

CLINICOPATHOLOGICAL STUDY OF PAEDIATRIC HEAD INJURY IN GANDHI MEDICAL COLLEGE BHOPAL FROM MAY 2011 TO JUNE 2013Sudhir Singh Pal¹, Archana Shukla², Amit Jain³**HOW TO CITE THIS ARTICLE:**

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ABSTRACT: A clinic pathological study of paediatric head injury was conducted in patients admitted in Gandhi medical college Bhopal during a period of 23 months from June 2011 to May 2013. This was done to assess various epidemiological parameters that influence that causation to trauma as well as the consequent morbidity and mortality in the paediatric age group. The study also included analysis of the paediatric age group head injury in relation to the age, sex, mode and type of injury. We also estimated differences in the incidence of recorded traumatic head injury by geographic area. Computerized tomography was done to diagnose the degree of the severity. Patients were classified by Glasgow coma scale and Scandinavian neurotrauma committee classification. It was found that 6-9 years of aged male patient with history of fall from height followed by RTA was the most common group. Closed injuries are more common and mostly associated with the orthopedic injuries. Chest infection is most common complication. Prognosis of open injury is better than closed.

KEYWORDS: Paediatric head injury, Clinicopathological, management.

INTRODUCTION: Head injuries are very common problem in the paediatric population. They are one of the primary causes of injury mortality and morbidity in childhood. As per National Crime Records Bureau (NCRB) report of 2006, there were 22,766 deaths (<14 years) due to injuries among children.^[1] According to the World Health Organization, falls ranked as the world's fifth most common cause of death in children aged 5-14 years in the year 2000, and falling is the most frequent type of accidental injury. It is interesting to note that these types of injuries mainly occur in the home, since it is the place where children spend most of their time.

There are very few studies from developing countries discussing the epidemiology of pediatric trauma. Our study aims to determine the frequency of various types of childhood injuries in different sex and age groups and also to find out the various modes and place of trauma among study subjects and their distribution according to different age and sex groups. It also gives an idea about the relative mortality in various types of childhood injuries.

AIMS AND OBJECTIVE: To assess the various epidemiological parameters that influence the causation of trauma as well as the consequent morbidity and mortality in the pediatric age group.

- Analysis of paediatric head injury in relation to age distribution, sex, mode of injury, and types of injuries.
- To estimate differences in the incidence of recorded traumatic head injuries by geographical area.
- To assess the various complications in comparison to closed v/s open injury and radiological parameters.

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The most common causes of head trauma in the first year of age are falls from parental arms, from changing tables or chairs, and are usually low-impact injuries. As the child learns to walk, falls from greater heights occur. As the child grows, due to the lack of research abilities, accidents of the car pedestrian type are becoming more common. In older children, there is an increasing prevalence of bicycle accidents, sports injuries, car accidents with the child as a passenger in a motor-car.

PATHOGENESIS OF BRAIN INJURY: After severe traumatic brain injury reduction in cerebral blood flow (CBF) begins almost immediately after injury lasting as long as 24 hours. This happens due to neurochemically mediated vasospasm, astrocyte swelling with compression of the microcirculation. This early hypoperfusion with normal metabolic requirement is a high risk setting and any associated hypotension or hypoxia leads to further hypoxic ischemic injury to the brain. As the injury evolves, blood brain barrier disruption occurs, vasogenic edema occurs. In addition cytotoxic edema is a key factor to secondary cerebral swelling. Enlarging hematoma also contributes to decreased cerebral perfusion pressure (CPP) by increasing the intracranial volume and intracranial pressure (ICP) and decreasing cerebral blood flow. Loss of cerebral auto regulation occurs frequently. Normal cerebral blood flow regulation in response to changes in blood pressure and cerebral vascular tone is absent. Eventually cerebral edema progressively increase the ICP further reducing the CPP (mean arterial pressure (MAP)-intracranial pressure). Aldrich et al^[2] reported that incidence of diffuse cerebral swelling was 41% in children as compared to 26% frequency in adults, possibly due to a hyperemic response (luxury perfusion). Lang et al reported incidence of diffuse cerebral swelling at 4 to 5 times that of adults.^[3]

CLINICAL EVALUATION AND MANAGEMENT IN EMERGENCY: In any child with multiple trauma, a quick primary and secondary survey is performed with prompt attention to airway, breathing and circulation (See chapter on multiple trauma). Pediatric patient with head injury may be brought unconscious, posturing (Decerebrate or decorticate), or actively convulsing. All patients should be presumed to be full stomach and oxygen therapy should be initiated. Comatose patients need to be intubated with rapid sequence intubation technique, with due attention to cervical spine stabilization. Jaw thrust maneuver can be performed during bag mask ventilation.^[4] Head tilt and chin lift maneuvers should be avoided. A cervical spine collar should be placed until cervical spine X-rays are obtained to rule out a fracture or dislocation.

