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Neuropsychiatric Sequelae of Head Injury

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Abstract: Almost half of people suffering head injury may later be diagnosed with neuropsychiatric disorders. Psychiatric disturbances associated with head injury are reviewed. Authors highlight the close link between head injury and psychiatry and provide an overview of the epidemiology, risk factors, and mechanisms of psychiatric sequelae including cognitive deficits, substance abuse, psychoses, mood disorders, suicide, anxiety disorders, dissociative disorders, post-concussion syndrome and personality changes following head injury. Various neuropsychiatric sequelae are briefly discussed and the respective treatments are outlined with emphasis on a multidisciplinary approach.

Keywords: Head injury; Psychiatric sequelae; Cognitive Sequelae; Personality change; Post-Concussional syndrome.

INTRODUCTION

Driving in the U.S. is on the right side,
Driving in the U.K. is on the left side,
Driving in India is Suicide!

This pithy aphorism graphically tells us the sad state of affairs on the roads of India. We have the highest accident rates per vehicle in the world. Our mortality and morbidity rates are also correspondingly high. Head injury accounts for a very high percentage of these figures and is emerging as one of the biggest killers. Needless to add that as the number of vehicles (especially two wheelers) goes up, the scenario will only worsen. Despite the emphasis placed on physical deficits during the early stages of recovery from a severe brain injury, it is cognitive and behavioral deficits that give rise to the major morbidity, which most impairs the capacity to return to work and maintain social activities[1]. Psychiatric disorders following head injury are important for a number of reasons. Firstly, head injury accounts for most cases of permanent disability after trauma. Given that trauma injuries occur most frequently in the young (<45 years), the cost, both to the individual and to society, is enormous[2]. Secondly, the psychological sequelae of head injury may frequently be overlooked, and as such, under treated. Nowhere is this more apparent than in people with mild head injury, who comprise over 85% of all head injuries, and who routinely do not receive adequate follow up care[3]. Improved clinical management of head injury has significantly reduced mortality. However, a large portion of these surviving patients will have cognitive or emotional sequelae and will need psychiatric interventions years after the injury. It is therefore essential to increase awareness of these sequelae so that psychological intervention is planned as early as possible, in hopes of improving function and limiting disability. In this paper we briefly review neuropsychiatric disorders following head injury.

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BIOLOGICAL MECHANISMS

Mechanical forces applied to the skull and transmitted to the brain lead to focal and/or diffuse brain damage. Focal lesions often result from a direct blow to the head and include brain laceration, contusion, intracerebral hemorrhage, subarachnoid or subdural hemorrhage, and ischemic infarct. Contusion occurs directly beneath or contralateral to the site of impact, commonly referred to as *coup* and *contre-coup* injury. It is most common in the orbital–frontal area and the temporal tips, where acceleration/deceleration forces cause the brain to impact on the bony protuberances of the skull. Diffuse brain injury also results from the differential motion of the brain within the skull, causing a shearing and stretching of the axons. This can produce a wide spectrum of injuries, ranging from brief physiological disruption to widespread axonal tearing, called diffuse axonal injury (DAI). Much of the damage sustained in head injury is immediate (referred to as primary injury), including DAI, cortical contusions, and disruption of small blood vessels. DAI after head injury is usually observed in the corpus callosum and in the brainstem[4]. It disrupts neuronal circuits directly, and may also disrupt neurotransmitter systems such as norepinephrine, serotonin, dopamine, and acetylcholine[5,6,7]. Hypoxia may lead to free radical and excitotoxic neurotransmitter release, which cause further neuronal damage to these systems.

