



POST TRAUMATIC BRAIN INJURY- NEURO BEHAVIORAL SEQUELAE: SEVERITY AMONG SURGICALLY MANAGED PATIENT

Neurosurgery

Dr.Sunilkumar B S	M. S, M. Ch., Associate Professor, Department of Neurosurgery, Government Medical College Trivandrum, Kerala, India.
Dr. Raja K Kutty*	M. S, M. Ch., Assistant Professor, Department of Neurosurgery, Government Medical College Thiruvananthapuram Kerala, india.695011 *Corresponding Author
Dr. Prasanth Asher	Assistant Professor, Department of Neurosurgery, Government Medical College Thiruvananthapuram Kerala, India. 695011
Dr. P.T. Baburaj	Associate Professor, School of Behavioral Sciences Mahatma Gandhi University Kottayam, Kerala, India
Dr. Anilkumar Peethambaran	Professor and Head, Department of Neurosurgery, Government medical college Thiruvananthapuram Kerala, India,695011.
Dr. Robert Mathew	Professor, Department of Neurology, Government Medical College Alleppey, Kerala, India

ABSTRACT

Background : Traumatic Brain Injury is an ever-enduring health hazard growing worldwide. Road traffic accidents are among the forefront to the cause of this problem. With this study, we aim to investigate the difference in the severity of neurobehavioral sequelae among survivors of TBI managed either surgically or conservatively.

Materials & methods : The patients were selected from Head Injury out-patient clinic of a tertiary care hospital between September 2012 to August 2013. There were 125 patients grouped into two depending upon the management. They were examined between six weeks to one-year post trauma using the tool Revised Neurobehavioral rating scale developed by Levin et.al. The data was entered in excel and analysed using standard statistical software. Statistical comparisons were made across five Domains of the NRS-R using the Mann Whitney U test.

Result : There were 65 patients in group 1 (conservative) and 60 patients in Group 2 (surgery). Each group were comparable with regard to age and severity of injury (mild, moderate and severe). Ist Domain of the NRS-R representing Executive cognition did not show any significant difference in severity between two groups. There were significant differences across all other domains including positive symptoms, negative symptoms, mood/affect and oral/motor between two groups. Emotional aspect of decision making motivation and impulsivity and mood were affected more significantly in surgically managed patients

Conclusion : Cognitive executive function is not significantly different in both groups while Behavioral executive functions are significantly severely affected in surgically managed patients.

KEYWORDS

Neurobehavioral sequelae, Traumatic brain injury, Surgery, Conservative

Background

Traumatic Brain Injury (TBI) is a universal public Health problem. The incidence of TBI is steadily increasing all over the world and India has dubious distinction of having highest incidence in the world of head Injury due to road traffic accidents(RTA) for 1000 vehicles or death per 1000 accidents. In Kerala, there have been 39420 cases of RTA claiming 4287 lives in the year 2016. Most of these deaths have been due to traumatic brain injury. The degree of disability among the survivors cannot be under estimated. Traumatic Brain Injury accounts for a great deal of chronic disability in terms of physical deficits due to neurological injury, neurobehavioral sequelae (NBS) and neuropsychiatric sequelae. Behavioral disturbances typically become obvious following recovery from the acute phases of the trauma such as acute confusion state and post-concussion syndrome.

Materials and methods

This is an inception cohort longitudinal descriptive study in which 125 traumatic brain injury patients were examined consecutively in the head injury clinic of neuro surgery Department at Medical College Kottayam during September 2012 to August 2013. The patients were between 18 and 60 years of age and were examined in the post head injury period of 6 weeks to one year. These patients include both operated and non-operated cases. Patients with history of previous behavioral abnormality or psychiatric symptoms before injury and vegetative state were excluded from the study. The study also excluded the Patient's with severe language dysfunction like Global Aphasia and inability to communicate.

Post traumatic NBS was assessed using neuro behavioral Rating Scale (NRS) by Levin et.al revised by himself and Mc Cauley et.al. Neuro behavioral rating scale - revised (NRS-R) has acceptable internal consistency and integrator reliability and validity. This is a 29-item

multidimensional clinical based assessment instrument designed and validated to measure NBS following head injury. NRS-R comprises of a brief structural interview which includes, test for orientation and memory for recent events, question regarding emotional state, post-concussion symptoms, focused attention and concentration, explanation of proverbs, tasks of planning and mental flexibility and delayed recall of three object presented at the beginning of session. One third of the questionnaire was based on examiner's observation which were graded in 4-point rating scale (Likert 4-point scale, absent, mild, moderate, severe). The rest of the points were rated according to the performance of the patient.

