Restless Leg Syndrome Associated with Aripiprazole

To the Editor,

The Restless Leg Syndrome(RLS) induced by aripiprazole is very unique albeit rare adverse effect[1,2]. The patient is a 55-year-old female who presented to our outpatient clinic with the diagnosis of major depressive disorder according to DSM-IV. Patient who has been receiving venlafaxine and quetiapine with doses of 75 mg/day and 300 mg/day respectively one year, was not being followed-up regularly. The patient had complaints such as suicidal thoughts and insomnia since the onset and gained 10 kg during the treatment. She was using ramipril 5 mg/day and her blood pressure follow-ups were normal. The venlafaxine dose of the patient was elevated to 150 mg/day and dose of quetiapine was remained same at 300 mg/day. Aripiprazol with a dose of 5 mg/day was added to the treatment of the patient due to complaints on the follow-up after a month. On the next one month follow up after the aripiprazole administration, patient stated that she suffered from insomnia, restlessness on legs, urge to move her legs and she had ceased the medication after 5 days. Her complaints were recovered after the cessation of the aripiprazole administration. The biochemistry, blood glucose, hemogram, iron, ferritine, transferrin level, renal hepatic test results, thyroid function tests, B12, and folic acid levels of the patient were all within normal range. Patient was evaluated by the departments of neurology and internal medicine and no peripheral neuropathic or vascular disease were found. There were no similar complaints before the aripiprazole treatment. Treatments of venlafaxine 150 mg/day and quetiapine 300 mg/day were continued. Venlafaxine dose was elevated to 225 mg/day but this dose was terminated gradually due to onset hypertension and depressive findings, duloxetine treatment with a dose of 30 mg/day was initiated, treatment of quetiapine 300 mg/day was resumed as unchanged. Duloxetine dose was increased up to 120 mg/day. RLS symptoms were not recurred during this period. Depressive symptoms of the patient remitted and treatment have been proceeding for one year.

In our case, the symptoms have been considered as a result of aripiprazole administration due to occurrence of the symptoms after the initiation of aripiprazole treatment, recovery of the symptoms after cessation of aripiprazole, absence of and organic disease causing RLS, normal lab results, and lack of similar picture in patient's history. The patient has been using venlafaxine and quetiapine for one year and no RLS symptoms were reported since then. Although the onset venlafaxine and quetiapine medication was proceeding, RLS symptoms were not observed after cessation of aripiprazole. In literature, a case of RLS related with the combination of venlafaxine and quetiapine was reported[3]. RLS symptoms were remitted when aripiprazole was added to treatment[4,5]. The inflammation of the RLS by D2 receptor antagonists and well response of the symptoms to dopaminergic drugs such as levodopa, suggest that the dopaminergic system acts as central role[1]. Both improvement and causation of the RLS symptoms by aripiprazole can be explained by the partial agonistic effect on D2 receptors.

RLS may cause depression or anxiety as well as sleeping disorder and affect the quality of life. Considering this reported side effect, especially in patients who was administered antipscyhotics, would be important for treatment follow-up and early diagnosis and treatment compliance in case this adverse effect is ensued.

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Psychotic Disorder or Complex Partial Seizure Disorder Due to a Sequelae of Herpes Simplex Encephalitis and Coincidental Parasagittal Meningioma

To the Editor,

Temporal lobe dysfunctions which are associated with the organic brain lesions or epileptic discharges can manifest as psychosis. Encephalitis due to herpes simplex virus infection often damage temporal lobe[1]. After herpes simplex virus encephalitis (HSVE), psychiatric and behavioral symptoms have been found approximately in 80% of the survivors[2]. Here, we describe a case presented with psychotic symptoms including paranoid delusions and olfactory hallucinations following the HSVE in a patient who concomitantly had a parasagittal meningioma.

