

## Bipolar Disorder after Traumatic Brain Injury: Ethiopian Perspective

Elias Tesfaye<sup>1\*</sup> and Selamawit Alemayehu<sup>2</sup>

<sup>1</sup>Assistant Professor of Psychiatry, Jimma University, Ethiopia

<sup>2</sup>Resident Physician in Psychiatry Department, Jimma University, Ethiopia

**\*Corresponding Author:** Elias Tesfaye, Assistant Professor of Psychiatry, Jimma University, Ethiopia.

**Received:** March 30, 2019; **Published:** May 20, 2019

### Abstract

**Introduction:** Traumatic brain injury (TBI) can result in a variety of neuropsychiatric disturbances and may cause vulnerability to psychiatric disorders, with latency periods of over 10 years. We report the case of a 26-year-old man with no psychiatric antecedents who developed manic and aggressive behavior after traumatic brain injury (TBI).

**Methods and Results:** Findings on psychiatric evaluation encompassing detailed history and mental state examination, neuropsychological test battery, baseline laboratory examination and imaging exams suggested the presence of Bipolar I Disorder; Current manic Episode, Severe, with psychotic features due to TBI.

**Conclusions:** This case demonstrates that TBI may cause vulnerability to psychiatric disorders, with long latency periods, and that its course may be exacerbated with existing psychosocial stressor. Evidences suggest valproate as the primary pharmacological intervention in conjunction with cognitive and physical rehabilitation, and family and personal support.

**Keywords:** Bipolar Disorder; Mood Disorder; Traumatic Brain Injury; Management

### Abbreviations

CT scan: Computed Tomography Scan; ETB: Ethiopian Birr; EEG: Electroencephalogram; FAB: Neuropsychological Test Battery Scored; GCS: Glasgow Coma Scale; ICU: Intensive Care Unit; LFT: Liver Function Test; MMSE: Mini-Mental Status Examination; PTA: Posttraumatic Amnesia; RFT: Renal Function Test; TBI: Traumatic Brain Injury

### Introduction

Traumatic Brain injury and psychiatric manifestations are often seen and treated as two entirely separate diagnoses, or sometimes confused as being the same thing. However, both can be true; brain injury is sometimes an entirely separate issue to mental health, whereas other times brain injury can lead to behavioral disturbance after the trauma incident. It can also be the case patients might have previous psychiatric illness prior to the injury, and that the brain injury may exacerbate the pre-existing psychiatric manifestations [1]. There are therefore different ways in which the two conditions can overlap [1]. This overlap can occur because all cognitive, psychological, emotional and behavioral skills come from the brain, and both brain injury and psychiatric illness occur because of some dysfunction of the brain [1].

Traumatic brain injury (TBI) can result in a variety of neuropsychiatric disturbances, ranging from subtle to severe intellectual and emotional disturbances, and may cause vulnerability to psychiatric disorders, with latency periods of over 10 years [2,3].

Mood disorders are more common in patients who sustained TBIs than in patients with similar background characteristics who underwent similar levels of stress but without sustaining brain injury [3,4], which would suggest that neuropathological processes

associated with TBI constitute an important contributing factor to the development of mood disorders [3,5]. In addition to the changes in cognition, behavior, and personality described above, a significant body of evidence suggests that TBI results in an increased relative risk of developing various psychiatric disorders, including mood and anxiety disorders, substance abuse and psychotic syndromes [6]. Major depression is the most common psychiatric disorder after TBI, with rates varying from 14 to 77% [3,7]. Mania after TBI is less common than depression, but occurs more frequently than in the general population, and can be seen in about 9% of patients [3].

Several studies argue strongly that TBI acts as a gateway for the development of many psychiatric disorders [8]. The consistent observation that individuals who sustain a TBI have higher base rates of psychopathology prior to injury also suggests that there is a reciprocal interaction: psychopathology predisposes to TBI, and TBI in turn predisposes the individual to develop psychiatric disorders [8]. Positive family history of affective disorder and subcortical atrophy prior to TBI are also considered risk factors [3,9]. Comorbid alcohol use disorders also affect the apparent, but not actual, risk for bipolar disorder among persons with TBI [6].

