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A Study of Traumatic Brainstem Injury – Clinical, Radiological and Pathological Correlation

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Abstract

Aim: To study the types of brainstem injury and their clinical and radiological presentation and to correlate the macroscopic and microscopic findings with the above clinical and radiological imaging. *Materials and methods:* 50 patients who sustained fatal head injury and died in our hospital were taken up for this study. Patient's age, time of injury, mode of injury, level of consciousness, the site of impact of head injury, other associated injuries and survival period were collected. Radiological evidence of fracture, hematoma, contusions, edema, and basal cistern obliteration were noted. At the time of autopsy, the brainstem was examined looking for areas of gross contusion, hematomas and edematous changes. The brainstem specimens were stained with haematoxylin eosin stain and the slides were studied under microscope with low and high power. Brainstem lesions were categorized as primary and secondary depending on macroscopic and microscopic appearance. *Conclusion:* The study has shown that the lateral impact during trauma has resulted mostly in secondary brainstem lesions. In majority of cases the primary brainstem lesions have been associated with basal skull fractures whereas majority of secondary brainstem lesions were associated with vault fractures. In primary brainstem lesions gross haemorrhagic lesions were seen in dorsal, dorsolateral aspect of midbrain and dorsal aspect of upper pons. In secondary brainstem lesions gross hemorrhagic lesions were seen in the midline and paramedian aspect of tegmentum of midbrain and pons.

Keywords: brainstem injury; diffuse axonal injury; traumatic brain injury

Introduction

Injury to the brainstem carries a poor prognosis both as regards life and neurological recovery. The brainstem may be injured at the time of accident (*Primary brainstem injury - PBSI*) or due to raised intracranial pressure because of brain shift or vascular involvement (*Secondary brainstem injury-SBSI*). With increasing severity of head injuries especially due to road traffic accidents the incidence of PBSI is increasing. Prior to the availability of CT scan the diagnosis of brain stem injury was presumptive, based on clinical criteria. Now with the availability of CT scans we can demonstrate the brainstem injury radiologically. CT scan is a common and reliable tool for diagnosis in severe head injury [1]. This study is to correlate the clinical, radiological, and pathological findings in patients of fatal head injury.

Our aim is to study the types of *Brainstem injury (BSI)* in fatal head injury patients and to analyze the frequent mode of accident and the site of impact producing brainstem injury. We have tried to find out the association between the type of skull fracture and brainstem injuries. We compared the survival period of primary and secondary brainstem injuries. We have discussed the CT scan findings of brainstem injury patients and also analyzed the macroscopic and microscopic findings in traumatic brain stem injury. We have correlated the autopsy findings with the clinical and radiological findings of those fatally injured patients.

Materials and Methods

This study was conducted in Institute of Neurosurgery, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai.

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Fifty patients, admitted in our Head Injury Unit who sustained fatal head injury and died were taken up for this study. The following details were recorded at the time of admission such as name, age, date and time of injury, mode of injury (road traffic accident, fall from height, assault) etc.

From the clinical examination the following details were collected (Glasgow coma scale, vitals, pupillary reaction, Doll's eye movement). The site of impact (anteroposterior, lateral and vertex) was noted. Associated injuries like fracture ribs, pelvis and long bones, abdominal visceral injury if present were also noted. From the CT scan brain, the site of fracture (frontal, temporal, parietal, occipital), hematoma (Extradural, subdural), subarachnoid hemorrhage, brainstem contusions, edema, level of basal cistern obliteration were noted. The patients who had a surgical procedure were recorded. Survival period (period between date and time of injury to date and time of death) was recorded.

At the time of autopsy, the macroscopic details were noted with special reference to the site of skull fracture, extradural and subdural hematomas, subarachnoid hemorrhage, evidence of edema and transtentorial herniation, parenchymal lesions like contusions and lacerations. Brainstem was examined in detail by taking sections at various levels, looking for areas of gross contusion, haematoma, edematous changes and the details were noted. The brainstem specimens were processed for paraffin embedding and the sections were stained with haematoxylin eosin stain and the slides were studied under microscope with low and high power.

