# Catatonia and Differential Diagnosis in Adolescence: A Case Report

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#### **ABSTRACT**

## Catatonia and differential diagnosis in adolescence: a case report

Catatonia is a motor dysregulation syndrome with main symptoms and signs such as mutism, posturing, stupor, motor rigidity, hyperalertness, refusal to eat or drink, and hypokinesis. Recognizing catatonia in children and making a differential diagnosis is important in terms of investigations required as well as for determining the treatment protocol. The present paper discusses the differential diagnosis of catatonia presentation and the clinical characteristics of a 17-year-old male case who was initially followed as inpatient in our clinic for catatonia presentation, though his diagnosis did not become definite within this period. Approximately one year later, he was readmitted to our clinic being pre-diagnosed with bipolar disorder-manic attack because of symptoms such as talking excessively, hyperactivity, irritability, and insomnia.

Keywords: Adolescent, bipolar disorder, catatonia

#### ÖZET

# Ergen yaş grubunda katatoni ve ayırıcı tanısı: Bir olgu sunumu

Katatoni, temel belirti ve bulguları mutizm, postür alma, stupor, motor rijidite, aşırı uyarılmışlık hali, yeme-içme reddi ve hipokinezi olan bir motor disregülasyon sendromudur. Çocuklarda katatoni tablosunun tanınması ve ayırıcı tanısının bilinmesi, yapılması gereken incelemeler ve tedavi protokolünün belirlenmesi açısından önemlidir. Bu yazıda ilk olarak katatoni tablosu nedeniyle servisimizde yatırılarak izlenen ancak bu yatışta tanısı netleşmeyen, yaklaşık bir yıl sonra da çok konuşma, hareketlilik, sinirlilik, uykusuzluk gibi belirtiler nedeniyle bipolar bozukluk-manik atak tanısıyla servisimize yeniden yatışı yapılan 17 yaşında erkek bir olgunun klinik özellikleri ve katatoni tablosunun ayırıcı tanısı tartışılmıştır.

Anahtar kelimeler: Ergen, bipolar bozukluk, katatoni



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## INTRODUCTION

Asubtype of schizophrenia, it is a motor dysregulation syndrome frequently seen together with mood disorders and medical or neurological diseases. Basic signs and symptoms include mutism, posturing, stupor, motor rigidity, hyperexcitability, refusal of eating-drinking, and hypokinesia (1). Catatonia appears as a separate entry in DSM-5 (2). Although catatonia and its differential diagnosis have been issues of interest for years in cases from the adult-age group, it is noticeable that awareness in this field regarding the pediatric and adolescent age group is

limited (3). Recognizing catatonia in children and making a differential diagnosis is important in terms of necessary investigations as well as for determining the treatment protocol. The present paper discusses the differential diagnosis of catatonia and the clinical characteristics of a 17-year-old male case who was initially followed as inpatient in our clinic for differential diagnosis and treatment of catatonia but was discharged upon his family's request before his diagnosis had become definite, and was then re-admitted to our clinic 9 months later being diagnosed with bipolar disorder-manic attack because of symptoms such as talking excessively, hyperactivity, irritability and insomnia.

# **CASE**

The case was a 17-year-old male, 3rd-grade student in high school, the older one of two siblings, living in Canakkale with his family. His neuromotor development had been in time with no history of problems in the perinatal or postnatal period. His family history revealed ongoing depression in the mother. He was first referred to a psychiatrist on May 2012 in Canakkale with symptoms of nonsensetalking, hyperactivity, irritability, and throwing stones at the neighbors following an event when his teacher had smacked him in school. Treatment was started with valproate 1000mg/day and olanzapine 10mg/day for pre-diagnosis of bipolar disorder. As the case became introvert and was not speaking, not moving and not eating after 1-2 weeks, drug therapy was discontinued and the family was referred to our hospital from Canakkale with recommendation of hospitalization. The picture at his admission to the emergency room was considered to present catatonia, and he was hospitalized. On mental state examination, he was reluctant to be interviewed, did not answer any of the questions, and wanted to keep lying down. He had restricted affect. Orientation, perception and memory could not be examined. Thought content could not be assessed as he did not speak. The patient was lying in his bed, eyes open, on his back, without changing his posture. He was not moving on his own in the bed unless his posture was changed by his father. He was going to the bathroom together with his father upon the latter's direction. After hospitalization, his vital signs were monitored, routine blood analyses, thyroid function tests, creatinine kinase (CK) level, urinalysis, substance analysis in the urine, vitamin B12 and folic acid levels, anti-HBs, anti-HIV, HCV-RNA, VDRL, CRP, serum ceruloplasmin level, cranial MRI, and EEG were evaluated. In order to exclude an organic etiology, consultation was requested from neurology and pediatrics clinics. The results of blood analysis, serum CK level, urine analyses, cranial MRI and EEG were within the normal ranges. Neurological examination and fundus examination were also unremarkable. Lorazepam 5mg/day was started