Head injury may cause contusional injuries affecting brain regions involved in the mediation of mood, especially "along the temporal lobes and frontal cortex"

In addition to brain damage occurring at the time of the impact, secondary damage from several processes may occur during the recovery period. These include hypoxia, hemodynamically significant cerebral vasospasm, anemia, metabolic abnormalities, hydrocephalus, intracranial hypertension, fat embolism, and subarachnoid hemorrhage. Other delayed effects include release of excitatory amino acids, oxidative free-radical production, release of arachidonic acid metabolites, nitric oxide synthase-2, and disruption of neurotransmitters like monoamines and serotonin[8,9]. Some of the changes to neurotransmitter systems may occur weeks after the initial head injury. Ciallella et al[10] found no changes in vesicular acetylcholine transporter protein or in M2 receptors at 1 day and 1-week post-head injury in rats. At 2 and 4 weeks, however, a 40%–50% increase in vesicular acetylcholine transporter protein and a 25%–30% decrease in M2 receptors were observed. These changes particularly involved the hippocampus and may have occurred in response to chronically lowered acetylcholine neurotransmission, which has been demonstrated after head injury in rats[11]. Hyperactivation of glutamate NMDA receptors after injury is short-lived (<1 h) and is followed by a profound and long-lasting (e"7 days) loss of function. Furthermore, stimulation of NMDA receptors by NMDA 24 and 48 h post-injury produced a significant attenuation of neurological deficits (blocked by co administration of NMDA receptor antagonist MK801) and restored cognitive performance 14 days post-injury. These results provide the underlying mechanism for the well known but heretofore unexplained short therapeutic window of glutamate antagonists after brain injury[12].

Risk Factors for Neuropsychiatric Disorders

The major risk factors for neuropsychiatric disturbances after head injury include increasing age, arteriosclerosis, and alcoholism. These delay the reparative process within the central nervous system[13]. Premorbid personality also a plays a significant role in the process of rehabilitation, as was pointed out by Symonds[14]: *"The late effects of head injury can only be properly understood in the light of a full psychiatric study of the individual patient... it is not only the kind of injury that matters, but the kind of head"*. Similarly, factors such as marital discord, poor interpersonal relationships, problems at work, or financial instability are important contributors to the neuropsychiatric disability.

Prevalence Neuropsychiatric Sequelae of Head Injury

Almost half of people suffering head injury may later be diagnosed with neuropsychiatric disorders. A 30-year retrospective follow-up study of 60 head injury patients showed that 48% had a DSM IV axis I disorder that began after head injury; 62% had an axis I disorder during their lifetimes. Common new disorders following head injury were major depression (27%), alcohol abuse or dependence (12%), panic disorder (8%), specific phobia (8%) and psychotic disorders (7%). Twenty three percent had at least one personality disorder[15]. A prospective study of 939 head injury patients revealed that the prevalence of any psychiatric illness in the first year was 49% following moderate to severe head injury and 34% following mild head injury**.** Whereas moderate to severe head injury is associated with a higher initial risk, mild head injury may be associated with persistent psychiatric illness[16]. Findings of few Indian studies are summarized in Table 1.

Table 1. Neuropsychiatric sequelae of head injury (all figures in percentages)

Sequelae	Chatterjee & Kishore [17] (n=37)	Keshavan et al [18] (n=60)	Sabhesan et al[19] (n=134)	Chaudhury et al [20] (n=146)
Cognitive	16.2	3	2.2	3.4
Personality	8	-	7.5	4.8
Psychoses	18.9	1	14.9	8.9
Neuroses & others	-	47	12.7	20.5
Total	**43.2**	**51**	**29.1***	**37.7**

* Total is less as some patients developed symptoms sequentially

A recent literature review concluded that major depression was the most common psychiatric disorders after head injury, at approximately 44%. Mania was much less frequent, at approximately 4%. The anxiety disorders were common, ranging from 6.5% for OCD to a high of 14% for PTSD. Substance abuse was also fairly common, at 22%, while psychosis was uncommon at less than 1%. Use of these data, however, generated estimates of relative risk (RR) as follows. The highest relative risk was for major depression, with an RR of 7.5. Bipolar disorder also had a high RR of 5.3. The RRs for the anxiety disorders was 2.0, with the exception of panic disorder with an RR of 5.8. The RRs for schizophrenia and substance abuse were close to or less than 1.0, suggesting either no, or a minor, increased risk for these disorders[4].

1. **Cognitive deficits:** Cognitive deficits are common after head injury and are classified as delirium, dementia, amnestic disorder, or mild cognitive deficit, depending on the variety of symptoms and their time of onset and resolution. They include impairment of arousal, attention, concentration, memory, language, and executive function. The cumulative effects of focal and diffuse

brain damage cause the cognitive deficits. Cognitive outcome depends on a number of factors, such as degree of diffuse axonal injury, duration of LOC and PTA, clinical clinical evidence of brain stem dysfunction at the time of injury, and presence and size of focal hemispheric injury.

