

# A Case of Bipolar Disorder, Premorbid Traumatic Brain Injury, and Comorbid Migraine Headaches

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Psychiatric and neurological disorders have been linked to head trauma such as traumatic brain injury (TBI).<sup>1</sup> Approximately 10% of persons with bipolar disorder (BD) have experienced a premorbid TBI.<sup>2</sup> Individuals with BD have a 2 to 3 times higher incidence of migraines than the general population.<sup>3</sup> The etiology of migraine and BD is unknown, but some studies have suggested that the conditions may share a common pathway.<sup>3</sup>

## CASE REPORT

A 36-year-old man was referred to a psychiatry practice for a years-long history of migraines that had been unresponsive to treatment with numerous medications. The patient had sustained a significant TBI at age 20 as a result of a motor vehicle accident that had required admission to an intensive care unit and intubation.

The patient works as a registered nurse. Despite the TBI, he had been able to graduate from nursing school and work without any performance issues. Recently, the patient and his wife had divorced after 10 years of marriage, and he had moved in with his parents. He cares for his 2 children every other weekend with his parents' support.

The man initially presented to the psychiatry practice 3 years prior for possible medical management of what he referred to as crippling migraines. He also stated that he felt "depressed and hopeless." He had been under the care of a neurologist for 6 years

for migraines that had not consistently been responsive to multiple medications and novel treatments, including tetrahydrocannabinol and narcotics.

The patient reported that his migraines fluctuate along with the changes in therapies, and he attributes the migraines to his TBI. He reported months of being migraine-free alternating with periods of debilitating migraines. He had missed excessive days of work, and he was looking into long-term disability benefits. When he is migraine-free, he reported being productive at home and work.

The patient provided a psychiatric history of depression without suicidal ideology or suicide attempts. He denied a family history of psychiatric disorders or insomnia. He denied a history of exposure to violence or emotional, physical, or sexual abuse. His medical history was unremarkable. The patient was being treated by his neurologist and primary care provider (PCP) for depression with venlafaxine and bupropion. He stated that this therapy did help initially, although he reported insomnia, poor appetite, a sense of worthlessness, and decreased interest in activities for the past month. The patient voiced concerns about the adverse effects of the medications, including body aches.

He had a history of insomnia, described as difficulty falling asleep. He was currently taking eszopiclone, prescribed by his PCP, to which he adds diphenhydramine nightly. He felt that the medication helps for a period of time, and that he has good restful nights. He also described periods of time during which he has very little or no sleep.

An initial mental status examination revealed a relaxed, well-dressed man who appeared to be his stated age. His body mass index (BMI) was 29.7 kg/m<sup>2</sup>. His mood was described as depressed, and his affect was consistent with his mood. His thought process was coherent and clear with goal-directed content. His thought content was without hallucinations or delusions. His insight and judgment were limited.

Mental status examination findings at various follow-up visits were not consistent with his initial presentation. He appeared well-dressed and agitated with hyperactive behavior. His speech pattern was pressured with euphoric mood and affect. Insight and judgment were limited.

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The patient's most current mental status examination revealed a relaxed, well-dressed man with consistent BMI. His speech was coherent and fluent, consistent with his relaxed mood and affect. Insight and judgment were intact. Throughout the patient's examinations, his memory testing remained intact, with impaired attention during the visits with euphoric mood.

While being followed by the psychiatry practice, mental status examinations have revealed elated mood, pressured speech, and heightened affect. Initially, the patient's venlafaxine was changed to desvenlafaxine for a mood disorder; depression was suspected. Various benzodiazepines had been prescribed but discontinued at the request of pain management consultants, since narcotics had been prescribed for his migraines.

After following the patient, who demonstrated cycling mood along with cycling migraines and insomnia, he was diagnosed with bipolar II disorder and insomnia. The patient was placed on carbamazepine, a mood stabilizer chosen for its efficacy with migraine headaches. Bupropion was tapered and stopped. Based on his partial response to carbamazepine, a second-generation neuroleptic, lurasidone, was carefully added to his regimen to achieve recovery, which was ultimately achieved. He will continue monthly visits to the psychiatrist to monitor for any exacerbation of symptoms.

The patient has been back to work consistently for 6 months. He and his ex-wife share custody of their children. Once the patient was diagnosed with BD and treated appropriately, his sleep improved, and his migraines had decreased to 2 days in the past 6 months.

## DISCUSSION

The patient's premorbid TBI is well documented to increase his risk of BD. The American Psychiatric Association has recognized that TBI can cause BD.<sup>2</sup> In a cross-sectional study, Drange and colleagues<sup>2</sup> found that symptoms of BD developed approximately 8.9 years after the TBI. The mean age of their study group was 42 years. They also found that premorbid TBI was associated with comorbid migraine headaches, which had been documented previously in the literature.<sup>2</sup>

An interesting theory proposed by Drange and colleagues is the possibility that patients with psychiatric symptoms and comorbid migraine headaches overestimate a history of TBI to explain their disruptive symptoms.<sup>2</sup> A meta-analysis by Perry and colleagues<sup>4</sup> revealed that a statistically significant number of cases of individuals with BD were associated with prior TBI. A registry-based study by Orlovska and colleagues<sup>1</sup> showed that the risk of BD increased by 28% following head injury. Their study also suggested that patients with BD often are initially diagnosed with another psychiatric disorder, which is consistent with our patient's case.

Pathologically, the effects of a TBI disrupt neurotransmitter systems, which negatively impacts mood.<sup>1</sup> Moreover, in one study in Taiwan, persons who had sustained a TBI after age 16

were more likely to develop BD, whereas those who had sustained a TBI before age 16 were more likely to develop depression.<sup>5</sup> The highest risk of developing a mood disorder was between 2 to 4 years after the TBI.<sup>5</sup>

Our patient's positive response to treatment suggests a positive prognosis, as long as he remains adherent to his medication regimen and scheduled office visits. His professional and personal life appears to have improved, as well, therefore representing a positive global response to treatment. His relationship with his ex-wife has benefited from treatment, increasing his support system beyond his parents with whom he lives. The patient has begun to socialize with other adults rather than remain secluded at home. He is active in his children's lives and volunteers in their school on occasion.

This case is an example of the importance of longitudinal follow-up to monitor for any signs of cycling mood, which may suggest a change in diagnosis and treatment plan. Also, based on the previously published literature, clinicians should be aware of premorbid and comorbid symptoms associated with BD. ■

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