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Post-Traumatic Bipolar Disorder: A Case Report

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Abstract | **Case Report**

Background: Traumatic brain injury (TBI) is increasingly recognized as a profound risk factor for neuropsychiatric sequelae, including secondary mood disorders. Post-traumatic bipolar disorder (PTBD) represents a complex phenotype characterized by unique structural pathology, high rates of treatment non-adherence, comorbid substance use disorders, and distinct management challenges. ***Case Presentation:*** We report the case of Mr. B.K., a 34-year-old unemployed male with a history of a severe TBI in 2015 requiring neurosurgical evacuation of an extradural hematoma. Two weeks post-intensive care unit discharge, he developed his first characterized manic episode. Over the subsequent decade, he experienced multiple recurrent manic episodes with psychotic features, consistently precipitated by poor treatment adherence. His most recent admission was triggered by severe psychomotor agitation, verbal and physical hetero-aggressiveness, grandiose delusions, disinhibited pedophilic impulses, and an addictive spiral involving cannabis, alcohol, tobacco, and alprazolam misuse. Structural brain magnetic resonance imaging (MRI) revealed extensive sequelae of cerebral contusions, focal necrosis/gliosis within the basomedial temporal cortex, and decreased fractional anisotropy in the uncinate fasciculus. The patient was successfully stabilized on a combination of quetiapine (600 mg/day) and sodium valproate (1000 mg/day), leading to full resolution of behavioral, delusional, and deviant sexual symptoms over a six-week period. ***Conclusion:*** This case underscores the pathophysiological role of ventromedial limbic networks in the genesis of secondary bipolar phenotypes and highlights the clinical necessity of maintaining aggressive, multimodal psychopharmacological adherence to mitigate high-risk behavioral decompensation.

Keywords: Traumatic brain injury; post-traumatic bipolar disorder; Uncinate fasciculus; Basomedial temporal cortex; Secondary mania; Case report.

INTRODUCTION

Traumatic brain injury (TBI) represents a major global public health crisis, contributing significantly to long-term disability and permanent neurobehavioral deficits [1]. Beyond immediate neurological impairments, TBI serves as a potent catalyst for late-onset psychiatric morbidity. Chronic neuroinflammation, diffuse axonal injury (DAI), and focal cortical contusions have been implicated in the pathogenesis of structural and functional circuit disruptions that mirror primary psychiatric conditions [2].

Among these post-traumatic sequelae, the development of a secondary bipolar phenotype frequently termed Post-Traumatic Bipolar Disorder (PTBD) or mood disorder due to a general medical condition with manic features is a relatively rare but profoundly debilitating consequence [3]. The clinical onset of mania following severe TBI typically implicates localized damage to orbitofrontal, ventromedial prefrontal, and basomedial temporal cortices, as well as the white matter tracts interconnecting these limbic regions [4].

Managing PTBD is exceptionally complex due to frequent comorbid substance use disorders, heightened impulsivity, cognitive deficits that impair insight, and secondary behavioral regularities such as severe treatment non-adherence [5]. In this report, we present the detailed longitudinal case of a 34-year-old male who developed severe, recurrent psychotic mania characterized by dangerous behavioral disinhibition following a critical neurosurgical TBI, and discuss the specific neuroanatomical networks mediating this presentation.

CASE PRESENTATION

History and Sociodemographic Profile

Mr. B.K., a 34-year-old divorced male and hairdresser by trade (currently unemployed), was

admitted under emergency protocols to our university psychiatric department. The primary indication for admission was severe psychomotor agitation associated with intense verbal and physical hetero-aggressiveness directed toward immediate family members.

Medical and Psychiatric History

- **Neurosurgical Trauma (2015):** The patient sustained a severe, life-threatening TBI secondary to a high-velocity motor vehicle accident (motorcycle). Acute neurosurgical intervention required the immediate evacuation of an extradural hematoma. Postoperatively, he required a 20-day stay in the intensive care unit (ICU) for management of a prolonged comatose state.
- **Psychiatric Evolution & Continuity of Care (2015–2019):**
  - *2015 (Rabat):* Two weeks following his discharge from the ICU, the patient developed his index characterized manic episode. He achieved clinical stabilization using a combination of olanzapine (10 mg/day) and sodium valproate (1500 mg/day).
  - *2017–2018 (Marrakech):* The clinical course was marked by complete treatment non-adherence. Frequent, unapproved discontinuations of both olanzapine and valproate precipitated two consecutive psychiatric hospitalizations for severe recurrent manic episodes.
  - *2018–2019:* The patient required two additional admissions to our university hospital structure due to acute manic episodes with marked psychotic features. In an effort to counter chronic non-adherence, his maintenance regimen was optimized via the introduction of a first-generation long-acting injectable (LAI) neuroleptic (pipotiazine 75 mg intramuscularly every four weeks), maintained alongside oral olanzapine (10 mg/day) and sodium valproate (1500 mg/day).

