical trials are currently under way to address this need, as listed in the US clinical trials database (www.clinicaltrials.gov: NCT02356003, NCT00965211).

Deep Brain Stimulation (DBS)

DBS is emerging as a treatment option for medically intractable patients. Due to its invasive nature and the possibility of remission or significant symptomatic improvement in TS with age, especially children and adolescent patients must be carefully selected. On the other hand, adolescence is a crucial time period, which identity formulation, psychosocial and academic development both occur. The chance for greater symptom control via DBS during this time might also would result with better psychosocial outcomes in these severe cases. In a recent meta-analysis, including 156 cases from 57 eligible studies, DBS resulted in a significant improvement of 52.68% (IQR = 40.74, $p < 0.001$) in the YGTSS. Analysis of controlled studies significantly changed stimulation versus off stimulation with a standardized mean difference of 0.96 (95% CI: 0.36– 1.56). Different target points revealed significant YGTSS reductions after stimulation of the thalamus, the posteroventrolateral part and the anteromedial part of the globus pallidus internus, the anterior limb of the internal

capsule and nucleus accumbens (Baldermann et al., 2016). There is limited data about the long-term efficacy of DBS for refractory TS, but existing reports are mainly positive. It was found that patients continued to demonstrate reductions in tic severity and obsessive-compulsive behaviors, and required less medication for TS and for psychiatric comorbidities. Adverse effects can be limited the use of DBT and can be divided into three categories, which are surgical, stimulation-related, or hardware-related. Reduction in energy levels, psychosis, hypomania and anxiety was reported as psychiatric symptoms. It is also suggested that patients with TS may be at increased risk of post-surgical infection due to compulsive touching or picking of the wound or scar.

Electroconvulsive Therapy (ECT)

ECT is shown to be effective and less invasive neuromodulation technique for treatment-refractory TS. Several case reports have subsequently been published with positive results (Kious et al., 2016). There are no controlled studies involving ECT for TS, and few reports involving TS that has not concomitant comorbid psychiatric illness. ECT should be considered for TS primarily in emergency situations (e.g., severe self-injurious behavior), or when there are comorbid psychiatric conditions that ECT is also effective.

Novel Pharmacotherapies

Newer treatments for TS with a growing evidence-base include anticonvulsant agents (Topiramate), vesicular monoamine transporter inhibitors (Tetrabenazine), glutamatergic agents including D-serine, riluzole, nacetylcysteine (NAC), and acamprosate (Kious et al., 2016).

Cases

Two treatment resistant TS cases (16 years old girl and 10 years old boy) and their follow up period (including DBS) will be presented and discussed.

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SYMPOSIA ABSTRACT: 598

Forensic Medicine Institution: History and Organization

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ABSTRACT

In Turkey, the first organization about forensic medicine was found in 1839. It was found a as 'Tıbbi Kanuni' (Forensic Medicine) in the educational program of 'Mekteb-i Tıbbiyye-i Şahane' (the name of the Istanbul School of Medicine in Ottoman period). In 1917, Forensic Medicine Association was separated from the Health Service General Management and joined with the Ministry of Justice and Adli Tıp Müessesi ve Meclisi was founded by the law number 225. February 19th, 2003 by law number 4810, April 4th, 1982 by law number 2659, it was established under the Ministry of Justice. Summary of the duties; to declare scientific and technical opinion about issues related with forensic medicine which were send by the courts and judicatures. Forensic Medicine Institution consists of; Forensic Medicine Institution Chairmanship, Forensic Medicine Chairmen Council, Forensic Medicine General Council, Forensic Medicine Specialized Council and Country Organization consist of Branch Offices and Forensic Medicine Group Chairmanships. Every specialized council consists of a chairman, two forensic medicine specialists, and other members whose number and specialties are defined separately for each council. There are 6 specialized councils, each have different members and duties. There are 6 Specialty Councils, each have different members and duties. The ones related to the psychiatry are 4th and 6th Specialty Councils and Observation Specialty Council (Gozlem Ihtisas Dairesi). Observation Specialty Council's duty is to observe the people who are sentenced to be observed by the councils and judiciaries and to produce official reports from the results of these observation.

KEYWORDS

Forensic medicine; specialized council; observation department; psychiatry

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SYMPOSIA ABSTRACT: 599

Psychotherapies: From Where to Where?