Neural changes (hypoxic neurons, neuronophagic cell death, axon retraction balls), vascular changes (focal edema, small hemorrhages, necrosis) were looked for to rule out PBSI. Changes of secondary traumatic effects (swelling, edema, global ischemia, haematoma of different sizes, duret hemorrhages) were also looked for. The brainstem lesions were categorized as primary and secondary depending on macroscopic and microscopic appearance and associated other lesions.

The injury was recorded as PBSI if there was

- Presence of contusion or haemorrhage in the brainstem in CT scans
- Presence of contusion or gross haemorrhage in the brainstem at the time of autopsy
- Evidence of focal hypoxic neurons, focal axonal injury with axon retraction balls, focal edema and haematoma in histopathological examination.

The injury was recorded as SBSI if there was

- Evidence of gross supratentorial traumatic mass lesion/edema with evidence of transtentorial herniation in CT scan
- Evidence of transtentorial herniation at the time of autopsy
- Presence of gross swelling and edema, global ischemia, diffuse haemorrhage and infarct in the brainstem in histopathological examination.

IBM SPSS version 20 was used for calculation of statistical significance. Continuous variables such as age and survival period were expressed as means and 'p' value calculated using single way ANOVA. Categorical variables such as sex, mode of injury, site of impact, GCS, pupil reaction, radiological features and autopsy findings were expressed as percentages with 'p' value calculated using Pearson chi square, likelihood ratio (if chi square assumptions are violated) and Krusker-Wallis independent samples test.

Results

The age of the patients range from 4-87 years (mean – 53.5 years). 6 patients were over the age of 60 while one patient was below 10 years of age. The mean age of PBSI was 55.7 years against 36.2 years for SBSI. There was a statistically significant correlation between age of the patient and histopathological finding ($p = 0.003$). With regards to gender, majority of the patients were male (92.6%). Only 3 women had brainstem injury. One had PBSI and the other two had SBSI. However there was no statistical significance ($p = 0.528$). Though RTA was the most commom mode of injury accounting for 35 cases of BSI, it did not have any statistical significance with HPE ($p = 0.841$). Of the 35 cases 14 had PBSI and 21 had SBSI. Fall from height accounted for 5 cases with 2 belonging to PBSI and 3 belonging to SBSI. Assault cases accounted for one SBSI.

All 41 cases with BSI had GCS ≤12. 11 patients had GCS >8, other 30 patients had GCS ≤ 8. GCS finding had no statistical significance ($p = 0.784$) with histopathological finding. Out of 41 BSI patients, 7 patients had retained pupillary reaction to light and oculocephalic response. The rest had an impaired or absent pupillary reflex and oculocephalic response. Among the seven patients with retained reflexes, one patient had PBSI and the rest had SBSI. Among the 6 with SBSI, 4 had anisocoria. However there was no statistical

correlation (p = 0.170) between the reflexes and histopathology.

Regarding the site of impact and histopathological examination (HPE) correlation, anteroposterior impact resulted in 4 PBSI and 6 SBSI. Lateral impact accounted for 36 patients where 11 patients had PBSI and 19 had SBSI. Site of impact could not be made out in 2 patients who had no brainstem injury. But there was no statistical significance (p = 0.127) between site of impact and HPE.

Among the 41 patients with HPE evidence of BSI, 15 had brainstem contusion revealed in CT and was not statistically significant (p = 0.672). 14 out of 15 patients had associated parenchymal contusions. Those with CT evidence of brainstem contusions, 6 had PBSI and 9 had SBSI. Cerebral edema was seen in 21 patients with SBSI as compared to 2 patients in PBSI group and was statistically significant (p = 0.000).

30 patients in the study had an associated hematoma (EDH/SDH/SAH), of which 8 had PBSI, 19 had SBSI and 3 had no BSI. Of the remaining 20 patients without an associated hematoma 8 had PBSI, 6 had SBSI and 6 had no BSI. There was no statistical correlation between the presence of fracture and histopathogy (p = 0.053).

