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Akathisia caused by traumatic brain injury in an adolescent

Bir ergende travmatik beyin hasarına bağlı akatizi

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Abstract
Akathisia is a clinical condition with subjective complaints such as restlessness, mental unease, an urge to move and dysphoria. It is usually caused by basal ganglion diseases and anti-dopaminergic medications. But rarely it may also occur due to traumatic brain injury (TBI). In this case report, a 17-year-old male patient was discussed who developed akathisia after TBI without any neuropsychiatric disorder or psychotropic drug use earlier. The patient was admitted to the child and adolescent psychiatry clinic with constant complaints of motor restlessness. Three weeks ago he was hospitalized with multiple fractures, subarachnoid and epidural hemorrhage, and cerebral edema after a car accident. Risperidone 0,5 mg/day was prescribed for psychomotor agitation. Two days later symptoms got worsened, consequently, the treatment was changed to propranolol 60mg and lorazepam 7,5 mg per day gradually, following the change of diagnosis to akathisia caused by TBI. There was a significant improvement in akathisia symptoms in days. Akathisia treatment was stopped at the end of three weeks without symptoms. Akathisia caused by TBI, which can be easily misdiagnosed as psychomotor agitation or delirium, is an important clinical condition that affects the treatment and the rehabilitation negatively. Therefore, it should be kept in my mind for differential diagnosis and its treatment should not be delayed.

Key Words: Akathisia, traumatic brain injury, adolescent, psychomotor agitation, lorazepam.

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Özet
Akatizi huzursuzluk, tedirginlik, hareket isteği ve disfori gibi yoğun öznel yakınmaları olan klinik bir durumdur. Genellikle bazal ganglion hastalıklarında ve anti-dopaminerjik ilaçlarda görülür. Ayrıca nadiren travmatik beyin hasarına (TBH) bağlı ortaya çıkabilir. Bu olgu sunumunda öncesinde bilinen bir nörolojik rahatsızlığı ve psikotrop ilaç kullanımı olmayan, travmatik beyin hasarına bağlı akatizi ortaya çıkan 17 yaşındaki erkek olgu tartışılmıştır. Olgu devamlı motor huzursuzluk şikayeti ile ayaktan çocuk ve ergen psikiyatrisi polikliniğine başvurdu. Üç hafta önce, araç içi trafik kazasına bağlı çoklu kırık, subaraknoid ve epidural kanama ve serebral ödem ile yatışı olmuştu. Psikomotor ajitasyona yönelik risperidon 0,5 mg/gün tedavisi başlandı. İki gün sonra semptomları kötüleşen hastada, tanı TBH'a bağlı akatizi olarak değiştirildi ve tedavi kademeli olarak propranolol 60 mg/gün ve lorazepam 7,5 mg/gün şeklinde düzenlendi. Günler içinde akatizi semptomlarında belirgin düzelme gözlendi. Semptomsuz geçen üç haftanın sonunda akatizi tedavi sonlandırıldı. TBH'a bağlı akatizi tedavi ve rehabilitasyon süreçlerini olumsuz etkileyen önemli bir klinik tablodur, psikomotor ajitasyon ve deliryum ile kolaylıkla karışabilmektedir. Bu nedenle ayırıcı tanıda akılda tutulmalı ve tedavisi geciktirilmemelidir.

Anahtar Kelimeler: Akatizi, travmatik beyin hasarı, psikomotor ajitasyon, ergen, lorazepam.

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Introduction

Akathisia, derived from the word "an inability to sit" in Greek, is a motor restlessness syndrome [1] first described by Haskovec in 1902 [2]. The common symptoms are subjective complaints such as restlessness, mental unease, an urge to move and dysphoria, which can be intense [3]. Although akathisia was first observed in patients with basal ganglion diseases, it is also a side effect of the antipsychotics [4, 5]. Other drugs reported causing akathisia are antidepressants serotonin antagonists lithium anti-epileptics [9], anti-emetics antibiotics and anti-hypertensives [6-12]. Furthermore, akathisia can occur following a traumatic brain injury (TBI) [13] and may easily

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be overlooked or misdiagnosed as psychomotor agitation or delirium [14]. In this case report, a 17-year-old male adolescent was discussed, who developed akathisia due to TBI after a car accident, without any psychotropic drug use or known neurological disorder earlier.

