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PROGNOSTIC FACTORS IN SEVERE HEAD INJURY AND THEIR ASSOCIATION WITH OUTCOME-OUR INSTITUTIONAL EXPERIENCE

Neurosurgery

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ABSTRACT

Severe head injury is leading cause of mortality and morbidity in Indian population especially among young men. Outcome prediction after severe head injury is very important especially for countries like India for better targeting of limited healthcare resources and implementation of specific methods of treatment to patients. This study was undertaken to assess prognostic factors and their association with outcome in severe head injury. This study is based on a prospective analysis of 100 patients admitted in emergency department of our hospital with severe head injury (Glasgow Coma Score ≤8). Basic demographic, clinical and radiological data at admission and during hospital stay were recorded according to Glasgow Outcome Score. Predicting outcome is an assimilative and integrative process of various pre-injury, injury and post-injury variables. CT scan should be done on urgent basis for planning further management.

KEYWORDS

severe head injury, Glasgow Coma Score (GCS), Glasgow Outcome Score (GOS).

Severe head injury is defined as brain injury resulting in loss of consciousness of greater than 6 hours and a Glasgow Coma Scale of ≤8, is a major cause of death and disability worldwide [1–5].The brain damage causes, hypoventilation leading to hypercarbia and hypoxia. Combined with brain swelling as a direct result of the trauma, this can lead to serious disability and death [6–9].

The management of severe head injury patients demands the dedication of expensive but limited intensive care resources for considerable length of time. In spite of these efforts, mortality and long term morbidity remains high. In all reported series of significant number of patients, a mortality rate in range of 30-40% was seen[10]. Outcome prediction after severe head injury continues to be an area of intense interest.

In part, this reflects the natural curiosity of the neurosurgeon, but as an increasing attention is paid to resource allocation in all societies, our ability or inability to accurately predict outcome becomes very important for targeting of scarce resources[11]. Commonly used predictors of outcome both individually or in combination includes-age, Glasgow coma scale score, pupillary reactivity, early hypoxia and hypotension,brain stem reflexes and CT findings[12]. This prospective study was undertaken to evaluate these factors as predictors of outcome in patients with severe head injury.

MATERIALAND METHODS

This study is based on the prospective analysis of patients admitted in our hospital from 1st October 2015 to 31st March 2017 with severe head injury with GCS of 8 or less. Patients with associated severe chest, abdominal or orthopaedic trauma were excluded in our study. After initial resuscitation these patients were evaluated and investigated. CT scan was done in all patients and those with significant operable lesions were operated upon immediately. Other patients were managed conservatively using ventilatory support, anti convulsants and anti oedema drugs. Clinical outcome was evaluated after six months of discharge, according to Glasgow outcome score:

1) Grade I (death)
2) Grade II (vegetative)
3) Grade III (mostly dependant)
4) Grade IV (minimally dependant)
5) Grade V (good recovery)

- GOS of I to III was considered as unfavourable outcome (U.O.).
- GOS of IV and V was considered as favourable outcome for statistical analysis.
- The data was analyzed as mean SD, non-parametric data was analyzed by chi square, Fischer's exact test and parametric test was applied to the interval data.

TABLE 1 EPIDEMIOLOGICAL PROGNOSTIC FACTORS IN SEVERE HEAD INJURY (n=100)m

Prognostic factors	Sub-groups	No.	Glasgow outcome score	% of UO	Significance/p value				
			I	II	III	IV	V		
Age	<20	20	2	2	2	3	11	30	S/<0.001
	20-40	40	15	5	0	10	10	50	
	>40	40	20	2	4	5	9	65	
Sex	Male	86	40	3	3	20	20	53.48	NS>0.05
	Female	14	3	1	1	4	5	35.71	
Mode of injury	RTA	82	34	6	10	16	16	60.97	NS>0.05
	Fall	14	4	4	0	2	4	57.14	

	Assault	4	1	1	0	1	1	50	
H/O alcohol intake	Yes	20	6	0	4	5	5	50	NS>0.05
	No	80	30	8	2	20	20	50	
Time of presentation	<8 h	80	30	6	10	4	30	57.50	NS>0.05
	>8 h	20	4	4	2	5	5	50	

U.O = unfavourable outcome, NS = not significant,S = significant.