We describe the case of a 26-year-old patient who presented with behavioral and mood symptoms after a severe head injury, and discuss the issues raised regarding the psychiatric manifestation, and the treatment implications for his illness.

### Case Report

HA is a 26-year-old male patient who is single. He came from Jimma. This is his second admission to psychiatric ward. He came to our hospital with his brother and father.

He had sustained motor bike accident 3 years back where he was unconscious for 1 month in ICU. As father reported, He had soft tissue trauma on his leg and hand. He also had brain surgery where they did elevation and evacuation as explained by the father. He was behaviorally well for 2 years working and doing his daily chores after the accident. He doesn't have any history of seizures, allergy and pain disorder. There is no history of allergies to medications. There is no reported history of Presence of psychiatric illness in family members or psychiatric treatment, suicidal attempt and history of substance abuse in the family.

He was functionally and behaviorally well for 2 years after the accident. His behavior started to change after 2 years from his discharge. In 2010 EC or 2018 GC, he started to forget stuff. He started to get annoyed towards his family without provocation. He further was aggressive towards his family verbally and physically. He always wants to make a fight. He also used to say that he has a power to finish many people. He also used to say he has a power to finish the whole country Ethiopia. Furthermore, he also has excessive explanation for question asked and he also shifted from one topic to other. He had sleep disturbance. For this he was having follow up in our hospital for 8 months. He was on sodium valproate 200 mg and diazepam 5 mg po at night. He discontinued his follow up before the onset of the illness. In addition, he didn't have any past history of suicidal thinking and attempt before the onset of the current illness. 2 years back he had fallen for a girl and wanted to take her hand in marriage. As his father reported he asked her hand in marriage for the family, but the family refused. He was devastated because of the response. He lost himself and gets angry at the situation. He used to say he should die instead of being refused and denied of his manly pride. After this incident his behavior started to change where he become easily annoyed and get angry easily.

### Current Presentation

This is a 26-year-old male patient who is known psychiatric patient for past 1year and on follow up.4 months back he stopped his medication because as his brother reported he said he will go to follow up alone and deceived his brother. He stopped the medication by himself. Afterwards he started to have sleep disturbance and he wakes up in middle of the night. At this time, he knocks doors of neighbors and orders them to get up to cut the khat plantation at the backyard. He also had urge to go out from home in the middle of the night. He sleeps on the average of 4 hour or less. As the father reported he also wanders around during the day. He usually goes to markets and disturbs people. If people aren't giving him attention or the willingness to hear him; He chases them away and beat them.

He claims that he has special power and he controls what people think. He also said that he can know about people by seeing. He further said that everyone loves him. He is special person not like the others or like me. He also said that even if he is chained that he can control what people can think and do. In addition, he claims that if I am beaten by anyone and I don't feel any pain because i have special capacity that anyone doesn't have.

Furthermore, he has urge to chase or interact females and try to take their clothes. He also has extravagant behavior where he took 70,000 ETB and wasted it by giving money to anyone he gets. He buys stuff for everyone he gets on the street. Things started to worsen for the past 2 months where he destructed the khat plantation at the back yard. In addition, also throws food at his mom when given food. He further calls them that they are "dogs". He verbalizes that he hates his father and mother. He has homicidal intent towards family members to the extent he verbally and physically threatens to kill his father.

He doesn't take care of himself as he used to. He doesn't change his clothes or take shower as frequently as he used to. In addition, he doesn't cut his nails. Otherwise He doesn't have history of flash back, night mares of the trauma, passivity phenomena, self-harming and seizure.

Routine laboratory tests including thyroid hormones, LFT, RFT and thyroid-stimulating hormone were within normal range. The first cranial computed tomography (CT) scan after the accident and current CT was not available.

Neuropsychological evaluation at admission at psychiatric ward showed psychomotor agitation but had no mannerism, no tics and no tremors. The quantity of speech was talkative, rapid rate with loud volume. He expresses his Mood "happy" claiming that he is rich. The general quality of affect was euphoric and depth of the affect was euphoric, appropriate. There was noted flight of ideas, Delusion of Grandiosity and ideas of influence. He had Suicidal/Homicidal Ideation. He is alert and oriented to place, person but not for date. Fund of knowledge and abstract knowledge was intact. He had Level 1 insight i.e. (Complete denial of illness). Social and tested judgment was poor.

