Malignant catatonia triggered by acute psychological traumatic experience in a patient with schizophrenia: A case report

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SUMMARY

Malignant catatonia is defined as catatonia accompanied by hyperthermia and/or autonomic instability. Catatonia can develop in association with psychiatric disorders such as schizophrenia, bipolar disorder, and major depressive disorder, as well as various medical and neurological conditions. However, our knowledge regarding the role of acute traumatic experiences in the development of catatonia remains limited. This case report discusses a presentation of malignant catatonia triggered by an acute psychological trauma in a patient with schizophrenia in remission. A 41-year-old male patient has been followed with a diagnosis of schizophrenia since 2003. Following an acute psychological traumatic event, the patient developed withdrawal, reduced speech, refusal to eat and drink, and generalized rigidity. Upon examination, the patient exhibited catatonic stupor, rigidity, mutism, negativism, and vital signs indicating hyperthermia and hypertension. Based on these findings, a diagnosis of malignant catatonia was considered. The patient was administered electroconvulsive therapy. After eight sessions of electroconvulsive therapy, a significant improvement in the patient's clinical symptoms was observed. Traumatic experiences may contribute to the development of malignant catatonia through mechanisms such as acute threat perception, inflammatory responses, autonomic instability, and dysregulated dopaminergic signaling

Key words: Schizophrenia, catatonia, trauma

INTRODUCTION

Catatonia is a syndrome characterised with rigidity, mutism, posturing, immobility, negativism, repetitive behaviours, echolalia, echopraxia and waxy flexibility (1). Catatonia can be conceptualised as a spectrum. At one end there are milder forms called benign and at the other end there are more severe forms called malignant (2).

Malignant catatonia is defined as catatonia accompanied by hyperthermia and/or autonomic instability. Autonomic instability may manifest itself with symptoms including tachycardia, high or irregular blood pressure, diaphoresis, tachypnoea and cyanosis (3). Although historically associated solely with schizophrenia, it is now known that catatonia can also be caused by other psychiatric disorders such as bipolar disorder, major depressive disorder, and psychotic disorders. Additionally, various medical conditions, neurological diseases, and the use of neuroleptics can also trigger catatonia (4,5). A systematic review by Oldham reported that approximately 20% of catatonia cases have a medical cause. Among these, two-thirds are related to conditions affecting the central nervous system, including encephalitis, neural injuries, developmental disorders, structural brain diseases, and seizures (6). Other contributing factors include infections, metabolic disorders, endocrinopathies, and sudden withdrawal from alcohol or benzodiazepines, all of which can lead to catatonic states (7-9).

Although many factors have been identified in the literature about the etiology of catatonia, our knowledge about the role of acute stressors or traumatic experiences is limited. Although a limited number of cases have been reported in the literature regarding the role of acute traumas in the development of catatonia, there has yet to be a case report identifying acute traumas as a triggering factor for malignant catatonia. In this case report, we

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aimed to discuss the presentation of malignant catatonia triggered by an acute psychological trauma in a patient diagnosed with schizophrenia who had been in remission for the past five years.

Case

A.H.K. is a 41-year-old male patient. The patient's complaints first emerged in 2003, presenting as persecutory delusions, visual hallucinations, agitation, and insomnia. Approximately five months after the onset of symptoms, the patient underwent inpatient treatment in a psychiatric clinic for two months. The predominance of positive psychotic symptoms, the absence of mood disorder features, and the exclusion of any identifiable organic cause that could explain the clinical presentation supported the consideration of a diagnosis of schizophrenia. The patient was treated with olanzapine at a dosage of 5 mg/day until 2019, during which positive symptoms diminished; however, negative symptoms such as social isolation and avolition partially persisted. It was reported that the patient voluntarily discontinued olanzapine treatment in 2019 and experienced no changes in his syptoms until August 2024.

In August 2024, the patient was threatened by unfamiliar individuals while walking alone on the street and was kicked in the leg. The trauma experienced by the patient did not result in any physical injury. However, based on information obtained from the patient's mother, it was learned that the patient perceived the incident as a death threat and subsequently experienced severe anxiety and fear. Following the incident, he began to exhibit symptoms such as withdrawal, reduced speech, refusal to eat or drink, and rigidity throughout his body. Consequently, his family took him to the emergency department. In the evaluation conducted by the neurology department in the emergency room, no abnormalities were detected during the neurological examination aside from rigidity, and both the brain computed tomography (CT) and magnetic resonance imaging (MRI) were assessed as normal. The psychiatric evaluation indicated that the patient was unresponsive to external stimuli and remained motionless. The patient's level of consciousness was assessed as catatonic stupor. Limited cooperation, mutism, and a flat affect were observed. Due to mutism, an assessment of thought content could not be conducted. Additionally, the patient exhibited widespread muscle rigidity and negativism. The values outside the normal reference ranges in laboratory investigations were determined as follows: Creatine kinase (CK): 7370 U/L (reference range: 30-200 U/L), C-reactive protein (CRP): 31 mg/dL (reference range: 0-5 mg/dL), aspartate aminotransferase (AST): 465 U/L (reference range: 5-34 U/L) and alanine aminotransferase (ALT): 266 U/L (reference range: 0-55 U/L). The patient's initial examination revealed the following vital signs: body temperature 38.5°C, blood pressure 140/90 mmHg, and pulse rate 101 beats per minute. Based on the assessment of the patient's clinical and vital findings, a preliminary diagnosis of malignant catatonia was considered, and the patient was admitted to the psychiatry ward. Upon admission, the Bush-Francis Catatonia Rating Scale (BFCRS) score for the patient was calculated to be 27. The fact that the patient had not used antipsychotic treatment for the last 5 years contributed to the exclusion of neuroleptic malignant syndrome (NMS) in the differential diagnosis.