TABLE 2 CLINICAL PROGNOSTIC FACTOR IN SEVERE HEAD INJURY (n=100)

Prognostic factor	Sub-group	No.	Glasgow outcome scale	% of UO	Significance/p value				
			I	II	III	IV	V		
Hyoptention	Present	30	10	5	0	7	7	50	NS/>0.05
	Absent	70	20	10	5	15	20	50	
GCS	3-6	45	30	5	0	5	5	77.77	S/<0.0001
	7-8	55	10	5	5	15	20	36.36	
Hypoxia	Present	20	5	5	5	2	3	75	s/<0.0001
	Absent	80	20	3	7	25	25	37.50	
Pupillary reflex	Normal	86	40	2	3	13	28	48.83	s/<0.001
	Both dialated	6	2	1	1	1	1	66.66	
	Anisocoria	8	4	2	1	1	2	75	
Treatment	Non-operative	60	30	2	3	5	20	58.33	S/<0.05
	Operative	40	5	5	1	10	20	27.50	

U.O = unfavourable outcome, NS = not significant, S = significant.

TABLE 3 PROGNOSTIC FACTORS ON CT SCAN IN SEVERE HEAD INJURY(n=100)

Prognostic factor	Sub-group	No.	Glasgow outcome score	% of UO	Significance/p value				
			I	II	III	IV	V		
MIDLINE SHIFT	Absent	45	10	5	5	10	15	44.44	S/<0.005
	<5mm	35	10	3	4	5	10	48.57	
	>5mm	20	14	0	0	4	5	70	
EDH	Absent	80	30	10	3	17	20	53.75	NS/>0.05
	<10mm	14	2	1	1	5	5	28.57	
	>10mm	6	1	0	0	0	5	16.66	
SDH	Absent	60	20	4	4	17	15	46.66	NS/>0.05
	<10mm	30	10	4	3	3	10	56.66	
	>10mm	10	3	1	1	2	3	50	
CONTUSION	Present	90	35	4	6	15	30	50	NS/>0.05
	Absent	10	3	2	1	2	2	60	

U.O = unfavourable outcome, NS = not significant, S = significant.

OBSERVATIONS

During the Eighteen-month (1st October 2015 to 31st March 2017) period, 100 consecutive patients of severe head injury were enrolled in this study. The influence of the epidemiological factors on the outcome is shown in Table 1. The majority of patients were males (86%) with road traffic accident (82%) as most common mode of head injury.

Increasing age of the patient had statistically unfavourable outcome (p value <0.001) relationship with age of the patient. In our study 65% of patients with ages more than 40 years had unfavorable outcome in comparison to 30% and 40% in patients below the age of 20 and between 20 to 40 years of age respectively

Mode of injury whether road traffic accident (60.97%), fall (57.14%) or assault (50%) did not had any significant relation with adverse outcome. In our study 20% patients had history of alcohol intake, but alcohol intake was not a significant predictor of outcome. 80% of patients presented to emergency within 8 hours.

The influence of various clinical factors on neurological outcome is shown in Table 2. 30% of patients were hypotensive when they presented in casualty, its relation with clinical outcome was not significant. Hypoxia, being one of the preventable secondary brain insults, greatly affected outcome in patients with severe head injury. 75% of patients who had hypoxia at the time of presentation had unfavourable outcome, as compared to 37.50% in non-hypoxic patients. So hypoxia was significantly associated with adverse outcome with p value <0.005. The GCS score at the time of admission has been shown to be a reliable predictor of clinical outcome after severe head injury. In our study 77.77% of patients with GCS of 3 to 4 had unfavourable outcome as compared to 52.63% in patients with 5 to 6 GCS and 36.36% in patients with 7 to 8 GCS. It was statistically highly significant with P value. <0.0001.

