Neuropsychiatric Aspects of Traumatic Brain Injury

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Each year, more than 2 million individuals in the United States sustain a traumatic brain injury (TBI), leading to more than 500,000 hospital admissions and 80,000 survivors with persistent neurologic disabilities. The population at highest risk for TBI is in the 15- to 24-year age range, with a male-to-female ratio of approximately 3:2. The most common causes of TBI include motor vehicle accidents, falls, assaults, and sporting accidents. In patients older than 65, the most common cause is a fall.

Risk factors for TBI include substance abuse and psychiatric conditions associated with impulsive behaviors, such as bipolar disorder, cluster B personality disorders, and attention-deficit/ hyperactivity disorder (ADHD). These pre-injury psychiatric conditions are associated with high-risk behaviors that can lead to TBI. More than 50% of TBIs involve alcohol intoxication, and 30% to 60% of patients are intoxicated at the time of injury.

TBI involves the application of force to the brain that results in structural or metabolic alterations manifested by altered consciousness or focal neurologic deficits of varying severity and duration. An initial score on the Glasgow Coma Scale of 13 to 15 constitutes a mild TBI, while a score of 9 to 12 represents moderate TBI, and a score below 9 is severe TBI. Mild TBI accounts for about 80% of all cases, with moderate and severe cases each being responsible for 10%.

The American Congress of Rehabilitation Medicine has further refined the definition of mild TBI, stating that it is any traumatically induced disruption of brain function with limited loss of consciousness (30 minutes or less) or post-traumatic amnesia for less than 24 hours (Table). Given the fact that most TBIs are mild, clinicians may encounter orbitofrontal and anterior temporal cortex tend to be selectively damaged. Additional diffuse axonal injury (DAI) occurs at the white-gray matter junction and in connecting pathways throughout the brain. Often undetectable through structural neuroimaging methods such as CT and MRI, DAI results from shearing forces applied to the brain during rapid acceleration and deceleration of the head. These acceleration-deceleration mechanics do not require the blunt force impact that occurs when a moving head strikes a stationary object or vice versa. They can also occur through rotational mechanics, such as whiplash injuries. Thus, the absence of external stigmata of injury or structural neuroimaging abnormalities does not rule out the presence of a TBI. It is these mild TBIs that while leaving few, if any, lasting neurologic or cognitive sequelae, may lead to disruptive psychiatric syndromes that are far from mild.

Secondary injuries from edema or hemorrhage result from increased intracranial pressure in severe trauma. However, TBI may also activate intracellular processes involving excitatory amino acids that lead to cell death. This process is initiated within hours of injury, although the cascade of events that ultimately cause cell death may take days or weeks. The precise mechanism that mediates excitotoxic cell death is not understood, but it is possible that prolonged excitation of neurons may secondarily cause intracellular metabolic disruption. While clinical trials of excitatory amino acid antagonists have been disappointing, these studies have targeted a more severely injured population. The clinical manifestation of this process in mild TBI may be the progressive emergence of neuropsychiatric symptoms over several days or weeks following injury.

Cognitive deficits

Given the reliance of higher cognitive functions on distributed rather than focal processing, DAI tends to profoundly affect the executive cognitive functions even in the absence of neurologic or focal cortical deficits. Acquired deficits in attention, concentration, and vigilance may resemble those of ADHD, although the postinjury onset or worsening of these deficits helps clarify the cause. Deficits
in executive cognitive functioning may particularly affect persons in occupations that are highly
dependent on rapid decision making and the complex use of information.
In uncontrolled studies, the cholinesterase inhibitor donepezil (Aricept) has demonstrated some
beneficial effects on TBI-related memory deficit. The psychostimulants have demonstrated efficacy
in improving attention, concentration, and vigilance, but patients should be monitored for increased
irritability. Clinicians must also take into consideration these acquired cognitive deficits when
providing instructions and education to patients, who may be easily overwhelmed by the information
exchange.

**Aggression and impulsivity**

Patients who sustain multiple head injuries over time appear to demonstrate increased irritability
with each subsequent injury, particularly when the injuries are associated with loss of
consciousness. Premorbid risk factors for aggression include a history of impulsive aggression,
arrest, and substance abuse. Aggression is also frequently encountered in post-TBI mania, which
occurs in 7% to 9% of patients independent of severity of injury, cognitive impairment, or physical
disability. While post-TBI manic syndromes may resemble the classic manic syndrome of euphoria,
elation, increased energy, and grandiosity, a more common presentation is a dysphoric mixed
bipolar syndrome. Treatment of post-TBI aggression aims to reduce disruptive behaviors without negatively impacting
other areas of functioning. Anticonvulsants appear to be effective and well-tolerated in treating
these disorders, although cognitive impairment may occur at higher doses. Traditional
antipsychotics have been associated with cognitive impairment in TBI patients, but
second-generation antipsychotics appear to be better tolerated.

**Depression**

The prevalence of major depression following TBI ranges from 15% to 61%. Estimates are limited
by the wide variety of methodologies and diagnostic criteria. Some investigators noted that fatigue,
frustration, poor concentration, boredom, and distractibility were common in depressed TBI patients,
but feeling sad or blue was not as common. On the other hand, Jorge and associates found that
feelings of depression and sadness did, in fact, discriminate between depressed and nondepressed
patients and suggested that cognitive impairment and fatigue were not useful diagnostic symptoms
in this population. Treatment of post-TBI depression with antidepressants appears to be effective, as
is psychotherapy and, when necessary, electroconvulsive therapy. Effective treatment is considered
crucial to maximizing cognitive and psychosocial functioning, which are often compromised by
depressive symptoms.

**Summary**

TBI is frequently complicated by neuropsychiatric symptoms that are multiply determined. The
complex interaction between neurobiologic changes and the external social environment may lead
to devastating psychosocial morbidity, even in the absence of profound neurologic or cognitive
impairment. Increased vigilance for previously undiagnosed or incidental TBIs in general mental
health populations may lead to more effective clinical management.

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