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Original Research Article

Post head injury neurobehavioral sequelae: severity among alcoholics

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ABSTRACT

Background: Alcohol use and traumatic brain injury (TBI) are closely linked public health problems. Alcohol intoxication is one of the major risk factor for TBI, and is a main determinant of prognosis in terms of mortality and functional outcome. The aim of the study is to find out the impact of alcoholism in the neurobehavioral outcome following TBI.
Methods: A total of 150 head injury patients were divided into two groups: alcoholics and non-alcoholics, and evaluated between six weeks to one year after injury using the revised neurobehavioral rating scale by Levin et al (NRS-R) for the evaluation of neurobehavioral sequelae and the outcome was compared between groups.
Results: The study showed significant difference between the groups indicating that the neurobehavioral sequelae were more in the chronic alcoholics group. In the comparison of individual factors, all except factors III (negative symptoms) and IV (mood and affect) were found to be significantly different. The factors I (executive), II (positive symptoms), V (oral and motor), and VI (not loading on any of the factors) were significant at 0.01 level.
Conclusions: Chronic alcoholism significantly increases the risk of developing neurobehavioral sequelae after traumatic brain injury.

Keywords: Traumatic brain injury, Neurobehavioral sequelae, Chronic alcoholism

INTRODUCTION

The global incidence of traumatic brain injury (TBI) is estimated at 939 cases per 100,000 people and is rising due to increased use of motor vehicles and increased reporting rate. The total volume of TBI in India is unknown, but estimates suggest that there are more than a million trauma related deaths in India per year, of which 50% are TBI related.[1] It is observed that alcohol intoxication is one of the major risk factor for TBI, and is a main determinant of prognosis in terms of mortality and functional outcome.[2] The common physical deficits after head injury include multiple cranial nerve palsies, motor and sensory disorders caused by cortical or brainstem lesions. The other more disabling effect of head injury includes cognitive incapacitation and personality changes which far outstrip the physical sequelae. These neurobehavioral sequelae refer to cognitive and behavioral effects of brain injury.

Chronic alcoholism results in characteristic structural and biochemical changes in the brain and results in accelerated brain atrophy.[4] Alcohol can also change the activity of neurotransmitter at synaptic receptor level and cause neurons to respond (excitation) or to interfere with responding (inhibition).[5] Both acute and chronic alcohol intakes appear to compound the brain damage and neurobehavioral sequelae caused by head injury. Alcohol potentiate the structural lesions of head injury. Alcohol

may acutely potentiate the brain injury via lipid peroxidation, vasodilatation, coagulation abnormalities and increasing plasma osmolality which in turns exacerbate brain edema. More and more structural injury to brain potentiates the severity of post traumatic sequelae of brain injury.

Many studies show varying influence of alcohol in causing neurobehavioral outcome following TBI with majority of studies showing an additive effect. Thus chronic alcoholism becomes a main determinant of functional outcome in TBI which affects the rehabilitation measures and quality of life. The purpose of this study is to evaluate whether chronic alcoholism affects the severity of neurobehavioural sequelae in head injured patients.

METHODS

The methodology followed in the study is a descriptive longitudinal quasi experimental study among the patients with traumatic brain injury in head injury outpatient clinic of Government Medical College, Thiruvananthapuram, during June 2016 to May 2017. A total of 150 head injury patients aged between 18-60 years and were evaluated between six weeks to one year after injury. Alcoholics were defined according to diagnostic and statistical manual of mental disorders-5 (DSM 5) criteria for alcohol use disorder and the total subjects were divided into two groups - alcoholics and non-alcoholics. Patient with history of previous behavioural abnormality or psychiatric symptoms before head injury, patients in vegetative state, patients who could not properly communicate and understand the questions, and patients with aphasia were excluded from the study.

Outcome was evaluated by neurobehavioral rating scale (NRS) by Levin et al revised by himself and Mc Cauley et al. Neurobehavioral rating scale - revised (NRS-R) is an internationally accepted multidimensional assessment tool for the evaluation of neurobehavioral sequelae.[7] It consists of a structured interview containing tests for emotional state, orientation, memory, attention and concentration, planning, explanation of proverbs, delayed recall and post-concussion symptoms. One third of the findings are categorized in a 4 point scale (Likert 4 point scale).[8] Rest of the points were assessed according to the performance of the patient.

