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Acute Post Traumatic 'Brain Oedema' in Children

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Abstract

Post-traumatic brain oedema has been considered to be one of the commonest and more important sequelae of head injury. To study its incidence, presentation and outcome a series of 252 children under the age of 15 years with head injury, admitted over a period of 2 years were analysed. Among them were 61 children who were diagnosed to have brain oedema as the dominant pathology based on CT scan appearance, operative findings or necropsy findings.

Contrary to the existing belief that children withstand severe head injuries better than adults, in children with brain oedema, the mortality was higher, half of the overall deaths (39 out of 252) were among these children. While most of the fatal cases with brain oedema were unconscious on admission they included 10 children who either talked (4 patients) or cried (6 patients) before lapsing into coma. Whether this is a true parenchymal oedema or a hyperaemic brain swelling is still controversial.

Key words -
Head injury,
Children,
Brain oedema,
CT,
Talked and died

Head injury in infants and children constitutes a major public health problem. Nearly five million children sustain head injury each year in United States [1]. Approximately 200,000 of these children are hospitalised, nearly 4,000 die [2], [3], [4]. Between 2 and 5 per cent remain severely handicapped for the rest of their lives [4]. An epidemiological study in South Delhi showed that of the head injured patients who needed hospitalization 40.8 per cent were below 20 years of age [5].

In this unit where a large number of head injured children are treated, the clinicians have been disturbed by the rapid deterioration and sudden death in some children with head injury without any surgically significant haematoma/contusion visible in the CT scan. The possibility of brain oedema being responsible for the clinical state of these patients was considered. Therefore, a study of analysing retrospectively the cases of head injury in children diagnosed to have 'brain

oedema' as a dominant lesion was undertaken.

Material and Methods

This is a retrospective analysis of a series of 252 consecutive cases of head injury in children under the age of 15 years, admitted to the Head Injury Unit of the Neurosciences Centre of All India Institute of Medical Sciences, from January 1980 through December 1981. The study includes all those children who required hospitalisation for longer than 24 hours, as well as those who died within 24 hours. Cases of minor concussion discharged fit within 24 hours of injury have been excluded. Besides routine clinical assessment all cases who failed to show expected continuous improvement or having shown improvement had deteriorated were submitted to investigations to rule out an intracranial mass lesion. A total number of 137 cases (54%) were investigated further. CT scan was done in 123 cases and carotid angiogram in 14 cases. For the purpose of this study cases diagnosed to have brain oedema as a dominant or significant lesion have been analysed.

"Brain oedema" was interpreted on CT scan in 51 children, when there was either diffuse lucency with or without collapsed ventricles, and/or compression of basal cisterns. Cases associated with sizeable intra and extra-cerebral haemotoma/contusions were excluded (Table I). Both carotid angiography and CT scan were done in 3 cases. Whereas the angiograms were reported as essentially normal, all three cases had CT scan evidence of 'Brain oedema'. In 10 cases in whom no CT was done the diagnosis was made at operation (6 cases) or medicolegal autopsy (4 cases) (Table I). In one of these later 4 cases there was associated laceration of tip of left frontal lobe associated with diffuse Brain oedema.

Table I - Brain Oedema : Diagnosis Criteria

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In another 5 cases who were diagnosed to have only 'Brain oedema' on the basis of the CT scan, medicolegal autopsy revealed additional lesions in two of them. In one case there was an extradural haematoma 3 cm × 3 cm × 1 cm and in another there was bilateral superficial parietal contusions. In the remaining three, there was brain swelling without any contusion or haematoma. The CT picture in both these cases showed bilateral diffuse lucency with obliteration of basal cistern and compressed ventricles. Of the 6 cases who were diagnosed to have brain oedema at operation in one there was a ½ cm thick extradural haematoma and in another there was a thin layer of subdural haematoma. In rest of the 4 cases there was only brain swelling.

Thus in a total of 61 children (25% of head injury in children) 'brain oedema' was diagnosed as the main lesion. Children with post traumatic 'brain oedema' were managed by routine intravenous mannitol 1 gm/kg of body weight 4 hours in divided doses and prophylatic anticonvulsants, phenobarbitone 3-5 mg/kg body weight. Patients were kept on intravenous fluids. Steroids, high dose barbiturate, ICP monitoring and hypothermia were not employed Patients were not managed in the intensive care unit routinely. Five of these children were put on controlled ventilation because of hyperventilation and no elective controlled ventilation was employed in patients with normal respiration.

