

**MODE, PRESENTATION,  
CT FINDINGS AND OUTCOME  
OF PEDIATRIC HEAD INJURY**

**M. Sharma  
A.K. Sharma**

**ABSTRACT**

*Twenty five to thirty per cent of all patients admitted with head injuries to any large hospital are children. Head injuries in children differ in several ways from those seen in the adult population. A standard management protocol is used in a service hospital and CT and neurosurgical consultation/transfer is based on specified criteria. The records of 312 patients admitted with head injury to Command Hospital (WC), Chandimandir were retrospectively analyzed to compare the mode of injury, initial presentation, CT findings and the outcome of management between those above and those below the age of 10 years.*

*Eighty seven (27.8%) of the 312 patients were children and 71% of them had sustained the injury due to fall from a height. One third of children were brought with vomiting and drowsiness and 9.2% with seizures, but lateralizing signs were found in only 5.7%. Nineteen children underwent a CT of the head and of these 74% had only cerebral edema. Only 4 of the 87 children were transferred to a neurosurgical unit. The mortality rate was lower in children (5.7%) compared to the older age group (11.6%) but the difference was not statistically significant. However, a significantly higher number (80.5%) of children recovered without any residual deficit. Majority of our children can be safely managed in a general sur-*

*Craneo-cerebral injuries constitute more than 50% of all trauma admissions and are also the commonest cause of mortality following trauma(1). Twenty five to thirty per cent of all patients admitted with head injuries to any large hospital are children(2). It has been well established that these injuries in children differ in several important ways from those seen in the adult population(3-5).*

**Patients and Methods**

The case records of 312 patients admitted to Command Hospital (WC), Chandimandir with head injury between July 1990 and June 1993 were retrospectively analyzed to compare the mode of injury, initial presentation, computed tomography (CT) findings and the outcome of management between those above and those below the age of 10 years, using the Chi-square test to establish their relative frequencies and significance. Children with birth trauma, and trivial scalp wounds were excluded from this report.

---

*gical unit provided a multidisciplinary approach is used and facilities of CT are readily available, since very few require active neurosurgical intervention.*

**Key words:** *Pediatric head injury, Trauma.*

---

*From the Departments of Pediatrics and Surgery, Command Hospital (Western Command), Chandimandir 134 107.*

*Reprint requests: Major (Mrs) Mukti Sharma, 73, Sector 5, Chandigarh 160018.*

*Received for publication: March 30, 1994;  
Accepted: April 15, 1994*

**Management Protocol**

All patients presenting to the Casualty Department of Command Hospital (WC), Chandimandir, with history of trauma to the head followed by loss of consciousness, bleeding from the ear, nose or mouth, vomiting and those with skull fracture, were admitted to the hospital. They were examined by a general surgeon, and a pediatrician and anesthesiologist were co-opted from an early stage in the management if the patient was below the age of 10 years or required ventilatory support. The baseline vital parameters, the neurological and pupillary signs and the state of sensorium based on the Glasgow coma scale (GCS)(6) (modified in children as shown in *Table 1*) were recorded. The GCS was reassessed after 6 hours of resuscitation and the patients divided into three groups after Miller(7). Those with GCS of less than 8 with no eye opening even to painful stimuli were classified as severe, those with a score of 9 to 12 or those who scored 7 or 8 but opened their eyes to pain were considered moderate and those who scored 13 to 15 called minor.

If the patient was conscious he was only observed for 48 hours for any symptoms of headache, vomiting, disorientation, drowsiness, bradycardia, hypertension, state of the pupils and any change in the neuro-

logical status. Prophylactic antibiotics were given only if he had a scalp wound or bleeding from nose, ear or mouth, or any other injury.

Patients who were unconscious, confused, irritable, resented examination or had seizures; or developed any of the above mentioned signs during the period of observation were started on dehydration therapy with intravenous frusemide (1-2 mg/kg/day in two divided doses). Mannitol infusion (0.25-1.0 g/kg) was used only if the patient's sensorium was rapidly deteriorating, to gain time for a CT scan or exploratory burr hole, or if the patient did not respond adequately to frusemide alone, provided CT had excluded any intracranial hematoma.

Seizures were controlled with slow intravenous phenytoin sodium (5-10 mg/kg) preferably with continuous electrocardiographic monitoring. It was also used prophylactically in patients who were extremely irritable and whose CT showed extensive cerebral contusion. In children, and if given prophylactically in adults, it was continued for a period of 8 weeks, otherwise for at least 2 years. Phenobarbitone and diazepam were avoided before a CT as the hypnosis caused by them could confuse the assessment of the sensorium.