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ABSTRACT

It is a fact that psychotherapy has been around as an art of healing since antiquity. In ancient times, shamans and sorcerers were amongst those who were instrumental as providers of "healing"(Bloch and Harari,2006,P.3) The modern journey of psychotherapy has an onset with Freud's psychoanalysis and following this comes psychodynamic psychotherapy .Another important branch of psychotherapy tree is behavioral psychotherapy based on learning theories and Pavlov's model of conditioning. Cognitive Behavioral Psychotherapy (CBT) followed the behavioral one. There are numerous schools of psychotherapy and there are new forms of psychotherapies such as Acceptance and Commitment Therapy(ACT),Narrative Therapy. Freud's patient, Anna O's (the pseudonym given to her) view of her treatment as 'talking cure' actually heralded the modern era of psychotherapies as talking therapies by the wider community (Bloch and Harari,2006,p.4). Starting from 1950s, psychotherapies involving more than one person emerged such as group therapies and family therapies (Bloch and Harari, 2006) İn the beginning of the modern psychotherapies, research was not on top of the agenda of psychotherapist. However nowadays many researches are going on in every aspects of psychotherapy. It seems psychodynamic approach makes up the main pillar of all modes of psychotherapies. After all, a psychotherapist needs psychodynamic framework to assess and understand a patient before deciding to apply a specific mode of psychotherapy. That is, therapeutic alliance, trust, exploring the "unconscious", free associations, defenses, transference, countertransference are some of the important points in assesment. Following this, it is not surprising to think that psychodynamic psychotherapy with empathy helps the patient to pinpoint and understand his or her inner world together with the patient's cultural repertoire, as background, upbringing and psychological development (Bateman, Brown and Pedder, 2000). The other important point to remember in psychotherapy is the possibility of side effects such as deterioration of mental state, boundary violations. It is good to see that more and more trainee doctors in psychiatry are enrolling in various modes of psychotherapy training. Some overseas formal psychiatry training programs offer advanced training in psychotherapies. Supervision is a must in psychotherapy training and requires serious attention. Last but not the least, the form and structure of the psychotherapy should involve the cultural values and beliefs in assessment and defining the psychotherapy for the patient (Jacob, 2013).

KEYWORDS

Psychotherapy; dynamic; inner world; culture; training

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SYMPOSIA ABSTRACT: 601

Autism and Autonomic Dysfunction

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ABSTRACT
Autism spectrum disorder (ASD) is a neurodevelopmental disorders that characterized with limited social interactions and restricted or repetitive behaviors. It has been estimated that 11.3 per 1000 children are ASD and the prevalence rates of ASD is increasing (Baio, 2012).

It is well known that autonomic nervous system (ANS) is one of our main behavioral regulator and it plays an essential role not only in regulating the heart, lungs, skin, and other visceral organs and glands but also it plays a paramount role in the regulation of emotions and social interactions (Benevides and Lane, 2013). The autonomic nervous system is responsible for multiple physiological responses, and dysfunction of this system is often hypothesized as contributing to cognitive, affective, and behavioral responses in children with ASD and examination of ANS activity may provide insight into behavioral dysregulation in children with autism spectrum disorders (Benevides and Lane, 2013). Although there is a body of growing literature investigating ANS dysregulation the autonomic in children with autism spectrum disorder, results are complicated. One reason about complicated results are proposed as the distinct measures used to document autonomic nervous system functionality (Benevides and Lane, 2013). In this study we aimed to investigate the relationship of ANS dysregulation with symptom severity and behavioral dysregulation of children with ASD.

KEYWORDS
Autism; autonomic nervous system; behavioral dysregulation; children; ASD

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SYMPOSIA ABSTRACT: 602

Growing up with Violence

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ABSTRACT
Everything is related to how is the World presented to the child said Donald Winnicott to point out the paramount importance of the environment on shaping human infants psyche and later life. War, violence and displacements affect every aspect of a child's life, growth and development. When a child born into and have to live with war torn area, all of the concepts and schemas about normality and abnormality will be shaped by the world enroll them. The formation of Ego, Superego, Ego ideal, role models affected by the world surround the child. Thus children who grow up in an environment that nothing is sustainable and predictable, it will not be easy to get baseline feeling of safety and trust to the world and others and the long-term effects of continuous exposure to violence and trauma could not be assessed with psychiatric disorders. Also it will not be easy to rehabilitate these children's confidence and trust to others and their future.