A 60 year-old female patient, admitted to hospital because of bad smelling, nausea, retching for hours, paranoid delusions, insomnia, hitting on her head hardly, and forgetfulness. The patient had HSVE history three years ago that ended up with bilateral temporal lobe damage. Insomnia and paranoid delusions emerged five months after the onset of acute encephalitis and olfactory hallucinations emerged during the last seven months. Patient was started olanzapine 5–30 mg/day with the initial diagnosis of psychotic disorder secondary to HSVE. Awake electroencephalography (EEG) exam showed excessive theta rhythms (5–7 Hz) on bilateral parasagittal regions that were consistent with parasagittal meningioma which was stable for ten years. In the diffusion-weighted magnetic resonance (MR) imaging of the brain, bilateral anterior temporal lobe edema was observed. In neurological examination, no focal motor or sensory deficits were found. Patient's paranoid symptoms and olfactory hallucinations persisted despite three antipsychotic trials (olanzapine 30 mg/day for two weeks, risperidone 2–8 mg/day for 10 days, and sulpiride 200–600 mg /day for three weeks). During inpatient hospitalization, we noticed that patient's paranoid delusions and olfactory hallucinations associated with agitation and hostile behaviors seemed to have a periodic pattern during the day. These periods almost emerged in the same way, two or three times a day, lasting for 2 to 4 hours, despite optimizing and/ or switching antipsychotic medications. Although patient did not manifest any clinical signs of seizure disorder, we started carbamazepine 200–800 mg/day and reduced sulpiride dose taking into consideration that intermittent nature of psychotic symptoms might be explained as an epileptic phenomenon. After two weeks of treatment, patient's psychotic symptoms and disruptive behaviors completely resolved.

At least 80% of survivors of acute encephalitis can manifest with neuropsychiatric symptoms such as mood disorders, delusions, hallucinations, anxiety, insomnia, sexuality changes, labile affect, memory impairment,

aberrant behavior, apathy, and disinhibition[2,3]. In this case report, patient's paranoid delusions and insomnia emerged five months after the onset encephalitis and behavioral abnormalities and olfactory hallucinations emerged during the last seven months, i.e., during the third year post-HSVE. In the literature, case reports that were characterized by late onset psychotic and behavioral problems as in our case are documented less[4,5]. Gaber and Eshietti reported a patient who had similar features with our case who was hospitalized for seven months with no response to antipsychotic medications. Particular episodes of abusive and disruptive behavior for periods of up to an hour once or twice daily were observed. Despite the absence of any EEG abnormalities, their patient responded dramatically to carbamazepine similar to our case[4]. Vallini and Burns reported another case who responded well to carbamazepine treatment for psychiatric symptoms. They reported successful treatment of an HSVE patient who presented with severe emotional lability and explosive emotional outbursts[5].

In sum, the episodic pattern of olfactory hallucination and paranoid delusions suggested the presence of post-encephalitic temporal lobe epilepsy rather than a psychotic disorder secondary to the HSVE. Although clinical or neurophysiological evidence of seizure disorder were not present, no response to antipsychotic treatment and improvement of the symptoms following carbamazepine therapy suggested that mood stabilizers such as carbamazepine would have been helpful for treatment-resistant episodic psychotic symptoms following the onset of HSVE. In this case report, the crucial role of psychiatrists and neuropsychiatrists in the care and study of individuals with these conditions was reported.

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Autoimmune Psychosis: Caveats in the Diagnosis

To the Editor,

I have read the review on "Autoantibodies to neurotransmitter receptors and ion channels in psychotic disorders", with interest1. The author made a commendable attempt to review the literature linking autoimmune system and psychosis. As the author pointed out, the discovery of autoantibodies specific for the neuronal cell membrane surface or synaptic proteins has yielded in paradigm shifts in diagnostic and treatment approaches to some neuropsychiatric disorders[1]. Recent evidences suggest

that around 6.5% of incidents of first episode psychosis could be due to autoimmunite system impairnents[2]. Moreover, population-based studies reported that a personal history of any autoimmune disease could increase the risk of schizophrenia by 45%, indicating a close link[2].

Diagnosing autoimmune psychosis is important as failure to do which may deprive the patient of treatment for a potentially curable entity. To keep autoimmune psychosis as a differential diagnosis in day to day psychiatric practice has been highlighted by the fact that, in one study, almost 75% of patients with limbic encephalitis consulted psychiatrist initially[3]. Unavailability of validated diagnostic criteria and validated diagnostic tests make the task of diagnosing autoimmune psychosis difficult.