NRS-R criteria are grouped into 5 factors: factor I (executive/cognition) related to difficulties in planning, mental flexibility, memory difficulties, orientation and comprehension; factor II (positive symptoms) related to distortions of normal function and consist of unusual thought, suspiciousness, agitation and disinhibition; factor III (negative symptoms) related to diminishment of normal function that reflected emotional withdrawal, blunted affect and hostility; factor IV (mood and affect) related to depressive mood, anxiety irritability, mental fatigability and mood lability; and factor V (oral and motor) related to difficulties in articulation, verbal speech and motor slowing.

Items that does not fit into the above factors but seem important in survivors of TBI like attention difficulty, decreased alertness, hallucination and guilt were considered as a separate group VI.

All the patients were examined by a single examiner and assessed by single neuropsychologist and each item in the scale graded with Likert-4 point scoring system. The total score and the score of each factors calculated and compared. Statistical analysis done with Statistical Package for the Social Sciences (SPSS)-16 trial version. Mann Whitney 'U' test was used to calculate the mean and difference between the groups.

RESULTS

Out of 150 patients investigated in this study 132 (88%) are males and 18 (12%) are females. Among these, 36% were aged less than 30 years, 32% were between 30 and 50 years and 32% were above 50 years. 4% were illiterate, 5% studied up to tenth standard and 44% beyond tenth standard. Out of 150 patients examined 36 patients (24%) never consumed alcohol, 20% occasionally consumed alcohol, 12% consumed less than 5 days per week, and 44% consumed more than 5 days per week. Among these 150 patients, 66 persons (44%) had alcohol intake prior to sustaining head injury. Out of the total number of patients 90 had mild head injury, 36 had moderate head injury and 24 had severe head injury. The mean Glasgow coma scale (GCS) was 12 in non-alcoholics and 11 in alcoholics. According to the initial computed tomography (CT) finding, bilateral brain injury was common in 28% patients followed by right frontal and right temporal lobe involvement of 20% each. Of the total patients 72 patients (48%) were operated and 78 (52%) were treated conservatively.

Out of the total patients in the study of population 60 were chronic alcoholics (40%) and 90 were non-alcoholics (60%). Comparison of the total score showed significant difference between the groups indicating that the neurobehavioral sequelae were more in the chronic alcoholics group. In the comparison of individual factors, all except factors III (negative symptoms) and IV (mood and affect) were found to be significantly different. The factors I (executive), II (positive symptoms), V (oral and motor) and VI (not loading on any of the factors) were significant at 0.01 level.

DISCUSSION

The results of our study prove that there is significant increase in neurobehavioral sequelae in the alcoholics group. This suggests that alcoholic group has more difficulty in cognition, planning and executive functions, more agitation, disinhibited behaviour and hallucinations, more speech difficulties, reduced attention, alertness and

motor slowing. This will have a great impact on job performance, interpersonal behaviour and social life.

Alcohol is a well-documented cause of road traffic accident and it is estimated that around 30% of patients with TBI are drunk at the time of accident and some studies showing values close to 50%.[9,10] In a study at NIMHANS, Bangalore, a study group containing 4731 head injury patients, 23.7% subjects agreed to regular alcohol intake and 18.4% were found to be under influence of alcohol at the time of injury.[11] In our study 44% had alcohol intake prior to sustaining head injury. It is noted that the prevalence of problem drinking in acute head injury with positive history of alcohol abuse or dependence is seen in 43% of all admitted patients.[12] Similar to this study, 40% of the subjects were chronic alcoholics in our study. Naturally head injury patient with history of chronic alcoholism are more likely to be intoxicated at the time of injury (57%) than those without such a history (31%).[13]

When analyzing the relationship between alcoholism and GCS scores at the time of hospital admission, there was a significant difference with lower values among alcoholic patients, compared to non-alcoholics.[14] Influence of alcohol significantly reduced the GCS score in a dose-dependent manner in patients with moderate and severe TBI.[15] In our study also the mean GCS was lower in alcoholics (11) compared to non-alcoholics (12).

Tien et al measured blood alcohol concentration (BAC) in patients admitted with severe brain injury and stratified into the following 3 levels: 0, no BAC; 0 to less than 230 mg/dl, low to moderate BAC; and 230 mg/dl or greater, high BAC and concluded that low to moderate BAC may be beneficial in patients with severe brain injury from blunt head trauma. In contrast, high BAC seems to have a deleterious effect on in-hospital death in these patients, which may be related to its detrimental hemodynamic and physiologic effects.[16]