Normal pupillary reflex was associated with good outcome.Normal pupillary reflex was associated with good outcome. It was found that 48.83% with normal pupillary reflex had unfavorable outcome as compared to 66.66% in patients with both dilated pupils and 75% in patients with anisocoria. Thus association of abnormal pupillary reflex with unfavorable outcome was statistically highly significant.

It was found that 40% of patients with severe head injury required operative intervention out of which 27.5% resulted in unfavorable outcome. In our study 60% of patients were managed conservatively out of which 58.33% resulted in unfavorable outcome.

This signifies the important role of early operative intervention in management of severe head injury.

The influence of the CT scan findings on the final outcome is shown in Table 3. Greater degree of midline shift on CT scan was associated with unfavorable outcome. It was seen that 44.44% with midline shift of <1 mm, 48.57% with midline shift of 1-5 mm and 70% with midline shift of >5 mm. This increase in unfavorable outcome with greater degree of midline shift is statistically significant. Presence of extradural or subdural hematoma did not significantly influence the outcome.

DISCUSSION

The glasgow coma scale (GCS) and the glasgow outcome score (GOS) have provided practical methods for assessment of the severity and the outcome in patients with severe head injury (GCS 8 or less). Aggressive management of these patients with elective ventilation compels to rely on the pupillary reflex , GCS and CT scan findings to guide his further decisions.[13-14] While it was predicted that aggressive management of severe head injury would decrease mortality, but it leads to increase the number of vegetative patients. Studies have shown that percentage of patients with functional outcome actually increases without significantly increasing the percentage of severe disabled patients.[14]However, very few studies have focussed exclusively on the outcome of patients with dilated unreactive pupils[15-19], thus giving a special significance to our study.

Patients were studied till the period of six months after discharge from hospital. A common Glasgow outcome scale (GOS) was used to compare the outcome. Many variables were analyzed to predict

prognosis, using GOS as dependent variable. Road traffic accident was commonest (83.64%) mode of severe head injury in our study. It was comparatively higher as compared to previous studies[19-20].It may be because of location of our Institute,which is on national higway-11C.
As in other previous studies, the increasing age had unfavourable outcome in our study also[20-21]. Livingston et al and Lewin et al[22-23] showed similar results. Although persons under the influence of alcohol are more likely to sustain head injury, it was not significantly associated with clinical outcome in our study.

Miller et al[24] reported associated polytrauma in 49% of cases in their series. Neufiled et al[27], reported a high mortality of 83% in patients with severe head injury in shock, while Andrews et al[28] had poor outcome in 95.55% of severe head injured patients with hypotension and cardiac arrest. A single episode of hypotension has been reported to cause an 85% increase in mortality.[24-28]

Majority of patients (80%) came after 1 h of injury. In this study, hypotension was not significantly associated with unfavourable outcome. This is in contrast to previous studies[28-29], probably because of selection bias as all patients with severe chest, abdominal or orthopedic trauma were excluded in our study. It has been seen that a large number of patients with severe head injury die, not because of primary brain damage but because of additional brain insult, hypoxia being one of the most important of them[28-30] . In our study majority of patients (82.62%) who were hypoxic at the time of admission had unfavourable outcome.

Trauma has been shown to increase the vulnerability of the brain to ischemia for a period that may last for upto 24 hours[31].The resulting ischemia, if present, occurs in the first few hours following the trauma[32-33].It has also been shown that the cerebral autoregulatory function and CO2 vascular reactivity was impaired in the injured brain[31-33]. Hence, it is not surprising that polytrauma patients with hypotension have been reported to be associated with a poor outcome[33].Children with polytrauma are especially vulnerable to hypotension, which is compounded by their need of comparatively higher cerebral blood flow and oxygen requirements. Brain stem blood flow of less than 40 ml / 100 g / min, in patients with bilateral unreactive pupils is associated with a poor outcome[34].However, Bouma et al[35] showed that, these patients in addition to a decreased brain stem blood flow also demonstrated a global cerebral hypoperfusion (cerebral blood flow of 18 ml / 100 gm / min or less).