Patients in whom a clear airway could not be maintained because of loss of laryngeal reflexes or associated faciomaxillary injury or those who had spontaneous hyperventilation, respiratory arrhythmia or ventilatory insufficiency (assessed by pulse oximetry), were intubated and ventilated with the Kimura or the East Radcliff ventilator. Elective hyperventilation was used as a therapeutic measure in patients with cerebral edema not responding to mannitol.

**TABLE 1**—*Glasgow Coma Scale. Modifications in the Responses in Children Under 5 Years.*

Age group	Best motor response	Best verbal response
<6 mo	Flexion	Smiles and cries
6-12 mo	Localization	Smiles and cries
1- 2 yrs	Localization	Sounds and words
2- 5 yrs	Obeys commands	Words and phrases

The criteria used to ask for a CT scan and a neurosurgical consultation/transfer are enumerated in *Tables II & III*, respectively.

Those patients who did not require neurosurgical intervention were continued to be managed with intravenous fluids or nasogastric tube medication and feeding to maintain their fluid and electrolyte balance; and care of the airway, bladder and bowel.

All patients with residual deficit or on anticonvulsants were followed up for a mean duration of 17 months after discharge.

**TABLE II—Indications for CT Scan in Cranio-cerebral Injuries**

---

GCS <8 with no eye opening to pain 6 hours after resuscitation
Deteriorating sensorium
Focal pupil/limb signs
Coma with unreliable history
Unresponsive to verbal commands for >24 hours
Seizures
Hyperpyrexia and neck rigidity

---

**TABLE III—Criteria for Transfer to Neuro-surgical Centre**

---

Compound depressed skull fracture
Penetrating skull injury
CT diagnosis of compressing intra-cranial hematoma*
CSF leak for > 10 days
Large aerocele

---

\*Giving rise to a mid-line shift of >5 mm.

## Results

Eighty seven (27.8%) of the 312 patients under study were children below the age of 10 years. Their mode of injury and clinical presentation is compared with that of the older age group in *Tables IV & V*, respectively. Seventy one per cent of the children had sustained the injury due to a fall from a height, and 27.6% were traffic accident victims, while on the other hand traffic accident was the commonest mode of injury in the older age group (69.8%).

Although the commonest presentation is a history of transient loss of consciousness, a significantly higher number ( $p<0.01$ ) of children (33.3%) were brought with, or developed vomiting and drowsiness during the period of observation. Children also had a significantly higher incidence ( $p<0.01$ ) of seizures (9.2%); however, laterdizing signs were found in only 5.7% ( $p<0.01$ ) and the incidence of severe head injuries was significantly lower ( $p<0.05$ ).

Of the 87 children, 19 underwent a CT of the head and of these 14 (16.1% of 87) had only cerebral edema. This was significantly higher ( $p<0.001$ ) than the incidence in 10 (4.4%) of the older age group, since most of them were associated with either cerebral contusion (23.1%) or intracranial hematoma (15.6%) or both. On the other hand, only 5 (5.7%) children had evidence of cerebral contusion and 3 (3.4%) had subdural hematoma. The incidence of intracerebral hematoma on the other hand was significantly lower in children ( $p<0.01$ ); and extradural hematoma was not found in any of the children who were scanned as compared to the 2.2% incidence found in the older age group (*Table VI*). There was a lower incidence of skull

**TABLE IV**—Comparison of Mode of Injury Below and Above 10 Years of Age

Mode of injury	< 10 years		> 10 years		Total	p value
	n	(%)	n	(%)		
Fall from height	62	(71.3)	15	(6.7)	77	<0.001
Traffic accident	24	(27.6)	157	(69.8)	181	<0.001
Assault	1	(1.1)	49	(21.8)	50	<0.001
Others	-		4	(1.8)	4	
Total	87		225		312	

**TABLE V**—Comparison of Clinical Presentation

Presentation	< 10 years (n=87)		> 10 years (n=25)		Total	p value
	n	(%)	n	(%)		
Transient loss of consciousness	36	(41.4)	79	(35.1)	115	>0.05
Headache/vomiting drowsiness GCS > 12	29	(33.3)	42	(18.7)	71	<0.01
Coma GCS 9-12	17	(19.5)	51	(22.7)	68	>0.05
GCS < 8	9	(10.3)	49	(21.8)	58	<0.05
Seizures	8	(9.2)	5	(2.2)	13	<0.01
CSF leak	3	(3.4)	11	(4.9)	11	>0.05
Depressed fracture	2	(2.3)	9	(4.0)	11	>0.05
Laterlizing signs	5	(5.7)	47	(20.9)	52	<0.01

fracture in the CT of children but the frequency of depressed skull fractures was not statistically different in the two groups.