KEYWORDS
Children; violence; war; idealization of violence

SYMPOSIA ABSTRACT: 603

When Adolescent Becomes Mother? Infant-Mother Interactions

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ABSTRACT
Teen pregnancy remains a public health problem of varying importance in developing and developed countries. The World Health Organization reports that there are 16 million births

annually to mothers aged 15–19 years. There are risks and consequences for teen parents and the child on the medical and socioeconomic level. We know that adolescence is a period in which many adolescents start exploring intimate relationships and sexual behaviors. Although the initiation of these relationship is a normative step in adolescents' sexual development, early sexual intercourse, sexual abuse, risky sexual behaviors (e.g., unprotected sex), sexually transmitted infections and unwanted pregnancy are common and can be problematic seriously. In Turkey 'unintended' or 'unplanned' pregnancy have often been consequences of sexual abuse. Many researches implicated that sexual abuse and unintended teenage pregnancies have a considerable impact on the individual well-being of teenage parents and their children. Unintended conceptions and being mothers can cause emotional, psychological and educational harm to young girls, often with enduring implications for their life opportunities. The teen mothers are more at risk for postnatal depression, school dropout and bad socioeconomic status. Teenage pregnancy is associated with an almost threefold risk of preterm delivery and stillbirth. Babies of teenage mothers have increased mortality in their first year and a significantly increased risk of living in poverty, developmental delays, educational under achievement and unemployment in later life, with associated societal costs and increased risk of mental and behavioral disorders across life span for both mothers and their children. Childhood negative life experiences in early life, insecure attachment, child abuse, violence within the family, during the critical first 3 years can have a profound influence on the brain development. Many studies also found that adolescent mothers may vocalize, touch, and smile at their infants less and may be less sensitive to and accepting of their infants' behavior. Teen mothers tend to hold less realistic developmental expectations of their children and apply harsh discipline. Unfortunately, this troubled mother-infant relationship impact negatively cognitive and social development of infants.

SYMPOSIA ABSTRACT: 604

Thyroid Hormone and Its Relationship with Cognition

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ABSTRACT

My presentation will represent thyroid function and cognition and also some general determinants of intelligence and cognition. The pituitary-thyroid axis is an evolutionary selected key regulator to optimize developmental processes and physiological responses to environmental stress. At the same time thyroid hormone might have a major role in the settlement of the life-history trade-off between growth, metabolism, and inflammation axes, which is required for homeostasis maintenance. From another biological point of view thyroid hormones have multiple pleiotropic effects, acting as an essential regulatory factor in numerous physiological systems, including the vascular tree and the heart , brain (including cognition and mood) , skeletal muscle and bone[1]. It is therefore feasible that survival beyond the reproductive phase is better off with lower levels of thyroid hormone signaling, away from the optimal characteristics that are generally required in the reproductive part of life, with a lower rate of aging as a result[2]. In the developing brain, TH is required for optimal neurogenesis, synaptogenesis, neuronal migration, plasticity, and myelination. Alternatively developmental TH insufficiency may negatively impact neurogenesis indirectly by its effects on neural activity. Neurogenesis is regulated by synaptic activity–it is enhanced by learning, long-term potentiation, exercise, environmental enrichment, and seizure activity. Cognition is a term referring to the mental processes involved in gaining knowledge and comprehension. These processes include thinking, knowing, remembering, judging, and problem-solving. These are higher-level functions of the brain and encompass language, imagination, perception, and planning. Evaluation of cognition or intelligence quotient (IQ) is a complex field[3]. The hippocampus maintains a capacity for neurogenesis throughout life, a capacity that is reduced in models of adult onset hypothyroidism[4]. Hippocampus is a region in the brain with dense concentration of thyroid hormone receptors, therefore, during lifetime thyroid hormones have much effects on this region. According to the studies, thyroid disorders, especially hypothyroidism, effect hippocampal formation and have a role in causing and development of dementia. Therefore, high levels of TSH in subclinical hypothyroid cases may be correlated with hippocampal degeneration and cognitive disorders, so we have to consider TSH levels and thyroid disorders in prevention and treatment of dementia disorders. Both overt hyper- and hypothyroidism are known to lead to cognitive impairment and clinical guidelines recommend screening for thyroid dysfunction among patients with cognitive disorders data on the association between subclinical thyroid dysfunction (SCTD) and cognitive function remained conflicting[5].