Executive functioning: Disturbances of executive functioning include poor planning, organizing, sequencing, and set-shifting, with impaired judgment and impulse control.

Alertness: may be impaired in severe head injury. The patient may be withdrawn, dull, and apathetic. Deficit of alertness often accompany deficits of motivation.

Attention: Our ability to maintain a goal-directed focus without support from the environment requires the endogenous control of behaviour. Fronto-parietal circuits modulate this control. This ability is compromised following head injury leading to increased lapses of attention[21].

Memory: Memory deficits are the most prevalent complaints. The degree of memory impairment is correlated with the length of PTA. Newly acquired knowledge is forgotten. Selective impairment of memory may persist depending upon the circumscribed damage to structures in diencephalon or medial temporal lobe structures. Both recent and remote memory may be impaired, but immediate memory may be spared[22].

Perception: Visual dysfunction affects about 50% of head injury patients. Visuo-perceptual disturbances such as impaired figure-ground perception and constructional abilities may be present in severe head injury as part of a general cognitive decline. Focal visuo-perceptual and visuo-constructive disabilities are rare[23].

Language: Anomia and word finding difficulties are present after head injury. Expressive aphasias are more common compared to receptive aphasias. Recovery from aphasia and related language disorder is greater than recovery from memory and other cognitive deficits when patients were tested after one year[24].

Intelligence: Both performance and verbal IQ are reduced in acute and chronic phases of severe head injury, but not after mild head injury. Recovery of verbal IQ is faster. Performance IQ may be lower even after three years. Approximately 10% of head injury patients with prolonged coma develop some degree of hydrocephalus, which may present as progressive intellectual deterioration. The "punch drunk" syndrome may be seen in boxers 5-40 years after retirement[25].

2. Substance abuse: Many head injury patients are intoxicated at the time of injury. Presence of high alcohol levels in blood not only has a negative impact on length of unconsciousness, behavioral changes, and neurocognitive changes but can also affect mortality. Tureci et al[27] reported a possible protective role for alcohol especially at lower doses in mice. Ethanol inhibition of NMDA-mediated excitotoxicity that predominates at lower doses is believed to be responsible for this protection.

3. Psychoses: There are several substantial clinical, epidemiological, and neurobiological differences between the post-traumatic psychoses and the primary psychotic disorders[28]. Psychosis secondary to head injury (PSTHI) occurs in 4% to 8.9% of individuals who sustain head trauma. Despite its rarity, PSTHI 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 PSTHI have family histories of psychotic disorder, while many others do not; and 3) the disorder has conceptual relevance to understanding schizophrenia spectrum disorders. The mean latencies between head injury and onset of psychosis is between four to five years, but can range from a few days to over 20 years. Despite the wide range of latencies, studies suggest that about half of patients with PSTHI demonstrate symptoms within the first year, and roughly 72% of patients have symptoms within the first five years[29]. There is some evidence suggesting that the duration of the latency between head injury and onset of psychosis may have clinical significance. Latencies of less than one year have been associated with diffuse injuries, paranoid symptoms and visual hallucinations. 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. In terms of prodromal symptoms, roughly half of patients with PSTHI 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[29,30].

Although PSTHI has been associated with many forms of delusions, including grandiose, referential, religious and Schneiderian symptoms, the most common symptoms are paranoid or persecutory delusions that are present in up to 80% of all patients. Auditory hallucinations are also common, with 60% to 93% of patients presenting with this symptom. Visual hallucinations, negative symptoms and

formal thought disorder are relatively rare, occurring in 8% to 32%, 15% to 22.2%, and 4.4% of patients, respectively[29,30]. Localization data from different populations and methodologies, including imaging, EEG and lesion location, consistently report abnormalities in the temporal areas[29,31]. Less consistent, but also common, are abnormalities in the frontal lobes[29,32]. There is no consistent finding for hemispheric laterality of lesions. Neuropsychologically, impairments in memory and executive functioning have been the most consistent finding. A review of the literature[31] revealed that 0.7%–9.8 % of patients with head injury develop schizophrenia-like psychosis. Most of these patients do not have a family history of schizophrenia. Paranoid psychoses can occur independently or as part of post-traumatic dementia. The psychotic features may be acute or chronic, transient or persistent, and may or may not be associated with mood disturbances. Chronicity was associated with premorbid schizoid personality[31]. A higher percentage of schizophrenic psychosis (63%) versus delusional disorders (40%) demonstrated a chronic course[32].