Only 4 (4.6%) of the 87 children were transferred to a neurosurgical centre (NSC). The mortality rate in children (5.7%) was lower than in the older age group (11.6%) but the difference was not statistically significant. However, a significantly higher number ( $p<0.01$ ) of children (80.5%) recovered without any residual deficit and post concussional syndrome was seen in only 9.2% (Table VII).

## Discussion

Accidental blunt head injuries in children are common but except for those caused by falls from extreme heights or due to motor vehicle accidents, they are almost always benign in their clinical consequences. Inflicted injury accounts for nearly 25% of children admitted for head injuries in the developed countries(5). These injuries are more severe, which perhaps explains the high incidence of skull fractures and intracranial hematoma reported in their literature(8,9). In our socio-economic

**TABLE VI—Comparison of CT Findings Above and Below Age of 10**

CT findings	< 10 years		> 10 years		Total	p value
	n	(%)	n	(%)		
Only cerebral edema	14	(16.1)	10	(4.4)	24	<0.001
Hemorrhagic contusion	5	(5.7)	52	(23.1)	57	<0.001
Intracranial hematoma	3	(3.4)	35	(15.6)	38	<0.01
Extradural	-		5	(2.2)	5	>0.05
Subdural	3	(3.4)	30	(13.3)	33	<0.05
Skull fracture	7	(8.0)	33	(14.7)	40	>0.05
Number of scans	19/87	(21.8)	64/225	(28.4)	83/312	

**TABLE VII—Comparison of Outcome Above and Below Age of 10**

Outcome	< 10 years (n=87)		> 10 years (n=225)		Total	p value
	n	(%)	n	(%)		
No residual deficit	70	(80.5)	138	(61.3)	208	<0.01
Post concussional syndrome	8	(9.2)	58	(25.8)	66	<0.01
Hyperactivity	2	(2.3)	6	(2.7)	8	>0.05
Dysphasia	1	(1.1)	15	(6.7)	16	<0.05
Motor paresis	1	(1.1)	13	(5.8)	14	>0.05
Transfer to NSC*	4	(4.6)	21	(9.3)	25	>0.05
Died	5	(5.7)	26	(11.6)	31	>0.05

\* NSC stands for neurosurgical centre.

setup, a "battered child" is still infrequently seen and the commonest cause of head injury is due to a fall from a window, balcony or the unguarded roof of a house(2), or due to a tumble down the stairway(10).

The skull of a child is better able to absorb the energy of physical impact due to its elasticity(2); thus cerebral contusions and lacerations are less frequent in chil-

dren than in adults, which explains the lower incidence of lateralizing signs seen in them. The classical temporal lobe contusion due to a contre-coup injury is reported in only 6.5% of children as compared to the 41.5% incidence in the older age group(11). Because of the close adherence of the dura to the inner table of the skull in children, extradural hematoma is also infrequent and was found in only 1% of 4465

children admitted for head injury in the Hospital for Sick Children, Toronto(12).

The most significant observation in children is a high incidence of cerebral edema which at times is acute and fulminant. The large number of children who present with or develop drowsiness and vomiting soon after injury and respond promptly to diuretics, confirms cerebral edema to be the cause of their symptoms. This feature has become more noticeable ever since facilities for CT have become easily accessible(2).

Cerebral edema develops as a result of the failure of the auto-regulatory mechanism of cerebral blood flow, hypoxemia and hypercapnea. The reduction of the systemic arterial blood pressure in turn further reduces the cerebral perfusion pressure and increases the cerebral edema. Hypotension leading to shock is invariably secondary to systemic injury, except in children under 2 years(13,14). Shock is an uncommon feature of head injury in adults but is a significant problem in children, as they are intolerant to blood loss(15), and this may be one of the "reasons for the higher incidence of cerebral edema in them.

The immaturity of the auto-regulatory mechanism of cerebral blood flow in children is perhaps the cause of the higher incidence of this clinico-pathological entity wherein fulminant brain edema develops in a child with head injury in the absence of any significant intracranial hemorrhage or brain contusion, leading to rapid deterioration of sensorium, seizures and decerebrate rigidity. A child recovering satisfactorily may suddenly deteriorate without any obvious reason or on withdrawal of diuretics. The CT almost invariably reveals only brain edema which if treated energetically

with diuretics and elective hyperventilation can arrest the rapid downhill course(2).

