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Mania As Sequelae of Traumatic Brain Injury- A Case Report

KEYWORDS: Traumatic brain injury (TBI), bipolar mood disorders, cognitive impairment

Dr. Asim Musa	Dr. Nisheet Patel	Dr. N.C.Parikh
Department of Psychiatry ,Smt. N.H.L Municipal Medical College and Sheth V.S. General Hospital, Ahmedabad	Department of Psychiatry ,Smt. N.H.L Municipal Medical College and Sheth V.S. General Hospital, Ahmedabad	Department of Psychiatry ,Smt. N.H.L Municipal Medical College and Sheth V.S. General Hospital, Ahmedabad

ABSTRACT

Abstract: *The neurobehavioral sequelae of traumatic brain injury consist of a spectrum of neurological (motor, sensory, cognitive) as well as neuropsychiatric manifestations.*

Objective: *We report a case of a 32-year-old female without psychiatric antecedents who developed manic episode after traumatic brain injury (TBI).*

Methods and results: *Findings on neurobehavioral examination, neuropsychological test, electrophysiological and imaging exams suggested the presence of a diffuse cerebral injury with a predominance of right parietal-temporal findings.*

Conclusions: *This case demonstrates that TBI may cause vulnerability to psychiatric disorders, with long latency periods, and that its course may be independent of cognitive impairment and recovery.*

Introduction

Traumatic brain injury (TBI) may predispose an individual to psychiatric disorders, with latency periods of over 10 years. Almost half of the people having history of traumatic brain injury (TBI) may later be presented with psychiatric disorders [1]. Psychiatric symptoms arising immediately after TBI may be etiologically related to the neurophysiological effects of the injury, as supported by an early relationship between TBI severity and psychiatric risk. Of all psychiatric disorders, mood and anxiety spectrum disorders increase the most following traumatic brain injury. Relapse of pre-existing affective or anxiety disorders is more likely, but de-novo mood and anxiety disorders arise with greater frequency after head injuries than in a comparative sample of the uninjured general population[7]Major depression is the most common psychiatric disorder after TBI, with rates varying from 14 to 77% [2,3,4,5]. Mania following TBI is less common than depression, but TBI is known to predispose a patient to have manic episodes by more than 9%, which is more than general population [6]. Patients with post-TBI related mania typically present with sleeplessness, grandiosity, impaired judgment, irritability, pressured speech, hyperactivity, and hypersexual behaviour. The majority of manic cases develop after at least 1 year post-TBI, and some studies have reported a delay of 4 to 5 years post-TBI [9] The confluence of either anterior sub-cortical atrophy and a focal lesion of a limbic or limbic-connected region of the right hemisphere, or genetic loading and a limbic-connected right hemisphere lesion may account for mania after TBI. Almost 50% of patients having secondary manic episode following TBI have abnormal electroencephalogram (EEG). [7]

CASE STUDY

Mrs R is 32 year old female right handed Hindu patient who studied up to 7th grade and worked as a labourer on daily basis on previously without significant past psychiatry illness. Her family history was negative from psychiatry side. She met with a road traffic accident and suffered traumatic brain injuries, which lead to loss of consciousness with bleeding discharge from right ear, followed by an epileptic seizure episode of tonic clonic movement. Routine investigations including thyroid function tests were normal. First cranial computer tomography (CT scan) showed Subdural haemorrhage over right tempoparietal region with compression to right lateral and 3rd ventricle. Neurological assessment reveals cognitive deficit and post traumatic amnesia. Patient had 4 seizure episodes in subsequent 24 hours. Loading dose of phenytoin with maintenance on 300 mg of oral phenytoin controlled epileptic seizure episodes. Subsequent neurological examination shows memory deficits in learning, short and long-term memory; problem-solving deficits. Subsequent CT scan brain shows hypo density in right tempoparietal region suggestive of old residual gliosis lesion.EEG appears to reveal slow theta wave but exact details of neurological assessment details at time of TBI were not available.

