

**A CLINICO-RADIOLOGICAL STUDY AND MANAGEMENT OF HEAD INJURY WITH
SPECIAL REFERENCE TO SEVERE HEAD INJURIES**Uday Gupta¹, Rangeel Singh Raina^{2*} and Sona Kaushal Bharti³¹Post Graduate M Ch Neurosurgery, Department of Neurosurgery, KMC, Manipal.²Professor & Head, Department of Pharmacology, Government Doon Medical College, Dehradun.³Associate Professor & Head, Department of Biochemistry, Government Doon Medical College, Dehradun.***Corresponding Author: Dr. Rangeel Singh Raina**

Professor & Head, Department of Pharmacology, Government Doon Medical College, Dehradun.

Article Received on 20/10/2018

Article Revised on 10/11/2018

Article Accepted on 30/11/2018

ABSTRACT

Background: Trauma is the leading cause of death of people from one to forty four (44) years of age and Traumatic brain injury is the main determinant of morbidity, disability and mortality in such patient. **Aims and objective:** To study the incidences, etiological factors and clinical presentation and neurologically assessment of severe head injuries patient. **Material and Methods:** A prospective study was carried out on the admitted patients of head injury and was followed up to 6 months after discharge. **Results:** Males are more prone for head injury with more frequent in (54.6%) in 20-40 years age group mainly due to road traffic accident. Among road traffic accidents two wheeler accident (56.2%) was the predominant mode of trauma in head injury patients and most of the patients were not wearing helmets (82.2%). CSF otorrhoea (20.1%) was more common than CSF rhinorrhoea (8.4%). History of unconsciousness (62.2%) was the most clinical finding in patient followed by vomiting (37.8%), ENT bleed (26.1%) and convulsions (8.4%). Incidence of GCS 3-8 was seen in 26.9 cases and between 9-12 was seen in 27.7% cases, whereas 13-15 score was seen in 45.4% cases. Mortality & morbidity in head injury patients has inverse relationship with GCS score. **Conclusion:** Head injuries are more common in Males of 20-40 years age group with road traffic accident as the predominant mode of trauma. However immediate intervention and proper post operative care and dedicated efforts improve mortality and morbidity.

KEYWORDS: Traumatic brain injury, Glasgow Coma Scale, intracranial pressure, hemiparesis.**INTRODUCTION**

Traumatic brain injury (TBI) is a non- degenerative, non-congenital insult to the brain from an external mechanical force. Possibly leading to permanent or temporary impairment of cognitive, physical and psychological functions with an associated diminished or altered state of consciousness.

Trauma is the most important cause of fatality in people from 1st year to 44 years of life. Traumatic brain injury is the main determinant of morbidity, disability and mortality in this group.^[1] Severe TBI is associated with 30 to 70% mortality rate^[2] and recovery of survivors is marked by severe neurological sequels thus impairing quality of life.^[3] Severity of TBI is generally established using the Glasgow Coma Scale (GCS). This scale was achieved by observation of three parameters: Eye opening, Motor response, and Verbal response.^[3] TBI is ranked as severe, with a GCS score of 3 to 8, moderate from 9 to 12 and mild from 13 to 15.^[4]

The GCS has been used as one of the most important predictors of TBI outcome, although other variables such as age, abnormal motor response, cranial computed

tomography (CCT) findings, pupillary abnormalities and episodes of hypoxia and hypotension have been subsequently introduced in an effort to reach a more precise prognosis. It often difficult to determine the GCS score in emergency room due to intubation or sedation during pre- hospital care.^[5] Interference of alcohol intoxication in decreasing the score of GCS is controversial.^[6] The Glasgow outcome Scale (GOS) is the most used score for assessment of functional prognosis after TBI.^[7] It has five levels, from death to good positive prognosis and is normally obtained at 3, 6 and 12 months after trauma^[8], However, it presents important shortcomings and early assessment Of brain damage may be very difficult during the patient's stay in intensive care unit.^[7]