KEYWORDS

Hippocampus size; thyroid-stimulating hormone; cognition

Moderate degrees of developmental TH insufficiency comparable to those used here, reduce excitatory synaptic transmission) long-term potentiation, and hippocampal-dependent learning in adult offspring. Moreover, neurotrophins, including nerve growth factor and brain-derived nerve growth factor, essential for the maintenance of neurogenesis and activity - dependent plasticity. Thyroid hormone regulates the hippocampal functions and also have main role in structural integrity of neurons in the hippocampal, so evaluation of thyroid function and TSH levels can help us in prevention, management, and treatment of dementia disorders and cognitive diseases. Thyroid hormones could also modulate the contents of b-amyloid precursor protein isoforms, which are important pathological processes for Alzheimer's Disease[6]. Most research showed that SHyper, but not SHypo, might be associated with a modestly elevated risk of dementia. Neither SHyper nor SHypo were significantly associated with a faster decline in MMSE over time, as compared to euthyroidism[7]. Thyroid hormone is an important neuroregulator in fetal development of the CNS and plays an important role in neurocognitive function after development. Overt hypothyroidism is a well-known reversible factor causing cognitive impairment including dementia. Overt hyperthyroidism or thyrotoxicosis has also been known to be associated with altered concentration and perception. Epidemiologically, these are thyroid dysfunction, depression, and cognitive impairment[8].

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SYMPOSIA ABSTRACT: 605

Traumatic Brain Injury and Neurorehabilitation

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ABSTRACT

Traumatic brain injury (TBI) is defined as a traumatically induced structural injury of the brain or physiologic disruption of normal brain function resulting from an external force. TBIs can be broadly classified by neurologic damage (diffuse, focal, or both), degree of penetration of the injury, and severity (mild, moderate, or severe)[1]. Symptoms of mild TBI be thought of as somatic, psychological, or cognitive. Examples of somatic symptoms that may occur with mild TBI include headache, drowsiness or fatigue, phonophobia, photophobia, insomnia, dizziness or difficulty balancing, nausea, and vomiting. Psychological symptoms associated with mild TBI may include irritability, anxiety, and depression. Cognitive symptoms that may occur after mild TBI include memory impairment, confusion, impulsiveness, difficulty concentrating, and problems with language or speed of processing. Various other more critical symptoms that may be associated with moderate to severe TBI include the following: diminished sensation (gustatory, olfactory, auditory, or visual), impaired depth perception, nystagmus, epileptic seizures, tinnitus, dementia, dysarthria, aphasia, peripheral neuropathy,

KEYWORDS

Traumatic brain ınjury; neurorehabilitation; cognitive rehabilitation; concussion therapy

and aggression. In addition, in a general population evaluated up to 10 years postinjury, patients with moderate or severe TBI had a substantially higher risk of developing and sustaining additional symptoms, such as abnormal social behaviors, abnormal executive functioning, increased aggression, and alexithymia[2]. The successful treatment of TBI patients with accompanying polytrauma generally requires lengthy physical rehabilitation, high levels of selfmotivation, and a strong psychological foundation. Psychological and neurologic impediments may create barriers to optimal rehabilitation from TBI during the acute and chronic phases of recovery[3]. Rehabilitation of the patient with TBI begins after he or she is stabilized, with the aims of improving independent functioning, social integration, and disability adaptation[4]. Clinicians should reevaluate patients for chronic symptoms of TBI and reassess treatment strategies. Routine follow-up evaluations and reassessments are recommended at 4-month intervals[5]. Depending on the complexity of the condition, some patients may require comprehensive rehabilitation regimens based on a range of therapeutic providers. Patients who remain incapable of independent living despite rehabilitation efforts may need to be monitored in supported living facilities or vocational programs[6].