assuming that he had catatonia. Several hours after the initial lorazepam dose, he spontaneously talked to his father and the nurse for half an hour. On the following days, the patient did not speak but ate his meals and partially followed instructions. The dose of lorazepam was increased to 6mg/day. His vital signs were normal over the course of hospital stay. On follow-up, he began to give one-word answers to the questions of the treatment team. Duration of staying in bed during daytime decreased and he began to walk around the clinic and sit next to the other patients of his own demand. He was assessed using the Clinical Global Impression-Severity of Illness (CGI-SI) and Clinical Global Impression-General Improvement (CGI-GI) scales; the score on CGI-SI decreased from 6 to 3 during follow-up and the score on CGI-GI was found to be 2. Although complete improvement had not been achieved and the diagnosis was not definite, the case was discharged on the 25th day of hospital stay with lorazepam 4mg/day therapy.

The patient did not come for recommended control visits after hospital discharge. He was admitted to our emergency psychiatry clinic in February 2013 with symptoms of skepticism, aggression, insomnia, talking excessively, spending a lot of money, running away from home, and refusing treatment. He was prediagnosed with bipolar disorder-manic attack and hospitalized for the second time. On the first mental examination in the clinic, he was conscious and showed correct orientation but partial cooperation. His self-care was decreased and he was reluctant to be interviewed. While interviewing, it was observed that speed and amount of his speech increased, he was perseveratively repeating the same sentences, his mood was elated, his affect was irritable, and psychomotor activity was increased. Thought content revealed themes such as that his mother wanted to poison him and people are talking about him. He displayed thought disorder with flight of ideas and loosening of associations. There were delusions of reference and persecution and auditory hallucinations. He had no insight and was refusing oral drug intake. Pharmacotherapy was commenced with valproate 500mg/day, haloperidol 20mg/day and biperiden 10mg/day via intramuscular (IM) route. On the fifth day of hospital stay, oral olanzapine 10mg/day was included in the treatment and IM haloperidol therapy was discontinued. During follow-up, the olanzapine dose was increased to 30mg/day and the valproate dose was gradually increased to 1500mg/day. Over the course of the follow-up period, it was observed that delusions of persecution and reference gradually decreased, speed and amount of association of ideas returned to normal, mood was euthymic, and he gained insight. Valproate dose was decreased to 1250mg/day while monitoring serum valproate concentrations. After a 60-day hospital stay, the case was discharged from the hospital with valproate 1250mg/day and olanzapine 30mg/day treatment and recommended policlinic follow-up. Scores of Young Mania Rating Scale (YMRS) over the course of his hospital stay were as following: on the day of hospitalization, 19; 1st week, 19; 2nd week, 23; 5th week, 11; 6<sup>th</sup> week, 10; and 8<sup>th</sup> week, 5.

# **DISCUSSION**

Presence of typical signs of catatonia in the present case such as not moving, not speaking and refusing to eat and drink at the time of hospital admission made it easier for us to recognize catatonia. In DSM-IV, catatonia is defined as a subtype of schizophrenia. However, catatonia can appear as a symptom also in diseases of different diagnostic categories. Recent studies in particular stated that mood disorder is the common underlying disease (4). Presence of bipolar disorder, which had been considered as a prediagnosis in the present case at an external center before admission to our hospital and thereafter became definite after the second admission, supports the literature.