4. **Mood disorders:** is a frequent complication that exerts a deleterious effect on the recovery process and psychosocial outcome of brain injured patients[33].

Major depression: occurs in approximately 25% of patients with head injury. It is associated with executive dysfunction, negative affect, and prominent anxiety symptoms. Depression occurs more frequently with left dorsolateral frontal and left basal ganglia lesions. The mechanism of depression following head injury is probably due to disruption of biogenic amine-containing neurons as they pass through the basal ganglia or frontal-subcortical white matter. Feelings of loss, demoralization, and discouragement seen soon after injury are often followed by symptoms of persistent dysphoria. Suicide potential should always be evaluated[34]. Fatigue, irritability, disinterest, and insomnia are seen in a substantial number of patients 6–24 months or even longer after head injury. Psychological impairments in excess of the severity of injury and poor cooperation with rehabilitation are strong indicators of a persistent depressive disorder. Depression commonly occurs during the period of recovery of other functions. The recovering patient must come to terms with his new physical and mental limitations, and may psychologically mourn those functions impaired or lost. The common symptoms of depression may be less pronounced in TBI patients due to overall personality flattening. Erratic or poor recovery, or worsening of a neurological deficit after initial recovery, may be signs of depression. Poor premorbid levels of functioning and past history of psychiatric illness are major risk factors for depression.

Mania: after head injury is less common than depression but much more common than in the general population supporting the contributory role of trauma in its etiology. It is seen in about 9% of patients. Mania occurs most often with lesions in right-sided limbic or limbic related structures. In post-traumatic mania irritable mood is more common than euphoric mood. Almost 50% of post-traumatic manic patients have abnormal EEG. Changes in mood, sleep, and activation may manifest as irritability, euphoria, insomnia, agitation, aggression, impulsivity, and even violent behavior.

5. **Suicide** is considerably increased after head injury and accounted for 14% of all deaths in an 18-year follow-up of those with war brain injuries. Change of character, alcoholism and interpersonal difficulties are frequently present. An association with lesions in frontal and temporal lobes has been reported[35].

6. **Anxiety disorders**, including generalized anxiety disorder, panic disorder, phobic disorders, posttraumatic stress disorder, and obsessive–compulsive disorder are common after TBI and range in frequency from 11%–70%. TBI patients often experience generalized "free-floating" anxiety associated with persistent worry, tension, and fearfulness. Increased activity of the aminergic system and decreased activity of the GABA inhibitory network is the proposed mechanism for the clinical manifestation of anxiety. Right-hemispheric lesions are more often associated with anxiety disorder than left-sided lesions.

Posttraumatic Stress Disorder (PTSD): Mayou et al[36] found that PTSD is "not associated with a neurotic predisposition" but is "strongly associated with horrific memories of the accident". PTSD did not occur, in their sample, in subjects who lost consciousness during the head injury or who were amnesic for the event. Similarly, while 6 (13%) of 47 veterans who had suffered a moderate head injury and who were amnesic for the event developed avoidance and arousal criteria of PTSD, none developed the full syndrome, and none met the criteria of re-experiencing the event[37]. Women were predisposed to develop PTSD: 6 of 10 women, versus 2 of 14 men, developed PTSD following head injury[38]. PTSD occurred in 82% of mild- head injury patients who had experienced acute stress disorder earlier (1 month post injury), but in only 11% of those who did not suffer acute stress disorder[39].

7. **Dissociative (Conversion) symptoms** including fits, fugues, amnesia, Ganser states, paralysis, anaesthesia, and disturbance of speech, sight or hearing are not uncommon. A neurasthenic reaction may incapacitate the patient for months or even years.

