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Psychosis Post-Traumatic Brain Injury: Causal Links and Nosographic Approaches about a Clinical Case

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Abstract **Case Report**

Psychotic manifestations, mainly consisting of delusions and hallucinations, are clinical entities that transcend nosographic boundaries and are present in various psychiatric and somatic disorders, particularly neurological and neurosurgical conditions. These psycho-behavioral manifestations can be isolated, coexist, or complicate a neurological deficit, thus raising questions about their imputability. Our goal is to shed light on the extent to which cranial traumas contribute to the development of psychiatric disorders and their specific characteristics. Ultimately, we aim to establish the foundations for an optimal biopsychosocial and legislative management approach if necessary. This will be illustrated through the clinical case of a patient who exhibited depressive symptoms shortly after the onset of delusional ideas with a psychotic nature, one year after experiencing a cranial trauma due to a public road accident. The somatic consequences of cranial trauma can cause psychotic symptoms. This is referred to as a post-traumatic psychotic disorder directly linked to localized brain damage, adhering to the structure-function paradigm. Such symptoms can appear up to four years later. The authors have reported that the onset of psychotic symptoms is frequently associated with depressive syndrome, which is a factor contributing to the chronicization of post-traumatic psychosis. Psychotic symptoms following Psychotic manifestations, such as delusions and hallucinations, can occur in various psychiatric and somatic disorders, especially neurological and neurosurgical conditions. These behaviors can occur alone, work in conjunction, or complicate a neurological deficit, raising questions about their connection to the condition. We want to understand how head injuries contribute to the development of psychiatric disorders and their specific characteristics. Ultimately, we aim to establish a comprehensive approach to managing these conditions, incorporating biological, psychological, social, and legal aspects if necessary. We will illustrate this through a clinical case of a patient who developed depressive symptoms shortly after experiencing delusions with a psychotic nature, a year after a head injury from a public road accident. The physical effects of head injuries can lead to psychotic symptoms, known as post-traumatic psychotic disorder, directly linked to brain damage. These symptoms can appear up to four years after the injury. It has been reported that the onset of psychotic symptoms is often associated with depression, which can contribute to the chronic nature of post-traumatic psychosis. Psychotic symptoms following head injuries are common and fall into several clinical categories that are similar both clinically and physiopathologically. More thorough investigations are needed for accurate diagnosis and better care, as well as to address any resulting legal implications. Cranial trauma is common and belongs to several nosographic frameworks that are quite similar clinically and physiopathologically. More appropriate investigations are necessary not only for a more accurate diagnosis and better-adapted care but also to facilitate the resulting medico-legal implications.

Keywords: Psychotic manifestations, Cranial trauma, Post-traumatic psychotic disorder, Depressive symptoms, Medico-legal implications.

INTRODUCTION

Traumatic brain injuries (TBIs) expose victims to damage both physically and mentally, triggering a range of disorders that can be psychological, neurological, or both in an intertwined manner. The psycho-behavioral consequences of TBIs are highly variable and can fit into several nosological frameworks, affecting purely psychiatric dimensions such as post-traumatic stress disorder (PTSD) or subjective syndrome. They can also manifest as psychotic

symptoms originating from neurological damage—whether anatomical or microbiological—secondary to the cranial impact.

These psychotic manifestations, primarily consisting of delusions and hallucinations, are trans-nosographic clinical entities present in various psychiatric and somatic conditions, particularly neurological and neurosurgical disorders. These psycho-behavioral manifestations can be isolated, coexist, or complicate a neurological deficit, raising questions about their causality.

METHOD

In this article, we will shed light on the extent of TBIs' involvement in the genesis of psychiatric disorders and their specificities through a clinical case followed in the psychiatry department of the Mohammed V Military Instruction Hospital. We will conduct a semiological and etiopathogenic analysis in comparison with what has already been described in the literature to ultimately establish the foundations for optimal biopsychosocial and potential legislative management.

