Psychotic Disorder and Traumatic Brain Injury

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Introduction
Traumatic brain injury (TBI) can result in serious and disabling neuropsychiatric disorders, such as cognitive deficits and personality change, as well as severe and chronic psychosis. This review focuses on the relationship between TBI and schizophrenia-like psychosis (SLP) including its epidemiology, diagnostic criteria, clinical presentation, psychopathology, risk factors, and pathophysiology. The relationships between post-traumatic epilepsy and SLP and brain trauma and schizophrenia are also discussed. The risk of SLP does increase after TBI. The clinical presentation has considerable overlap with primary schizophrenic disorder, with a prominence of persecutory and other delusions and auditory hallucinations, as well as a lack of negative symptoms. The onset is often gradual, with a subacute or chronic course. More severe and diffuse brain injury, especially of the temporal and frontal lobes, is the most prominent risk factor. Genetic load may also play a role, but presence of epilepsy could be a protective factor. Further large and systematic longitudinal studies are needed.

Epidemiology
Published data on the occurrence of psychosis in patients who had TBI are limited and suffer from the absence of a standard definition of psychosis, which makes comparisons of studies difficult. This is reflected in the wide range of incidence rates for SLP-TBI reported in the literature. Ota [5] studied 1168 adults who were admitted to hospital after TBI and reported psychosis, which could be “functional” or “organic,” in 2.7%. Miller and Stern [6], in a long-term follow-up of 100 patients with TBI, found 10 with psychosis, all of whom suffered from dementia. A study by Lishman [7], which used contemporary diagnostic criteria, identified only five patients with schizophrenia-like illness in 670 patients with penetrating head injury. Achte et al. [8], in a large Finnish series, reported psychosis in 8.9% of 3552 brain-injured men, with 24% schizophrenia-like, occurring more commonly with mild injuries in younger persons. In a subsequent report, Achte et al. [9] studied a sample of 2907 war veterans in Finland who sustained brain injury, and found that 26% had psychotic disorders. In a detailed evaluation of 100 of these veterans, the authors found that 14% had paranoid schizophrenia. Violon and De Mol [10] found that of 530 patients with head injury, 3.4% developed psychosis 1 to 10 years after injury. The lack of appropriate noninjured control individuals in most of these studies makes it difficult to put these figures in perspective, except that most studies report rates of SLP-TBI that are much higher than rates of schizophrenia in the general population. The survey by Davidson and Bagley [11], which included data comprised of a comprehensive...
review of the published literature, suggested a two- to threefold increase in the risk of schizophrenia in individuals with head injury. In conclusion, the evidence so far suggests that SLP, some of which resembles schizophrenia, is more likely to occur in individuals who sustain head TBI, but more systematic studies are needed to yield incidence figures using current criteria.

Clinical Presentation
This description is based on the review by Davidson and Bagley [11] and two recent reports of SLP-TBI [12•,13•], one of which [12•] was an analysis of 69 published cases.

The majority of the subjects are young, with a male preponderance. The mean age of onset of psychosis was 26.3 (SD=10.2) years and 33.4 (SD=15.4) years in the two studies, with 80% and 90% being men. Because TBI is more common in young men, it is uncertain whether the SLP rates merely reflect this. It has been suggested [12•] that men may be overrepresented after accounting for the base rates for TBI, but this is far from established. The mean interval between the TBI and the development of psychosis was 4.6 [13•] and 4.1 [12•] years in the two reports, with a wide range (0 to 34 years). Although most TBI is caused by motor vehicle accidents, assaults, gunshot injuries, and falls are all represented.

Characteristics of head injury
The TBI is more likely to be closed, but an open injury is not necessarily protective against future SLP, as has sometimes been suggested [8]. The severity of the injury is usually moderate to marked. The severity of the head injury, by neuroimaging and neuropsychologic criteria, was greater in the SLP-TBI group than the TBI group without psychosis [13•]. Although many SLP-TBI occur after head injury in childhood, early head injury was not over-represented in one large study [13•]. In approximately 40% of cases in this study [13•], the head injury was followed by personality or behavioral change, the main characteristics of which were impulsivity, aggressiveness, loss of social graces, moodiness, and, less commonly, apathy. There was evidence for brain damage in the temporal, parietal, and frontal lobes, more often unilateral than bilateral, on the basis of neuroimaging, clinical, and neuropsychologic data. The patients with SLP-TBI had more widespread neuropsychologic deficits than the control group [13•].

Psychopathology
Prodromal symptoms are common, often lasting for months, and include bizarre or antisocial behavior, social withdrawal, affective instability, and deterioration in work. Depressive symptoms are often present at the time of presentation, but confusional symptoms at onset are unusual.

The psychosis is delusional-hallucinatory in nature. A range of delusional symptoms, similar to that seen in schizophrenia, is present and includes first rank Schneide- rian symptoms. In the study by Sachdev et al. [13•], one or more delusions were present in all subjects, with persecutory (55.5%), referential (22.2%), control (22.2%), grandiose (20%), and religious (15.4%) delusions the most common. Delusions of thought alienation, thought insertion, withdrawal, or broadcast were present in six patients (13.3%) and somatic passivity in three patients (6.7%).

The review by Fujii and Ahmed [12•] emphasized the presence of persecutory delusions. Organic themes, described by Cutting [14•], were absent in the Sachdev et al. [13•] study. However, the review of published cases [12•] did find five cases with the Capgras delusion and three cases each with reduplicative amnesia, erotomania, and stealing. Delusions relating to misidentification, stealing, or hiding, which are prone to occur in dementia patients with psychosis, are not generally seen in SLP-TBI. Hallucinations are predominantly auditory (more likely) or visual. Formal thought disorder and catatonic features are usually absent. The psychosis is, therefore, predominantly a positive syndrome, with only 22% and 15% patients demonstrating negative symptoms such as flattening of affect, avolition, or asociality in the two studies. Agitated behavior and aggressive behavior are common.

Diagnostic criteria
As is apparent from the previous description, the clinical picture of TBI resembles schizophrenia; the difference is that like other secondary schizophrenia, the emphasis is on a paranoid-hallucinatory psychosis with few negative symptoms. To meet the criteria for a psychotic disorder caused by traumatic brain injury, it is crucial to establish that 1) the psychosis is a direct physiologic consequence of a brain disorder, and 2) the psychosis is not better accounted for by a primary psychiatric disorder such as schizophrenia. The pathophysiology of SLP is discussed herewith, but the association between TBI and SLP cannot be considered proof of causality even though it is a recognized association, psychosis as a consequence of brain injury is plausible, and many of the patients are not otherwise vulnerable to schizophrenia.

Course
The long-term course of SLP-TBI is poorly studied. Fujii and Ahmed [12•] reviewed 69 cases and found some follow-up information in 39. Of these, 25 patients were reported to have improved, 11 not improved, and three were worse. The authors’ group [13•] did not follow patients systematically, but it is not unusual for clinicians to encounter TBI-related psychosis that, despite its prominence of positive symptoms, responds poorly to neuroleptic medication.