GLASGOW COMA SCALE (GCS): For clinical evaluation, as much as possible, GCS,^{[5][6]} for adults should not be used considering the anatomical, physiological and developmental differences of pediatric age group, a modified GCS is available.^{[7][8][9][10]}

INDICES OF GOOD OUTCOME: Single most reliable examination for evaluating outcome in children less than 3 years of age is ocular examination, as oculomotor functions are fully developed by two months of age, while cortico spinal myelination and optic pathway myelination develop much later. Child with open fontanel and ocular score of 3-4 generally has good outcome. Similarly children with motor score of 4 and closed fontanel will have good outcome. Those with closed fontanel and verbal score of 3 have good outcome.

INDICES OF POOR OUTCOME: Evidence of retinal hemorrhage indicates poor outcome. Radiographic evidence of post-traumatic splitting of suture indicates poor outcome and high incidence of seizures. A bilateral linear skull fracture correlates with poor outcome. Incidence of post-traumatic seizure is 10% in children with depressed fracture and 7% without depressed fracture. Occurrence of generalized tonic clonic seizure indicates poor outcome to the extent of 27 to 66%. Irrespective of closed or open fontanel, children with motor neurological deficit like hemiparesis have poor outcome in about 25% of cases.

The child has suffered a severe head injury irrespective of child coma score, if any of the following is present: Unequal pupils (A difference of ³ 1 mm.) Asymmetric motor examination, Open head injury with CSF leak or exposed brain, Neurological deterioration (GCS decreased by ³ 2 points).^[11]

TYPES OF INJURIES:

1. **Injuries to Scalp:** These range from abrasion, subgaleal or sub-periosteal hematoma to laceration of various size, shape and depth, depending upon mode and nature of injury.
2. **Fractures**^{[13][14][15]}
 - a. **LINEAR FRACTURE:** Single fracture line passes through entire thickness of skull. A simple linear fracture in itself does not require any intervention. Patient should place in PICU observation specially when the fractures lines are crossing over trajectory of meningeal artery or dural sinus.
 - b. **DIASTATIC FRACTURE:** Linear, Comminuted, Stellate common in children < 3 years. A diastatic linear fracture is break in skull continuity, more specifically sutures, with coarse definite separation of bone edges by > 5 mm over distance of 2 cm. They are potential source of epidural or subdural haematoma or meningocoele.
 - c. **BASILAR SKULL FRACTURE:** Basilar skull fracture may present as raccoon eyes (anterior fracture) or battle sign (Petechial hemorrhage over mastoid) in middle cranial fossa fracture). Basilar skull fractures may lead to cerebrospinal fluid (CSF) leak or cerebrocele. With CSF leak chances of meningitis are very high and appropriate meningeal coverage with antibiotics.
 - d. **Depressed Skull Fracture:** Outer table of one or more of the fracture edges lie below normal anatomic level of the inner table as compared to surrounding intact skull. It could be closed or open. An open depressed fracture is surgical emergency. Surgery for closed depressed fracture is usually cosmetic but if radiological appearance suggest dural laceration, brain penetration, mass effect, or underlying subdural hematoma, extradural hematoma or Intracranial hematoma exploration required. In neonates and infants depressed fracture are green stick fracture located in frontal or parietal areas. They are called "Ping-Pong" fracture because they show indented bone without fracture in cortical layers. They may resolve spontaneously, fracture > 5 mm may require surgical intervention.
 - e. **COMPOUND SKULL FRACTURE:** This is a neurosurgical emergency. Once diagnosed clinically, a skull X-ray^[16] and CT scan of the head is mandatory. Appropriate management can be undertaken based on findings.

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- 3. Intracranial hematoma:** Extradural (epidural), subdural, intradural, contusions may be encountered. CT scan of the head is diagnostic and the investigation of choice.

EPIDURAL HEMATOMA: Epidural hematoma occurs mainly at the convexity or posterior fossa.^[17] It could occur due to middle meningeal arterial bleed or tearing of bridging veins. It occurs due to compression /decompression injury, acceleration/deceleration injury or direct trauma and usually present as:

Initially normal child coma score followed by progressive deterioration. Initial loss of consciousness, followed by recovery (Lucid interval), followed by loss of consciousness.