Cranial Computed Tomography (CCT) is the image of choice for management of TBI in an emergency room. With this exam, hematomas can be speedily diagnosed and as required, support early surgical treatment.^[9] Marshall et al^[10] developed a classification for CT finding in TBI. CT alterations used for the classification are oedema (assessed by compressions or absence of cisterns 0, volume of high or mixed density injuries

(blood collections), midline deviation and evacuation of mass injury.^[8] Various studies confirmed the prognostic predictive value of Marshall Classification in TBI patients.^[11] Recovery after TBI was related to severity of initial damage (primary lesion) and presence of secondary injury.^[12] An important cause of development of secondary injury is development of intracranial hypertension (ICH) which may be due to intracranial hematoma or cerebral oedema.

The traditional goal of management of patients with TBI has been to limit the secondary damage by manipulation of intracranial pressure and of cerebral perfusion pressure as well as to avoid aggravating factors such as hypoxemia and hypotension to ameliorate the prognosis of the patients.^[3]

AIMS AND OBJECTIVES

1. To determine the incidence of age, sex, distribution, etiological factors, clinical presentation, neurologically assessment and mode of head injuries with particular reference to severe head injury.
2. To assess the mortality & morbidity in the patient of head injury in form of clinical review & type of intracranial lesions.

MATERIAL AND METHODS

This prospective study was carried out in Subharti Medical College and associated CSSH hospital, Meerut in the patients of head injury who were admitted during September 2011 to December 2012 and were followed up to 6 month after discharge up to July 2013.

Inclusion criteria

1. All patients of mild to severe head injury admitted in CSSH in emergency department are included. Severity was based on different GCS levels.
2. Patients of all age groups & all sexes were included.
3. Patients of poly- trauma with head injury were included.
4. Those were having skull fractures & cases with intra – cranial air on CT were included.
5. Emphasis was given to patients with severe head injury (GCS3-8).

Table 1: Age and sex distribution of cases.

| S.NO. | Age Groups | Male (n=85) | Female (n=34) | Total |
|-------|------------|-------------|---------------|-------|
| 1. | 0-5 | 4 | 3 | 7 |
| 2. | 6-10 | 4 | 3 | 7 |
| 3. | 11-15 | 3 | 3 | 6 |
| 4. | 16-20 | 7 | 1 | 8 |
| 5. | 21-40 | 49 | 16 | 65 |
| 6. | 41-60 | 12 | 6 | 18 |
| 7. | >60 | 6 | 2 | 8 |

Maximum number of head injury was caused due to road traffic accidents (Table 2). The above table shows that road traffic accident is the main cause of head injury in majority of cases (62.5%) with male preponderance (62 cases: 52.1%). Incidence of head injury due to fall from

Exclusion criteria

Those patients who refused investigations or were not available for a 6 month follow up were excluded.

All the details about patient's profile (Name, Age, Sex, Date of admission and discharge, Short history: mechanisms of injury, unconsciousness, vomiting, convulsions, ENT bleeds) was taken into account of patients of head injuries and examinations and investigations were conducted.

1. General examination

- a. External injury: laceration, abrasion or contusion.
- b. Systemic injury: long bone or pelvic fracture, maxillary or mandible fracture chest injury, abdominal visceral injury or spinal injury.

2. Clinical examination: Patients were assessed clinically in terms of:

- a) Glasgow coma scale – which include best motor response, verbal response & eye- opening
 - b) Pupillary examination – size & reaction were noted.
 - c) Planter reflexes : flexor or extensor
 - d) Pulse & Blood pressure
 - e) Localizing sign – mono paresis & quadric paresis or sign of any cranial nerve injury
 - f) Evaluation of discharge from ear & nose.
3. Radiological investigations : x- ray Skull- A.P./ Lateral view, CT scan/ MRI
 4. Management : Conservative / Operative
 5. Final outcome

RESULTS

This was a prospective study was carried out in Subharti Medical College and associated CSSH hospital on patients of head injury who were admitted during September 2011 to December 2012 and were followed up to 6 months after discharge. There were 823 head injury patients which were admitted in the hospital 119 were found in this group.

