Neuropsychiatry of Psychosis Secondary to Traumatic Brain Injury

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Psychotic episodes following brain injuries can often be mistaken for schizophrenia. How can the presentation of psychotic episodes reframe our understanding of this complex phenomenon?

Psychosis secondary to traumatic brain injury (PSTBI) is reported to occur in anywhere from 4% to 8.9% of individuals who sustain traumatic brain injury (TBI) (Achte et al., 1991; Violon, 1988). Despite its rarity, PSTBI is of interest to clinicians and neuroscientists for three reasons: 1) there is usually a latency between the head injury and presentation of psychotic symptoms, thus the appearance of psychosis is often unexpected and puzzling; 2) there are diagnostic issues as some people who develop PSTBI have family histories of psychotic disorder, while many others do not; and 3) the disorder has conceptual relevance to understanding schizophrenia spectrum disorders (Fujii and Ahmed, 2001).

The purpose of this paper is threefold: 1) to review the literature on PSTBI; 2) to describe the shortcomings of the DSM-IV criteria for PSTBI; and 3) to provide a conceptual framework for understanding PSTBI.

Review of PSTBI Literature

Onset. The age of onset for PSTBI appears to be highly variable, but most studies report means in the mid-20s to mid-30s (Fujii and Ahmed, 2001). Onset before 20 years of age appears to be relatively uncommon and may be associated with a more severe presentation of psychosis (Davison and Bagley, 1969).

The latencies between TBI and onset of psychosis are also variable. Although several researchers report means between four and five years, latencies can range from a few days to over 20 years (Achte et al., 1991; Fujii and Ahmed, 2001; Sachdev et al., 2001). Despite the wide range of latencies, studies suggest that about half of patients with PSTBI demonstrate symptoms within the first year, and roughly 72% of patients have symptoms within the first five years (Davison and Bagley, 1969; Fujii and Ahmed, 2002a).

There is some evidence suggesting that the duration of the latency between TBI and onset of psychosis may have clinical significance. Latencies of less than one year have been associated with diffuse injuries (Davison and Bagley, 1969), paranoid symptoms (Achte et al., 1991) and visual hallucinations (Fujii and Ahmed, 2002a). By contrast, patients with longer latencies before the onset of symptoms were found to have localized damage to the temporal lobe and presence of epilepsy (Davison and Bagley, 1969). In terms of prodromal symptoms, roughly half of patients with PSTBI demonstrate bizarre behaviors, affective instability and antisocial behaviors. Academic or vocational deterioration were reported in roughly one third of the patients, and about one third of patients also demonstrated social withdrawal (Sachdev et al., 2001).

Risk factors. Several risk factors for PSTBI have been reported. They include male gender, premorbid neurological abnormalities such as early head injury or neurological disorder (Fujii and Ahmed, 2001), previous psychological disturbance (Violon, 1988), family history of psychotic illness, or mental retardation (Achte et al., 1969). A family history of schizophrenia was reported in 2.9% to 18% of patients with PSTBI, thus appearing to be higher than in normal patients, but less than in patients with schizophrenia (Davison and Bagley, 1969).

Injury characteristics. Patients with either open- or closed-head injuries can develop PSTBI. Severity of injury, as measured by duration of loss of consciousness and posttraumatic amnesia, varies from mild to severe (Fujii and Ahmed, 2002a).

Epilepsy. The relationship between epilepsy--particularly temporal lobe epilepsy--and psychosis is well documented, and epilepsy is also a common comorbid condition in PSTBI (Lishman, 1998). Rates of epilepsy have ranged from 9% to 34% (Fujii and Ahmed, 2002a; Sachdev et al., 2001). Patients with open-head injuries appear to be more susceptible to comorbid epilepsy, with one study...
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The development of a seizure disorder that, in turn, generates the development of a psychosis. As such, the diagnosis of PSTBI may be missed. In the second category, TBI contributes to the development of psychosis. One way this can occur is if the patient has a history of schizophrenia and/or a family history of psychosis. In this case, TBI can act as a precipitating factor. Another way this can occur is if the patient has no family history of schizophrenia and no or low genetic risk for psychosis. As such, TBI can act as a precipitating factor.

To address the complex relationship between TBI and psychosis, Fujii and Ahmed (2002b) developed a classification scheme for PSTBI that was adapted from Lishman’s (1998) description of the possible relationships between TBI and psychotic disorder. This conceptual framework is based on the assumption that psychosis is a neurobehavioral cognitive syndrome that results with sufficient damage to frontal and temporal structures and the dysregulation of the dopamine system (Fujii and Ahmed, 2002b). Tenets of this framework are presented in the Table and discussed below.

New Classification Scheme

In the first category, the development of psychosis is directly caused by the TBI. In these cases, there is no family history of schizophrenia and no or low genetic risk for psychosis. In the second category, TBI contributes to the development of psychosis. One way this can occur is if the patient has a history of schizophrenia and/or a family history of psychosis. In this case, TBI can act as a precipitating factor. Another way this can occur is if the patient has no family history of schizophrenia and no or low genetic risk for psychosis. As such, TBI can act as a precipitating factor.