SUBDURAL HEMATOMA: SDH can be acute, subacute or chronic. Acute subdural hematoma is associated with poor outcome and is clinically characterized by severe depression of level of consciousness with focal deficit and cerebral injury. Sub-acute, presents as progressive deterioration of consciousness and/or neurological function, associated with good outcome with appropriate and timely neurosurgical intervention. Chronic subdural hematoma is the most common entity in pediatric traumatology, and is amenable to surgical treatment.

INTRA CEREBRAL HEMATOMA AND CONTUSIONS: These have variable and multiple presentations ranging from perfectly normal to mildly depressed level of consciousness, to severe neurological deficit, seizures and coma. They may or may not require surgical intervention, may or may not respond to surgical management depending upon extent and nature of damage.

DIFFUSE BRAIN INJURY: Depending upon severity of impact, head injury could lead to minor concussion or diffuse axonal injury. A concussion is temporary loss of neurological function immediately after trauma with no radiological findings and associated with complete recovery. While diffuse axonal injury is usually due to shearing movements of the brain during acceleration deceleration injuries, it is almost always associated with poor outcome. Patient with poor neurological status whose clinical status does not correlate with CT scan findings should be subjected to MRI which usually will reveal lesion in corpus-callosum, rostral part of pons or in the internal capsule.

- 4. Other injuries:** Some of the other injuries commonly seen are CSF leak, growing fracture of skull or cerebral vascular injury.

RADIOLOGICAL INVESTIGATIONS:

- 1. Skull X-ray:** lateral view and AP view of skull should be taken. It can reveal fracture, diastasis and other bony abnormalities. If a CT scan is easily available X-ray skull can be avoided.
- 2. CT Scan:** CT^{[18],[19],[20]} is the investigation of choice. It reveals bony injury, hematoma (Appears hyperdense when compared to brain parenchyma), evidence of cerebral edema (Hypodense compared to normal brain parenchyma and isodense compared to CSF) or mass effect (midline shift). CT is necessary for operative planning.
- 3. MRI:** had no role in acute management of a patient with head injury. It can be used in cases of diffuse axonal injury and in follow up for prognostication. Some-time a small lesion in vital

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areas of brain may not be seen on CT but seen on MRI. MRI is usually reserved for later detailed evaluation after acute problem has been addressed.

4. **Cervical Spine (Lateral View):** A lateral view of the cervical spine is obtained in the emergency room to rule out cervical bony injury or dislocation. This must be followed by full cervical spine series with anteroposterior and oblique views to rule out any fracture or dislocation

SURGICAL MANAGEMENT: Neurosurgical management includes operative removal of extradural (Epidural) hematoma or subdural hematomas as soon as possible after the diagnosis is made. ICP monitor needs to be placed in most cases for ICP and CPP monitoring and further management in the pediatric intensive care unit.(PICU).

NON-SURGICAL MANAGEMENT IN THE PICU: The basic aim is to minimize the primary injury and prevent secondary insult to the brain. Treatment remains supportive. As discussed in pathogenesis the key to management of a head injured patient with cerebral edema or diffuse axonal injury is to maintain cerebral perfusion pressure by control of ICP and hemodynamic status.

Primary cerebral injury is due to trauma per se, accumulation of blood clot and tissue damage leading to decreased cerebral blood flow, increased cerebral blood volume, increased intracranial pressure and decreased cerebral perfusion pressure.^{[21],[22]} Secondary injury is mainly due to failure of auto regulation and biochemical changes, which lead to cerebral edema (vasogenic, cytotoxic, hydrostatic or osmotic.) Tissue damage is mainly due to direct injury or apoptosis changes.

SEIZURE CONTROL: Midazolam may be used to control seizures in patients with status epilepticus. Propofol or Thiopentone may also be used, however hypotension associated with the use of these agents should be treated with fluid therapy. Phenytoin should be initiated in patients with post traumatic seizures, although use of prophylactic phenytoin in all head injured children is not supported by clinical evidence. In patients who are sedated and muscle relaxed, EEG should be monitored and nonconvulsive seizures should be controlled to reduce the cerebral oxygen consumption.