For analysis, the NRS-R is divided into 5 factors.

Factor I (executive cognition) which include items in mental flexibility, difficulty in planning and memory difficulties.

Factor II (positive symptoms) distortions and exaggeration of normal function consist of unusual thought content suspiciousness, agitation and dis inhibition.

Factor III (Negative symptoms) a decrease or absence of normal function like emotional withdrawal, blunted affect and hostility.

Factor IV (Mood and affect) include items measuring depressive mood, anxiety irritability, mental fatigability and lability of mood

Factor V (oral and motor) consist of items rating difficult in perceptive and expressive speech and motor slowing.

Items not loading an any of the above factors and important to the survivors of TBI were also considered. This consisted of attention difficulty, reduced alertness, guilt and hallucination. These 4 items are

considered separately because they are important in post head injury patients with Neurobehavioral sequelae. Depending upon the management i.e. surgical or non-surgical, 125 patients were divided into two categories. The choice of these treatments is dependent on the clinical scenario and radiological findings. Conservative management mostly consists of medications to reduce Intracranial Pressure and Hyperventilation. The surgical procedure may vary depending upon the characteristic of the lesion induced by trauma on the radiography. Surgical maneuvers include craniotomy and evacuation of hematoma viz – Extradural or Intradural (subdural or intracerebral). Other procedures include a decompressive craniectomy and surgery for compound/depressed fracture and CSF leak.

All patients were examined by a single examiner and each item was quantified by assigning appropriate grade using Likert 4-point scoring system. Sum Score for all of these total items and for each factor were calculated separately and compared between groups. Statistical analysis was done by SPSS-16 trial version. Mann Whitney 'U' test was calculated for each of 5 domains comparing surgical and non-surgical. The NRS(R) score of total 29 Items are also calculated. Man Whitney 'U' test were used to calculate the mean and the difference between two groups.

Results

Out of 125 patients 110(80%)were males and 15 females (12%). 75(60%) had mild head injury 30 (20%) have moderate and 20(16%)had severe head injury. 45(36%)were less than 30 years of age and 40 (32%) each were between 30-49 years age and 50-60 years of age. 60(48%) were operated for their head injury while 65(52%) were managed conservatively. Study group has almost equal number of patients is operated (60) patients and non-operated (65) groups.

The calculated mean was found to be high for operated patients indicates all these behavioral symptoms were categorically more severe in operated patients.

Except for Executive/cognitive (Factor I), all the other factors including total neuro behavioral rating score were significantly different between two groups. Positive symptoms (Factor II), Negative symptoms (Factor III), Mood/Affect (Factor IV) and item not loading on above factors were significant at 0.01 level. Oral/motor (Factor V) were significant at 0.05 level while we compare the post traumatic Brain Injury Neuro behavior sequelae between these two groups. The factor for which NBS has no significant difference between two groups was Executive/ cognitive function which includes factors for planning and execution of a decision. This factor I corresponds to intentional or goal oriented behavior, in which decreased initiative and motivation, difficulty in planning and conceptual disorganization produce defect in the high order cognitive abilities causing predominantly executive dysfunction. They are implicated in job performance, social relationship and both basic and instrumental activities of daily living. Regarding, other factors; factor II (positive symptoms) show emotional state, Factor III (Negative symptoms) show survival oriented behavior and factor IV show mood and affect of the patient while factor V indicate language function.

Discussion

Neurobehavioral sequelae evolution or resolution depends on the time elapsed after Traumatic Brain Injury and has as individual variation. It also depends on localization and lateralization of the injury, severity of injury, medical and physical co-morbidities and pre-psychosocial and post-psychosocial factors. As many as 65 % of moderate to severe Traumatic Brain Injury patients has long term cognitive impairment. Some acute concussion or post-concussion symptoms occur in most TBI patients and the other Neurobehavioral changes are evident in about 10% of mild, 67% of moderate and 100% severe Traumatic Brain Injury patients².

The focal injuries to the brain in TBI are mainly sub frontal region, bilateral temporal cortex and frontal cortex. But in case of diffuse axonal injury it can be sub frontal white matter, deep midline structure including basal ganglia, rostral brain stem, hippocampal region of temporal lobe and corpus callosum. Scheid R et.al.³ in their study proved that severity of the post head injury cognitive disturbances depends on the extent of diffuse axonal injury as well as the volume and location of focal brain injury. Although the damage may be diffuse or multifocal, certain brain regions are highly vulnerable to injury and account for the high rate of challenging behavioral and consequently the increased rate of psychiatric illness that are associated with TBI⁴.