Sakaset al[36] reported that 17.5% of patients in their series had bilateral unresponsive pupils and localizing motor response. While lack of pupillary responsiveness and motor abnormalities are thought to reflect brain stem ischemia, frequent involvement of the third nerve nuclei in the brain stem suggests its higher vulnerability to ischemia than the corticospinal pathway.

Marmarouet al [37-40] reported that besides the age, motor score and pupillary responses at admission were the factors most likely to affect the outcome. It should, however, be noted that the ICP may be normal for the first 24 hours and may rise as late as 3 days following the trauma.[41]

Mortality and morbidity rates in patients sustaining severe head injuries remain high. In spite of various combinations of predictors, no model has satisfied all the requirements of an ideal model. This was a clinical study to determine and evaluate factors predicting outcome in patients with severe head injury. It should not be the reason to delay procedures like CT scan, ventriculostomy or other treatment plans.

Majority of previous studies have shown that GCS at the time of admission is a reliable predictor of final outcome. In our study, unfavourable outcome was significantly increasing with decreasing GCS. In our study, none of the surviving patients with both fixed pupils at admission was in grade V of GOS after six months. Various other studies have also proved that impaired pupillary response have a well documented correlation with unfavorable outcome.[41] CT scan is of utmost importance to guide further management as shown in our study and various previous studies. Prognostic role of CT scan in predicting outcome is also undisputed. Among all the CT findings which we studied, midline shift is the most important factor that influences the outcome[41-42].In our study, there was increase in mortality with increase in midline shift, with mortality reaching up to 55% in patients with midline shift of more than 5 mm. One out of five patients who had normal CT scans died.
Usually, a patient classified as functional outcome will have achieved this status by the first 6 months[43].Heiden et al[44] reported that 68% of the patients classified as severe disability at 1 month, had improved to the category of moderate disability or a good recovery at 6 months and 72% had achieved this status at 1 year. Interestingly, while the initial linguistic impairment is largely due to a dominant hemisphere injury, long term recovery of language function is most dependent on the duration of the coma[44].

It can be concluded from this study that road traffic accident was the commonest mode of severe head injury. Increasing age had unfavorable outcome. Hypoxia should be avoided on an absolute basis. Urgent CT scan of the head should be done to look for operable mass lesions as early detection and evacuation of the mass lesions saves life. Thus predicting outcome following traumatic severe head injury is an assimilative and integrative process of various pre-injury, injury and post-injury variables.