Males were more prone to head injuries than females in age groups above 16 years. Below 16 years incidence was same (Table 1).

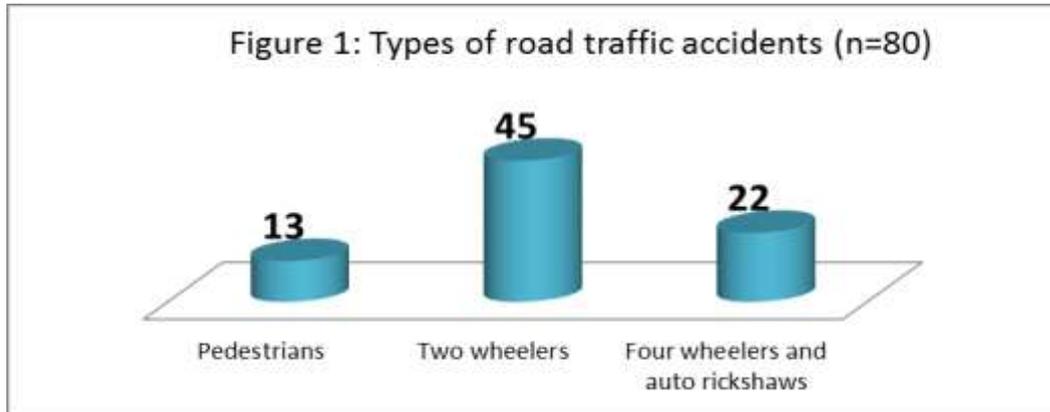
height was nearly equal in both sex. It was found that maximum number of head injury patients belonged to the age group of 21-40 years (54.6%).

Table 2: Distribution of cases according to mode of injury.

| S.No. | Mode of head injury | No of patients | Incidence(%) |
|-------|-----------------------------|----------------|--------------|
| 1. | Road traffic accidents(RTA) | 80 | 67.2 |
| 2. | Fall from height | 23 | 19.3 |
| 3. | Non-fire arm assault | 10 | 8.4 |
| 4. | Firearm injury | 6 | 5.04 |

Majority of the accident victims of head injury were two-wheeler riders (56.2%), followed by four-wheeler/auto-

rickshaw travellers (27.6%.) (Figure 1). All the patients on two wheelers were not wearing helmets.

**Figure -1: Types of road traffic accidents (n=80).**

Long bone or pelvic fracture was the most common associated injury (9.24%) in these patients. Road traffic accident was the most common cause of associated

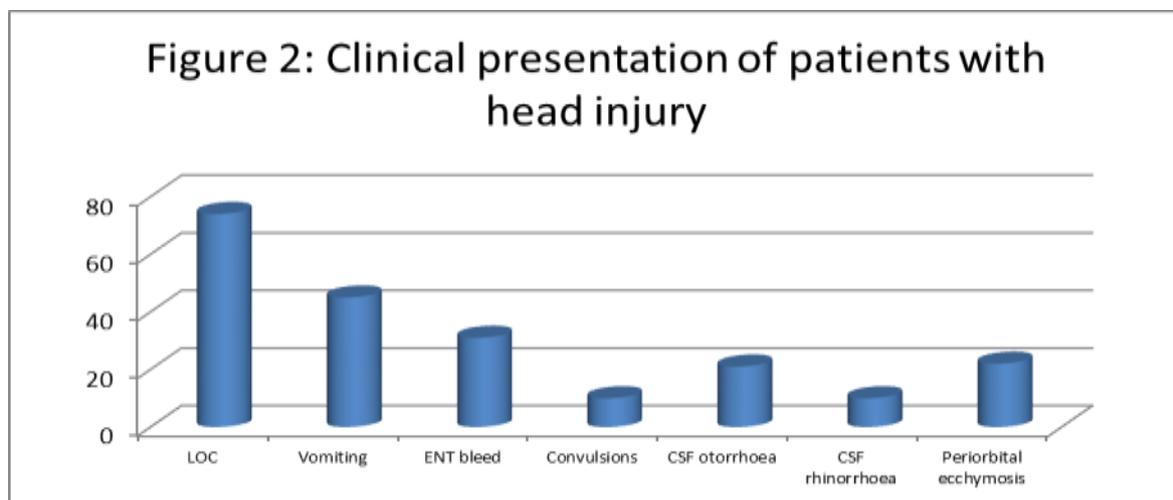
systemic injury affecting the long bone or pelvis (81.8%) followed by major chest injury and maxillary or mandibular fracture. (Table 3).