The rapid development of alteration of mood, behavior, and memory with altered consciousness and seizure, in a few days or weeks, sometimes with headache or mild hyperthermia indicates autoimmune psychosis in general[3]. There are specific clinical descriptions related to different autoimmune psychosis that can help in the differential diagnosis. Anti-NMDA receptor encephalitis is seen mainly in children or younger woman manifesting as paranoid delusion, hallucinations, agitation, speech abnormalities and bizarre behaviour, whereas anti-AMPA receptor limbic encephalitis patients are mostly seen in women aged 50-70 years with subacute memory loss and confusion and atypical psychosis[2]. Atypical presentation of Voltage-Gated Potassium Channel (VGKC -complex) antibody-associated encephalitis is characterized by a person of middle age with memory deficits, confusion, apathy, and irritability, often accompanied by excessive sweating and salivation[2].

Assessment of antibodies can help in differential diagnosis between autoimmune vs. non autoimmune psychosis as well as in differentiating specific autoimmune syndromes. NMDAR antibodies in patients with schizophrenia were studied, resulting in inconsistent results. Overall, findings indicate limited clinical significance of serum IgA or IgM NMDAR antibodies in patients with schizophrenia, but the absence or rare detection of GluN1 IgG antibodies confirms the specificity of these antibodies for anti-NMDAR encephalitis[3].

A recent study from India, assayed serum autoantibodies of six neuronal proteins, namely NMDAR, AMPA1, and AMPA2 receptors; VGKC complex proteins Lgi1 and Caspr2; and GABAB1 receptor with the Autoimmune Encephalitis Mosaic 1 kit from EUROIMMUN, Luebeck, Germany, in patients with suspected for autoimmune encephalitis. The results showed that a third of the suspected cases of autoimmune encephalitis were positive for NMDAR antibody and the remaining two-third being antibody negative[4]. The study failed to show any strong association with specific clinical characteristics, natural history or outcome. The study results raised pertinent questions regarding the diagnostic reliability of the serum antibody assessments. Recent evidences demand autoantibodies should be examined in both serum and CSF due to the fact that those with clinical autoimmune encephalitis who did not have detectable antibodies in serum had antibodies in the CSF[3].

In conclusion, specific clinical features with serum and/or CSF positivity for specific antibodies and a response to immunotherapy could be the best diagnostic criteria at the moment. As the author pointed out, there is an urgent need for validated diagnostic criteria and biomarkers for autoimmune psychosis[1].

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Acute Urinary Retention Related with Sublingual Buprenorphine Administration

To the Editor,

Suboxone, a sublingual tablet consisting of buprenorphine/naloxone is an effective and well-tolerated treatment option in the opioid dependence[1]. Rate of urinary retention with buprenorphine is less than 1%, particularly with intrathecal and epidural delivery[2]. However, it is rare with sublingual administration.

We decided to write this report after our observation of a 20-year-old male patient, who was admitted to AMATEM (Alcohol and Substance Treatment and Education Center) clinic with the diagnosis of opioid addiction. The patient had a 2-year-history of abusing heroin. His use was progressed to daily intravenous pattern within 6 months. His medical history was not significant. In his urine analysis, benzodiazepine and opioid levels were over the normal range. In order to prevent fatal drug interactions between suboxone and benzodiazepine, suboxone treatment was not started on until benzodiazepine vanished in urine sample. As a result, suboxone treatment could be started on the seventh day of admission. The initial dose was 4 mg/1 mg, titrated up to 8 mg/2 mg on day 2. Since the patient complained of inability to urinate and suprapubic discomfort on day 4, urethral catheterization was performed with 16" Foley catheter and 2000 cc urine was drained. The microscopic examination of the urine was normal and urine culture was negative. Result of the urine drug screen was also negative. The serum creatinine level was in normal range. He denied any previous urinary symptoms. Urology was consulted and Suboxone treatment was stopped. The patient was observed with catheter for three days without trouble. After removal of catheter, the patient was able to complete a trial void. No further urological complaint of the patient was present after discontinuation of suboxone treatment. After the event, the patient refused a retrial of buprenorphine and detoxification was completed with symptomatic treatment.

It is known that one of the significant adverse events related with opioids is urinary retention. Although urinary retention mechanism is not completely known, it was shown in the literature that both mu and delta opioid receptors are involved in bladder realization and impaired sensations by inhibiting the sensory input at the level of the dorsal horn and periaqueductal gray matter (PVG)[3]. As being a partial agonist of μ-opioid receptor, buprenorphine may cause diminishing of bladder sensations and delay in urination threshold, thus increasing compliance and bladder capacity via μ-opioid receptor activation. The mode of opioid delivery also appears to play a role in the risk of urinary retention; which is found higher with intravenous and epidural administration[4]. However, urinary discharge problems associated with sublingual buprenorphine are rarely reported. The first case was a 66-year-old male patient reported by Murray *. Recently, acute urinary retention after sublingual buphrenorphine administration of a 49-year-old male patient with a past history of benign prostatic hyperplasia (BPH) has been reported[5]. In our case; unlike others, the patient is rather young and has no risk of BPH. Management of acute urinary retention in patients without BPH involves Foley catheterization to allow the offending agent time to be excreted. Immediate catheterization is recommended; otherwise the bladder may excess its functional capacity. This information is important for physicians who prescribe buprenorphine to monitor for acute urinary retention and treat appropriately.