Overall mini-mental status examination scored 22/30 showing mild neurocognitive derangement (See table 2). Neuropsychological test battery scored 10/18 showing significant executive dysfunction (See table 1).

FAB Items	Score out of 3
Similarities (conceptualization)	2
Lexical fluency (mental flexibility)	0
Motor series (luria test)	3
Conflicting instruction	1
Go-No-GO (inhibitory control)	1
Prehension behavior (environmental autonomy)	3
Overall	10/18

Table 1: FAB.

N.B. FAB less than 12 suggests frontal lobe dysfunction.

Category MMSE	Score
Orientation to time	0
Orientation to place	5
registration	3
Attention and calculation	5
recall	3
Language	2
repetition	1
Complex command	3
overall	22/30

Table 2: MMSE.

N.B. MMSE less than 24 suggest cognitive disorder.

### Physical examination

**Current VS:** BP 100/70, PR 76, RR 18, T ATT, he has more than 6 cm long visible old scar on the right side of his forehead.

### Course of the illness and management

4 months back he stopped his medication. At which time he started to have loss of sleep, irritability, excessive talking, excessive singing and dancing, excessive happiness and laughing alone, grandiose delusion, elated behaviors and aggression towards the family member. For this a diagnosis of Bipolar I Disorder; Current manic Episode, Severe, with psychotic features due to TBI was made and he was started on sodium valproate (800 mg in divided dose), haloperidol (2 mg/day) with PRN IV diazepam. After 1 week; he developed drug adverse effect. For this reason, tranquilization was hold and the medications were changed to resperidone 1 mg per day and the dose of sodium valproate increased to 1200 mg in divided dose. In the mean time his symptoms started to subside within 3 weeks. During this time low dose of the drugs were maintained with close follow up. Finally, He was discharged after 41 days stay in our hospital with improvement with sodium valproate 1200 mg in divided dose and resperidone 1 mg at bed time.

### Discussion

The patient have suffered severe TBI 3 year back after sustaining motor vehicle accident; justified by being unconscious for more than 1 month during his stay in ICU. Despite this, we failed to secure his previous medical record to determine his initial score on Glasgow Coma Scale during his stay in ICU. The diagnosis of bipolar disorder due to head injury was supported by the evidence that the patient developed psychiatric symptoms 2 years after the head injury and He has no family or pervious personal history of mental illness.

The absence of any behavioral disturbance 2 years after the accident which can suggest doubt to the diagnosis of the bipolar disorder due to TBI. Furthermore, the presence of stressor 1 year after the accident might have fastened the behavioral changes. However, longer latency periods after trauma have been associated with psychiatric manifestation as reported in the literatures [2]. Since TBI can cause permanent vulnerability to psychiatric disorders, contributing substantially to long-term disability [3] and quality of life [3], psychiatric evaluation and (long-term) monitoring should be included in the routine follow-up of TBI.

The evaluation of persons with TBI and suspected bipolar spectrum disorders follows the general principles and components of a complete psychiatric evaluation as outlined in the American Psychiatric Association's Practice Guideline for Psychiatric Evaluation of Adults [6]. Physical examination, including vital signs and a complete neurological exam, is a requisite element of the initial evaluation.

As mentioned above, structural neuroimaging is a useful component of the evaluation of persons with TBI generally. However, more recent and refined neuroimaging techniques should be reserved for research at the present time. Video-EEG monitoring and 24-hour ambulatory recordings may be useful in the differential diagnosis of patients presenting with paroxysmal behavioral disturbances of unclear etiology or those that are associated with intra- or post-episode alterations of consciousness [6]. This is particularly relevant to the evaluation of persons with TBI and mixed affective episodes in light of the possible associations between such disorders and posttraumatic epilepsy [6]. Otherwise, neurophysiologic studies are not presently regarded as useful elements of the clinical evaluation in this context [6].

If the clinical history or examination suggest other endocrine or concurrent physical conditions, then performing problem-focused laboratory studies is appropriate [10]. In light of the relatively high frequency of neuroendocrine abnormalities in this population [11] screening for thyroid dysfunction is encouraged as part of the initial evaluation. As with the evaluation of persons with depression, screening for human immunodeficiency virus infection as well as performing urine and/or serum toxicology screening for alcohol and other substances of abuse is encouraged [6].