Table 3: Relationship between mode of head injury and associated systemic injuries.

| S No. | Type of Injury | Mode Of Injury Road traffic accident | Fall from height | Non-firearm injury | Firearm injury |
|-------|----------------------------------|---|---------------------|-----------------------|-------------------|
| 1. | Long bone or pelvic fracture | 9 (81.8%) | 1 (9.1%) | 1 (9.1%) | - |
| 2. | Maxillary or mandibular fracture | 2 (50.0%) | 1 (25.0%) | 1 (25.0%) | - |
| 3. | Major chest injury | 3 (75.0%) | 1 (25.0%) | - | - |
| 4. | Abdominal visceral injury | 3 (100%) | - | - | - |
| 5. | Spinal injury | 1 (50.0%) | 1 (50.0%) | - | - |

History of unconsciousness is found in 62.2% of patients of head injury followed by vomiting (37.8%), ENT bleed (26.1%) and convulsions (8.4%). At the time of

admission hemiparesis was found in 16.8% of head injury patients (Figure 2).

**Figure 2: Clinical presentation of patients with head injury.**

Bone deficit/skull fracture was present in 35% patients. Generalized cerebral oedema was observed in 34.4% cases. 36.9% cases had hemorrhagic contusion/Intracerebral hematoma and 21.1% cases had acute subdural hematoma. In 45.2% cases, skull fracture was present in frontal region followed by temporal or temporo-parietal region (33.3%) and parieto-occipital region (21.5%). Extradural hematoma was maximally present in temporo-parietal region in 65.2% cases and

coup injury was greater in all the site of skull fracture. Haemorrhagic contusion /intracerebral hematoma was maximally present in frontal region(48.9%) of cases and at all sites coup injuries were greater than contre-coup injuries.

The study showed that the patient with low GCS (3-8) has got the highest mortality while patient with high GCS (13-15) has got the highest survival (Table 4 & 5).

Table 4: GCS in patients with compound head injury and outcome.

| S.No. | GCS | No. of patients | Incidence (%) | Outcome (Good Recovery) |
|-------|-------|-----------------|---------------|-------------------------|
| 1. | 3-8 | 32 | 26.9 | 0 |
| 2. | 9-12 | 33 | 27.7 | 24 (20.1%) |
| 3 | 13-15 | 54 | 45.4 | 52 (43.7%) |

Table 5: Outcome associated with GCS in compound head injury.

| Outcome (GCS) | Severe Head Injury 3-8 | Moderate Head Injury 9-12 | Mild Head Injury 13-15 |
|---------------------|---------------------------|------------------------------|---------------------------|
| Good recovery | 0 | 24 (20.1%) | 52 (43.7%) |
| Moderate disability | 9 (7.6%) | 6 (5.5%) | - |
| Severe disability | 3 (2.5%) | 2 (2.5%) | 1 (.9%) |
| Vegetative | 11 (9.5%) | - | - |
| Dead | 9 (7.6%) | 1 (.9%) | 1 (.9%) |

In this study wound infection was the most common sequel (4.4%) of head injury. Post-traumatic lepto-

meningeal cyst and intracerebral abscess were found in 0.9% cases and 1.7% respectively.(Figure 3).