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SYMPOSIA ABSTRACT: 606

The Use of Heart Rate Variability in Psychiatric Research

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ABSTRACT

The emotions that we experience while interacting with our environment elicit varying degrees of physiological arousal. A crucial component involved in the generation of this physiological arousal is the autonomic nervous system (ANS). The ANS is basically subdivided into an excitatory sympathetic nervous system (SNS) and an inhibitory parasympathetic nervous system (PNS) that often interact antagonistically to produce opposite effects over various structures, including the sino-atrial node controlling heart rate. Heart rate variability (HRV) is a measure of this continuous interplay between sympathetic and parasympathetic influences on heart rate that provides information about autonomic flexibility, hence it represents the capacity for regulating emotional responses. The first observations concerning the existence of physiological rhythms imbedded in the heart rate signals date back to 1960's, whereas the clinical importance of HRV became apparent in the late 1980s when it was confirmed that HRV was a strong predictor of mortality following acute myocardial infarction. In the last two decades, a growing body of research demonstrated that the diminished HRV is associated with many psychiatric conditions such as anxiety and depressive disorders. For instance, recent studies have found that depressed patients with coronary artery disease (CAD) have decreased HRV compared with non-depressed CAD patients and that the reduced HRV is associated with increased mortality and morbidity in CAD populations. Bereaved individuals and patients receiving treatment for melancholic major depression showed decreased resting parasympathetically mediated HRV, and patients with bipolar depression exhibited reduced overall resting HRV. A common finding in the research on anxiety and autonomic control is diminished heart rate variability in anxiety disorders such as panic disorders and specific phobias, especially in the parasympathetically mediated HRV. This has been considered to largely reflect a reduction in vagal control and an associated loss of autonomic flexibility. In addition to the research conducted on clinical population, many experimental

psychological studies made use of HRV parameters in determining problems with emotion regulation and vulnerability to stress. For instance, it has been shown that during the presentation of stimuli triggering negative emotions such as fear and anger, the use of adaptive coping strategies (cognitive techniques such as distancing and reappraisal) lead to significant increases in HRV compared to maladaptive strategies such as suppressing emotional responses. Decreased HRV scores have also been associated with low social competence, neurotic personality features, and vulnerability to stress. In conclusion, research support the utility of HRV as a noninvasive, practical and objective index of the individual's ability to regulate emotional responses both in clinical and non-clinical populations.

SYMPOSIA ABSTRACT: 607

Benzodiazepine Use in Anxiety Disorders: Risks and Benefits

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ABSTRACT

The large class of central nervous system depressant medications -the benzodiazepines- have been extensively used for over 50 years, anxiety disorders being one of the main indications. Inappropriate prescription rates vary from 2.2 to 17.6%. With that being said female, elderly and patients with poor physical health are at greater risk of exposure to such unjustified use of benzodiazepines. The reason behind these high percentages of inappropriate prescriptions is the insufficient training of health care providers for managing patients with psychosocial problems, high level of stress and anxiety. Desperate against these patients, clinicians often think benzodiazepines are the 'lesser evil' despite their risk of addiction and misuse[1]. Benzodiazepine anxiolytics should be prescribed primarily either for the short-term (4–6 weeks) relief of severe anxiety symptoms, or where anxiety disorders are disabling and severe and causing both significant personal distress and substantial impairment of daily activities. In rare instances longer-term prescriptions of benzodiazepines may be seen as a form of harm reduction in patients who would otherwise consume illicit benzodiazepines or abuse alcohol to 'cope' with anxiety, again, efforts should be made to reduce the dosage over time, wherever possible. There are other situations where anxiety is complicated by other medical conditions, or where the risk of dependence with benzodiazepine use may be considered acceptable because of the severity of illness and potential hazards associated with other treatment approaches, such as may occur in some patients with schizophrenia. Patients who may need maintenance therapy are those who are on a high diazepam equivalent dose, have a range of aberrant drug-related behaviors (especially doctor shopping) and have a chaotic social setting or unstable psychiatric diagnoses[2]. Withdrawal symptoms can be physical or psychological. Symptoms can be prolonged and are sometimes hard to distinguish from those of underlying anxiety disorders, although perceptual disturbances are relatively infrequent in untreated patients with anxiety disorders. Withdrawal reactions are generally short-lived, typically lasting less than one month, although duration is influenced by individual pharmacokinetic factors. There is controversy about whether symptoms persisting for many months are withdrawal reactions, or simply the features of an underlying disorder, or worsening of that condition triggered by treatment withdrawal[3]. Many patients are able to take short courses of benzodiazepines quite safely and to stop them when no longer needed. If treatment courses lasting longer than four weeks are required, this should not necessarily be regarded as a deviation from good clinical practice, although continuing vigilance of potential hazards is needed throughout treatment.