Any alteration in the neurotransmitter level in the brain following TBI can cause cognitive dysfunction and behavioral disturbances in the patient. The neuro transmitters implicated in this alteration are Acetylcholine, serotonin glutamate and dopamine. Genesis of mood disorders particularly depression and decline in cognitive functions especially memory and attention are influenced by change in the balance of cholinergic system⁵. The serotonergic system is activated in TBI and can alter the serotonin level in areas of tissue damage may be other reason for these behavioral changes like emotional changes disinhibition and aggression⁷.

Cognitive defects particularly attention, memory information processing, speed and problems in self-perception are very common following TBI especially Severe injury⁸. Change in personality is seen in majority of post TBI survivors. This can be of two types, exaggeration of pre- injury traits or fundamental changes in response patterns. Several common clusters of symptoms are included as personality changes like impulsivity, irritability, affective disability, apathy and lack of awareness.⁴

NBS after TBI can be due to the profile of region of brain injury as well as alteration in the neurotransmitter level in the brain. Along with NBS, Neuro psychiatric symptoms also co-exist in post TBI patients. Both of these together can produce a significant behavioral change in the patients.

The severity length and clinical manifestation of this post TBI, Neuro behavioral sequelae have large variability between patients. Some factors influencing these sequelae are localization and lateralization of injury extent and type of injury, medical and psychiatric comorbidities. Some of these factors are modifiable while most of them are not. The more influential factors include, young age, education level, low score in Glasgow outcome scale, previous history of TBI or psychiatric diseases⁹ and alcohol use as well as low Glasgow coma scale(GCS) score (severity of head injury).

Diffuse axonal injury in more often associated with generalized cognitive dysfunction such as attention disorder, whereas focal injuries cause specific defects such as aphasia. Because of difficulties with selective and divided attention patients with Traumatic Brain Injury have difficulty with multitasking. Declarative memory for events is more affected than implicit memory¹⁰ working memory and prospective memory are impaired particularly after frontal lobe injury.

Executive function are directed for establishing goals, planning initiating, sequencing and inhibitory response¹¹. Frontal lobe components like basal ganglia and thalamic circuits (Papez circuit) are responsible for this function is injury to Frontal lobe can cause impaired executive function, defective conceptual reasoning, difficulties in decision making and have impaired self-monitoring and regulation. Orbito frontal and medial frontal cortical injuries as well as ventral pallidum and ventral Tegmentum injuries result in Abulia of varying degree¹². Personality changes such as emotional lability disinhibition, aggression, apathy and paranoia are most often exaggeration of premorbid personality traits which was dormant during pre-injury period. Orbito frontal injury causes personality changes with mania, euphoria and impulsivity while medial frontal damage may cause personality disorder with severe apathy. Damage to limbic system, Orbito frontal cortex, left anteromedial frontal lobe and anterior cingulate has been particularly associated with aggressive behaviour³

Cognitive deficits in mild TBI can be observed in patients even in the absence of history of loss of consciousness or post traumatic Amnesia¹⁴. Behavioral symptoms of traumatic brain injury can be a secondary abnormality due to emotional response to injury, its physical limitations or fear of the impact on function, weak defense mechanism, poor social support medication, and drug use can all complicate the presentation of the behavioral symptoms¹⁵. Some of the risk factors that have been identified for development of Neuro behavioral sequelae include female gender advanced age, pain and prior affective or anxiety diagnoses¹⁶.

Post traumatic brain injury behavioral changes can affect their interpersonal relationship¹⁷ and behavior in society at large¹⁸ (antisocial/criminal aspect). Cognitive impairment is among the most commonly occurring sequelae in Traumatic Brain Injury and post traumatic cholinergic deficit are thought to contribute to the development of post traumatic cognitive impairments¹⁹.

Moderate to severe Traumatic Brain Injury are associated with more severe and persistent cognitive deficits such as awareness, reasoning, language visuospatial processing and general intelligence¹⁹ cognitive and behavioral changes are more closely associated with long term disability than sensory, motor and other sequelae of traumatic brain injury.

Conclusion

Neuro behavioral sequelae of varying severity occurs in majority of patients with moderate and severe head injury and in small proportion of patients with mild head injury. The severity of this is more pronounced in surgically managed patients compared to non-surgical group. In the surgical group, the behavioral executive function is significantly worse while cognitive executive functions are not significantly affected by surgery.

