Traumatic Brain Injury–Induced Mania

To the Editor: Several studies have shown that the incidence of aggression in post–traumatic brain injury is between 35% and 90%.[1,2] Chronic irritability and aggression are seen in approximately 40%–70% of patients with traumatic brain injury. Aggression in patients with such an injury is generally reactive without premeditation and is nonpurposeful, explosive, periodic, and ego dystonic (ie, the patients are genuinely remorseful after the event).[3] Damage to the limbic system, orbitofrontal cortex, left anteromedial frontal lobe, and anterior cingulate gyri has been particularly associated with aggressive behavior.[4]

This is a case of a 60-year-old man with no prior personal or family psychiatric history who presented to the emergency room with manic symptoms. He had been doing well until he suffered a head injury, after which his personality changed dramatically. In addition to newly developed manic symptoms, he began to display very aggressive and violent behavior. It is important to diagnose these new cases of mood disorder secondary to traumatic brain injury.

Case report. Mr A is a 60-year-old white man with no prior psychiatric history who presented to the emergency room accompanied by his wife to get help for newly developed aggression and legal trouble. Mr A had been in his normal state of health until 5 months ago when he began to have syncopal episodes. He fell and hit his head during 1 of these episodes and required surgical staples for a scalp laceration. A computed tomography (CT) head scan failed to demonstrate acute brain injury at that time. He was eventually diagnosed with sick sinus syndrome and ultimately had a pacemaker placed. Mr A's wife told us that he had a brief period of depression following his head trauma and later developed behavioral and personality changes characterized by elevated and irritable mood, grandiosity, and decreased need for sleep (2 to 3 hours per night). He also began to watch pornography, which he had not been interested in prior to his head injury. He began spending money excessively ($10,000–$20,000 per month) on clothing. He became paranoid about people stealing water from the water company he owned and accused his close friends of taking advantage of him and trying to kill him. He grew so paranoid that he hired a private detective to investigate his suspicions that his wife was cheating. Mr A was arrested multiple times over a 1-month period for aggravated assault. On 1 occasion, he pulled a gun on a police officer. He began to see a private psychiatrist and was started on valproic acid and olanzapine. His psychiatrist ultimately stopped seeing Mr A because he was noncompliant with medications, was noncooperative, and displayed aggressive and threatening behavior during follow-up visits. Mr A subsequently went to the emergency room to seek psychiatric help.

At the emergency room, Mr A was alert and oriented and had good eye contact. His speech was pressured, and he was very hypervigilant. He described his mood as "okay" with constricted affect. His thought process was logical with some tangential thought and grandiosity. He denied suicidal or homicidal ideations or hallucinations and demonstrated intact memory but poor insight and judgment. His laboratory results including complete blood count, basic metabolic panel, thyroid-stimulating hormone, and urine drug screen were normal, and the electrocardiogram showed mild tachycardia. We restarted valproic acid and added olanzapine. He was disruptive, distracted, and uncooperative and filed multiple complaints against the hospital administrators and staff during his stay. He requested to leave while in an acute phase of mania, and an order of protective custody was filed. As Mr A continued to be agitated, threatening, combative, and paranoid despite being medicated, a valproic acid level was obtained and demonstrated a subtherapeutic level. The treatment team began to suspect that Mr A was cheeking medications or that the metabolism of the medication was somehow accelerated. The neurology department was consulted, and all neurologic workup including an electroencephalogram and CT scan of the brain were unremarkable. Gradually, we increased the dosage of valproic acid from 1,000 mg daily to 2,500 mg. His mood stabilized; he was no longer violent and aggressive but, instead, was interactive and cooperative. Mr A was discharged after 16 days of inpatient stay under the care of his wife. He was diagnosed with bipolar disorder, mania secondary to traumatic brain injury per *DSM-5* criteria.

Mr A was arrested several times for possession of a deadly weapon and aggravated assault—knowingly and recklessly causing serious bodily injury to another person, or using or exhibiting a deadly weapon in the course of committing any assault crime. He had no prior history of psychiatric illness and developed symptoms after head trauma. In this case, the CT was normal. Magnetic resonance imaging (MRI) was not done, as Mr A had a pacemaker implanted. The CT and MRI cannot illustrate diffuse axonal shearing. Diffuse axonal injury is commonly caused in head injury.[5] Mr A had significant head injury requiring stitches. The association between the head injury and the later development of bipolar mania and aggression could possibly be temporally but not causally associated. Or, an underlying yet to be diagnosed event might have led to the syncope as well as to the aggressive behaviors purported to be secondary to disinhibition. It is important to understand the presentation of these cases. It can be challenging to get to an accurate diagnosis, and it is important to stabilize such patients in an inpatient setting.

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