KEYWORDS

Benzodiazepine; pharmacology; risk-benefits ratio

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SYMPOSIA ABSTRACT: 610

Imaging Modalities in Traumatic Brain Injury (TBI): Future Perspectives

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ABSTRACT

Traumatic brain injury (TBI) remains a major cause of death and disability, occurs from numerous mechanisms. Due to the heterogeneity that each injury may have completely different pathophysiology from another and the numerous molecular mechanisms involved in TBI, it has been difficult to develop therapeutic strategies to treat the devastating consequences[1,2]. But also, optimal imaging technique has been a subject of debate due to the difficulty in consistent and accurate detection of injury. The patient who sustains a traumatic brain injury (TBI) typically undergoes conventional neuroimaging studies, usually computed tomography (CT) and magnetic resonance imaging (MRI). Except patients with moderate to heavy TBI, majority of the patients present with no findings on conventional studies. Structural damage may be present at a microscopic level. Moreover, there may be functional and metabolic abnormalities in the absence of structural damage. Diffusion tensor imaging (DTI) measurements gaining interest because of its ability to detect axonal injury. But under the absence of detectable structural damage, the use of functional and metabolic imaging modalities such as positron emission tomography (PET), single photon emission computed tomography (SPECT) and functional magnetic resonance imaging (fMRI) can provide information on TBI-related neuropathology[3]. Metabolic and functional imaging modalities used in combination with structural imaging are important to provide additional information and insight into the neuropathological effects of TBI potentially useful in better understanding TBI sequelae as well as providing additional information in the assessment and treatment of it[4].

KEYWORDS

Neuroimaging; Imaging modalities; Traumatic Brain Injury

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SYMPOSIA ABSTRACT: 700

Insight in Bipolar Disorder Patients: The Schrodinger's Cat

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ABSTRACT

There are several ways of defining and conceptualizing insight. Clinical concept of insight refers to awareness of a mental disorder and its consequences, awareness of need for treatment, awareness of symptoms and attribution of the symptoms to the disorder. Numerous conceptual models have attempted to explain insight: as a symptom, as a defense, as psychological (misattribution) or neuropsychological phenomenon, or from a sociocultural perspective. Neurobiologically, insight has been associated with frontal lobes and other cortical structures implicated in the highest mental functions. Several instruments exist for assessing insight. Clinical correlates of insight are many but always consistent: severity of

illness, psychotic symptoms, depressive symptoms, treatment adherence, quality of life, functioning, violence, and personal qualities such as conformism. In the past ten years there have been more than 200 studies of people "who do not think they are ill". Most of these studies are involving patients with psychotic disorders. This is a serious issue since these people do not take their medications or participate in programs that could help them, have higher rates of involuntary commitments, have more contacts with the criminal justice system through misdemeanors and disorderly conducts, and finally have a lower quality of life. Awareness is one of the top two predictors of which patients will take their medication-the other being a social support system, friends and loved ones, who respect the patient's point of view and work with them without anger or blame. According to studies, awareness of the signs and symptoms of illness is fully present in about a quarter of patients, moderately or intermittently present in another quarter, and missing in half of patients. Denial of illness does not in itself indicate bad judgment. If you are not ill, then taking medications which have side effects and might harm you is not bad judgment but just common sense. Research has shown that insight into a person's illness is not necessarily associated with the symptoms getting better. Insight in bipolar patients is still not sufficiently studied, especially how insight is fluctuating in different phases of the illness. While in depressive phase patients easily admit their suffering and are motivated to take treatment, in hypomanic or manic states insight is questionable or missing. In conclusion; in recent years, there has been a surge of research into the conceptualization and assessment of insight, as well as its relationships with prognosis, compliance, neuropsychological impairment and severity of psychopathology in psychotic disorders, but there is no enough scientific data about the insight in bipolar disorder. However, these studies have yielded inconsistent results. Different dimensions of insight are probably related to different aspects of outcome, and this needs to be reflected in the individual treatment planning phase. Although important advances have been made in this area, many questions remain unanswered, and these need to be addressed in future research.