History of Present Illness and Current Episode

The current psychiatric decompensation began two months prior to the index admission, developing insidiously following a self-initiated, total cessation of all psychiatric medications lasting several months. The initial presentation evolved to include severe psychomotor instability, unremitting logorrhea, intense irritability, reckless pathological spending, a drastic reduction in sleep duration without subjective fatigue, and erratic, unconstructive hyperactivity driven by grandiose and unrealistic projects.

Concurrently, the patient experienced a profound escalation in addictive behaviors, including increased tobacco consumption, daily heavy cannabis and alcohol use, and the erratic misuse of unprescribed alprazolam, which he claimed was an attempt to self-medicate his escalating anxiety and intractable insomnia. This substance use pattern rapidly exhausted his financial resources. Following his family's refusal to provide further monetary funds, the patient underwent acute behavioral decompensation, exhibiting dangerous hetero-aggressiveness that necessitated emergency intervention and involuntary transport by his father and sister.

Clinical and Paraclinical Assessment at Admission

Upon presentation, the psychiatric examination revealed a disheveled, unkempt male demonstrating severe psychomotor agitation and continuous, pressured logorrhea. Formal thought process evaluation revealed marked tachypsychia with flight of ideas, alongside complex grandiose and omnipotent delusions. His affect was deeply irritable, hyperthymic, and accompanied by free-floating anxiety. Judgment was critically impaired.

Notably, the patient exhibited severe sexual behavior disinhibition characterized by pedophilic impulses and tendencies, which he verbalized in a highly disinhibited, unconcerned manner. Somatic evaluation confirmed total insomnia without subjective exhaustion, and marked anorexia.

Diagnostics & Imaging

- **Neuroimaging:** A brain structural magnetic resonance imaging (MRI) scan performed at admission demonstrated prominent chronic sequelae of prior cerebral contusions. Prominent areas of focal necrosis and gliosis were visualized via hyperintense Fluid-Attenuated Inversion Recovery (FLAIR) signals within the basomedial temporal cortex. Furthermore, diffusion tensor imaging sequences revealed a significant decrease in fractional anisotropy (FA) within the uncinate fasciculus.
- **Laboratory Panels:** A comprehensive metabolic and infectious workup was conducted. Complete blood count (CBC), serum electrolytes, renal function (BUN/creatinine), liver function tests (AST/ALT/bilirubin), lipid panel, C-reactive protein (CRP), thyroid-stimulating hormone (TSH), and infectious serologies (syphilis, HBV, HCV, and HIV) were all within normal physiological parameters.
- **Toxicology:** Urine toxicology screening was explicitly positive for cannabinoids and benzodiazepines, corroborating his recent substance misuse history.

Therapeutic Management and Outcome

Due to the severity of the psychomotor agitation and hetero-aggressiveness, the patient was placed under close, continuous psychiatric observation. He was initiated on an integrated, high-dose atypical antipsychotic and mood stabilizer regimen consisting of quetiapine (gradually titrated over several days to a target dose of 600 mg/day) and sodium valproate (optimized to 1000 mg/day).

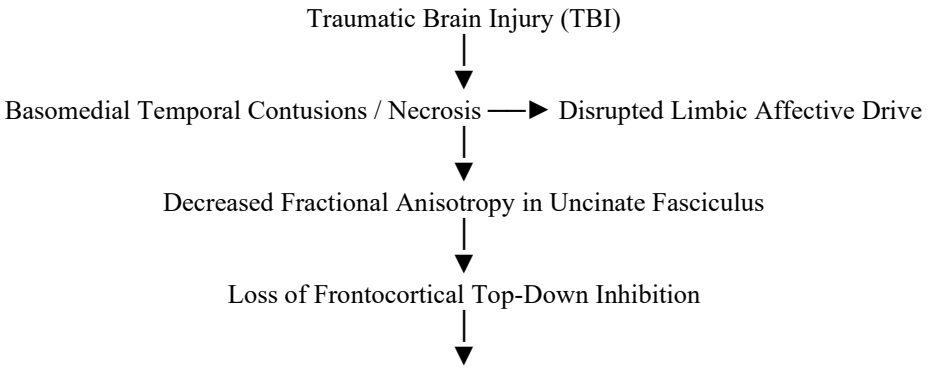
The clinical response to this pharmacological combination was highly favorable. Over the course of the six-week hospitalization, we observed a progressive sedation of psychomotor agitation, stabilization of core hyperthymic mood parameters, complete resolution of the grandiose delusions, and absolute cessation of the deviant sexual impulses. Normal sleep architecture and appetite were successfully restored. At the time of discharge, the patient demonstrated full insight into the nature of his neuropsychiatric illness, his prior non-adherence, and the dangerous aggressive behaviors that precipitated his emergency admission.