8. **Post-Concussional Syndrome (PCS):** Individuals sustaining mild head injury often report a constellation of physical, cognitive, and emotional/behavioral symptoms referred to as PCS. The most commonly reported symptoms are headache and other pain, dizziness or light headedness, memory and concentration difficulty, amnesia, sleep disturbance, frustration, irritability, fatigue and weakness, visual disturbances, sensitivity to light, sensitivity to noise, tinnitus, periods of confusion or mental dullness, emotional and behavioral changes, loss of self-confidence, slow reactions, judgment problems, depression, and anxiety[40]. PCS with an incidence of 43% was the commonest neuropsychiatric sequelae after head injury in a prospective Indian study[41]. Onset of PCS is usually during the first month after head injury. Most patients recover within 3–6 months after injury. However, about 15% of patients will have symptoms lasting longer than 1 year. Though, non-organic factors are certainly important in causation, the underlying pathogenesis is thought to be diffuse axonal injury from acceleration and deceleration forces. PCS patients have a reduction in the volume of blood flowing through the brain as well as prolongation of mean cerebral circulation time. Abnormal audiologic examination has been reported in PCS patients complaining of dizziness, hearing disturbances and tinnitus. MRI is more sensitive to abnormalities after head injury than CT scan. However, neuropsychologically significant abnormalities on MRI may not become fully apparent until some time after head injury. PET has demonstrated changes in cerebral glucose metabolism greater than would be expected by the lesions detected by CT and MRI scans. SPECT studies have shown focal abnormal regional cerebral blood flow.

9. **Personality change**: Commonly reported changes include excessive tiredness, indifference, concentration and attention disorders, inflexibility, tendency towards perseveration, absence of ability to anticipate, behavioral disinhibition irritability, a change in quality of relationship with more shallowness, and obsessive-compulsive symptoms. van Reekam[4] reported on DSM personality disorders, with avoidant, borderline, and narcissistic personality disorders being the most common. The numbers, however, are very low. Another study involving 60 patients assessed on an average thirty years after TBI showed that (23.3%) had at least one personality disorder. The most prevalent individual disorders were avoidant (15.0%), paranoid (8.3%), and schizoid (6.7%) personality disorders. Nine patients (15.0%) had DSM-IIIR organic personality syndrome[15].

Personality changes may occur due to direct result of disturbances of neural tissue, or due to indirect effects of the brain injury such as the individual's reactions and responses to impairments, environmental factors, premorbid personality and mental constitution and so forth. These two etiologies of personality changes are not necessarily mutually exclusive, and the relative contribution of organic and psychogenic factors can be particularly hard to disentangle[13]. Some localization of personality change due to frontal lobe injury has been reported. Luria describes two main variants of frontally-mediated changes in personality and emotions: (i) *pseudopsychopathic* seen with lesions in the orbitobasal aspects of the frontal lobes manifesting with euphoria, impulsiveness and inadequate actions, which may be superimposed on a background of disinhibition, and (ii) *pseudodepressed* after lesions of the convexity regions of the frontal lobes manifesting with narrowing of interests and generalised emotional indifference, superimposed on a background of general inhibition and torpidity. Medial frontal syndrome is characterized by akinesia, sparse verbal output and incontinence. Clinically a mixture of these syndromes is more commonly seen. Behavioral sequelae are very common and largely account for the distress to care givers much more than physical disabilities. A greater severity of behavioral sequelae is found in patients who have an abnormal EEG or compressed ventricles on early CT scans indicating significant brain edema. Behavioral sequelae are known to correlate with severity of injury, extent and degree of disturbed behaviour during recovery, though again such relationship is not linear.

Aggression: Physical/verbal aggression and impulsiveness are particularly difficult for family members to manage. It may occur alone or as a manifestation of another psychiatric disorder. Sometimes it merely represents an exaggeration of previous personality.

Apathy: Ten percent of patients tend to have apathy without depression, and 60% have some degree of apathy and depression following head injury. Apathy refers to a syndrome of disinterest, disengagement, inertia, lack of motivation, and absence of emotional responsivity. The negative affect and cognitive deficits seen in patients with depression are not seen in patients with apathy. Apathy may be secondary to damage of the mesial frontal lobe.