Results and Discussion

A great stride has been made in the diagnostic assessment of head injured patients with the advent of computerized tomography scanning. It is possible to diagnose and treat promptly intracerebral and extracerebral haematoma with better results. It is also possible to distinguish between parenchymal lesions of increased and decreased radio-density. To extrapolate from this and distinguish between intracerebral haematoma/contusion, oedema/ischemia/haemodynamic swelling, however remains controversial.

During the period of study the total number of head injuries in all age groups admitted to the centre was 931 and children thus accounted for 27 per cent of the head injury population. The type of lesions seen in these children is listed in Table II.

Table II - Head injury in Children

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The age and sex distribution of the head injury children is given in Table 3. There was no statistical difference between the age and sex distribution of the patients in the oedema group as compared to others.

Table III - Age and Sex Distribution

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There was no difference in the mode of injury between the two groups. Fall was the commonest mode of head injury in child-ten both with oedema (67%) and the others (64%) (Table IV). However, in an unselected series of head injury in all age groups, the commenest mode of injury was Road Traffic Accident (RTA).

Table IV

Table IV

There was CT evidence of 'brain oedema' in 51 cases. CT was done within 48 hours of in 40 cases and after 48 hours in 11 cases (Table V). Brain oedema was diagnosed on the basis of the following findings in the CT scan:

Table V - CT Evidence of Oedema

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Diffuse lecency;
(2) Ventricular compression;
(3) Obliteration of basal cisterns.

The earliest evidence of brain oedema in CT was seen at 2 hours from the time of injury. Identical findings were observed in a patient whose CT scan was done 10 days after injury. No serial CT scan was done because of unmanageable load on the scanner for more acute problems.

The various CT findings in the 51 cases who were diagnosed to have brain oedema is shown in Table IV. Diffuse lucency alone without any evidence of space occupation in the form of ventricular compression or compressed cisterns was seen in 14 cases (Group 1). In spite of any evidence of brain

swelling, 3 out of these 14 children died (21.4%). In 36 cases there was evidence of space occupation in the form of compressed ventricles and/or compressed or obliterated cisterns. Of these, 8 died (22.2%). These 36 cases were subdivided into two groups, those with basal cisterns compressed but not obliterated (Group 2-24 cases) and those with obliteration df perimesencephalic cisterns in their CT scan (Group 3 -12 cases). There was a highly significant difference (Fischer's exact test applied $p < 0.001$) in the mortality of group-2 and survived and 8 out of 12 cases of the second group died (66.6%). It was interesting to note that only 1 patient had compressed ventricles without associated diffuse lucency in the CT scan.

Table VI - CT : Mortality Correlation

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In the 61 children with brain oedema the best coma scale [6] within 6 hours of admission was 8 or more in 26 cases, of these 6 died (mortality 23.0%). Coma scale was less than 8 in 30 cases, of whom 14 died (mortality 46.6%). All the 9 children with coma scale of 3 to 4 died. Sixteen cases (26%) had convulsions in the acute stage (Table VII). The mortality in ' Brain oedema' children with convulsion was higher (43.7%) than those without it (28.8%).

Table VII - Clinical Parameter - Outcome

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The four grave clinical signs were dilated fixed pupils, bilateral decerebration, hyperventilation and hyperpyrexia (Table VIII). The results of caloric and oculocephalic response were not available in all cases and hence cannot be commented upon. All the patients with bilateral dilated and fixed pupils died and only one patient each with bilateral decerebration and hyperventilation survived.

Table VIII - Clinical Parameter - Outcome

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Out of the total 252 children with head injury admitted, 39 children died (15.4%). Half the deaths (20/39) were in the 'brain oedema' group (Table IX). The mortality of the 'brain oedema' group was 33 per cent whereas in the other 191 head injuries it was 10 per cent. The 20 cases of 'brain oedema' who died were analysed further (Table X). Four of these had talked and six had cried sometimes during their clinical course following injury. The most disturbing element was that 3 out of the 4 cases who talked and died had come to us conscious (with a coma scale of 14 or 15).

