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CASE REPORT

Bipolar disorder after traumatic brain injury

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Abstract
Objective. We report the case of a 47-year-old man with no psychiatric antecedents who developed manic and depressive symptoms after traumatic brain injury (TBI). *Methods and results*. Findings on neurobehavioral examination, neuropsychological test battery, electrophysiological and imaging exams suggested the presence of a diffuse cerebral injury with a predominance of left fronto-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.

Key Words: *Bipolar disorder, cognitive dysfunction, traumatic brain injury*

Introduction

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 [1].

These include problems with attention and arousal, concentration, executive function, intellectual changes, memory impairment, personality changes, affective and anxiety disorders, psychosis, sleep disorders, aggression and irritability [2].

Despite the emphasis placed on physical deficits shortly after severe brain injury, it is cognitive and behavioural deficits that give rise to the major morbidity that most impairs the capacity to return to work and maintain social activities [3], contributing to long-term disability [4] and compromising the quality of life [5].

TBI is the result of mechanical forces on the skull and transmitted to the brain leading to focal and/or diffuse brain damage, as well as secondary effects (cerebral oedema, hydrocephalus, increased intracranial pressure, infection, hypoxia, neurotoxicity, and others) [2]. The differential motion of the brain within the skull also causes shearing and stretching of the axons [6], with injuries ranging from brief physiological disruption to widespread axonal tearing, called diffuse axonal injury [7]. Delayed effects include the release of excitatory amino acids, oxidative free-radical production, the release of arachidonic acid metabolites, and disruption of neurotransmitters like monoamines and serotonin [8–10].

Mood disorders are more frequent in patients with sustained TBIs than in patients with similar background characteristics who underwent similar levels of stress but without sustaining brain injury [11], which would suggest that neuropathological processes associated with TBI constitute an important contributing factor to the development of mood disorders [12].

Major depression is the most common psychiatric disorder after TBI, with rates varying from 14 to 77% [1,4,13,14]. 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 [15]. Positive family history of affective disorder and subcortical atrophy prior to TBI are also considered risk factors [16]. Rates for post-TBI bipolar disorder have ranged from 1.7 to 17% [1,17], some authors stating that post-TBI bipolar disorder appears only in individuals with a previous history of axis I psychopathology, usually remaining chronic [17], but such data have come primarily from case reports.

We describe the case of a 47-year-old patient who presented with behavioural and mood symptoms after a severe head injury, and discuss the issues

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raised regarding the physiopathological mechanisms, and the treatment implications for his illness.

Case report

JPF is a 47-year-old divorced, right-handed, male patient who was educated up to 6th grade and who had previously worked in security. He had antecedents of operated mitral cardiopathy, auricular fibrillation and HTA, and a history of tobacco use and alcohol abuse. According to the patient and his ex-wife, there were no personal or family histories of psychiatric disorders. He suffered a traffic road accident, with polytraumatisms and TBI, after which he remained in a coma for 4 days. Subdural haematoma drainage was carried out and he was medicated with phenytoin and corticoids. Routine laboratory tests including thyroid hormones and thyroid-stimulating hormone were within normal range. The first cranial computed tomography (CT) scan revealed left temporo-parietal craniotomy, and haematoma substitution by hypodensity in the left cortico-parietal area of the left cerebral hemisphere. Five months later the CT scan revealed no anomalies. The EEG revealed incidence of slow waves (theta and delta) over the left fronto-temporal region. Short latency auditory evoked potentials showed no significant changes and the cognitive evoked potentials (P300 wave) were compatible with cognitive dysfunction.

Neuropsychological evaluation 5 months post-TBI revealed attentional deficit (sustained, divided and selected), mental tracking deficit, slowing of information processing speed, verbal fluency deficit (semantic subtype), anomic specking and dysarthria, impaired repetition, reading and writing deficits (semantic alexia and phonological alexia), secondary dyscalculia, ideokinetic apraxia, melokinetic apraxia, ideomotor apraxia, writing apraxia and constructional dyspraxia, memory deficits in learning, short- and long-term memory; problem-solving deficits, planning deficit, and flexibility capacity impairment. Although he started neuropsychological rehabilitation, he has not been able to return to work.

A year and a half after the accident he was referred for inpatient psychiatric treatment because of behavioural symptoms, namely, disinhibition, pressure of speech, aggressive behaviour and irritability, but with no psychotic symptoms. Corticoids were slowly withdrawn and he was discharged a week later with the diagnosis of post-TBI organic personality disorder and was medicated with gabapentin (1600 mg/day), haloperidol (2 mg/day), quetiapine (200 mg/day), propanolol, digoxin, lisinopril, spironolactone, furosemide and citicoline.

The following month he started complaining of depressive symptomatology, with sadness, apathy, abulia, anedonia and suicidal ideation, being medicated with escitalopram (20 mg/day) and lamotrigine (100 mg/day). A month later he became dysphoric, with pressured speech, coprolalia, excessive spending and psychotic symptoms (delusions of persecution and jealousy), but without hallucinations or significant changes in sleep patterns. Escitalopram was suspended, and haloperidol was titrated to 10 mg/day. His symptoms abated after 2–3 weeks, but by the fourth week he started complaining again of depressive symptoms; escitalopram was re-prescribed, and a dose of 1200 mg/day of carbamazepine was added. A week later he was again disinhibited and slightly euphoric, with no psychotic symptoms.