REFERENCES

- Whiteneck GG, Gerhart KA, Cusick CP. Identifying environmental factors that influence the outcomes of people with traumatic brain injury. *J Head Trauma Rehabil.* 2004;19(3):191-204. doi:10.1097/00001199-200405000-00001.
- SB Sorenson JK. Occurrence, severity and outcomes of brain injury. *J Head Trauma Rehabil.* 1991;6(2):1-10.
- Scheidt R, Walther K, Guthke T, Preul C, von Cramon DY. Cognitive sequelae of diffuse axonal injury. *Archives of neurology.* 2006;63(3):418-424. doi:10.1001/archneur.63.3.418.
- McAllister TW. Neurobehavioral sequelae of traumatic brain injury: evaluation and management. *World psychiatry : official journal of the World Psychiatric Association (WPA).* 2008;7(1):3-10. doi:10.1002/j.2051-5545.2008.tb00139.x.
- Shytle RD, Silver AA, Sheehan KH, Sheehan D V., Sanberg PR. Neuronal nicotinic receptor inhibition for treating mood disorders: Preliminary controlled evidence with mecamylamine. *Depression and Anxiety.* 2002;16(3):89-92. doi:10.1002/da.10035.
- Perry EK, Perry RH. Neurochemistry of consciousness: Cholinergic pathologies in the human brain. *Progress in Brain Research.* 2004;145:287-299. doi:10.1016/S0079-6123(03)45020-6.
- Tsuiki K, Takada A, Nagahiro S, Grdi?za M, Diksic M, Pappius HM. Synthesis of Serotonin in Traumatized Rat Brain. *Journal of Neurochemistry.* 1995;64(3):1319-1325. doi:10.1046/j.1471-4159.1995.64031319.x.
- Lippert-Gruner M, Kuchta J, Hellmich M, Klug N. Neurobehavioural deficits after severe traumatic brain injury (TBI). *Brain Injury.* 2006;20(6):569-574. doi:M8U8323711Q16J0U [pii]r10.1080/02699050600664467.
- Koutzoukis C, Sc M, Ali I, Psych MRC, Mccarthy G, Psych MRC. *After Traumatic Brain Injury.* 1999;(March).
- J Nicholl WL. Neuropsychiatric sequelae of traumatic brain injury. *Seminars in neurology.* 2009;29(3):247-255. doi:10.1176/appi.psy.41.2.95.
- Cummings J. Frontal-subcortical circuits and human behavior. *Archives of Neurology.* 1993;50(8):873-880. doi:10.1001/archneur.1993.00540080076020.
- Mega MS, Cummings JL. Frontal-subcortical circuits and neuropsychiatric disorders. *The Journal of neuropsychiatry and clinical neurosciences.* 1994;6(4):358-370. doi:10.1176/jnp.6.4.358.
- Grafman J, Schwab K, Warden D, Pridgen a, Brown HR, Salazar a M. Frontal lobe injuries, violence, and aggression: a report of the Vietnam Head Injury Study. *Neurology.* 1996;46:1231-1238. doi:10.1212/WNL.46.5.1231.
- McCrea M, Guskiewicz KM, Marshall SW, et al. Acute Effects and Recovery Time Following. *The Journal of the American Medical Association.* 2003;290(19):2556-2563. doi:10.1001/jama.290.19.2556.
- Wong M RM. Neurobehavioral Sequelae of Traumatic Brain Injury. *The Mount Sinai journal of medicine.* 2009;76(2):163-172. doi:10.1002/MSJ.
- Meares S, Shores E, Taylor A, et al. Mild traumatic brain injury does not predict acute postconcussion syndrome. *Journal of Neurology, Neurosurgery, and Psychiatry.* 2007;79(3):300-306. doi:10.1136/jnnp.2007.126565.
- Weddell R a, Leggett J a. Factors triggering relatives' judgements of personality change after traumatic brain injury. *Brain injury.* 2006;20(12):1221-1234. doi:10.1080/02699050601049783.
- Sarapata M, Herrmann D, Johnson T, Aycock R. The role of head injury in cognitive functioning, emotional adjustment and criminal behaviour. *Brain Injury.* 1998;12(10):821-842. doi:10.1080/026990598122061.
- Rabinowitz AR, Levin HS. Cognitive Sequelae of Traumatic Brain Injury. *Psychiatric Clinics of North America.* 2014;37(1):1-11. doi:10.1055/s-0029-1223878.