Psychosis following stab brain injury by a billiard stick

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Abstract
Traumatic brain injury sometimes can lead to psychotic disorder which resembles schizophrenia. We report a 17-year-old boy, admitted to psychiatric department for psychotic symptomatology. He had suffered penetrating craniocerebral injury after stabbing by a billiard stick, three years earlier. On admission, he expressed delusions with paranoid and religious content. The magnetic resonance imaging of the brain showed a 10 cm large tubular area of posttraumatic encephalomalacia of the left hemisphere, whereas the electroencephalography revealed slow left temporal activity. The patient’s recovery was uneventful with clozapine at a dosage of 100 mg daily. This case shows the diagnostic challenge in differentiation between schizophrenia and psychotic disorder due to traumatic brain injury. The authors emphasise the importance of imaging of the brain, especially magnetic resonance, in establishing the diagnosis of psychotic disorder due to traumatic brain injury.

Key words: head injury, psychiatry, radiology, MRI scan, traumatic brain injury

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Psychotic disorder secondary to traumatic brain injury (PDTBI) is specified by clinical signs of hallucinations or delusions when there is a direct relation between psychosis and traumatic brain injury. Although there are no published epidemiological data about stab brain injuries in the territory of Southeast Europe, in western countries stab brain injuries are relatively uncommon, because the adult calvarium usually provides an effective barrier. We report the unusual case of PDTBI in relation to stab brain injury by a billiard stick.

Case report
A 17-year-old boy was admitted to the psychiatric department due to psychotic symptomatology. He had been intentionally stabbed by a billiard stick, which passed through his left orbit deeply into the brain three years earlier. At that time, the wound was treated conservatively. Meanwhile, he gradually started exhibiting social withdrawal, anhedonia, disinhibited behaviour accompanied by delusions and visual hallucinations. In his family history, there were no similar symptoms.

On admission, he expressed delusions with paranoid and religious content. The patient showed signs of bradydysopia, his mood was depressed and he had no insight of his condition. Except for the left eye mydriasis, the patient’s neurological findings were unremarkable.

The magnetic resonance imaging of the brain showed a 10 cm large tubular area of posttraumatic encephalomalacia (Figure 1a) surrounded by a peripheral gliosis (Figure 1b) and hemosiderin rim (Figure 1c). The affected brain areas were left orbitofrontal region, insula, putamen, deep white matter and parietal lobe with consecutively slightly enlarged left lateral ventricle. The electroencephalography (EEG) showed slow left temporal activity. Abdominal ultrasound, chest radiography, blood and urine analysis showed no relevant abnormality.

The patient remained in the hospital for five weeks. He was initially given 12.5 mg of clozapine to the maximum dosage of 100 mg/day, which was reached by the second week. By the end of the third week his visual hallucinations had ceased and his delusions had become less intensive. Moreover, his depressed mood was not so pronounced. No side effects were recorded and the blood tests showed no alterations either.

Discussion
According to Bauer and Patzelt, penetrating cranio-cerebral injuries are frequently inflicted with a knife. They occur almost exclusively in cases of homicide and only 13 cases were documented during a period of 30 years. However, to the best of our knowledge, only two cases of stab brain injury by a billiard stick were reported before. Orbitae and temporal regions are areas of thin bone where forcefully thrust sharp objects may penetrate easily. Usually, brain injury is limited to the wound tract with a good prognosis of recovery unless the brain stem is damaged. Although reactive gliosis is the universal reaction to brain injury, the precise origin of the glial cells...
reacting to injury are unknown.

It has been estimated that traumatic brain injury (TBI) increases the risk of psychosis two- to three-fold over that of general population. Commonly, the latency period between TBI and new-onset psychosis is about four years, which makes establishing the diagnosis of PDTBI more difficult. Nonetheless, Kim et al. argued that no firm conclusions could be made regarding the incidence or prevalence of PDTBI. As pre-injury risk factors for PDTBI, Arciniegas et al reported male gender, neurodevelopmental and neuropsychiatric problems, and family history of schizophrenia. In the present case, the risk factor was male gender, while personal and family history were not significant.

Traumatic lesions of temporal and frontal lobes are the most common injury risk factor. Neuropsychologically, posttraumatic behavioural changes in our patient could be attributed mostly to the lesion of the frontal lobe, precisely prefrontal cortex that is responsible for executive functions such as initiation, mental flexibility, planning, working memory, problem solving, verbal reasoning, inhibition and mental flexibility. Therefore, clinical symptoms are divided according to the affected cognitive function. The orbitofrontal syndrome, prominent in our case, is characterized by disinhibition, impulsive behaviour, poor judgment and insight, irritability and emotional lability. The Vietnam Head Injury Study concluded that subjects with lesions limited to the frontal lobes tended to show more aggressive and violent behaviours compared with patients with non-frontal head injury and controls without head injury. Moreover, a countless cases treated with abandoned infamous psychosurgical procedure called ‘frontal leucotomy’ testify in favour of the mentioned role of the prefrontal cortex. While left hemispheric injury is associated with the development of schizophreniform posttraumatic psychosis, like in the present case (Figure 1), right hemispheric injury is more frequently associated with the development of delusions alone.

Figure 1: Sagittal T2W (a, arrows), Axial T2 FLAIR (b) and Axial T2* GRE (c) MR images show a large tubular area of post-traumatic encephalomalacia. Note the rim of peripheral gliosis (b, arrow) and hemosiderin (c, arrow).

Electroencephalographic abnormalities together with posttraumatic epilepsy and cognitive impairments are the most common post-injury risk factors associated with PDTBI. About 70% of the persons with PDTBI have EEG abnormalities that are manifested mostly (55%) as asymmetric temporal slowing, which was found in our patient as well. According to the Diagnostic and statistical manual of mental disorders, the criteria for establishing the diagnosis of PDTBI are: prominent hallucinations or delusions; evidence from the history, physical examination, or laboratory findings that the disturbance was the direct physiological consequence of a medical condition; lack of evidence suggesting that the disturbance occurred exclusively during the course of a delirium. Analysing 60 published cases of PTDBI, Fujii and Ahmed found that most patients experienced delusions (47 out of 60), while almost half the subjects experienced hallucinations (28 out of 60).

As far as medications are concerned, the ‘start-low, go-slow’ approach is recommended. Anti-psychotic medications with potent anti-histaminic, anti-dopaminergic and anti-cholinergic properties should be avoided due to their tendency to produce significant adverse effects in these patients. Regarding the benefits and potential side effects, the atypical anti-psychotics such as clozapine, olanzapine, risperidone, quetiapine and ziprasidone are advised.

Although many questions remain about how traumatic brain injury is associated with psychotic symptoms, this case certainly supports their relation. Among the abovementioned diagnostic modalities, magnetic resonance imaging of the brain plays a significant role and represents a contemporary method of establishing the diagnosis of PTDBI.
References