Case

The patient was admitted to the emergency department with multiple fractures after the car accident. Subarachnoid and epidural hemorrhage, cerebral edema with midline shift and compression of midbrain structures were observed in the brain CT; and Glasgow Coma Scale was scored E: 1 M: 3 V: 1. Initially, he was treated conservatively in a neurosurgery intensive care unit, then in inpatient ward for a total of two weeks. No symptoms of psychomotor agitation or akathisia were reported during that time, only his state of consciousness was noted as confused and loss of orientation was indicated. The patient was admitted to the child and adolescent psychiatry outpatient clinic by his parents three days after discharge from the neurosurgery inpatient ward. His parents stated that he was constantly moving, restless, had physical aggression towards himself and others, and could not sleep due to these complaints. At the examination, even though the patient was conscious and some cooperation could be established, he had aphasia. He partially understood what was said and showed his requests by pointing. Prior to the accident, he had no history of psychiatric disorder or impairment in social or academic functioning. Treatment started with risperidone 0.5 mg daily, as the symptoms thought to be caused by psychomotor agitation. Two days later, aphasia began to improve, and the patient could be able to communicate verbally with short sentences. Nevertheless, the symptoms of restlessness and fidgeting got worsened and the diagnosis was changed to akathisia caused by TBI. Risperidone was discontinued and propranolol 60 mg daily was initiated. Five days later, the patient was hospitalized for physical rehabilitation and frequently assessed by child and adolescent psychiatrist for akathisia symptoms. His symptoms improved only minimally after propranolol treatment in the first assessment following the hospitalization. But symptoms of motor restlessness, mental unease, an urge to move, physical aggression and abdominal pain were present. Lorazepam 2.5 mg/day was added to treatment. The diagnostic tests regarding abdominal pain showed no pathology. The next day symptoms including abdominal pain improved moderately. Abdominal pain was thought to be a subjective complaint of discomfort caused by akathisia. Lorazepam dosage was increased to 7.5 mg daily gradually in the following days. During six days follow-up, there was a significant improvement in the symptoms and the only side effect was sedation. Then, both propranolol and lorazepam medication gradually stopped in three weeks without any akathisia symptoms.

Discussion

The most notable hypothesis attempting to explain the pathophysiology of akathisia is the dopamine receptor blockade in the mesocortical and mesolimbic areas [15]. A possible mechanism which causes akathisia can result from the different functions of the core and the shell parts of nucleus accumbens [16]. It is thought to involve the interaction of more than one neurotransmitter system. As a consequence, the complexity of this disorder makes it difficult to explain it with a single neurotransmitter [15].

There is a small number of reported akathisia caused by TBI cases. A 61-year-old man developed motor restlessness due to serious bilateral prefrontal cortex damage occurred after trauma. Symptoms were assessed as akathisia and alprazolam 2 mg/day was started. Due to the lack of improvement in symptoms, treatment was changed to diazepam 15 mg/day and bromocriptine 7.5 mg/day. In the following days, symptoms completely disappeared [17]. After a bicycle accident, agitation and continuous walking behavior occurred in an adult female patient with TBI. She was diagnosed with delirium and treated with risperidone 2 mg/day and temazepam 20 mg ante noctum. At the re-examination, the agitated behavior was worsened. Following a change of diagnosis to akathisia, antipsychotic medication was discontinued and clonidine was prescribed 0.05 mg/day. Clonidine dose was attempted to be reduced after 2 weeks, but the symptoms recurred. The patient had to be discharged with clonidine treatment. Only after 3 months, clonidine treatment could be stopped [14].

Our case, to our best knowledge, is the only adolescent case with akathisia caused by TBI without prior antipsychotic treatment. In two other adult case reports [14, 17], similar to ours, no antipsychotic medication was present before the akathisia symptoms. A 13-year-old adolescent was treated with haloperidol for agitation after a bicycle accident and the next day patient developed symptoms of akathisia [1]. Also, a 17-year-old adolescent female with bifrontal contusions and multiple injuries after an accident was reported. Following haloperidol treatment due to her agitation, symptoms of akathisia occurred [13]. However, it is not clear whether the akathisia was caused by TBI or antipsychotics in these cases.

In our case, the patient had aphasia concomitantly which complicated the diagnosis. Both in our case and the case reported by Wielenga-Boiten et al. [14], psychomotor agitation was not considered as akathisia initially and risperidone treatment was started towards agitation. After a change in diagnosis, treatment was changed to focus on akathisia. Diagnosing akathisia can be difficult due to unspecific criteria [18]. Differential diagnosis also includes affective or psychotic disorder, restless legs syndrome, tics, substance withdrawal and movement disorders, such as dystonia or tardive dyskinesia [19]. The patient had no neuropsychiatric disorder, psychotropic medication or substance addiction background. His symptoms were not restricted to leg stereotypes or worsened at night, as would be expected in restless legs syndrome [20]. In accordance with akathisia, he did not present any symptoms of motor or phonic tics [21].

Although treatment suggestions for akathisia are often based on case reports and small randomized studies [14], clonidine [22], propranolol [4] and benzodiazepines [1] are shown effective have been reported to be effective. In our case, improvements in the symptoms were observed on the first day, after lorazepam was added to propranolol treatment. No deterioration in the cognition or other side effects were observed other than the sedation with the maximum dose of 7.5 mg per day. No clinical scales were used for the patient's assessments, this is thought to be a limitation of this case report.

It is noted that if akathisia is misdiagnosed as delirium, and with the neuroleptic treatment symptoms may not improve. This may challenge the clinician to use higher doses of neuroleptics, causing symptoms to deteriorate [14]. In addition to the discomfort of the patient, akathisia is an important clinical condition that affects the treatment and the rehabilitation negatively. Therefore, it should be kept in my mind for differential diagnosis and its treatment should not be delayed.

Conflict of Interest: No conflict of interest was declared by the authors.

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