Catatonia after minor head trauma in an adolescent

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Abstract

Catatonia is a poorly understood and underrecognized psychomotor condition characterized by three or more catatonic symptoms, commonly including abnormalities in speech, affect, and movement. Catatonia is generally associated with psychiatric disorders such as bipolar disorder and schizophrenia but may be seen in general medical conditions and rarely after physical trauma. Here we present a case of catatonia in an adolescent patient as a result of minor head trauma. It is essential that ED physicians recognize this condition as delay in diagnosis may lead to patient harm.

Introduction

Catatonia, or catatonic behavior, is a poorly understood psychomotor syndrome typically characterized by lack of movement and lack of communication. Although classically associated with schizophrenia, it most commonly occurs in patients with bipolar disorder. Changes from the DSM-IV to DSM-5 expanded on the diagnostic application of catatonia, recognizing that it can occur in patients with other general health conditions independent of depressive, bipolar, or psychotic disorders [1].

The diagnostic criteria for catatonia, which remain unchanged since the DSM-IV, require three or more of the following: stupor, waxy flexibility, catalepsy, mutism, posturing, negativism, stereotypy, mannerisms, grimacing, agitation, echopraxia, and echolalia. There are multiple types of catatonia: akinetic, excited, and malignant. Akinetic catatonia is the most common. A patient who is akinetic may mimic coma and stare blankly, without response to voice or noxious stimuli. Excited and malignant catatonia share significant overlap. Patients may display pointless and impulsive movements or seem combative and agitated. This psychomotor agitation can lead to life-threatening complications such as autonomic dysfunction, rhabdomyolysis, or hyperthermia [2]. Although less commonly recognized, catatonia has been described in the setting of endocrine abnormalities, electrolyte derangements, neurologic conditions, and drug-related illness [3].

Rarely, catatonia can occur in association with physical injury. To date, there have been few reported cases in the literature describing catatonia immediately following trauma [4, 5, 6, 7]. Herein we describe a case of akinetic catatonia following minor blunt head trauma in a teenaged patient.

Presentation:

A 16-year-old male with no reported medical history presented to a Level 1 trauma center as a trauma alert via Emergency Medical Services (EMS) following a fall while playing basketball. Per paramedic report, the patient was pushed while jumping and subsequently hit his head against the metallic support pole of the basketball hoop. He reportedly lost consciousness for one to two minutes, after which time he opened his eyes. A cervical collar was placed, and the patient was transported on a backboard. En route to the Emergency department (ED), the patient had no verbal or motor response to painful stimuli with a reported Glasgow Coma Scale (GCS) of 5. EMS vitals included heart rate of 114 beats per minute (bpm), blood pressure (BP) 139/77 mmHg, and point-of-care glucose of 134 mg/dL.

Upon arrival at the ED, vitals were within normal limits, including temperature and repeat glucose. Advanced Trauma Life Support (ATLS) protocol was initiated. Primary survey revealed a young male who appeared reported age without external evidence of trauma. Initial GCS was 6 (eyes opened spontaneously (4), no verbal response (1), and no motor response (1)). Further examination revealed blank staring with evidence of tearing. When the patient's upper extremities were moved against gravity by the examiner, the patient maintained the fixed positioning in which he was placed. No other physical exam abnormalities were identified on primary or secondary surveys. Discussion of intubation for airway protection was had between the ED and trauma team given depressed GCS. Ultimately, it was decided to forgo advanced airway placement as the patient was deemed to be protecting his airway. Chest x-ray and pelvis x-ray were performed after initial assessment and showed no acute abnormalities. Laboratory and additional imaging studies were initiated. Results of a complete metabolic panel and blood count were unremarkable. Computed tomography (CT) studies of the head and entire spine were negative for acute injury, as were CT angiography studies of the chest, abdomen, and pelvis. The patient remained closely monitored both physically and on continuous telemetry and was frequently reassessed.

Approximately thirty-three minutes after arrival, the patient was found to be whispering "help me", repeatedly, in response to questioning. It appeared that his postural findings had resolved unilaterally on the right, and he could follow commands in the right upper and lower extremity. His left upper extremity continued to remain in fixed positions in which the examiner placed it. He appeared to be tracking staff with his eyes. He remained tearful. His GCS had thus improved to 13 (opened eyes spontaneously (4), voiced words (3), and followed commands (6)). Two hours after arrival, the patient's mental status and neurologic exam normalized. He could follow commands in all four extremities and answered questions appropriately. He corroborated the paramedic report of initial events but did not recall the events following his head strike.

The patient's mother arrived at the ED and reported that the patient had a past medical history significant for anxiety and depression, for which he had been prescribed aripiprazole and citalopram. The patient reported that he had not been taking the medication for approximately one week. He had never experienced catatonia before. After demonstrating he could ambulate and tolerate oral intake without difficulty, he was discharged home in the care of his mother with return precautions and instructions to follow up with his psychiatrist and psychologist within the week.

