Psychosocial Impact of Brain Injury: A Review

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Abstract

Traumatic brain injury (TBI) is known as "silent epidemic" because of lack of knowledge about it and its delayed symptoms of memory and cognitive problems. The incidence is approximately 1.4 million per year in the United States, and in the developing countries, it is approximately 341 per 100,000. In the United States, the annual productivity loss due to TBI and its delayed effects is more than $56 billion. TBIs occur mainly due to traffic accidents and falls. All the age groups are affected, but the rates are higher in men than in women. The causes vary as per the age groups as traumas affect mainly children and elderly, and traffic accidents and violence affect more of adolescents and young adults. Data from various studies indicate wide variation in the incidence and prevalence of psychiatric disorders after TBI, but they are definitely higher as compared with general population. We also noted different psychiatric disorders in post head injury cases during follow-up at our university hospital, which included depression, behavioral disorders, cognitive disorders, memory impairment, anxiety disorders, stress-related headache, vertigo, irritability and sleep disorders, etc. Therefore, this review was undertaken to assess the psychosocial impact of brain injury on an individual.

Keywords
- traumatic brain injury
- psychiatric disorders
- psychological effects
- social effects

Introduction

Traumatic brain injury (TBI) is known as "silent epidemic" because of lack of knowledge about it and its delayed symptoms of memory and cognitive problems. The incidence is approximately 1.4 million per year in the United States,[1] and in the developing countries, it is approximately 341 per 100,000. In the United States, the annual productivity loss due to TBI and its delayed effects is more than $56 billion.[2] TBIs occur mainly due to traffic accidents and falls.[3] All the age groups are affected, but the rates are higher in men than in women. The causes vary as per the age groups as traumas affect mainly children and elderly, and traffic accidents and violence affect more of adolescents and young adults.[3] Data from various studies indicate wide variation in the incidence and prevalence of psychiatric disorders after TBI, but they are definitely higher as compared with general population. Therefore, this review was undertaken to assess the psychosocial impact of brain injury on an individual.

Also, the unpublished data of 56 TBI cases at our university hospital with 3 weeks to 9 months follow-up revealed that 30 (53.5%) of 56 patients had one or other psychiatric disorders. This incidence seems to be very high and is important because our university catches patients from rural areas of Uttar Pradesh; thus, it appears that the people in rural areas are more vulnerable for emotional and psychological disturbances.

Impact on Individual

Depression

The incidence rate of depression post TBI vary from 15.3 to 33% and prevalence rate from 18.5 to 61%.[4] Symptoms vary from transitory responses to stressing situations to pathologic conditions, and also there is coexistence of symptoms of depression, adjustment disorder, and grief.[5] Also, it is difficult to differentiate somatic symptoms of depression from symptoms directly caused by TBI. The symptoms include fatigue, less involvement in activities, insomnia, decreased appetite, and concentration.[6]

Depression after TBI may occur due to reduction in left prefrontal gray matter volume,[7] lesions in dorsolateral prefrontal cortex and left basal ganglia,[8] and also in lateral and medial frontal lobe,[9] leading to rupture of neural circuits of the prefrontal cortex, amygdala, hippocampus, basal ganglia, and thalamus. This leads to mood disorders[10] and

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also the low levels of serotonin encountered after TBI may cause emotional changes, disinhibition, and aggression seen after TBI. The lesion in hippocampus and alteration in its morphology has also been reported in cognitive and depressive disorders.[11,12]

Poorer social functioning,[8,13] high levels of work dissatisfaction, unemployment,[7,14,15] low economic status,[7] less education,[15] and lack of close personal relationships[13] have been reported in patients who develop depression following TBI. Also, psychiatric comorbidity is common in depression after TBI. Frequently depression and anxiety coexist mainly related to injuries in the right hemisphere.[16] Isolated depression was more in lesions in the anterior areas on the left.[7] Also in our findings, 3 of our 28 cases having left-sided lesions had depression whereas 4 had behavioral disorders, and further, the behavioral disorders and depression coexisted in 2. However, 1 of 17 right-sided lesions had depression. Thus, it appears that psychiatric disorders are encountered more in left-sided lesions as compared with right.

