

Mood disorder following traumatic brain injury: a case report

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ABSTRACT

Traumatic brain injury is a clinical situation that generally affects young people aged 45 years or younger and causes mortality and critical functional losses. The most common psychiatric disorder following traumatic brain injury is depression. Although the relationship between depression and organic diseases has been studied a lot, there is less data about mania. Secondary mania differs from primary mania with advanced beginning age, absence of family history, more difficult and slower response to treatment; and secondary mania usually has no recurrence. In this report, secondary mania and its clinical features are discussed in light of a mood disorder following a trauma case. The case is still followed with mood stabilizer treatment and the patient is euthymic.

Keywords: Traumatic brain injury, secondary mania

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Traumatic brain injury (TBI) affects young people aged 45 years and younger and causes mortality and morbidity [1]. The most essential factors that affect quality of life in the long term are cognitive and behavioral changes in these patients. Trauma has primary and secondary impacts; while primary impacts are contusion and diffuse axonal injury emerging at the moment of accident, changes emerging as a response to primary injury are considered secondary impacts (intracranial pressure increase, parenchymal edema, inflammation, glial proliferation, etc.) [2]. Neuronal degeneration and the repair process following trauma cause changes in neuronal activity and neuronal circuits in the long term. Posttraumatic changes in the neurotransmitter system are also associated with mood disorders following trauma [3].

Various psychiatric disorders may develop after trauma depending on the affected region of the brain [1]. The lateral orbitofrontal cortex is associated with irritability, impulsivity and emotional lability; anterior

cingulate cortex with apathy and akinetic mutism; temporal lobe with lability, aggression and psychosis; and hypothalamus with sexual behavior and aggression [4]. In one case changes in the right hemisphere were detected on MR imaging of a 17-year-old patient with mania after TBI and in another case left hemisphere lesion was associated with mania. Additionally, a case of mania following bilateral temporal hemorrhage was also reported [5].

In this case report, mood disorders following trauma and clinical features of secondary mania are discussed in the light of a case about a patient with bipolar disorder following trauma.

CASE PRESENTATION

The patient was a 25-year-old single male, working as a salesman in a company. He had graduated from university and lived with his family in



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a small city.

He was taken to hospital involuntarily by family members with complaints of insomnia, increased self-confidence, and excessive passion for spending money. According to the family members and the patient himself, he had no previous psychiatric disorder and drug use. He had no psychiatric family history. He had subarachnoid hemorrhage after falling from a height two years previously. He was treated in the intensive care unit for a period. After discharge from hospital, he had a period of depression accompanying unhappiness, pessimism, and unwillingness to live which lasted 4-5 months. He was treated with sertraline 50 mg and entered a remission period. He had a period of increased mobility, self-confidence and uncontrolled behaviors thereafter, treatment was completed and medicines were stopped. The patient was treated with low dosage risperidone. In follow-up, he had one more manic period that subsided in a short time. The patient whose complaints recurred in spite of medical treatment in the last 15 days was admitted to our clinic.

In his psychiatric examination psychomotor activity was increased, he exhibited grandiose attitude and he was irritable. He had flighty ideas and his speaking was fast and speed was increased. Young Mania Rating Scale (YMRS) was 32 points. Laboratory studies were in normal range.

On MR imaging, posttraumatic sequelae were detected in the right temporal lobe, bilateral basal ganglion levels, right corpus callosum splenium, thalamus, bilateral frontal lobe and subcortical white substance. The patient's treatment was arranged as 30 mg/day olanzapine and 1000 mg/day valproic acid. In the second week of treatment, clonazepam 6 mg/day and haloperidol were added because he did not respond to treatment with olanzapine and valproic acid. Haloperidol was increased to 30 mg and olanzapine was decreased step by step and stopped. The patient subsided and his YMRS regressed to 6 points. The patient was discharged in the end of the fourth week. After discharge from hospital, akathisia occurred due to haloperidol, so haloperidol treatment was stopped and his treatment was resumed with trifluoperazine 2 mg. The patient is still euthymic.

DISCUSSION

Depression is the most common psychiatric disorder after trauma [4]. Anxiety disorders and mania follow depression. Although correlations between depression and organic diseases have been commonly studied, there is less data about mania. In a previous study, the rate of mania was detected as 9% in one-year follow-up of TBI patients [6].

Psychiatric symptoms after trauma vary according to the affected region of the brain. Prefrontal cortex, temporal cortex and hypothalamus are the most common regions associated with psychiatric disorders following trauma [4]. Bilateral orbitofrontal and right temporoparietal, right basal and medial temporal lobe, basal ganglions, thalamus and right frontotemporal lesions have been associated with mania [5-7]. In our case, there were posttraumatic lesions in the right temporal lobe, basal ganglions and bilateral frontal area, in line with literature.

Although mania is usually associated with bipolar disorder, there are many etiological causes [6]. However mania induced by bipolar affective disorder is called primary mania, whereas mania induced by neurological, metabolic or pharmacological causes is called secondary mania [8].

Secondary mania comprises 1.75% of all admissions for psychiatric reasons, and affects 4.67% of all manic patients [9]. It is more prevalent than primary mania in later ages. Usually these patients have no family history of primary or secondary mania. In psychiatric examination of these patients, irritability is more common than euphoria. Even though the response to treatment is more difficult, the total disease period is shorter than primary mania. Treatment protocol is similar to primary mania. For this patient, treatment was started with olanzapine. It is known that in bipolar affective disorder long term use of first generation antipsychotics is associated with increased risk of dyskinesia and neuroleptic malignant syndrome. However, we needed sedation for the patient. Haloperidol was added after insufficient response to olanzapine. Haloperidol was then changed with trifluoperazine because akathisia occurred with haloperidol treatment. Maintenance treatment is not

necessary in secondary mania because secondary mania usually does not recur [10]. In this case, mood stabilizer was used due to both depression and recurrent mania. Valproic acid was selected due to its neuroprotective effect to protect patient from potential epileptic seizures.

CONCLUSION

This case report differs from other cases due to longer disease and treatment period, more difficult response to treatment, recurrence and necessity of maintenance treatment [8]. A confusing factor is whether the use of antidepressants caused mania in this patient.

Informed consent

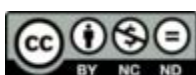
Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of interest

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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