The literature describing pharmacotherapy agents used to treat idiopathic manic and mixed mood states are used to treat bipolar spectrum disorders among persons with TBI. Clinicians are encouraged to refer to each medication's product information sheet as well as other reference materials for complete reviews of dosing, side effects, drug-drug interactions, treatment risks, and treatment contraindications before prescribing these or any other medications [6].

Valproate may exacerbate cognitive impairments in some persons with TBI, but it appears less likely to do so than either carbamazepine or lithium [12]. Nonetheless, use of any of these agents necessitates careful and continuous assessment for the development of treatment-related motor (e.g. tremor, ataxia, gait disturbances) and cognitive impairments as well as other adverse side effects (e.g. weight gain, gastrointestinal problems, hematologic abnormalities, hepatotoxicity, alopecia, etc). Additionally, the risk of polycystic ovarian syndrome requires consideration of alternate treatments in females.

Given that lithium carbonate is used often as a first-line treatment among persons with idiopathic bipolar disorder, it merits special comment as a treatment of mixed states among persons with TBI. Intolerance of lithium carbonate appears to be more common among persons with TBI than with primary mania or mixed mood episodes [6]. This intolerance is often attributable to the adverse cognitive and motor effects of lithium carbonate, which appears more likely to produce nausea, tremor, ataxia, and lethargy in persons with neurological disorders than in the general psychiatric population [6]. Additionally, lithium carbonate lowers seizure threshold; in light of the risk for posttraumatic epilepsy as well as the potential comorbidity between posttraumatic epilepsy and mania, this effect is concerning with respect to lithium's use in this population. As such, partial response, relapse of symptoms, or need for a second mood-stabilizing medication are common limitations of the use of this agent among TBI patients [6].

Several of the newer anticonvulsants (e.g. lamotrigine, oxcarbazepine) and the atypical antipsychotics (e.g. risperidone, olanzapine, ziprasidone, aripiprazole, etc.) may be useful in the treatment of posttraumatic manic, hypomanic and mixed states, but there are few published reports of their use. Clinicians interested in using these agents for this purpose are advised to undertake such treatments cautiously and with careful monitoring for adverse neurological, cognitive, motor, cardiac, and metabolic side effects [6].

In the absence of evidence demonstrating the superiority of one of these agents over the others, we generally recommend either valproate or quetiapine as first-line treatments given their effectiveness for acute mania, rapid-cycling bipolar disorder, and anti-manic prophylaxis as well as their reasonable tolerability in persons with TBI with minimal side effects [6,13,14].

The TBI literature provides no clear guidance regarding the psychotherapeutic approach to persons with mania or mixed mood states after TBI. However, Education and supportive interventions regarding both TBI and the mood disturbance with which the patient presents are reasonable. And the goal of therapy should focus on helping the patient to accepting his fate, to encourage the patient on the benefit of adherence to his medication to control his behavior, to engage in physical and cognitive rehabilitation and to educate the family about his disability and how they can help the client to maximize his recovery. Additional psychotherapeutic interventions are modeled after those used in the management of persons with idiopathic bipolar disorders, as described in the American Psychiatric Association's practice guidelines for the treatment of patients with bipolar disorder [6,13,14].

### Conclusion

Traumatic brain injury (TBI) may cause vulnerability to psychiatric disorders even with latency periods of over 10 years. The condition is further exacerbated on those who have previous psychiatric illness and those with psychosocial stressor. Bipolar disorder is a rare outcome after TBI, when compared to major depression which is the most common disorder. In treatment of bipolar disorder after brain injury; it should include psychopharmacological management, namely valproate and low dose atypical antipsychotics as the primary pharmacological intervention with high emphasis on minimizing drug side effects. In addition, giving cognitive and physical rehabilitation, family and personal support will be crucial.

## Acknowledgements

I would like to thank all the people who contributed in one way or the other to the work described in this case report. First and foremost, I would like to thank my professor Dr. Elias giving me an assignment to clerk this rare case. Secondly, James Peter Akabawi a colleagues who give me a constructive comments and recommended me to submit for publication.

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## Volume 3 Issue 6 June 2019

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