Due to the unavailability of lorazepam in Turkey for some time, the patient was started on a treatment regimen of diazepam at a dosage of 20-30 mg/day. During the first days of the patient's admission, the body temperature fluctuated between 38.5°C and 39.5°C, while the blood pressure ranged between 130/90 mmHg and 150/110 mmHg. The patient was consulted to the Internal Medicine department, hydration was recommended and antipyretic treatment was initiated. Due to elevated ALT and AST levels, a hepatobiliary ultrasound was performed. Following the absence of any pathological findings, N-acetylcysteine (NAC) 900 mg/day treatment was initiated upon the recommendation of the internal medicine department. The patient's ALT and AST levels gradually decreased within two weeks. On the fourth day of the patient's admission, in light of the lack of improvement in catatonic symptoms despite diazepam treatment, it was decided to proceed with electroconvulsive therapy (ECT). The patient was evaluated by the Neurology, Cardiology, Pulmonology, and Anesthesiology and

Reanimation departments. The patient underwent a total of eight sessions of bilateral ECT, administered three times a week. Following the second application of ECT, a significant improvement in the patient's catatonic symptoms was observed. The patient began to speak in short sentences, exhibited improvement in affect, and started consuming liquid foods. Following the eighth session of ECT, the patient began to walk and speak fluently, exhibited a reduction in rigidity, and normalized oral intake. During the patient's hospitalization, hemogram, electrolyte levels, liver function tests, and kidney function tests were closely monitored. The CK level decreased to 108 U/L, the CRP level to 5 mg/dL, the AST level to 28 U/L, and the ALT level to 41 U/L, all returning to normal reference ranges. The patient, who exhibited a reduction in catatonic symptoms and was assessed with a BFCRS score of 0, was discharged on the 30th day of hospitalization.

DISCUSSION

Particularly, it has been discussed through various cases that acute stressors and traumatic experiences can trigger catatonic states. Such conditions are commonly associated with psychiatric disorders such as post-traumatic stress disorder (PTSD) or acute emotional stress (10-13). However, there have been no case reports in the literature regarding the development of malignant catatonia based on acute traumatic experiences. This case underscores the potential of acute traumatic stress to trigger severe psychiatric disorders, including malignant catatonia.

The association between traumatic experiences and the onset of malignant catatonia is complex and not yet fully elucidated. Several mechanisms have been proposed to explain this relationship; however, these explanations primarily focus on how acute stress factors contribute to the development of catatonia (14). Traumatic experiences, may heighten vulnerability to catatonia through pro-inflammatory mechanisms and dysregulated threat processing in the amygdala (15). Increased cytokine activity is thought to be a significant factor in the emergence of sickness behavior by suppressing dopamine production and function in the reward system (16). Sickness behavior is a behavioral response pattern characterized by symptoms such as apathy, psychomotor slowing, loss of interest and appetite, and occasionally fever, reflecting a strategy aimed at conserving energy. This model shares similarities with certain aspects of conditions such as catatonic stupor (17). Another explanation is that in situations of acute fear or stress, an individual may enter a catatonia-like state when perceiving that there is no possible escape to ensure safety. This phenomenon resembles an acute fear response observed among mammals and birds, known as the 'freezing' or 'playing dead' reaction. This response has been associated with a mechanism involving the activation of the vagus nerve (18). In addition to these mechanisms, autonomic nervous system dysfunction, manifesting as sympathoadrenal hyperactivity, is considered a potential underlying mechanism in the relationship between acute traumatic experiences and malignant catatonia. Acute traumatic experiences can lead to autonomic instability by overactivating the sympathoadrenal system, dysregulating the HPA axis, disrupting neurotransmitter homeostasis, triggering neuroinflammatory processes, and impairing vagal tone. These physiological alterations collectively contribute to an increased susceptibility to malignant catatonia (19). In individuals with a history of trauma, the interplay between acute threat perception, inflammatory responses, autonomic instability and dysregulated dopaminergic signaling may trigger malign catatonia as a maladaptive defensive mechanism.

In conclusion, acute trauma or traumatic life events can trigger catatonic presentations, particularly in individuals who are already at psychiatric risk. Given the serious risks associated with malignant catatonia, particularly the high rates of morbidity and mortality, it is crucial to recognize and assess the presence of acute traumatic experiences in patients, as well as to intervene early in such situations.

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