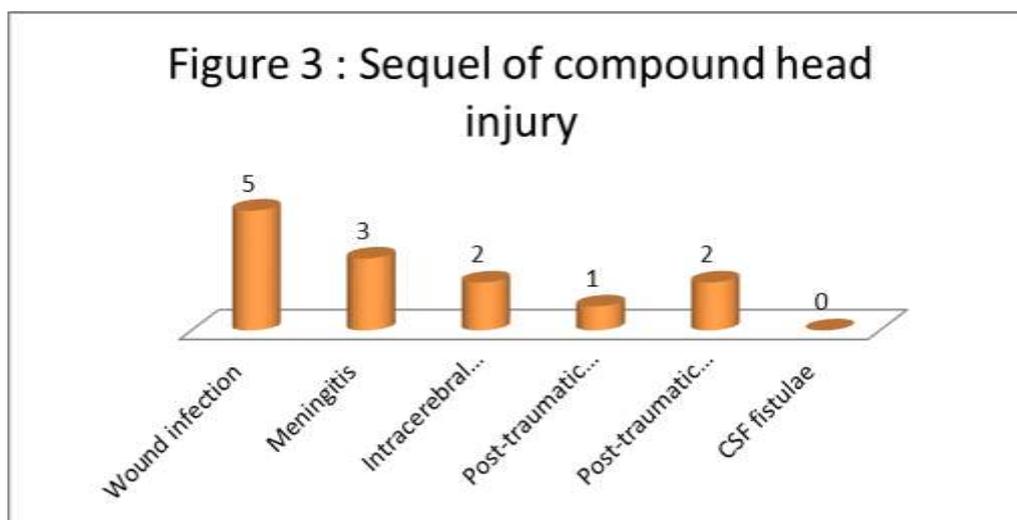


Figure 3: Sequel of compound head injury.

In the study the most frequently reported symptom is headache(5.76%) and in 3.84% cases it is still present after 2 months, followed by weakness, dizziness, fits, hearing problem (deafness) rashes, memory problem (forgetfulness). The incidence of all symptoms decreased further after 6 months.

DISCUSSION

Head injury is a nightmare for our society. It is a problem of enormous magnitude with great medical, social, economical and epidemiological significance. The present study was carried out on 119 patients of head injury who were admitted and treated, discharged and

followed up to six months in neurosurgery unit of this hospital during study period. In this study head injury constituted 46.3% of trauma patients admitted to this admitted. The incidence of severe head injury was 27% of all the head injuries admitted.

Mechanism of head injury

Commonest cause of head injury is road traffic accidents. This is mainly due to urbanization and increasing number of vehicular population. In a study by Kraus^[13], 60% of injuries were due to road traffic accidents and 20% to 30% of all injuries were due to fall. In our study road traffic accidents accounted for 67.2% of head injury

followed by fall from height in 19.3% cases. This slight increase in road traffic accidents were due to poor design of roads, poorly maintained vehicle with no safety facilities and poorly trained drivers in this part of the country. Another study has also reported large number of patients with head injury in road traffic accidents (64.0%).^[14]

Gender and Head injury

In this study male to female ratio was found to be 3:1. The overall ratio of males is more because of the excessive exposure of males to traffic and outdoor activities than females. Bharti *et al*^[14] has reported that it was predominantly males who were involved with head injuries (85.0) since they are actively involved with outdoor activities like driving and property disputes, etc. Similar results were observed by Kraus^[13] that overall head injury was less common in females, male to female ratio of incidence being 3:2.

Cause of Injury and Gender

Male and females are nearly equal. i.e. 12 males (10.08%) in comparison to 11 females (9.24%) in fall from height group. This is due to the fact that in this group most of the patients had fall from roof and among them majority are children and their activities are not influenced by sex.

Incidence of Head injury in different Age groups

In a study conducted by Reverdin *et al*^[15] head injury is most frequent in young people between 20-40 years and around 60-70% head injury occurs in human benign in 30s and 40s. In another study by Mahapatra.^[16] 25-27% of all head injury victims were children of 23.5%, but 54.6% patients belonged to the age group of 20-40 years. This is the age group (20-40) years that is maximally involved in activities the house, driving and quarrels and hence most susceptible.

Type of Road traffic accidents

Among road traffic accidents two wheeler accidents accounted for 56.2% of head injury patients and most of the patients of two wheelers were not wearing helmets (82.2%) as it is compulsory by law in most states but not followed by most people. Bharti *et al*^[14] has also observed that two wheeler was the common vehicle involved in accident (43.3%) and among these majority of them were not wearing helmets (75%).