Mania

The reported prevalence is 4.2% for mania directly caused by TBI[17] and increases to 9% within 12 months of follow-up.[18] The symptoms include aggression, irritable moods, and euphoria.[19] It is associated with lesions in the temporal basal poles and orbitofrontal cortex mainly in the right hemisphere.[18–21] Also, it has been reported that there are focal lesions in areas connected to the limbic system in the right hemisphere, or anterior subcortical atrophy, lack of inhibitory function of the frontal cortex on subcortical limbic structures due to abnormalities in these circuits.[22] The bipolar disorders are common following TBI.[23]

In our study, mania was not documented in any of the 30 cases, although there were four pure temporal lesions, two on either side barring frontotemporal which were six.

Obsessive-Compulsive Disorder

The prevalence varies between 0.7 and 1.6% and is similar to the general population.[17] The main symptoms include obsessive slowness and compulsive exercise practice. Patients with obsessive slowness had compromised performance in neuropsychological tests for executive function, memory, and language.[24,25] Obsessive-compulsive disorders (OCDs) are mainly related to lesions in frontal and subcortical areas, mainly the orbitofrontal cortex, caudate nucleus, and anterior cingulate cortex.[25,26]

There is lack of study related to psychosocial factors in OCD after TBI. It is a condition with a biological basis, but it disrupts the rehabilitation process. Psychiatric comorbidity is common in patients with OCD after TBI.[27]

Posttraumatic Stress Disorder

The reported prevalence is 13.9% in post TBI cases mainly influenced by the severity of TBI and posttraumatic amnesia due to the formation of pathologic memories. Posttraumatic stress disorder (PTSD) symptoms were not reported in patients who had brief episode of unconsciousness and but had developed amnesia after TBI.[28–30] The increase in the astrocytic protein S-100B has been reported in PTSD cases following TBI 1 year later[31] in patients in whom traumatic memories involved amygdala, hippocampus, and other related structures.[32]

Patients with post TBI PTSD had reduced quality of life, poorer productivity functioning,[33] and reduced insight.[34] Also, the comorbidity with depression and anxiety is common in PTSD after TBI.[33–36]

Psychotic Disorders

Post TBI psychosis is rare, and the reported incidence is 0.1 to 9.8%.[37] The studies revealed that psychotic patients are more predisposed to suffer trauma[38] and genetic background for schizophrenia increases this.[39] In the acute phase of TBI, the psychotic symptoms are delirium, delusions, reference, control, and grandiosity[40] whereas the hallucinations appeared later (i.e., > 2 years after the TBI) and may be auditory or visual. Aggressive behavior along with negative symptoms, disorganization, and catatonia were also reported. Prodrome symptoms include depression, antisocial and inappropriate social behavior, social withdrawal, and deterioration at work.[41]

Post TBI psychoses had electroencephalographic abnormalities in the temporal lobes that may be accompanied with seizures,[42] also the focal lesions or brain atrophy in the frontal and temporal lobes.[40–44] The increased susceptibility has been reported in patients with neurologic diseases or previous TBI.[42]

The psychotic symptoms result from impairment of neural circuits in the frontal and the temporal lobes, leading to an increase in the temporal limbic activity. There is lack of studies related to psychosocial factors in psychosis after TBI.[45]

Disorders Related to Alcohol

Alcohol is a psychoactive substance and is a leading cause of traffic accidents, falls, and violence leading to TBI. Alcohol dependence was reported in 24.1% and abuse in 10.8% of the TBI cases.[46] The brain atrophy has been reported in TBI patients with history of moderate or heavy use of alcohol[47] along with reduction in prefrontal gray matter volume.[10] The neuronal loss related to alcohol has been reported in the frontal cortex, hypothalamus, cerebellum, hippocampus, amygdale, and locus coeruleus.[48]

Traumatic brain injury and alcohol use individually produced mild alterations in event-related potential testing, but changes were greater when both conditions coexist.[49]

