excitatory neurotoxicity, might be responsible for insufficient follow-up FA changes.

On the other hand, the existence of white matter changes even in first-episode drug-naive schizophrenia patients supports the view that these problems occurs in stages of development, because the degree of FA changes refers to the fiber tract organization's degree of function[1]. They have a positive correlation. Moreover, the reduction of FA values directly indicates histological abnormalities. Also, our findings overlap significantly with those described by Wang et al.[2] who reported that there was a significant decrease in absolute FA in the white matter in 35 first-episode drug-naive patients with schizophrenia and after 6 weeks of antipsychotic treatment that did not correlate with symptom reduction.

As a result of white matter studies, distensions were detected in schizophrenia patients, particularly in axonal atrophy and periaxonal oligodendrocyte in the prefrontal cortex. This was deemed compatible with increased radial permeability and decreased FA values in the white matter of schizophrenia patients. This also suggests a cause from changes in axons' skeletal structure or demyelinization rather than a big degeneration in axons[3].

These findings show that the CC, which is the main determiner of interhemispheric connection, is affected distinctly in schizophrenia patients. When all these findings are considered, all of them probably result in a neuro-developmental defect that creates a shortage in neurons' modulator capacity paving the way to changes in cellular morphology; then abnormal synaptic circuits come into existence.

Consequently, we report FA reductions especially in the posterior region, also insufficient FA increase in white matter after antipsychotic treatment in patients experiencing a first episode of psychosis. However, prospective collaborative studies are needed to clarify the potential long-term effects of antipsychotics on white matter microstructure and also its reversibility.

Keywords: corpus callosum, first episode schizophrenia, fractional anisotropy

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[Abstract:0146] ***Anxiety, stress, and adjustment disorders***

Levels of Cortisol, Oxidative Stress, and DNA Damage in Victims of Childhood Sexual Abuse

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INTRODUCTION: Brain tissue continues to develop throughout childhood and during adolescence. Trauma experienced during these periods has been reported to have particularly serious consequences. With a few exceptions, most studies reported elevated cortisol levels in non-stressed circumstances in the child and adolescent victims of sexual abuse compared to controls[1]. Prolonged exposure to elevated cortisol levels has been shown to cause an increase in reactive oxygen species (ROS) at the cellular level and increased oxidative stress[2].

The aim of the present study was to evaluate cortisol levels, oxidative stress, and DNA damage in child and adolescent victims of sexual abuse versus healthy controls who did not have a history of trauma.

MATERIALS AND METHODS: The study was conducted in the Department of Child Psychiatry at Dicle University. Study data were collected between May 2012 and November 2012. The study included a total of 38 children (10 males and 28 females) aged between 9 and 17 years who had experienced childhood sexual abuse and 38 age- and gender-matched children as the control group. Children who reached an intelligence score below 70 points, who had a significant neurological or medical disorder, who received oral contraceptives, had previous or current cortisol therapy or used vitamins, and those who had morbid obesity or active infection were excluded in order to prevent interference with the biochemical parameters. In addition, patients with a history of psychiatric disorder before the latest trauma and those with a history of alcohol or substance abuse were excluded from the CSA group. Parents of all participants signed consent forms regarding their voluntary participation in the study. Approval for the study was obtained from the Non-Interventional Clinical Research Ethics Committee at Dicle University Faculty of Medicine. Sociodemographic features of the participants were obtained and

a clinical data form was completed. This was followed by the collection of 2 ml venous blood samples for biochemical tests. The blood samples were obtained in the morning between 10.00 and 12.00 am. Cortisol, glutathione peroxidase (GPx), Coenzyme Q, 8-Hydroxy-2-Deoxyguanosine (8-OHdG), and Superoxide dismutase (SOD) were tested using the ELISA method and commercial kits. The statistical analysis was performed using the SPSS 15.0 software package.

RESULTS: The mean age was 13.4±2.5 years (range 9-17 years) among the victims of sexual abuse. In the control group, the mean age was 13.5±2.6 years (range 9-17 years). There were ten males and 28 females in the CSA and control groups. The duration of education was lower in the victims of CSA and their parents compared to the control group. The number of siblings was higher. There was no significant difference between the groups in terms of their family history of psychiatric disorder and smoking/substance abuse. There was also no significant difference between the groups in terms of age at menarche and menstrual cycle.

Regarding the parameters related to sexual abuse, 61% (n=23) of the victims experienced sexual abuse involving penetration. Of those victims, 55% (n=21) experienced a single assault and 45% (n=17) experienced multiple assaults. Of the victims, 24% (n=9) experienced familial sexual abuse (incestuous) and 76% experienced sexual abuse committed by non-related persons.

Cortisol levels were significantly higher in the CSA group compared to the control group ($p<0.01$). There was no significant difference between the groups in terms of the levels of oxidative stress parameters (GPx, SOD, and coenzyme Q). Likewise, 8-OHdG levels as an indicator of DNA oxidation were not significantly different between the groups (Table 2). The mean time elapsed since the first sexual abuse until the date of examination was 20.6±22.4 months (3-95 months). The evaluation of the relationship between this time span and cortisol levels revealed that cortisol levels decreased as the time interval increased ($r=-0.279$, $p=0.04$). Similarly, 8-OHdG level decreased as the time elapsed since the sexual abuse increased ($r=-0.252$. $p=0.04$).

In the CSA group, there was no significant relationship between the sexual abuse involving penetration and the levels of GPx, SOD, coenzyme Q, and 8-OHdG. The coenzyme Q level was lower in the victims who sustained multiple assaults than the victims of a single assault ($p=0.04$). Cortisol and SOD levels were lower in the victims of familial sexual abuse ($p=0.03$ and $p=0.04$, respectively).

DISCUSSION: Studies conducted during childhood and adolescence on the victims of CSA report elevated cortisol levels; conversely, when studies on CSA victims are conducted after a significant amount of time has elapsed, cortisol levels are reported lower. This decrease in cortisol levels over time is referred to as attenuation hypothesis[3]. Consistent with the literature data, the present study reported higher cortisol levels in the CSA group. Furthermore, cortisol levels decreased as time since the sexual abuse increased.

In the present study, there was no significant difference between the control group and CSA group in terms of oxidative stress and DNA damage. In a recent study, increased oxidative stress has been shown in rats that were exposed to stress. In another study, oxidative stress was suggested to play a critical role in the development and exacerbation of post-traumatic stress disorder (PTSD). Consistent with the current results, a study conducted on 14 patients with PTSD reported no significant difference in terms of GPx and SOD levels when compared to the control group[4].

Both cortisol and 8-OHdG levels were found to be decreased as the time since sexual abuse increased. Although we did not find any difference between the groups in terms of 8-OHdG concentrations, this finding was considered to be a reflection of the relationship between cortisol and DNA damage. In addition, the decrease in cortisol levels over time was suggested to be reflective of an adaptive process preventing harmful effects of prolonged exposure to high cortisol levels on brain structures such as the hippocampus and frontal cortex[5].

In conclusion, no significant difference was found in children and adolescents who experienced sexual trauma in terms of oxidative stress level and DNA damage. Furthermore, some factors related to the trauma, such as sexual abuse within the family and multiple assaults, were found to have affected the level of oxidative stress. Because this is the first study that has been conducted in child and adolescent victims of sexual abuse, longitudinal studies on a larger scale are needed to confirm the results of the current study.

Keywords: DNA damage, oxidative stress, sexual abuse

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