Discussion

Catatonia following minor head trauma is a rare clinical phenomenon, with few cases published in the literature to date. Here, we described the first pediatric case of traumatic catatonia in a teenager following mild traumatic brain injury (TBI). Although rare, it is important that clinicians are aware of this psychomotor syndrome, as failure to recognize or a delay in diagnosis may place patients at risk for iatrogenic harm and even death. Given the significant variability in presentation, as well as overlap with other pathologic states, clinicians must maintain a high degree of suspicion when approaching patients with perturbations in behavior or mentation. Particularly in undifferentiated patients, catatonia must be considered along with TBI, electrolyte or metabolic derangements, delirium, post-concussive syndrome, neuroleptic malignant syndrome, serotonin syndrome, cerebrovascular accidents, and drug effects.

In our case, had the patient not demonstrated obvious waxy flexibility, catatonia may not have been considered prior to addressing his airway in the primary survey. It is likely that the patient would have been intubated for airway protection in the setting of trauma and low GCS given the

dogma surrounding ATLS protocols, which suggests a GCS of 8 or less predicts loss of airway reflexes and warrants intubation. Strict adherence to this protocol could have led us astray and caused untold harm to our patient.

The pathophysiology of catatonia is not well-described and likely complex. Some authors suggest it involves disruption of the basal ganglia at sites along the thalamocortical tracts, which thereby disrupt motor function, similarly to parkinsonism. [8] On a synaptic level, this disruption is thought to result in decreased downstream dopamine transmission, under stimulation of GABA-A receptors, and overstimulation of NMDA receptors. Additionally, newer neuroimaging studies suggest there is increased blood flow to motor areas in patients with catatonia, as well as abnormal hyperactivity in the supplementary motor area (SMA) and pre-SMA in functional studies. These structures and pathways are thought to be critically involved in motor control, movement selection, initiation, timing, and inhibition. [9]

Other authors suggest that catatonia is an outward response of fear or intense anxiety; however, not all patients with catatonia report anxiety during their episode. [10] Some authors cite a potential evolutionary basis for this fear response, quoting the tonic immobility seen in some animal species in response to movement-driven predation. They suggest catatonic features in response to fear-inducing stimuli in man are misexpressed remnants of this originally adaptive behavior selected by our ancestors. [11] In addition, catatonia has been implicated as a rare manifestation of post-traumatic stress disorder. [12, 13, 14] Studies have shown that patients with post-traumatic stress disorder (PTSD) exhibit overall decreased cortical GABA activity and increased glutamate production. [15] Decreased GABAergic tone is associated with anxiety and cognitive impairment, whereas increased glutamatergic tone is thought to promote derealization and depersonalization. This may suggest that patients with PTSD are more prone to develop catatonic symptoms, partly owing to an underlying neurochemical imbalance.

TBI patients are predisposed to development of psychiatric disorders. In one study, 43 of 60 TBI patients developed a new axis i or ii psychiatric disorder during the 30 year follow up. [16] Catatonia after traumatic or anoxic brain injury has been described in the literature [17, 18, 19]. Unlike in our patient, the severity of each of the TBIs described in these cases resulted in severe limitation of function requiring prolonged rehabilitation or resulting in permanent disability. Given the suggested pathophysiology of catatonia, it is unclear whether our patient's catatonic symptoms were triggered by a fear-based response to the event or the minor head trauma that he sustained.

The gold standard treatment of catatonia is benzodiazepines, and purely catatonic patients characteristically have a robust response to administration. [20] In patients with suspected catatonia, a lorazepam challenge is often issued to confirm the diagnosis. Patients are given 1-2mg of lorazepam intravenously (IV) and reassessed after 5 minutes. If the patient's symptoms are unchanged, the dose is repeated. A positive response to the challenge is defined as a 50% reduction in catatonic symptoms. [20] Intramuscular (IM) and per os (PO) administration has been described. [8] If given IM, it is recommended that the second dose should be given after 15 minutes, and if given PO, the second dose should be given after 30 minutes. Although lorazepam is classically used, there is no high quality evidence suggesting its superiority to others in the class. A zolpidem challenge has also been described. [21] Similarly to the lorazepam challenge, 10mg of zolpidem is given to the patient PO and symptoms are assessed after 30 minutes. Maintenance dosages of benzodiazepines are often required to prevent recurrence of symptoms. [20] Electroconvulsive therapy (ECT) is employed for severe or resistant cases.

Conclusion

Catatonia is an underrecognized condition, and may be particularly unrecognized in the ED. It is essential for clinicians to consider catatonia in the patient who presents with alterations in mentation or motor function, as catatonia is generally reversible, and prognosis improves with early treatment [20]. In addition, patients may be spared unnecessary interventions if catatonia is recognized. Although the pathophysiology of catatonia is incompletely understood, the treatment is often simple and reliable.

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