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Risperidone and Lithium for Pathological Laughing

To the Editor,

Pathological laughing and crying (PLC) is a situation that emerges as exaggerated and inappropriate laughing or crying episodes or both of them. It suddenly appears without any stimuli[1]. We diagnosed a case with pathological laughing who was previously treated with sufficient dose and duration of clomipramine, sertraline, and aripiprazole. However, no response was obtained from these treatments until choosing lithium and risperidone.

The case was a 21-year-old female graduated from high school; married, housewife with one child. She presented to our clinic with the complaints of four years duration and were unable to control involuntary laughing attacks and anxiety attacks. All these resulted in boredom, restlessness, irritability, with additional causes to family and social problems. Her neurological and psychiatric examination was not remarkable. She was laughing during the interview and examination. She used to laugh at times, but there was no sign of manic thought content during her laughing episodes. She was even laughing in times that she did not feel good. She did not have any medical disease nor reported use of any illicit drugs. There was no history of psychiatric or neurological illness in the family history. Magnetic resonance imaging (MRI) of the brain and electroencephalography (EEG) tests were evaluated as normal. Routine blood tests were normal, as well. The detailed background history of the patient did not show any signs of bipolar disorder with manic or hypomanic episodes. There were no impulse control problems pointing any pathological disorder. Treatment with clomipramine did not provide any positive response despite adequate time and dose. Afterwards, the treatment was replaced with sertraline. Despite of adequate dose and duration, there was just a partial response. Aripiprazole was then added to the treatment. Despite of adequate dose and duration, there was only a

partial response. Therefore, we changed the therapy with lithium 900 mg/day and risperidone 2 mg/day. Rigid clinical follow-up brought us an improvement with this treatment process in about a year. Regular follow-up examinations in patients continue currently.

Laughing and crying in such patients can be seen as both forms, transition from laughter to crying or crying to laughter. However, pathological crying seems to be more common than laughter[1]. Our rare phenomenon is patient's laughing attacks. Pathological laughing and crying episodes may overlap with euphoria, but may be non-euphoric[2], if the presence of an existing clinical care is not kept in mind, it may interfere with manic or depressive episodes. In this case, we neither observed manic episodes nor depressive episodes during treatment. PLC particularly coexists with neurological diseases such as ischemic stroke, multiple sclerosis, Parkinson's disease, amyotrophic lateral sclerosis, and Alzheimer's disease[1]. In this case, additional neurological diseases did not exist and the brain MRI was normal.

So far, role of 5-HT system in PLC etiology is only researched on paralyzed patients. Partial destruction of serotonergic neurons and damaged raphe nucleus were proposed ones[3,4]. There are neuroanatomical changes as decrease of 5-HT transporter and serotonergic 5-HT1A receptors in the pons, limbic areas, and nucleuses of raphe for PLC etiology[3]. In double-blind placebo-controlled trials, there are many reports indicating efficiency of selective serotonin reuptake inhibitors (SSRIs) (citalopram, sertraline, fluoxetine, and paroxetine), tricyclic antidepressants (amitriptyline, imipramine, and nortriptyline) in pathological laughing and crying treatment[4]. In a case reported by Ozturk et al., they did not report any benefits with sertraline, but was good outcome with citalopram for pathological crying[5]. However, our case did not yield in positive response with the SSRI treatment.

To the best of our literature knowledge, treatment of PLC with serotonin agents came to the forefront more recently. However, due to a positive response from both antipsychotics and mood modulator drugs instead of two different serotonin drugs, we believe that a different receptor system might have played a role in PLC etiopathogenesis. Although it is extremely rare, PLC may emerge by neurological diseases or without any lesion on the brain imaging. Therefore, possible beneficial effects of lithium and risperidone should be considered in the treatment of such patients.

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