There is a significantly higher incidence of early post traumatic seizures in children associated with deterioration of sensorium(16). This has been attributed to the "immaturity" of the brain at this age, whereas the "plasticity" of the young brain is said to minimize the morbidity and long term sequelae of head injury in children(2), who have a much lower incidence of post traumatic epilepsy and post concussional syndrome than in adults(17). Both these features can be better explained if they are correlated with the higher incidence of cerebral edema, which when rapidly increasing can give rise to seizures without any lateralizing signs and if treated quickly is reversible and does not have any sequelae.

The principles of resuscitation are the same for adults and children. However, children differ in the normal values of their vital parameters and their tolerance to blood loss. Their smaller size imposes differences in equipment size, drug doses, fluid and electrolyte requirements, and the difficulty of carrying out procedures(15). A multidisciplinary approach by co-opting a pediatrician and an anesthesiologist at an early stage in the management of these children can prevent secondary brain damage due to hypoxemia, hypotension, cerebral edema and seizures(18). Very few children have intracerebral hematoma and majority of depressed fractures in children do not require elevation except for cosmetic reasons(2).

There has been a 46% reduction in the number of head injury admissions to neurosurgical centres in UK after guidelines for admission and referral were implemented(12). However, the criteria for

transfer of patients with head injury to neurosurgical centres in developed countries<sup>^</sup>) may not be relevant in our set up as yet, especially in children. Majority of our children can be safely managed in a general surgical unit provided a multidisciplinary approach is used and facilities of CT are readily available. Since very few require active neurosurgical intervention, this approach reduces the load on the already overtaxed, few neurosurgical facilities available to us.

#### REFERENCES

1. Mahboob S, Muralidhar B, Kashikar D, Krishna Rao CV. Head injury and general surgeon. *Indian J Surg* 1992, 54: 415-418.
2. Tandon PN. Head injury in infancy and childhood. *Indian Pediatr* 1985, 22: 255-258.
3. Brooks M, MacMillan R, Cully S, *et al.* Head injuries in accidents and emergency departments: How different are children from adults? *J Epidemiol Community Health* 1990, 44:147-151. \*
4. Luerssen TG, Klauber MR, Marshal LF. Outcome of injury related to patient's age: A longitudinal prospective study of adult and pediatric head injury. *Neurosurgery* 1988, 68: 409-416.
5. Duhaime AC, Alario AJ, Lewander MD, *et al.* Head injury in very young children: Mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 1992, 90: 179-185.
6. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. *Lancet* 1974,11: 81-84.
7. Miller JD. Minor, moderate and severe head injury. *Neurosurg Rev* 1986, 9: 135-139.
8. Krause JF, Fife D, Cox P, Ramstein K, Conroy C. Incidence, severity and external causes of pediatric brain injury. *Am J Dis Child* 1986,140: 687-693.
9. Chan K, Yue CP, Kirpal SM. The risk of intracranial complications in pediatric head injury: results of multivariate analysis. *Child Nerv Syst* 1990, 6: 27-29.
10. Joffe M, Ludwig S. Stairway injuries in children. *Pediatrics* 1988, 82: 457-461.
11. Tandon PN, Prakash B, Banerjee AK. Temporal lobe lesions in head injury. *Acta Neurochirurgica* 1978, 41: 205-221.
12. Hendrick EB, Nash DC, Hudson AR. Head injuries in children. A statistical survey of 4465 consecutive cases at the Hospital for Sick Children. Toronto, Canada *Clin Neuro Surg* 1965,11: 46-65.
13. Illingworth G, Jennet B. The shocked head injury. *Lancet* 1965, 2: 511-514.
14. Newfield P, Pitts LH, Kaktis JV. The influence of shock on the mortality after head trauma. *Crit CareMed* 1980,8: 254-260.
15. Gentleman D, Dearden M, Midgley S, Maclean D. Guidelines for resuscitation and transfer of patients with serious head injury. *Br Med J* 1993,307: 547-552.
16. Mahapatra AK, Tandon PN, Bhatia R, Banerji AK. Head injured patients who talked and died. *Indian J Surg* 1993, 55: 361-366.
17. Tandon PN. Post-traumatic epilepsy. *In: Proceedings on the National Seminar on Epilepsy.* Eds Tandon PN, Mani KS, Walker AE. 1975.
18. Gentleman D. Preventing secondary brain damage after head injury: A multidisciplinary challenge. *Injury* 1990, 21: 305-308.
19. Miller JD, Jones PA, Dearden NM, Tocher JL. Progress in the management of head injury. *Br J Surg* 1992, 79: 60-64.