CLINICAL CASE

Ms. F.B., a 23-year-old law student with no known medical history, was the victim of a road traffic accident two years ago, resulting in a loss of consciousness and various injuries to her limbs. She was taken to the emergency department of the Mohammed V Military Instruction Hospital, where an initial radiological assessment revealed a temporo-parietal fracture, a right parietal subgaleal hematoma, and a fracture of the petrous bone (Figure 1).

She was then transferred to the neurosurgery department, where she underwent additional assessments and an ENT consultation, with no need for surgical intervention (Figure 2). The patient was kept under observation for about ten days, showing good clinical and radiological progress before being discharged in a stable condition.

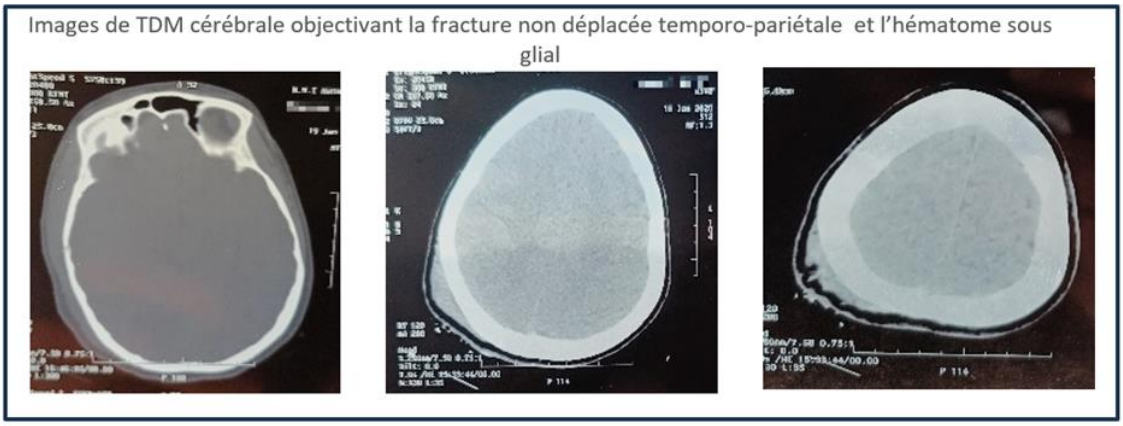

Figure 1: CT images showing the non-displaced temporo-parietal fracture and subgaleal hematoma

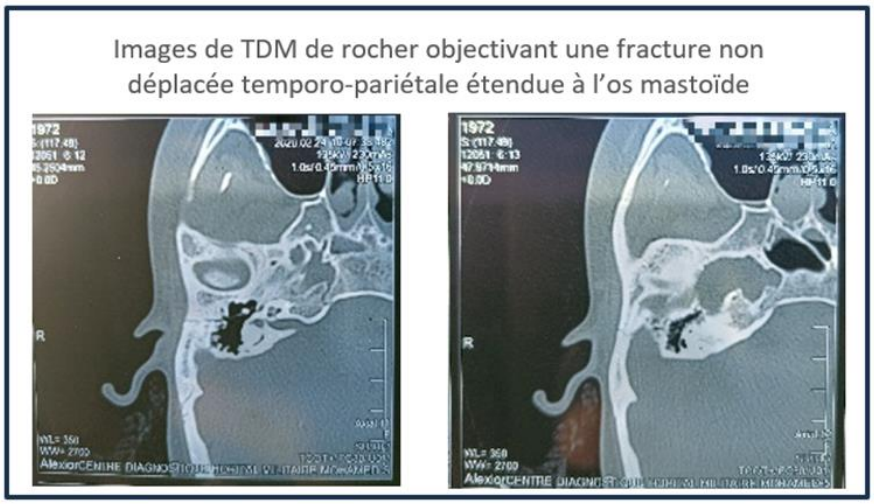

Figure 2: CT images of the petrous bone showing the non-displaced temporo-parietal fracture extending to the mastoid bone

A few months later, F.B. presented to the psychiatry department of the Mohammed V Military Instruction Hospital, requesting a medical certificate to support a complaint against the motorcyclist who hit her, at the request of a lawyer she had hired for legal proceedings. Irritable and verbose, she described the accident as intentional and demanded justice. The treating physician requested that she return with a family member for a hetero-anamnesis, but the patient did not follow up on the doctor's request.