MANAGEMENT OF INTRACRANIAL HYPERTENSION AND CEREBRAL PERFUSION PRESSURE (CPP): Intracranial hypertension is defined as ICP >20 mm Hg for >5 min Recommendations for adults include maintaining an ICP of less than 20 mmHg and perfusion pressure (CPP) of >60 mmHg. These may be applicable for older children. Although no normal data is available through clinical evidence, in infants with severe traumatic brain injury it would be logical to suggest targeting for an ICP of <15 mm Hg and a CPP of >45 to 50 mm Hg. Careful use of inotropic agents such as dopamine may be necessary to maintain these parameters, however induced hypertension with phenylephrine is not recommended since cerebral autoregulation is lost and further increase in cerebral blood flow may exacerbate edema formation by hydrostatic effect.^{[23][24]} CSF drainage by venticulostomy has been shown to be as effective as mannitol therapy in reducing ICP^[25] Hypoxia and hypotension at any point of management is associated with poor outcome, should, therefore be avoided or promptly treated.

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MECHANICAL VENTILATION: In all children with Child Coma Score is less than 8, intubation and mechanical ventilation is indicated, head should be kept in midline and 30 degrees elevated position,^[26] Arterial blood gases should be monitored to maintain pCO₂ levels between 31 to 32 rather than 26 to 27 mm Hg for better outcome 6 months after trauma.^[27] Hypercarbia must be avoided. Positive endexpiratory pressures (PEEP) in the range of 4 to 5 cm of H₂O are harmless as far as ICP is concerned and should be used to prevent atelectasis.

SEDATION AND MUSCLE RELAXATION: Sedation and muscle relaxation is recommended for adequate control of ICP. Fentanyl, midazolam and vecuronium infusion is a good combination as long as hypotension is avoided. Subject to availability, pentobarbitone, phenobarbitone, propofol may be used. Intermittent thiopentone and Intravenous lidocaine is recommended to blunt raised ICP response while suctioning the endotracheal tube. Alternatively instillation of lidocaine in the endotracheal tube may be as effective.

FLUID THERAPY: Initial resuscitation should be done with normal saline or ringers lactate solution to support hemodynamic status in a hypotensive patient. A central line should be placed to guide fluid therapy by central venous pressure monitoring. Glucose should be maintained at normal level. Hypoglycemia should be avoided in infants and neonates. More commonly, due to stress of the head injury, serum glucose is high; glucose containing fluids should be avoided initially. Ringers lactate or half normal saline may be used. Hypertonic saline has been used by some centers in presence of hyponatremia due to cerebral salt wasting syndrome. Hypertonic saline used for reducing refractory ICP, has however not been shown to affect survival in clinical studies.^[28] If hyponatremia is due to SIADH (Syndrome of inappropriate anti-diuretic hormone secretion) fluid restriction may be required. Blood and plasma are used as guided by the hematocrit and coagulation profile.

MANNITOL: Mannitol can reduce ICP by two mechanisms. Reduction of viscosity which is transient, and dependent upon autoregulation being intact. More potent action, however is by its osmotic effect. Mannitol in dosages of 0.5 to 1 gm/kg may be used intravenously at 6 hourly interval with monitoring of serum osmolality (To be kept under 320). Mannitol should not be used if serum osmolality is >330, patient is hypotensive, patients with renal failure. Rapid pushes of mannitol can transiently increase ICP by causing transient systemic hypertension, therefore should be avoided. Mannitol has a theoretical risk of enlarging a hematoma by rapid shrinkage of brain and tearing of bridging veins. Therefore a CT may be necessary to rule out a hematoma before mannitol therapy is initiated.

STEROIDS: Always controversial, but there is no clinical evidence to use in cerebral oedema due to head injury. Steroid use should be reserved for patients with brain tumours.

NUTRITION: Early institution of enteral feeds is recommended if there is no associated intraabdominal injury to major organs such as liver, spleen, Rehabilitation Tracheostomy may be indicated in patients in prolonged comatose state who cannot protect their airway or require long term ventilation. Once patient is recovered from acute injury in the PICU, Early physical therapy for prevention of deep vein thrombosis and prevention of contractures may be necessary. Arm, leg hand and feet splints should be used as indicated. Evidence based international guidelines.^[29]

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COMPLICATION AND SQUEALAE:

EARLY: Transient cortical blindness, Seizures, Cranial Nerve palsy, Diabetes insipidus Cortical venous occlusion, Hemiparesis.

LATE: epilepsy, Post traumatic aneurysm, Meningitis, Hydrocephalus Memory loss Disability Muscle contractures.

Outcome after pediatric head injury^[7] I] Child Outcome Score Excellent recovery. II] Mod but non-disabling deficit, III]. Either a secure motor or cognitive deficit, IV]. Vegetative V]. Death. I and II - Good outcome III to V - Poor outcome.