Clinical presentation of Head injury patients

History of unconsciousness is found in 62.2% of patients of head injury followed by vomiting (37.8%), ENT bleed (26.1%) and convulsions (8.4%). Bricolo^[17] reported that 5% of patients with head injuries have seizures in post traumatic period. At the time of admission hemi paresis was found in 16.8% of head injury patients as compared to 19% by Becker *et al*.^[18] Pupillary changes were found in 38.6%, which were higher in comparison to 21% reported by Becker *et al*. Facial palsy was found in 5.8%

of head injury patients, bradycardia in 15.9% of patients and hypertension in 21.8%.

Treatment modalities in patients of Head injuries

As stated by Carey^[19], the aim of surgery in open compound head injuries is to convert an open injury to a close injury by debriding the scalp and damaged brain, by removing the bone fragments and elevation of depressed fractures and dural repair either by using the available adjacent dura or by grafting. Wylen and Nanda^[20] in a series of 52 patients of depressed skull fracture and carried out elevation and repair within 72 hours in 32 cases with good results.

Ommaya *et al*^[21] observed that maximum number of post-traumatic CSF leakage, i.e CSF rhinorrhoea and CSF otorrhoea will cease within one week with conservative treatment. In the present study, conservative treatment was given to all cases of CSF rhinorrhoea and CSF otorrhoea and all cases of CSF rhinorrhoea stopped within one week which was similar to the study of Ommaya (1977)^[21], which stated that maximum number of CSF rhinorrhoea will stop within one week.

Effect of GCS on the Outcomes of the patients

According to Marshall *et al* (1991)^[22] GCS provides simple grading of the arousal and functional capacity of the cerebral cortex. He found that as the GCS rises there is significant decline in mortality. In the present study there is 7.6% mortality among the patients having GCS 3-8 in comparison to 68.0% in the study of Marshall *et al* (1991).^[22] In the group of patients, vegetative patients were 18.8% total being 26.4%. As the GCS rises to 13-15 the mortality declines to 1.9% which too is nearly similar to 4%.^[5] Bharti *et al* (1993)^[14] reported that mortality was 100% in patients with GCS 4 and 23% with GCS between 5-8. The result is probably due to improve facilities and quick transportation to the hospital.

Sequel of Head injury

According to the study by Miller and Jennett^[23] on the complication of depressed skull fracture, wound infection was found in 4.2% of cases. This decrease in infection rate is probably due to the more precautions taken regarding sterility in pre and post op period. In the present study the most common symptom one week after discharge was headache (5.04%) which was similar to the study by Alves and Jane^[24] and the percentage of patients with headache remained almost constant up to 2 months after discharge, with further improvement at 6 months follow up.

Weakness was the next common symptom at time of discharge followed by forgetfulness, dizziness fits, deafness and rashes. All of them gradually improved over the period of time. One patient with rashes the cause was phenytoin induced allergy for which patient drug was changed to carbamazepine.

SUMMARY AND CONCLUSION

Males are more prone to head injury due to road traffic accident with maximum in two-wheeler accidents as most of the patients were not wearing helmets. CSF otorrhoea was more common than CSF rhinorrhoeas. Extradural hematoma was more common in tempero-parietal region and Coup injuries were more common than contre coup injuries.

Mortality & morbidity in head injury patients was found to increase rapidly with fall of GCS, however immediate intervention with proper post-operative care and dedicated efforts improve morbidity. There is a distinct advantage of reducing pressure by decompressive craniotomy with duraplasty. Early and aggressive management always gives good results.