Sexuality: Limbic structures particularly amygdala, septal

nuclei, hypothalamus, and various cortical areas which form part of the neuroanatomic and physiologic substrate of human sexual behaviour may be damaged in head injury, resulting in impaired sexuality. Frontal lobe injuries with resultant disinhibition, lack of social judgment, difficulty in modulating and initiating sexual overtures can profoundly affect the sexual responses.

Sleep: disturbances after a head injury have received very little scientific attention despite the fact that several studies indicate that they may occur in 30% to 70% of patients. For individuals with head injury, problems falling asleep or maintaining sleep can exacerbate other symptoms such as pain, cognitive deficits, fatigue, or irritability. Sleep disturbances can thus compromise the rehabilitation process and the ability to return to work.

Headache: is a common symptom and at times a persistent and disabling sequelae of head injury. It may occur alone or as part of a syndrome.

MANAGEMENT

The goals of management of head injury patients include a variety of domains including drugs and rehabilitative techniques. One has to acknowledge that management of such cases is by no means a quick one but requires a sustained effort. The aim of the management is to help the patient achieve his optimal level of functioning. It is important to understand the nature of head trauma to ascertain the severity of injury and the likely sequence of recovery. The level of disability at various stages following the injury needs to be assessed very clearly to look for the amount of recovery made in various spheres. For example if a patient after severe TBI continues to have severe memory problems, it is likely that he may require a shift from his occupational status as he may not be able to cope with his work and may end up feeling frustrated. At the same time it is of paramount importance to keep the family members informed about his clinical status and the level of disability. It helps the patient in adjusting to his social and occupational spheres. The duration of service need is also extremely variable. Those with relatively mild injuries may require a brief period of rehabilitation and then may return essentially to their premorbid status. Others, however, will have severe, lifelong deficits that require a shift from a medical treatment model to a psychosocial and environmental support model over time.

1. **Rehabilitation:** It encompasses cognitive rehabilitation, behavioral treatment, social skills training, vocational training, individual therapy, group therapy, and family therapy. Rehabilitation should begin on the day of the injury and continue until the patient is stable or has recovered fully. Value of early mobilization is well recognized. Appropriate period of rest away from work is necessary depending upon the severity of injury and complications. Change of employment may be warranted if the job prior to head injury was intellectually demanding. Successful rehabilitation improves a patient's productivity in a variety of activities, including vocational, educational, family participation and community service activities.

Cognitive Retraining: Functional skill development is the hallmark of cognitive retraining programs. Each patient's disability is analyzed in the context in which that particular patient would have to work. The therapist helps the patient to structure the tasks, which is encountered in day-to-day life. Strategies are thought to overcome the deficits of the patient, which would impair performance on these tasks. Functional skill development is a useful approach in rehabilitation of the head injured patients. A computer based cognitive retraining program has been developed. It aims to improve the information processing capacity of the patient using visual stimuli.

Psychological therapies: Personality changes are often resistant to treatment. Psychotherapy at superficial levels may help. Behavioral techniques like social skills training may help in reducing disruptive behaviors. Operant conditioning by rewarding self-helping behaviour while belligerent and manipulative behaviour are ignored has proved its utility. Relaxation training is useful in anxiety disorders.

Family Therapy: Family therapy is a particularly important part of patient's recovery. The most stress for relatives of head injury patients occur in the first month after injury with gradual lessening until six months. The degree of stress on family members is related to magnitude of behavioral, personality and affective change in the patient. The patient's disability may inflict social isolation on the family. Families need reassurance that anger, frustration and sorrow are natural emotional reaction for caregivers of head injured patients. Organizing self-help groups, educating the family in handling the patient, providing trained helpers, and much needed respite for temporary periods during holidays and supportive psychotherapy will help in rehabilitation

2. **Pharmacological intervention:** The need may arise for control of certain symptoms and behavior that may not resolve over time. Psychotropic agents may be used for these conditions. They are summarized as below

Anticonvulsants: Anticonvulsants are used to treat seizure disorder, mood lability, mania, impulsivity, aggression, and rage. Carbamazepine and valproic acid are most commonly used and found to be equally beneficial. Phenytoin and

barbiturates are not recommended as they decrease cognitive function and motor performance.