PAEDIATRIC HEAD INJURY MANAGEMENT GUIDELINES:

MINOR HEAD INJURY: Despite the extensive research in the management of head injuries in adults, the paediatric guidelines for minor head injury was not introduced until 1999, the same year Homer and colleagues stated that there were not enough scientific studies to produce evidence based recommendations for management of paediatric.^[30,31,32] The American Association of Pediatrics (AAP) was the first to publish recommendations for management of closed head injury in children.^[33,34] The defined a group of children older than 2 Years with normal mental status at first examination, no focal neurological deficits or physical evidence of skull fracture. LOC was set to less than 1 minute. Due to an estimated low risk of developing an intracranial injury and an even smaller risk of a neurosurgical relevant injury in this patient group, observation for at least 24 hours was mainly warranted. In Case of LOC <1 min the option of additional CT scanning was to be considered.^[35] For children <2 years CT was to be promptly considered for patients at an intermediate risk-level (LOC < 1 min, extensive vomiting, history of lethargy, non-acute skull fracture, or parental concern) or otherwise admitted for observation and CT after 4-8 hours if symptoms had not resolved.^[34]

In the UK, modified NICE guidelines had been applied to children due to lack of other paediatric guidelines. In 2006, the "children's head injury algorithm for the prediction of important clinical events" (CHALICE) was recommended instead, however with certain caution since the decision rule had not been validated. This decision rule by Dunning and colleagues is a large multi Centre study including more than 2000 children younger than 16 years old, that lists 14 criteria, among others, vomiting 3 times, LOC > 5 minutes, GCS < 14 or fall from > 3 metres, as separate risk factors for intracranial pathology after head trauma. A CT was required if any of the 14 criteria were present. The sensitivity for a clinically important head injury was 94-99% and the specificity about 87%. The negative predictive value for clinically significant intracranial pathology was 99.9%. The CT ordering rate was estimated to 14%; an increase from the earlier management guidelines, but a decrease in the admission rate. There have also been reports that application of the decision rule could double the use of CT.^[35,36] In 2009, the North American PECARN group published a prediction rule for identifying children at very low risk of TBI for whom CT could routinely be obviated.^[37]

MODERATE HEAD INJURY: The definition of moderate head injury varies in literature with regards to the level of consciousness of GCS 13. In most adult recommendations, an initial GCS of 13 is considered to belong to mild head injuries. In the SNC recommendations, patients with GCS 9-13 are regarded as having a moderately increased risk of intracranial complications and are therefore recommended a routine CT scanning and hospital admission.^[38] This is more in line with other studies on paediatric trauma patients with mild alterations in consciousness (GCS 13-14) who have a

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relatively high incidence of intracranial injuries (19-27%) and for which routine CT is recommended. Deterioration can occur quickly with confusion, development of focal neurology or decrease in the level of consciousness. Moderately head injured patients are therefore recommended hospitalization and close observation. The majority of moderately head injured patients will recover with 24-36 hours without further treatment. However, according to earlier recommendations for paediatric head injuries, the effect of an epidural hematoma might be overtly expressed first after 48 hours (The phenomenon commonly known as "talk and die"), where as in adults it is expressed often within 24 hours.^[40]

SEVERE HEAD INJURY: Previous management strategies and decision making were mainly based on adult treatment recommendations or clinical experience.^[40] In 2003 the first evidence based pediatric guidelines for the management of severe TBI were published by Adelson et al.^[41] During the past decades, advances in the management of severely head injured children have been made, especially regarding treatment strategies within the intensive care setting to optimize outcome and outcome prediction.^[42,43] Severe head injuries are defined as GCS 8 or less (RLS 4-8). Immediate resuscitation, establishment of free airway, oxygenation and restoration of normal blood volume are crucial factors to assure adequate perfusion to the brain and hence, minimize secondary intracranial complications before referral to a trauma center.

All therapeutic strategies post trauma focus on the prevention or reduction of secondary brain injury by optimizing cerebral blood flow (CBF). Severely head injured children receive an urgent CT scan to identify those in need of immediate neurosurgical evacuation of an intracranial hematoma. There after the identified children are referred to a neurointensive or pediatric intensive care unit for optimal treatment. Cerebral perfusion pressure (CPP) is so far the best surrogate marker for estimating the CBF, measured by mean arterial pressure (MAP). The ideal level of CPP for children is not fully known, however there are two studies that show favorable outcomes associated with CPP between 40 and 65 mm of Hg and poor outcomes with CPP < 40 mm of Hg. The pediatric guidelines for severe head injury (2003) recommend a CPP > 40 mm of Hg and a slightly higher CPP with increasing age.^[44] There are two different schools for the treatment of intracranial hypertension: the CPP targeted therapy and the ICP targeted therapy (Lund Concept). The CPP targeted therapy aims lowering at lowering the ICP by adding vasopressors, to increase the CPP by increasing MAP, which only works as long as autoregulation is intact. The ICP targeted therapy uses aggressive treatment for keeping ICP to 20 mm of Hg or below by usage of systemic anti-hypertensive agents and maintenance of normovolemia, which has shown favorable outcomes.^[42]