A year later, the patient returned for a second psychiatric consultation, accompanied by her sister, complaining of concentration and memory problems that had affected her academic performance, which had not progressed since the accident. She attributed these deficits to the head trauma, still convinced of its criminal intent, with a negative impact on the quality of her relationships with family members who did not believe her version of events. The interview revealed depression with more elaborate persecutory ideas than a year ago, now involving not only the motorcyclist but also the medical team she claimed had mishandled her care, the national security officials she accused of evidence concealment and corruption, and her father, whom she accused of colluding with the alleged assailant to cover up the incident.

Given the new semiological elements observed by the psychiatrist and the hetero-anamnesis with the sister, the patient was prescribed Risperidone 2 mg/day and Sertraline 50 mg/day with close follow-up appointments. The evolution was marked by a gradual distancing from her delusional ideas with decreasing emotional involvement, leading to encapsulation and an improvement in her mood, a recovery of her memory capacities, and a relatively satisfactory return to social and academic functioning.

DISCUSSION

The somatic consequences of traumatic brain injury (TBI) can cause psychotic symptoms. These are referred to as post-traumatic psychotic disorders, directly linked to localized brain damage and following the structure-function paradigm. Some authors call this "Schizophrenia-like psychosis," a schizophrenic syndrome secondary to TBI, indicating diffuse or localized brain damage. This clinical entity is differentiated from post-traumatic schizophrenia, which refers to primary schizophrenia decompensated following TBI. These symptoms can appear up to 4 years later [3].

One of the first descriptions and correlations between TBI and psychotic manifestations is attributed to Bleuler, who introduced the notion of schizophrenia secondary to TBI with a clinical presentation and evolution identical to idiopathic schizophrenia. Kraepelin later classified childhood TBIs as predisposing factors for "early-onset dementia in adulthood" in 1919, and Schultz in 1932 noted that TBI facilitates the onset of psychosis [4].

In the late 1960s, a study by Davidson and Bagley on TBI victims found that the risk of developing psychotic symptoms after TBI is three times higher than in the general population. They identified several risk factors, such as frontal lesion location, left hemisphere involvement, closed head injuries, and coma duration over 24 hours—most of which were present in our patient. Depressive symptoms are also frequently encountered and could contribute to the chronicity of post-traumatic psychosis [5, 6].

Typically, psychotic symptoms following TBI are associated with confusional symptoms and post-traumatic amnesia. However, in some cases, similar to our patient, psychotic symptoms can appear long after the trauma. According to a study by Fujii and Ahmed in 1997, the incidence of such disorders was twice as high in men as in women; 72% of patients developed psychosis on average 4 years after severe TBI with post-traumatic amnesia lasting more than 7 days. The symptomatology often includes persecutory delusional syndrome and mainly auditory hallucinations, neuropsychological test disturbances, attention disorders, as well as memory, executive, and visuospatial function impairments [7, 8].

Biochemically, other studies have shown that cognitive disorders are very frequently found after TBI, regardless of its severity. These deficits particularly affect arousal, attention, memory, and executive functions and involve numerous neurotransmitters (glutamate, dopamine, serotonin, and acetylcholine). However, in the context of chronic cognitive disorders secondary to TBI, as in our patient, only the cerebral cholinergic system seems to be involved [9].

After mechanical shock, these cholinergic neurons and their ascending projections are particularly vulnerable and susceptible to damage. In animal-induced TBI models, there is initially an acute phase of excessive cholinergic system activation followed by a persistent reduction in cholinergic function [10].