Table IX - Mortality : Comparative Analysis

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Table X - Expired Cases

Table X - Expired Cases

Case illustration (talked and died):

M. A., 7-year old male child fell from 10 feet height. He was admitted to peripheral hospital. On regaining consciousness 1½ hours later, he was found to have right hemiparesis for which he was

referred to us. On admission to our unit, 14 hours after injury, his coma scale was 15. X-ray skull showed a linear fracture in the left temporoparietal region. A CT scan showed bilateral diffuse lucency, compressed ventricles and obliteration of perimesencephalic cisterns and sylvian fissures. Child was kept under observation and given intravenous mannitol and prophylactic phenobarb. He had a generalised seizure at 22 hours after injury. Postictally he remained in coma and started decerebrating. Sudden respiratory arrest developed at 24 hours. He was put on respirator but showed no improvement and died on the 3rd day.

The injury respiratory arrest interval among the fatal cases had two peaks:

Within 24 hours of injury - 11 cases

On 3rd day - 5 cases

Only 2 cases had arrest after 3rd day (Table X).

It is highly probable that the cause of death in the group of patients whose CT appearance showed bilateral diffuse lucency with compressed ventricles and obliterated cisterns (Group 3, Table VI) was massive raised pressure. The temporal course also suggests that massive oedema had caused the enormous pressure as all of them had deteriorated by 3rd day. This is also supported by Clasen's [7] (1980) observation, on experimental and autopsy studies, that oedema leading to raised pressure reaches its height by 3rd day and after that day there starts resolution of oedema [7]. It is possible that this group of patients may be helped by ICU care with continuous ICP monitoring and maximal therapy directed at decreasing brain oedema and ICP namely elective controlled hyperventilation, Osmotic therapy, and barbiturates for at least 3 days and may be decompressive surgery. However, no definite proof is available that these cases could be helped by surgical decompression [8], [9].

The patients in our series who had only bilateral diffuse lucency in their CT scans without a mass effect (Group 1, Table IV) had a mortality of 21.4%. What are these lesions? It is possible that this lesion represented mild oedema which had not caused any significant mass effect at the time of scan. Oedema could have progressed with time, resulting in massive ICP and thus could explain the 3 deaths in this group. As we don't have the serial CT scans of these patients, a definite opinion cannot be given on this aspect.

- ***Bilateral diffuse lucency in plain CT scan***
- ***Compressed ventricles in plain CT scan***
- ***Compressed basal cisterns in plain CT scan***

The lucent areas in CT which we have interpreted as brain oedema was similarly interpreted by New [10] (1974), Marindo de Villasante and Taveras [11] (1976), Ambrose [12] (1976), and Penn [13] (1980). The most common CT appearance of head injured children in our series without an intracranial haematoma was diffuse lucency associated with increased brain bulk manifested as compressed ventricles and compressed cisterns. However, Langfitt and his School (1965, 1982), [9], [14] Zimmerman et al (1978) [15] and Bruce et al (1981) [16] argue that the most common cause of raised ICP, brain swelling and neurologic deterioration is vascular engorgement or haemodynamic swelling and not brain oedema as strictly defined and the CT appearance was that of compressed ventricles and obliterated cisterns without white matter lucency. Moreover, the Philadelphia group felt that lucency in the CT scan representing oedema appeared after 48 hours of injury when there was resolution of haemodynamic swelling [17]. This was not the case in our material as 40 out of the 50 cases with diffuse lucency had their CT scan within 48 hours of injury. Penn [13] (1980) arguing in favour of post-traumatic brain oedema as the cause of secondary deterioration opined that brain swelling with

increased brain bulk without associated lucency in the CT scan was due to proteinaceous oedema fluid and not due to haemodynamic swelling [13]. Yet another interpretation of the lucent areas in the CT scan of a head injured patient was put forward by Miller et al (1980) as ischaemic lesions [18]. They interpreted CT picture as ischemia when the lucency in the scan did not produce any mass effect or injured children with a CT scan picture of bilateral diffuse lucency without mass effect represent ischemic lesions?

To answer these unanswered queries, a prospective study is being planned.

Summary and Conclusion

1. Diffuse lucency in CT in the absence of significant intracranial haematoma presenting as increased bulk of brain has been considered as brain oedema. And when mass effect leads to obliteration of basal cisterns in the CT scan it is a grave sign.
2. This is a frequent occurrence (25%) in children with head injury and has serious prognostic connotation (33% died).
3. It can affect children who have recovered consciousness, yet may end fatally (10/20 fatal cases).
4. Injury deterioration interval amongst fatal cases had two peaks:
   a) within 24 hours (11 deaths)
   b) on third day (5 deaths)
5. Pathogenesis and management remain an enigma.

A prospective study for further elucidation of this problem and to solve it is being planned.

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