The main counter argumentation for the Lund Concept is the potential risk of hypotension, which could consequently lead to secondary brain injury and worse outcome. For continuous ICP monitoring on sedated children, intraparenchymal ICP monitoring or external ventricular drains (EVDs) are possible options, which aid in estimating CPP and CBF. An EVD can sometimes be difficult to place correctly in case of diffuse cerebral edema and very small ventricles. However, an EVD has the advantage of the possibility of CSF drainage, which decreases the intracranial volume and possibly the ICP. Elevation of the head, intravenous analgesics and increase in sedatives are non-invasive methods of lowering the ICP. Rapid infusion of hypertonic saline, osmotic agents (e.g. mannitol) and hypocapnia (pCO₂ 3.5 - 4.0 kPa) are options when other measures have not affected the ICP, or when there are signs of cerebral herniation (Cushing Effect). Prophylactic use of osmotic

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agents or hyperventilation is not recommended because of the risk of a rebound effect, increased brain edema and excessive cerebral vasospasm. This could consequently lead to reduced cerebral blood flow and ischemia.^[45] The use of steroids has been controversial for a long time and does not have a place in current head injury management.^[46] Anti-convulsive therapy should be given only in presence of seizures.^[47] Second line treatment when intracranial pressure seems resistant to the above regime, is change of anesthesia to e.g. barbiturate coma and prolonged sedation. According to the pediatric guidelines from 2003 continuous infusion of propofol (prolonged for 24 hrs) is not recommended in the treatment of pediatric TBI, due to the risk of metabolic acidosis and death (the so called propofol infusion syndrome).^[48,49]

Decompressive craniectomy can be meaningful in such situations.^[50,51] The use of hypothermia in severe head injury has been one of the last possibilities in former guidelines,^[40] although there have not been any evidence of a favorable effect in children with TBI. In previous studies there have even been a tendency towards worse outcome in patients receiving hypothermia treatment after TBI,^[52,53,54] which has recently been verified in a large randomized multicenter study on adults.^[55] Hence, early hypothermia cannot be considered as a neuroprotective strategy in patients with severe traumatic brain injury. Sharma and Lahoti et al in a study of 791 patients reported that most mortality occurred in the 1-3 year age group.^[1] Bener et al. also reported the same result in his study.^[56] Mortality was higher in males. RTA was most common cause of death, followed by burn and fall from height.^[1] These results are similar to those of the studies done in developing countries,^[57,58] whereas studies from developed countries reveal RTA to be the most common cause of death followed by gun-shot injuries.

METHOD AND MATERIAL: This study comprised all children and adolescents 0–12 years old, consecutively recorded for head injury, at Hamidia hospital Bhopal, during June 2011 to May 2013.

- Practically all patients diagnosed with or suspected of moderate or severe brain injury were brought by ambulance, or self-vehicle. When the diagnosis and degree of severity was uncertain, CT scan done and inform to expert on telephone he come to see the patient.
- Classified patient according to Scandinavian Neurotrauma Committee (SNC) classification into minimal, mild, moderate and severe. Minimal head injury is defined as a patient with GCS 15 at admission and with no Loss of consciousness (LOC) & focal neurological deficits. Mild head injury is defined as initial GCS of 14 to 15, brief LOC (< 5 min) and no focal neurological deficits. The definition of moderate head injury defines patients with initial GCS of 9-13 and/or focal neurological deficits or LOC > 5 min after head trauma, while severe head injury includes all patients with a GCS score of 8 or below.
- Filling of proforma after stabilization of patient.