REFERENCES

1. Bruns J Jr, Hauser WA. The epidemiology of traumatic brain injury: a review. *Epilepsia*, 2003; 44 Supple 10: 2-10.
2. Kraus JF, Macarthur DL, Silverman TA, Jayaram M. Epidemiology of brain injury. In: Natrajan RK, Wilberger JR, Povlishock JT, editors. *Neurotrauma*. New York: Mc Graw Hill, 1996; 13-30.
3. Finfer SR, Cohen J. Severe traumatic brain injury. *Resuscitation*, 2001; 48(1): 77-90.
4. Gharjar J. Traumatic brain injury. *Lancet*, 2000; 356(9233): 923-9: Review.
5. Balestreri M, Czosnyka M, Chatfield DA, Steiner LA, Schmidt EA, Mielewski P, et al. Predictive value of Glasgow coma scale after traumatic brain injury: Change in trend over past ten years. *J Neurol Neurosurg Psychiatry*, 2004; 75(1): 161-2.
6. Styke L, Diaz-Arrastia R, Gentile L, Shafi S. Effect of alcohol on Glasgow Coma Scale in Head injured patient. *Ann Surg*, 2007; 245(4): 651-5.
7. Da Richa AB, Schneider RF, de Freitas GR, Andre C, Grivicich I, Zanoni C, et al. Role of serum S100B as a predictive marker of fatal outcome following isolated severe head injury or multi-trauma in males. *Clin Chem Lab Med*, 2006; 44(10): 1234-42.
8. Zink BJ. Traumatic brain injury outcome: concepts for emergency care. *Ann Emerg Med*, 2001; 37(3): 318-32.
9. Toyama Y, Kobayashi T, Nishiyama Y, Satoh K, Ohkawa M, Seki K. CT for acute stage of closed injury. *Radiat Med*, 2005; 23(5): 309-16.
10. Marshall LF, Eisenberg HM, John A, Thomas G L, Antony M, Mary AF. A new classification of head injury based on computed tomography. *J Neurosurg*, 1991; 75: s14-s20.
11. Mass AI, Hukkelhoven CW, Marshall LF, Steyerberg EW. Prediction of outcome in traumatic brain injury with computed tomographic characteristics: a comparison between the computed tomographic classification and combinations of computed tomographic predictors. *Neurosurgery*.
12. Doberstein CE, Hovda DA, Becker DP. Clinical considerations in the reduction of secondary brain injury. *Ann Emerg Med*, 1993; 22(6): 993-7.
13. Rendell JB, Livy, Dask PK. Traumatic brain injury clinical management and research. Elizabeth Frost (eds) AIREN – Geneva, Switzerland ISSN-1012-9871: 113-24.
14. Kraus J.F. Epidemiology: In MINS head injury clinical management and research. Elizabeth Frost (eds) AIREN- Geneva. Switzerland ISSN-1012-9871: 113-24.
15. Reverdin A. Head injury in children. In NINS. Head injury, Clinical Management and Research. Elizabeth Frost (Eds), Airen Publisher, Geneva Switzerland, 1990: 193-204.
16. Mahapatra AK, Jaiswal A. Epidemiology of Head Injury. *Neuroscience Today*, 2003; 7: 29-32.
17. Natrajan M. Emergency care and initial evaluation of head injury. *Antiseptic*, June 1987; 312.
18. Strangl, MacMilan R, Jennett B. Head Injuries in accident and emergency department at Scottish Hospitals. *Injury*, 1988; 10: 154-5.
19. Wylen EL, Nanda A. Infection rate with replacement of bone fragment in compound depressed skull fractures. *Surg Neurol*, 1999; 4: 452-457.
20. Stephanov S. Brain abscess from neglected open head injuries. Experience with 17 cases over 20 years. *Swiss Surg*, 1999; 6: 288-92.
21. Ameen A, Ameen et al. Variables for post traumatic meningitis. *J Neurosurg*, 1998; 88: 471-477.
22. Lobato RD, Cordobes F, Rivas J J, et al. Normal computerized tomography scans in severe head injury. Prognostic and clinical management implications. *J Neurosurg*, 1996; 67: 648-656.
23. Kreider GN: Repair of cranial defect by a new method. *JAMA*, 1920; 64: 1024.
24. Becker D.P., et al: The outcome of severe head injury with early diagnosis and intensive management. *J Neurosurgery*, 1977; 47: 491-502.