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Differential with schizophrenia. Fujii and Ahmed (2002a) reported several potentially discriminating variables between PSTBI and schizophrenia. Patients with PSTBI appear to have fewer negative symptoms and are more likely to present with positive findings on magnetic resonance imaging (MRI) and EEG. The most common MRI findings for PSTBI are focal abnormalities in frontal and temporal areas, while for schizophrenia, the most common abnormalities are enlarged ventricles. The most common EEG finding in PSTBI is temporal slowing versus frontal slowing for schizophrenia.

Course and treatment. There is much variability in course of illness and treatment responsiveness, with some patients demonstrating relatively short courses and others being more chronic. Chronicity was associated with premorbid schizoid personality (Davison and Bagley, 1969). A higher percentage of schizophrenic psychosis (63%) versus delusional disorders (40%) demonstrated a chronic course (Achte et al., 1991). One study reported that neuroleptics were the most widely used treatment, followed by anticonvulsants and lithium (Fujii and Ahmed, 2002a). Considerations for selection of antipsychotic medication include increased sensitivity of the brain-injured to the sedating, anticholinergic and seizure threshold-lowering side effects of these drugs (Ahmed and Fujii, 1998). For example, chlorpromazine (Thorazine) has been reported to produce a delusional state along with worsening of cognition (Sandel et al., 1993). Although unsupported, some pharmacological strategies include the use of antikindling anticonvulsants for the treatment of late-onset psychosis and functional pharmacological approaches to treat specific functions such as attention and memory.

Shortcomings of Criteria

Ahmed and Fujii (1998) argued that the DSM-IV criteria for PSTBI are inadequate, as they do not provide guidance to determine whether psychotic symptoms are a direct physiological consequence of a previous head injury, or to aid in differential diagnosis. Problems in linking TBI to psychosis include determining what severity of TBI is significant enough to trigger a psychosis and how long after TBI is the brain injury considered etiologically significant. Differential diagnostic issues include determining diagnosis when there are potential multiple etiologies for psychosis such as substance abuse, epilepsy, TBI and schizophrenic illness.

Perhaps the most difficult differential diagnostic problem is discriminating patients with PSTBI from those with schizophrenia. As the aforementioned review of the literature suggests, there is much overlap in presentation. In addition, it is likely that many patients diagnosed with schizophrenia have sustained recent head injuries that are undocumented or actually meet the criteria for PSTBI (Burg et al., 1996; Fujii and Ahmed, 1996). To address the complex relationship between TBI and psychosis, Fujii and Ahmed (2002b) developed a classification scheme for PSTBI that was adapted from Lishman’s (1998) description of the possible relationships between TBI and psychotic disorder. This conceptual framework is based on the assumption that psychosis is a neurobehavioral cognitive syndrome that results with sufficient damage to frontal and temporal structures and the dysregulation of the dopamine system (Fujii and Ahmed, 2002b). Tenets of this framework are presented in the Table and discussed below.

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mentioned previously, the relationship between seizure disorder and psychotic disorder is well-established, and seizure disorder is a common sequelae of TBI. Traumatic brain injury may also contribute to the development of a psychosis by increasing biological vulnerability or risk. Vulnerability can be increased by damage to frontal and temporal structures or dysregulation of the dopamine system. These structures have been implicated in schizophrenia and many disorders associated with secondary psychosis. Damage to these structures may render the person vulnerable to developing a psychosis with additional damage or changes to these areas. In addition to direct structural damage, the sequelae of TBI—for example cognitive deficits, behavioral dyscontrol and emotional problems—can contribute to risk by increasing the individual’s psychological vulnerability in dealing with stress. A reduction in coping skills would render one vulnerable to stress that is associated with increases in the release of dopamine (Roth et al., 1988). Reduced coping abilities may also foster behaviors that would increase the risk for psychosis such as substance abuse and damage from future TBI.

Traumatic brain injury can also contribute to the development of a psychosis by triggering a psychotic episode in patients who have biological risk such as those with a genetic vulnerability for schizophrenia or those with a pre-existing seizure disorder. In these cases, the person already has significant vulnerability to develop a psychosis due to frontal systems and temporal abnormalities. Traumatic brain injury is the factor that raises them above the threshold for psychosis. For these patients, the development of psychosis may have been inevitable with additional damage or changes. Furthermore, it is possible that they may never develop a psychosis if good health is maintained.

The proposed subcategories for how TBI contributes to the development of a psychosis are not mutually exclusive. Thus, these subcategories should be conceptualized as illustrations of different ways that TBI can contribute to meeting the threshold for psychosis.

In the final category, the episode of TBI and the onset of psychosis are coincidental. The TBI may, however, exacerbate cognitive deficits or the severity and treatability of the psychotic condition. In these cases, there is likely a high genetic loading for schizophrenia. Traumatic brain injury may also be very mild without loss of consciousness, or it may be sustained after the onset of psychotic symptoms.

### Table

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<td>increasing biological vulnerability to the damage or changes to frontal systems and temporal abnormalities, the dopamine system will trigger a psychosis.</td>
<td>3. Ahmed II, Fujii D (1998), Posttraumatic psychosis. Semin Clin Neuropsychiatry 3(1):23-33.</td>
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