Also, patients who did not sustain abstinence after TBI had more mood disorders[10] whereas depression, anxiety, suicidal thoughts, violent behavior, difficulties for concentration, and use of cannabis were reported in the individuals with previous TBI along with higher rates of depressive and anxiety symptoms, antisocial personality, and suicidal attempts.[50,51]

Personality Changes

Apathy

Apathy means disorders of decreased motivation, abulia, and akinetic mutism. The incidence varies from 10 to 46.4%.[52,53] It is mainly seen in the lesions involving the subcortical areas or right hemisphere, causing damage to cortico-striatal-pallidal-thalamic pathways involving the anterior cingulate cortex, accumbens nucleus, ventral pallidum, and medial dorsal thalamic nucleus, which are the mediators of motivation leading to akinetic mutism, abulia, and apathy.[54] Further, the orbitofrontal cortex, amygdala, hippocampus, and tegmental ventral area are also involved in the motivational state related to rewards. Lesions in these structures also produce apathetic symptoms. Dopamine is the main neurotransmitter linked to apathy as it has role in the mechanisms of novelty seeking, reward, and response to unexpected events.[55]

Affective Lability

These include emotional instability or rapid mood changes, involuntary emotional expression disorder, and pathologic laughing and crying,[56] which is unrelated to the subjacent mood or independent from usual provoking stimuli.[55,57] Also associated are aggression and anxiety in the left-sided frontal lobe lesions. It has been reported that impairment of cerebro-ponto-cerebellar pathways leads to incapability of the cerebellar structures to get adjusted to the execution of laughing or crying according to the environmental stimuli, thus leading to inappropriate or chaotic emotional expression.[58] The various neurotransmitters involved in these expressions are serotonin, dopamine, and glutamate.[59]

Aggression

Aggression is damaging, threatening, or intimidating behavior that may be impulsive or premeditated or episodic dyscontrol, with recurrent crises of out-of-proportion fury due to provocation or frustration[60,61] along with the antisocial behavior with-the inconsideration for moral and social principles.[62,63] The main characteristics of aggression after TBI are impulsivity and anger[64] with the incidence of 14.4 to 33.7%.[16,65] Also, it is associated with substance abuse, male sex, TBI severity, intelligence level, and low socioeconomic status.[5,66–70]

The impulsive aggression may result from failure to regulate negative emotions, such as anger. Threatening environmental stimuli are transmitted to amygdala, from where they are relayed to the basal ganglia where they are integrated with social information from the orbitofrontal cortex. The behavioral responses are then initiated through projections toward the other cortical areas, hypothalamus, or brainstem. The orbitofrontal cortex, dorsolateral prefrontal cortex, and anterior cingulate cortex inhibit the activity of amygdala, thus forming a regulatory mechanism that is lost in patients with injuries in these areas leading to the propensity to impulsive aggression.[71] The studies have reported the role of serotonin in aggressive behavior[62,72] along with polymorphisms in the tryptophan-hydroxylase enzyme gene.[71]

Other Personality Changes

The personality changes reported in post TBI cases are behavioral disinhibition, aberrant sexual behavior, hypersexuality, moria, and self-awareness impairment.[21,73–75] These are attributed to the frontal lobe impairment as it modulates the primary responses that come from other regions, such as the limbic system and motor cortex.[20]

Conclusion

Reviews of various studies reveal that TBI affects the individual adversely psychosocially. We also noted different psychiatric disorders in post head injury cases during their follow-up, which included depression, behavioral disorders, cognitive disorders, memory impairment, anxiety disorders, stress-related headache, vertigo, irritability and sleep disorders, etc. The incidence was as high as 53.5% in a rural university catering mainly rural population.

In the Indian context, TBI and psychiatric disorders related to it have special importance as the road traffic accidents are higher in India, so psychiatric disorder follow-up is also common. Main psychiatric disorders are impulsive or irritable behavior followed by cognitive changes, depression, and behavioral problems that may be stress related. Often, these things are ignored, which may hamper productivity of the country. A detailed analysis with well-planned study will reveal better outcome.

Conflict of Interest
None.

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