The alternation of manic and depressive symptoms led to the diagnosis of post-TBI bipolar disorder; however, the patient has attended psychiatry consultation only sporadically, with irregular treatment adherence.

Discussion

The patient can be considered to have suffered severe TBI, even though some medical notes were unavailable for scrutiny, such as his initial score on the GCS (Glasgow Coma Scale), and the length of PTA (posttraumatic amnesia) which created difficulty in assessing the severity of the injury. The long latency period might have caused some of the uncertainty around whether the bipolar disorder was due to TBI, although longer latency periods have been reported in the literature [1,18].

Although some authors consider that post-TBI bipolar disorder appears only in individuals with previous personal [17] or family [16] history of axis I psychopathology [17], in contrast to the findings of other researchers, besides alcohol abuse we found no evidence of other personal or family histories of psychiatric disorders [19,20].

Lishman [21] hypothesises that psychiatric symptoms following a head injury are precipitated initially by organic factors, but in some patients are maintained by socioeconomic and psychosocial factors that can predict cognitive dysfunction after TBI [22]. It is well documented that patients with TBI are often young men who come from lower socioeconomic backgrounds, who tend to misuse alcohol and drugs, and possess certain premorbid personality traits. However, some authors have not found enough evidence to suggest that persons who sustain mild TBI show substantially different premorbid personality to that of their peers [23]. Even though it was not possible to adequately assess the level of our patient's premorbid personality, emotional problems are usually exacerbated after injury [2]. Alcohol consumption is considered a predisposing factor to head injury [4], and may continue afterwards, delaying the reparative process within the central nervous system [21].

Although corticoid treatment has been considered as one cause of the appearance of mood symptoms [24], this does not explain our patient's symptoms, since they persisted even after corticoids were withdrawn.

Patients with post-TBI bipolar disorder show predominantly subcortical 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) [15,16,25,26], suggesting that subcortical and cortical right hemisphere lesions may produce different neurochemical and/or remote metabolic brain changes that may be the underlying cause of either a bipolar disease or a unipolar mania. Our patient's CT scans and EEGs revealed a lesion on the temporo-parietal area of the left cerebral hemisphere. The neuropsychological evaluation revealed deficits in the areas affected by the lesion, but also changes suggesting dysfunction in other areas, such as the left frontal dorsolateral, right parietal cortex, the supplementary motor area, and in the right frontal dorsolateral and orbito-frontal area.

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–deceleration forces [7]. These forces commonly are the origins of certain brain injury profiles including orbitofrontal, anterior and inferior temporal contusions, with diffuse axonal injury. The latter particularly affects the corpus callosum, superior cerebellar peduncle, basal ganglia, and periventricular white matter [6]. 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.

However, some of the cognitive deficits might also be due to, or aggravated by, the mood disorder itself, since it has been reported that there may be a subgroup of bipolar patients who develop cognitive deficits due to the disease process, and which are independent of the mood state [27].

The complex processes of cognition and mood are not mediated by any specific brain region, but require the coordinated activity of several areas; a compromise of neural connectivity may result in attenuation of the functions regulated by these areas and result in clusters of signs and symptoms currently recognised as psychiatric disorders [28].

Another proposed mechanism is the "diaschisis model", whereby the loss of function produced by acute focal brain damage, even without axonal shearing, may produce dysfunction in adjacent or remote regions connected through fibre tracts [29].

The severity of the neuropsychiatric sequelae of the brain injury is determined by multiple factors, but, in general, prognosis is associated with the severity of injury [2].

Since TBI can cause permanent vulnerability to psychiatric disorders, contributing substantially to long-term disability [4] and quality of life [5], psychiatric evaluation and (long-term) monitoring should be included in the routine follow-up of TBI.

Treatment should include cognitive and physical rehabilitation, family and personal support, and psychopharmacological management of mood and other behavioural syndromes [30]. Pope et al. [31] described a case of bipolar disorder after TBI was successfully treated with valproate and lithium, and Monji et al. [19] reported a case where valproate was effective in monotherapy. Although the exact mechanisms of action for valproate have not yet been determined, postulated theories include an "anti-kindling" effect in the limbic system on emotion, cognition, and behaviour; an enhancement of GABAergic-mediated inhibitory control; and action as a general CNS stabiliser, making it a primary pharmacological intervention for the treatment of neuropsychiatric symptoms after brain injury [19].

Future research should also focus on the efficacy and timing of psychological interventions in combination with medications, in the treatment of post-TBI Axis I disorders, to create standards of care, and most importantly, enhance the quality of life of individuals after TBI [17,32].

Key points

- Traumatic brain injury (TBI) may cause vulnerability to psychiatric disorders, with latency periods of over 10 years.
- Bipolar disorder is a rare outcome after TBI, major depression being the most common disorder.
- The complex processes of cognitive and mood requires the coordinated activity of several brain areas, and lesions in one area may influence the functioning of adjacent or remote ones.
- Treatment of bipolar disorder after brain injury should include psychopharmacological management, namely valproate as the primary pharmacological intervention, cognitive and physical rehabilitation, and family and personal support.

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Statement of interest

The author has no conflict of interest with any commercial or other associations in connection with the submitted article.

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