DISCUSSION

This case illustrates a profound clinical presentation of Post-Traumatic Bipolar Disorder (PTBD) occurring downstream of a severe neurosurgical TBI. The close temporal proximity between the patient's discharge from the ICU and his index manic episode (two weeks) strongly supports a secondary, organically driven structural etiology rather than a coincidental primary psychiatric disorder [6]. While primary bipolar I disorder typically manifests in late adolescence or early adulthood, secondary post-traumatic mania characteristically presents later in life, directly following structural neurological insults, and frequently features atypical elements such as profound executive dysfunction, high-risk behavioral disinhibition, and rapid cycling [7].

The neuroanatomical findings in this patient provide striking physical evidence of the structural substrate underlying post-traumatic mania. The presence of focal necrosis and gliosis within the basomedial temporal cortex highlights direct structural damage to core limbic structures responsible for emotional regulation, impulse control, and affect modulation [8].

Of particular pathophysiological relevance is the documented decrease in fractional anisotropy within the uncinate fasciculus. The uncinate fasciculus is a critical white matter tract that structurally couples the amygdala and anterior temporal lobe with the orbitofrontal and ventromedial prefrontal cortices [9]. Disruptions in this pathway as quantified by reduced fractional anisotropy reflecting loss of microstructural white matter integrity uncouple frontocortical top-down inhibition from subcortical limbic drive [10]. This structural disconnection syndrome explains the emergence of the patient's severe tachypsychia, flight of ideas, grandiose delusions, and the profound behavioral disinhibition that culminated in pedophilic impulses.



Furthermore, this case illustrates the bidirectional relationship between TBI and substance use disorders. Patients with structural frontobasal damage frequently exhibit high novelty seeking and diminished risk aversion, predisposing them to an addictive spiral [11]. Mr. B.K.'s escalating abuse of cannabis, alcohol, and alprazolam represents both a manifestation of damaged prefrontal inhibitory controls and a maladaptive attempt to self-medicate underlying anxiety and structural insomnia. This poly-substance abuse subsequently undermined medication adherence, lowering the seizure and psychiatric threshold, and resulting in severe hetero-aggressiveness when financial barriers impeded drug acquisition [12].

From a therapeutic perspective, the patient's long-term instability underscores the absolute failure of basic oral regimens due to poor insight and chronic non-adherence, which previously required the introduction of long-acting injectable (LAI) neuroleptics. In the acute stabilization of this index episode, the combination of quetiapine (600 mg/day) and sodium valproate (1000 mg/day) proved highly efficacious.

Quetiapine's potent antagonistic activity at dopamine D2 and serotonin 5-HT2A receptors, combined with its active metabolite norquetiapine's norepinephrine reuptake inhibition, provides simultaneous management of acute psychotic agitation, irritable hyperthymia, and severe insomnia [13]. Sodium valproate complements this by augmenting γ-aminobutyric acid (GABA)-ergic neurotransmission and downregulating protein kinase C pathways, stabilizing post-traumatic cortical hyperexcitability secondary to prior tissue gliosis [14].

CONCLUSION

Post-Traumatic Bipolar Disorder is a severe neuropsychiatric condition rooted in the structural disruption of frontotemporal and ventromedial limbic networks, prominently mediated by white matter degradation in pathways such as the uncinate fasciculus. As demonstrated by Mr. B.K., these structural lesions frequently yield highly volatile phenotypes characterized by psychotic mania, profound behavioral disinhibition, and severe substance use comorbidities.

Successful long-term management requires a proactive clinical approach that pairs comprehensive neuroimaging (such as diffusion tensor imaging) with assertive, compliance-focused psychopharmacological strategies to prevent dangerous behavioral decompensation and optimize functional recovery.

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