6 month later, she admitted in patient psychiatry department with behaviour abnormality as over talkativeness, irritability, hyper religious and hypersexual behaviour, aggression, pressure of speech. Psychotic features were also noted as overvalued ideas of persecution over nearby relatives. Young mania rating scale was 43 out of 60 suggestive of severe mania. Sodium valproate was introduced with gradual removal of phenytoin. Sodium valproate was given 1000 mg in divided doses. Olanzapine was started to control psychotic symptoms and lorazepam was given 4 mg to maintain sleep and restlessness. 2 weeks later she was discharge with 1000 mg sodium valproate, 10 mg olanzapine, 2 mg of lorazepam. She achieved baseline mood level within 8 weeks of treatment with YMRS score 8. On subsequent follow-ups, she was maintained on 1000 mg sodium valproate only with gradual tapering of olanzapine and lorazepam depending upon presented symptoms. Subsequent next 4 months she continues sodium valproate with baseline mood. However, after that she consults psychiatry sporadically with irregular adherence to the treatment.

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Discussion

Patient's initial psychological and neurological assessment was not feasible at time of traumatic brain injury. Traumatic brain injury was classified as moderate level traumatic brain injury by definition although initial Glasgow coma scale and initial medical management and complete neurological assessment were not available to asses. [10]. Latency period in this patient was supported by literature as well as case reports of psychiatry manifestation with latency period of 10 year after traumatic brain injury [1].

In our study patient did not have any past significant positive psychiatry morbidity. Family predisposition was ruled out from history. History also suggested no recent or past substance abuse. Social and environmental factors were also against psychiatry manifestation. Studies suggested that premorbid psychiatry condition and positive family history is strongly considered for posttraumatic brain injury psychiatry manifestation, which is contrast to present study [11, 12]

The possible mechanisms involved in the dysfunctions found in our patient, not exclusively explained by the location of the lesion, are possibly due to widespread axonal tearing and diffuse axonal injury caused by acceleration and deceleration forces, which is also supported by various literatures. [11]

Patients with post-TBI bipolar disorder show predominantly sub cortical lesions (right head of caudate and right thalamus), while patients with post-TBI unipolar mania more often show cortical involvement (mainly right orbitofrontal and basotemporal cortices) [11, 13], suggesting that sub cortical and cortical right hemisphere lesions may produce different neurochemical or remote metabolic brain changes that may be the underlying cause of either a bipolar mania.[13]Our patient's CT scans and EEGs revealed a lesion on the temporal-parietal area of the right cerebral hemisphere. The neuropsychological evaluation revealed deficits in the same areas affected by the lesion, but also changes suggesting dysfunction in other areas, such as the right parietal cortex, right frontal dorsolateral.

Cognitive impairment is often diffuse with more prominent deficits in the rate of information processing, attention span, memory, cognitive flexibility, problem solving, impulsiveness, affective instability, and disinhibition. These symptoms are frequently observed characterizing the changes in TBI patients as seen in our patient. [14]Cognitive impairment is our patient can be explained by diffuse axonal injury as well as co morbid bipolar mood symptoms. The lack of strong evidence for cognitive impairment prior to illness onset in bipolar disorder appears to be inconsistent with classically defined neurodegenerative illness causation. [15]

Treatment should include cognitive and physical rehabilitation, family and personal support, and psychopharmacological management of mood and other behavioural syndromes.[16]Education, non-pharmacologic interventions, and some symptom-targeted pharmacotherapies, as well as encouraging patience during the time required for spontaneous recovery after mild to moderate TBI may afford substantial reductions in post TBI symptoms and improvements in everyday function.[17]There is limited evidence in the literature about specific treatments for mania post-TBI.

Mood-stabilizing antiepileptic drugs have proved effective, in particular valproate and carbamazepine. Atypical antipsychotics are another treatment option; especially in patients who have manic symptoms accompanied by psychotic features. Lithium carbonate should be avoided as it lowers the seizure threshold, may worsen cognitive impairment and has a low therapeutic index. Although the exact mechanisms of action for valproate have not yet been determined, postulated theories include an ''antikindling'' Effects of sodium valproate in the limbic system on emotion, cognition, and behaviour and an enhancement of GABAergic-mediated inhibitory control, as well as action as a general CNS stabiliser is making it a primary pharmacological intervention for the treatment of neuropsychiatric symptoms after brain injury. [18]

To enhance quality of life and future perspective of social as well as occupational rehabilitation of patients having psychiatry co morbidity following traumatic brain injury there is a basic need of guideline of psychological as well as pharmacological management of patients. [20]

Conclusion

The key to the diagnosis and management of post-TBI complaints is to avoid premature closure on a diagnosis, to coordinate care through a multidisciplinary team, and to involve the patient and his or her family in decision making. The incidence of TBI is increasing worldwide, and this is creating an urgency to delineate best practice strategies needed to improve outcomes.

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