Indeed, this decrease in alpha-7-nicotinic receptors (involved in sensory filtering in the frontal lobe) has been demonstrated and could constitute a substrate for the emergence of delusional ideas and perception disorders, as well as defective social functioning. This explains the lower efficacy of classical neuroleptics (which do not act on the cholinergic system) compared to atypical antipsychotics, which antagonize both dopaminergic D2 and serotonergic 5HT2 receptors. This dual action increases acetylcholine release in the medial prefrontal cortex, improving cognitive functions and significantly attenuating deficit symptoms. Risperidone and Olanzapine, which increase acetylcholine in the medial prefrontal cortex, have shown

these effects according to the works of Schreiber et al., Butler, and Umansky [11].

Similar to our patient's case and previous studies over the past fifty years, the results remain highly controversial. However, they all agree on the inability to identify clear diagnostic criteria for post-traumatic delusional disorder, differentiate it from other forms of non-traumatic psychoses, and establish a direct and clear causal link between TBI and psychotic symptoms. This complicates not only the management of these patients but also the medico-legal implications. In most cases, the diagnosis of schizophrenia (which is not attributable to the trauma) is the most common [2]. In an expert context, only the repair of bodily damage is considered, without taking into account post-traumatic psychosis, which still does not constitute an exclusive entity due to the significant overlap and diversity of explanations for its clinical and pathophysiological dimensions [1].

CONCLUSION

Given these psychotic manifestations, shared by both post-traumatic psychotic disorder and other non-traumatic psychoses, current scientific advances still cannot conclude a direct causal link between TBI and potential subsequent psychotic manifestations. One hypothesis is that in some patients who would have developed schizophrenia but did not yet present prodromal or pre-clinical symptoms, the occurrence of TBI reduced cognitive reserve, thus limiting cognitive remediation possibilities.

REFERENCES

1. Auxéméry, Y., & Fidelle, G. (2011). Psychose et traumatisme psychique. Pour une articulation théorique des symptômes psycho-traumatiques et psychotiques chroniques. *L'Encéphale*, *37*(6), 433-438.
2. Sinders, M., & Benoudiba, F. Imputabilité des symptômes psychotiques dans les suites d'un traumatisme crânien: De La Doctrine À La Pratique.
3. Auxéméry, Y. (2011, April). Les troubles psychotiques post-traumatiques: un modèle physiopathologique de la schizophrénie. In *Annales Médico-psychologiques, revue psychiatrique* (Vol. 169, No. 3, pp. 155-159). Elsevier Masson.
4. Schultz, B. (1939). La schizophrénie traumatique. *Ann Med Psychol*, 99, 1-2.
5. Schneider, K. (1918). Schizophrene Kriegpsychose. *Zeit Neurol*, (43), 420-439.
6. Davison, K. (1969). Schizophrenia-like psychoses associated with organic disorders of the central nervous system: review of literature. *Br J Psychiatry*, *4*, 113-184.
7. Fujii, D., Ahmed, I., & Hishinuma, E. (2004). A neuropsychological comparison of psychotic disorder following traumatic brain injury, traumatic brain injury without psychotic disorder, and schizophrenia. *The Journal of neuropsychiatry and clinical neurosciences*, *16*(3), 306-314.
8. Arciniegas, D. B. (2003). The cholinergic hypothesis of cognitive impairment caused by traumatic brain injury. *Current psychiatry reports*, *5*(5), 391-399.
9. Dixon, C. E., Ma, X., & Marion, D. W. (1997). Reduced evoked release of acetylcholine in the rodent neocortex following traumatic brain injury. *Brain Research*, *749*(1), 127-130.
10. Fujii, D., & Ahmed, I. (2002). Characteristics of psychotic disorder due to traumatic brain injury: an analysis of case studies in the literature. *The Journal of neuropsychiatry and clinical neurosciences*, *14*(2), 130-140.
11. Bennouna, M., Greene, V. B., & Defranoux, L. (2007). Théorie cholinergique dans les psychoses après un traumatisme crânien et dans la schizophrénie: un lien?. *L'Encéphale*, *33*(4), 616-620.