Feature	Responses	Score
Eye opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Verbal response	<ul style="list-style-type: none"> • Alert, Babbles, coos, words or sentences normal for age 	5
	<ul style="list-style-type: none"> • Less than usual ability, irritable cry. 	4

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	<ul style="list-style-type: none"> • Cries to pain. • Moans to pain. • None 	3 2 1
Motor response	<ul style="list-style-type: none"> • Normal –Spontaneous. • Withdraws to touch. • Withdraws from nail bed pain. • Flexion to supraorbital pain. • Extension to supraorbital pain. • None. 	6 5 4 3 2 1
Total coma score		3/15-15/15
GLASGOW COMA SCORE FOR CHILDREN		

DISCUSSION & RESULTS: The present study "Clinicopathological study of Paediatric Head Injury patients" conducted on 165 cases admitted from June 2011 to May 2013 in Department Of Paediatric Surgery, Gandhi Medical College, Bhopal. We included all head injury patients of 0-12yrs age. However, maximum number of patients were found to be in 6-9yrs age group. The Male to Female ratio was found to be 1.89:1 i.e. Males were having higher incidence of head injury. Most common mode of head injury was "Fall from height" 2nd and 3rd being RTA and Fall of Heavy object respectively. Rare modes were Sports injury and Assault. In our study, most of the patients at the time of presentation were conscious but had H/O Unconsciousness or Vomiting at the scene of trauma.

We had divided the patients on the basis of- Glasgow Coma Score in three groups Mild, Moderate and Severe. On the basis of type of injury in Open & Close type. Associated injuries included Limb, Chest and Spine injuries, and abdominal injuries. We measured outcome on "Glasgow Outcome Score"

In our present Study 46.66% patients of 6-12 years of age & is the most common age group of paediatric head injury. According to Lahoti et al (2010) most common age group of paediatric head injury is 6-12 yrs group which is 52.33% of total patients. In our study majority of patients were male; which was found to be 65.45% while females were 34.55%. In our study fall from height found in 50.90% patients, RTA in 32.72% of Cases. According to Lahoti et al 2010 fall from height was found in 39.44% patients and RTA in 27.81% patients.

In our study fall from height found to be most common mode in all age group but in 6-12 yrs age RTA was found to be most common mode. Lahoti et al 2010 also found this. In our study Scalp Injury with Fracture was found to be most common type of Head Injury other Studies also support this.

In our study orthopaedic injuries are associated with 28.48% of patients. In our study most mortality occur in 1-3 years and 9-12 years age group which was found to be 37.5% in both group.

Lahoti et al (2010) - 39.21% Deaths occur in 1-3 yrs age group. In the present study, RTA was most common mode of injury in mortality patient which was found to be 75%. This is also supported by other studies in different countries.

In our study 75% of cases mortality occurred in Close Wound patients because early gain of attention occurs in Open Wound patients. Other studies also support this.

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In our study most common Pathology for Death is Diffuse Axonal Injury which was found to be 50% of died patients. Other Studies also found this.

CONCLUSIONS: 6-9 years found to be most common age:

- Males are more affected than females.
- Fall from height followed by RTA was found to be most common mode of Injury.
- Close injuries are more common than open injuries.
- Orthopaedics injuries are most common associated injuries.
- Chest complications were the most common and increased the hospital stay.

OBSERVATIONS:

TYPE OF HEAD INJURY:

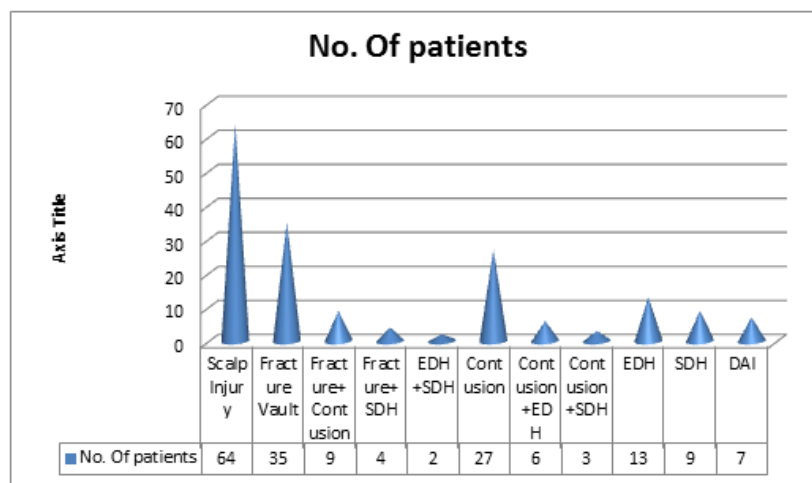


Table 1

- Table I Suggest Scalp Injury and Fracture are Most Common

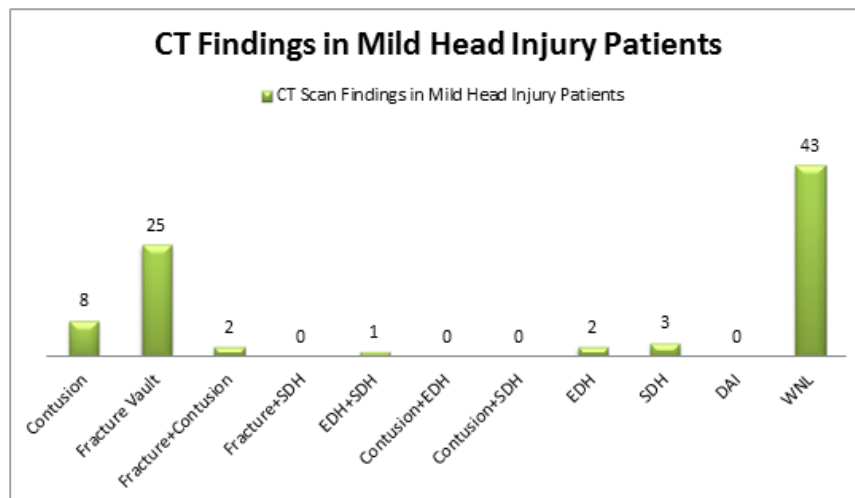


FIGURE II: CT Findings

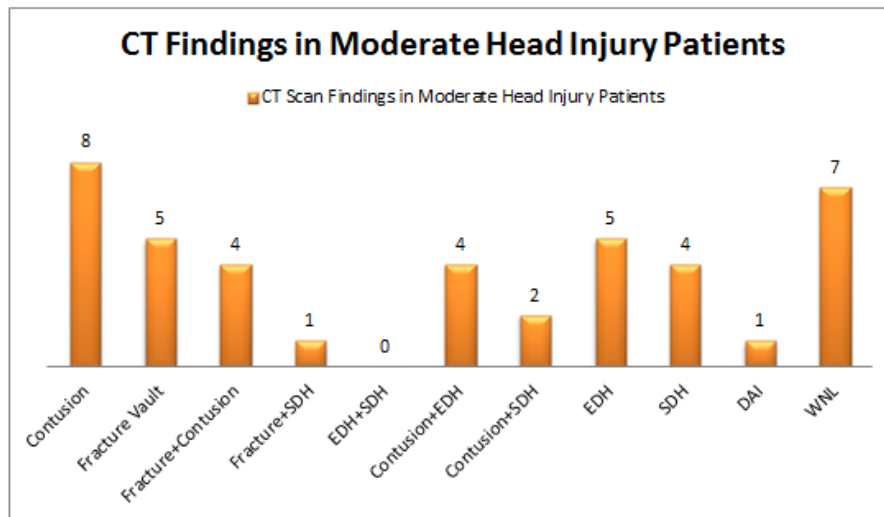


FIGURE III

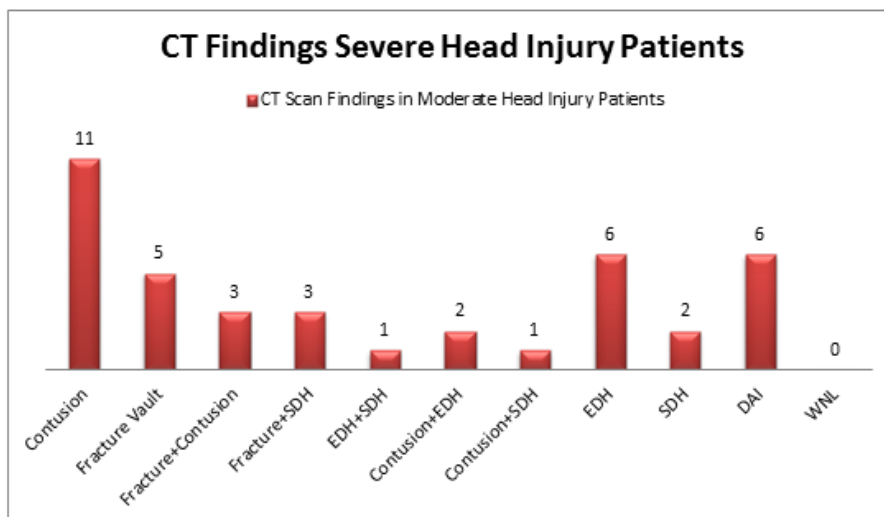


FIGURE IV

Complications	Mild	Moderate	Severe
Chest Complication	10	14	18
Electrolyte imbalance	5	4	6
UTI	0	5	10
Bed sore	0	3	8
Wound complications	5	2	4
None	64	22	12

TABLE V

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