Delirious mania is a neuropsychiatric syndrome difficult to diagnose that is characterized by suddenonset delirium, mania, and psychosis. Catatonia is usually one of the critical features of the syndrome and is seen in about 15% of acute mania patients (5). It is usually difficult to distinguish from excited catatonia, and some authors propose delirious mania

as a subtype of catatonia. However, it is important to distinguish these two clinical pictures, as their treatments and courses are different (6). The condition may be life-threatening with increasing severity unless recognized and treated properly. In the present patient, too, sudden and severe onset of the clinical picture with symptoms such as incoherent speech, hyperactivity, leaving home, irritability and throwing stones at the neighbors and the subsequent appearance of catatonia are consistent with delirious mania mentioned in the text. However, since the case showed catatonia at the time of admission to the emergency room and his family stated that those disorganized behaviors displayed before the onset of catatonia had started suddenly, primarily the examinations focusing on the exclusion of organic causes were prioritized.

Medical and neurological diseases besides psychiatric conditions and, in addition, neuroleptic malignant syndrome (NMS) in those with a history of antipsychotic drug use should be kept in mind in the differential diagnosis of catatonia. NMS is a catatonialike condition that usually presents itself with extrapyramidal signs, alterations in blood pressure, and alterations in consciousness, and hyperreflexia; it is a rare but serious side effect of neuroleptic therapy. In the majority of cases, symptoms appear in the first weeks of treatment (7). NMS can be seen with the use of any antipsychotic drug including atypical antipsychotics, even with small doses (8). In the literature, it is mentioned that cases displaying catatonia symptoms and receiving antipsychotic medication before catatonia is improved are more prone to develop NMS (4,9). The present case had a history of short-term olanzapine use before his first hospital admission. Vital signs of the case were monitored at frequent intervals and complete blood count and serum CK levels were checked at certain intervals to exclude olanzapine-associated NMS. On follow-up, vital signs, routine blood analyses and serum CK level were within the normal ranges and consciousness was always clear with no alteration. For all these reasons, NMS was not considered in the present case.

In the literature, it is emphasized that catatonia can also develop in many medical and neurological diseases (9,10). The prevalence rate of underlying medical and neurological diseases in children and adolescents that present with catatonia is considerable. In prospective prevalence studies, the prevalence rate of catatonia associated with general health status in psychiatry clinics was 20-25% (1). Catatonia may also develop due to head trauma, epilepsy, metabolic and endocrine disorders, hepatic failure, hepatic encephalopathy, systemic lupus erythematosus, infections (Ebstein-Barr, hepatitis C) and various drugs (11-14). Again, it is reported that catatonia may appear in children with pervasive developmental disorder, particularly in adolescence (15,16). The present case had no symptoms that support pervasive developmental disorder or delay in developmental stages. Detailed medical examinations, which were performed to exclude other systemic diseases, revealed no positive signs. EEG and detailed neurological examination, performed to exclude non-convulsive status epilepticus, were considered to be within the normal limits, and no leukocytosis was determined. Relatively recent publications, however, mention encephalitis cases associated with anti-N-methyl-Daspartate (anti-NMDA) receptor antibodies (17,18). In this type of encephalitis, catatonia may accompany seizure, dyskinesia, autonomic instability, and hypoventilation. Diagnosis becomes definite by demonstrating anti-NMDA antibodies in the cerebrospinal fluid. In the present case, NMDA encephalitis was considered as one of the differential diagnoses, but the antibody could not be studied because of limited facilities for analysis. In the present case, the absence of other symptoms that suggest this type of encephalitis, unremarkable neurological examination, and a gradually improving clinical picture with benzodiazepine treatment led us away from this diagnosis.

In the literature, it is reported that catatonia accompanying mood disorders in particular shows rapid response to high-dose sublingual or intramuscular benzodiazepine therapy; the catatonia picture resolves within a few hours after the first or second dose in most of the cases; however, in some cases resolution may only occur after weeks, or even months (4). In the present case, catatonia gradually improved after long-term benzodiazepine use, although the underlying diagnosis was a mood disorder. However, the patient's talking with the nurse of his own accord for about a half an hour after the first benzodiazepine dose was considered consistent with dramatic improvement mentioned in the literature.

Catatonia is not a rare condition in children and adolescents who are admitted to acute psychiatry clinics and should be considered in the differential diagnosis. It seems that catatonia being under a separate title in DSM-5 will enhance recognition of this picture by clinicians. Detailed medical examination in children and adolescents that present with catatonia is of great importance for the exclusion of underlying medical diseases and neuroleptic malignant syndrome. In addition, considering the prevalence of concurrency of mood disorders and catatonia presentation, taking a detailed anamnesis of mood disorders in cases thought to be suffering from catatonia would be beneficial in follow-up and during treatment.

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