Hsing-Lin Lin et al assessed the BAC of head injury patients and was then stratified into four levels: none (less than 8 mg/dl), low (8 to less than 100 mg/dl), moderate (100 to less than 230 mg/dl), and high (≥230 mg/dl). A group of patients not tested for BAC were categorized as a compared group, and the patients with BAC test with level less than 8 mg/dl were added as the none group and the inference was that, in patients with blunt head injury, patients in the none group had less severity and mortality, and the mortality was higher in patients with BAC between 8 and less than 100 mg/dl group than other intoxicated groups.[17] Acute neuro-inflammation is a pathophysiological feature of TBI.[18] Brain parenchymal injury leads to increased cerebral blood flow due to the action of pro-inflammatory cytokines and cause increases in endothelial permeability and infiltration of circulating leukocytes. Alcohol can potentiate this immune response by activating transcription factors involved in inflammatory signalling pathways, expression of cytokines and inflammatory mediators, expression of adhesion molecules, and recruitment of inflammatory cells leading to sustained post-traumatic inflammation which in turn leads to adverse outcomes.[19]

Neurobehavioral sequelae of TBI may be due to primary brain damage or due to secondary factors such as sleep disturbance. Reduction of verbal and non-verbal intelligence quotient (IQ) and intelligence is a commonly cited change following TBI.[20] Difficulties in planning, judgment, and cognitive aspects of decision making leads to diminished executive functioning.[21,22] In behavioral domain, patients who suffered TBI may have irritability, aggressive behavior, and agitation.[23] It may be due to focal or diffuse brain injury.[24] Aggression can range from mild irritability to outbursts of violence which may end up in destruction of property or assaults on others. A study was conducted on TBI inpatients, which showed that there is a significant correlation of aggression with other conditions such as new-onset major depression and impaired psychosocial functioning.[25] Memory acquisition and retrieval are also negatively impacted following a TBI.[26] Behavioral components such as low motivation and poor emotional regulation are also found in individuals following a TBI.[22]

There is scarcity of literature on the impact of chronic alcoholism in neurobehavioral sequelae of head injury. Sparadeo and Gill reported lower global cognitive and neurobehavioral status at hospital discharge in persons with TBI who were intoxicated at admission compared to those not intoxicated.[27] At 1 year after TBI, the alcohol-abusing individuals had increased cerebral atrophy, electroencephalogram impairments, and reduced rates of return to work.[28] Alcoholism leads to accelerated brain atrophy mainly in the region of cerebral cortex and white matter and there is a preferential involvement of frontal lobes resulting in cognitive and executive dysfunction and uninhibited and impulsive behaviour.[29] Functional magnetic resonance imaging (fMRI) has provided additional evidence of reduced activation in the amygdala and hippocampus of chronic alcohol abusers when presented with emotional words and expressions.[30] Magnetic resonance imaging (MRI) studies have also revealed volumetric deficits in the brain's right hemisphere.[31] Some studies show greater right hippocampal atrophy in chronic alcohol abusers.[32] Dikmen et al also reported that neuropsychological performance in TBI patients was related to the degree of pre-injury alcohol use, with impairments in those individuals with more chronic pre-TBI alcohol use.[28] This is comparable with the results of our study which also showed significant neurobehavioral sequelae in the alcoholics group. Kelly et al studied severe TBI patients who had a positive BAC at the time of hospital admission during inpatient rehabilitation (mean 48.8 days post injury) and found them to be worse on measures of intelligence, verbal and visual memory, attention and concentration, and delayed recall compared with non-intoxicated TBI patients.[33] Wilde et al compared TBI patients who were BAC-positive or BAC-negative at the time of injury and found neuroimaging

evidence for greater brain atrophy in patients with a positive BAC at the time of injury and/or a history of moderate to heavy alcohol use.[34] However Turner et al also found that pre-injury consumption of alcohol and admission BAC were not consistently related to any post-TBI cognitive outcomes.[35] Ponsford et al reported that harmful or hazardous pre-injury alcohol use was associated with poor memory performance and slower processing speed at 6-9 and 12-15 months after moderate to severe TBI.[36] It is clear that the results of majority of these studies are comparable with the results of our study supporting the evidence that chronic alcoholism increases the severity of neurobehavioral sequelae in TBI patients.

CONCLUSION

Several factors influence the severity of these neurobehavioural sequelae which occurs in the post head injury periods. Alcohol abuse both increase the risk for head injury and potentiate the resulting brain injury, which can lead to negative neuropsychological consequences. Alcohol abuse and head injury may interact in same patient to influence neurobehavioural status. Almost similar to head injury, the alcoholism also causes neurobehavioral changes in the affected patients independently. This study proves that chronic alcoholism potentiates the severity of neurobehavioral sequelae in head injured patients.

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