References:-

1) Maas AIR, Stocchetti N, Bullock R. Moderate and severe traumatic brain injury in adults. Lancet Neurol. 2008;7(8):728–41.
2) Finfer SR, Cohen J. Severe traumatic brain injury. Resuscitation. 2001;48:77–90.
3) Martins ET, Linhares MN, Sousa DS, Schroeder HK, Meinerz J, Rigo LA, Bertotti MM, Gullo J, Hohl A, Dal-Pizzol F, Walz R. Mortality in severe traumatic brain injury: a multivariated analysis of 748 Brazilian patients from Floriano´polis city. J Trauma. 2009;67:85–90.
4) Hukkelhoven CW, Steyerberg EW, Farace E, Habbema JD, Marshall LF, Maas AI. Regional differences in patient characteristics, case management, and outcomes in traumatic brain injury: experience from the tirilazad trials. J Neurosurg. 2002;97:549–57.
5) Harris OA, Bruce CA, Reid M, Cheeks R, Easley K, Surles MC, Pan Y, Rhoden-Salmon D, Webster D, Crandon I. Examination of the management of traumatic brain injury in the developing and developed world: focus on resource utilization, protocols, and practices that alter outcome. J Neurosurg. 2008;109:433–8.
6) Ghajar J. Traumatic brain injury. Lancet. 2000;356(9233):923–9.
7) Brain Trauma Foundation; American Association of Neurological Surgeons; Congress of Neurological Surgeons; Joint Section on Neurotrauma and Critical Care, AANS/CNS, Bratton SL, Chestnut RM, Ghajar J, McConnell Hammond FF, Harris OA, Hartl R, Manley GT, Nemecek A, Newell DW, Rosenthal G, Schouten J, Shutter L, Timmons SD, Ullman JS, Videtta W, Wilberger JE, Wright DW.
8) Guidelines for the management ofsevere traumatic brain injury. I. Blood pressure and oxygenation. J Neurotrauma. 2007;24(Suppl 1):S7–13.
9) Jones PA, Andrews PJ, Midgley S, Anderson SI, Piper IR, Tocher JL, Housley AM, Corrie JA, Slattery J, Dearden NM, Miller JD. Measuring the burden of secondary insults in head-injured patients during intensive care. J NeurosurgAnesthesiol. 1994;6:4
10) Marmarou A, Anderson RL, Ward JD, Choi SC, Young HF. Impact of ICP instability and hypotension on outcome in patients with severe head trauma. J Neurosurg. 1991;75:S59–66.
11) Stuart GG, Merry SG, Yelland JDN. Severe head injury managed without intracranial pressure monitoring. J Neurosurg. 1983;59:601e605.
12) Pillai SV, Kolluri VR, Praharaj SS. Outcome prediction model for diffuse brain injuries: development and evolution. Neurol India. 2003;51:345e349.
13) Narayan RK, Greenberg RP, Miller JD, Enas GG. Improved confidence of outcome prediction in severe head injury. J Neurosurg. 1981;54:751e762. Marshal LF, Smith R, Shapiro HT : The outcome with aggressive treatment in severe head injuries. Part-I The significance of intracranial pressure monitoring. J Neurosurg 1979; 50 : 20.
14) Becker DP, Miller JD, Ward JD et al : The outcome from severe head injury with early diagnosis and intensive management. J Neurosurg 1977; 47 : 491-502.
15) Braakman R, Gelpkca GJ, Habbemma JDF et al : Systematic selection of prognostic failure in patients with severe head injury. Neurosurgery 1980; 6 : 62-79.
16) Guterman P, ShenkinHA : Prognostic features in recovery from traumatic decerebration. J Neurosurg 1970; 32 : 330-335.
17) Jennett B, Teasdale G, Braakmann R et al : Prognosis of patients with severe head injury. Neurosurgery 1979; 4:283-289.
18) Miller JW, Butterworth JF, Gudeman SK et al : Further experiences in the management of severe head injury. J Neurosurg 1981; 54 : 289-299.
19) Teasdale G, JennettB : Assessment of coma and impaired consciousness – a practical scale. Lancet 1974; 2 : 81-84.
20) Hukkelhoven CW, Steyerberg EW, Rampen AJ, France E. Patient age and outcome following severe traumatic brain injury: an analysis of 5600 patients. J Neurosurg. N 2003;99:666e673.
21) Pfenninger J, Kaiser G, Lutschg J, Sutter M. Treatment and outcome of severely head injured child. Intensive Care Med. 1983;9:13e16.
22) Livingstone DH, Lavery RF, Mosenthal AC, Knudson MM, Manley GT. Recovery at one year following isolated traumatic brain injury: a western trauma association prospective multicentre trial. J Trauma. 2005;59:1298e1303.
23) Lewin W, Marshall TFDE, Roberts AH. Long term outcome after head injury. BMJ. 1979;2:1533e1538.
24) Miller JW, Butterworth JF, Gudeman SK et al : Further experiences in the management of severe head injury. J Neurosurg 1981; 54 : 289-299.
25) Teasdale G, JennettB : Assessment of coma and impaired consciousness – a practical scale. Lancet 1974; 2 : 81-84.
26) Jennet B, Bond M : Assessment of outcome after severe brain damage, a practical scale. Lancet 1975; 1 : 480-484.
27) Newfield P, Pitts LH, Kaktins J et al : Influence of shock on survival after head injury. Neurosurgery 1980; 6 : 596-597.
28) Chestnut RM, Marshal LF, Marshal SB : Medical management of intracranial pressure. In Cooper PR (ed.) Head injury 3rd edition, Williams & Wilkins, Baltimore. 1993; 225-246.
29) Jenkins LW, Moszyniski K, Lyeth BG et al : Increased vulnerability of the mildly traumatized rat brain to cerebral ischaemia: use of controlled secondary ischaemia as a research tool to identify common or different mechanisms to mechanical and ischaemic brain injury. Brain Res 1969; 477 : 211-224.
30) Bouma GJ, Muizelaar JP, Chou SC et al : Cerebral circulation and metabolism after severe traumatic brain injury the elusive role of ischaemia. J Neurosurg 1991; 75 :685-693.