Antidepressants: SSRIs are useful in the treatment of depression, mood lability, and impulsivity. Tricyclics and monoamine oxidase inhibitors are not preferred in the treatment of head injury patients because of their anticholinergic side effects and drug–food interactions, respectively. Trazodone is useful for agitation and sleep.

Psychostimulants: Psychostimulants (e.g. methylphenidate 5 mg bid) are useful in the treatment of inattention, distractibility, disorganization, hyperactivity, impulsivity, hypoarousal, apathy, hypersomnia, mood and cognition.

Dopaminergic Agents: Head injury is frequently associated with disturbances of dopamine transmission, which persists for many years. The frontal lobes are especially rich in dopamine, and their frequent involvement in head injury is associated with decreased dopamine activity. Dopaminergic agents (amantadine, bromocriptine, and levodopa) have been used in the treatment of cognitive symptoms[28].

Hypnotics:Zolpidem is an imidazopyridine nonbenzodiazepine hypnotic indicated for short-term management of insomnia. It has rapid onset, and short half-life (2-3 hours). Zopiclone is a longer acting alternative. Trazodone, an antidepressant with no anticholinergic side-effects and little cardiotoxicity, in doses of 50-100 mg is frequently used as a hypnotic because it is sedative with little carry-over to the next day and has excellent physiological effects on slow wave sleep. Given the biological sleep disturbance component and disturbances in diurnal rhythms, melatonin remodulation appears physiological. The dose is 0.5 mg - 1 mg taken an hour before dusk and should take several weeks to work fully. The benzodiazepine may relieve symptoms non-specifically and incompletely but should be avoided, if possible, because of their addictive qualities and impairments at the psychomotor, cognitive, amnesic and drug interaction levels.

Other Agents: Naltrexone in doses of 50 mg–100 mg/day is useful in treating self-injurious behavior. Buspirone, a post-synaptic serotonin -1A partial agonist is also useful in the treatment of aggression in doses of 45 mg–60 mg/day. However, because of the organicity, even buspirone is sometimes associated with paradoxical reactions, such as irritability instead of anti-aggressive effects. Beta-blockers, such as propranolol, have also been used to treat aggression and violent behavior. Starting with 20 mg twice daily the dose of propranolol can be increased by 20mg to 30mg a day till therapeutic effects begin to emerge. Sometimes dosages up to 800 mg /day are required for the treatment of aggressive behavior.

OUTCOME

The outcomes that are of greatest importance to injured individuals, their families, and their communities are located at the disability and handicap levels, whereas many of the causes of these outcomes are at the levels of pathology and impairment. The correspondence among these levels is highly complex, making it difficult to identify the cause(s) of particular functional problems or to predict the outcome of treatment directed at improving the individual's competence[42]. This problem is particularly intense in head injury because of the large number of interacting impairments that may occur in an individual. A related practical problem is the question of how to focus treatment or service efforts. At one extreme, one can treat a long list of specific impairments in the hope that the sum total of this work will translate into high-quality global outcomes (an approach directed at the injured individual). At the other extreme, one can accept an individual with a current constellation of disabilities and handicaps and examine how social and environmental supports can be woven in relation to the individual's strengths and weaknesses to improve the outcome (an approach directed at the environment)[42].

CONCLUSION

Patients with head injury, whether mild, moderate, or severe, often experience enduring emotional and cognitive consequences. Evidence to date suggests that depression is the most common comorbidity, occurring not merely as a coincidence, but seemingly intrinsic to the neuropathologic process. However, there is no single factor—psychological, physiological, somatic, or demographic—that uniformly predicts psychiatric comorbidity. Of interest is that the frequency of minor depression/dysthymia is not significantly different among patients with head injury or general trauma. Furthermore, the emergence of psychiatric disorders appears to have no direct relationship to the severity of brain trauma. Ideally, treatment of these patients should involve a multidisciplinary approach, with the psychiatrist working in close collaboration with the patient, family, neurologist/neurosurgeon, psychologist and social worker. Neurologists evaluating patients with head injury should consider psychiatric and cognitive evaluations and follow-up as part of their routine treatment.

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