31) MuizelaarP : Effect of manitol on ICP and CBF and correlation with pressure autoregulation in severe head injured patients. J Neurosurg. 1984; 61 : 700.
32) Nordstrom CH : Cerebral blood flow, vasoreactivity and oxygen consumption during barbiturate therapy in sever traumatic brain lesions. J Neurosurg 1988; 68 : 424.
33) Kennedy C, SokoloffL : Normal values for cerebral blood flow and cerebral metabolic rate in children. J Clin Invest, 1957; 36 : 1130. .
34) Ritter AM, Muizellar JP, Barnes T et al : Brain stem blood flow, pupillary response and outcome in patients with severe head injury. Neurosurgery 1999; 44 : 941-948.
35) Bouma GJ, Muzzelaar JP, Stringer WA et al : Ultra early evaluation of regional cerebral blood flow in severely head injured patients with xenon enhanced computerized tomography. J Neurosurg 1992; 77 : 360-368.
36) Marion DW, Darby J, YonasH : Acute cerebral blood flow changes caused by the severe head injury. J Neurosurg 1991; 74 : 407-414.
37) Eisenberg HM, Gary HE, Aldrich EF et al : Initial CT findings in 753 patients with severe head injury. A report from the NIH traumatic Coma Data Bank. J Neurosurg 1990; 73 : 688-698.
38) Ross DA, Olsen WL, Ross AM et al : Brain shift, level of consciousness and restoration of consciousness in patients with acute intracranial hematoma. J Neurosurg 1989; 71 : 498-802.
39) Toutant SM, Klauber MR, Marshal LF et al : Absent or compressed basal cisterns on first CT scan. Ominous predictor of outcome in severe head injury. J Neurosurg 1984; 61 : 691-694.
40) Marmonou A, Anderson RL, Ward JD et al : Impact of ICP instability and hypotension on the outcome in patients with severe head trauma. J Neurosurg 1991; 75 : S59-66.
41) Kishore PRS, Lipper MH, Becker DP et al : Significance ofCT in head injury: correlation with intracranial pressure.AJNR 1981; 2 : 307-311.
42) Chan K, Dearde NM, Miller JD : Intracranial blood flowvelocity after head injury. Relationship to severity of injury,neurological states and outcome. J NeurolNeurosurgPsychiatry 1992; 55 : 787-791.
43) Jennett B, Snoek J, Bond MR et al : Disability after severe head injury, observations on the use of the Glasgow Outcome Scale. J NeurolNeurosurg Psychiatry 1981; 44 : 285-293.
44) Heiden JS, Small R, Calon W et al : Severe head injury .Clinical assessment and outcome. PhysTher 1983; 63 : 1946-1951.