Table 1: Demographic, clinical, radiological and pathological profile and their correlation

Parameters	No brainstem injury	Primary brainstem injury	Secondary brainstem injury	'p' value (< 0.05 is significant)
Number of cases	9	16	25	
Mean age (in years)	43.8	55.7	36.2	0.003
Sex				0.528
Male	9	16	23	
Female	0	1	2	
Mode of injury				0.841
RTA	8	14	21	
Fall from height	1	2	3	
Assault	0	0	1	
Site of Impact				0.127
Anteroposterior	1	4	6	
Lateral	6	11	19	
Vertex	0	1	0	
No impact	2	0	0	
GCS				0.784
3-5	4	8	11	
6-8	3	3	8	
9-12	2	5	6	
Pupillary and oculocephalic reflex				0.170
Present	3	1	6	
Absent	6	15	19	
Fracture				0.068
Absent	7	5	10	
Skull base	0	4	1	
Frontal	2	2	1	
Temporal	0	3	7	
Parietal	0	1	6	
Occipital	0	1	0	
Brainstem contusion in CT				0.007
Present	0	6	9	
Presence of cerebral edema	5	2	21	0.000
Presence EDH/SDH/SAH				0.053
Present	3	8	19	
Absent	6	8	6	
Surgical intervention	0	4	8	
Mean survival (in hours)	47	41.55	103.2	0.133
Gross appearance of brainstem hemorrhage				0.033
Present	0	7	12	
Absent	9	9	13	

Patients with skull fractures had PBSI in 11 patients, SBSI in 15 patients and no BSI in 2 patients. Patients without skull fractures had no BSI in 7 patients, PBSI in 5 patients and SBSI in 10 patients. There was no statistical correlation between the presence of skull fracture and HPE findings ($p = 0.068$). In those patients with PBSI and skull fractures, 4 cases had skull base fractures, 2 cases had frontal bone fracture, 3 cases had parietal bone fracture and one patient had occipital bone fracture. Those patients with SBSI and skull fractures 6 cases had parietal bone fracture, 7 cases had temporal bone fracture, one patient had skull base fracture and one patient had frontal bone fracture.

Autopsy revealed gross brainstem hemorrhage in 19 cases of which 7 was PBSI and 12 was SBSI and was statistically significant ($p = 0.033$). 22 patients with HPE proven brainstem injury had no gross evidence of brainstem hemorrhage.

The duration of survival ranged from 6-600 hours with a mean survival of 73.42 hours. While patients with PBSI had a mean survival of 41.55 hours, those with SBSI had a mean survival of 103.2 hours. In spite of this difference in survival duration, no statistical significance could be established ($p = 0.133$).

Discussion

The extent and type of brain injury resulting from head injury depend on the physical mechanisms involved. Components include the nature of the force (contact or inertial loading), the type of injury (rotational, translational, angular), the magnitude and duration of impact [2]. Inertial injuries are commonly called acceleration and deceleration injuries. Three types of acceleration may occur [3].

- Translational acceleration
- Rotational acceleration
- Angular acceleration.

At longer acceleration duration, inertial effects are maximum and the resulting strains are able to propagate deeper into the brain. This can cause deep diffuse axonal injury involving the brainstem. Diffuse axonal injury is caused by angular acceleration or rotational acceleration. The extension of focal neuronal damage is centripetal from the cortex inward to the brainstem as the injury force increases. Most of the cases of diffuse axonal injury arise from vehicular accident in which acceleration is long. The direction of acceleration is important in the production of axonal injury. Angular acceleration in coronal plane has a high incidence of severe form of diffuse axonal injury involving the brainstem.

In our study the incidence of PBSI was 39.02% and it was relatively very high when compared to similar studies [4] in the literature. The incidence of SBSI was 60.98%. Most of the victims of head injury in this study were middle aged people, the mean age of patients with PBSI and SBSI were respectively 55.7 and 36.2 where as in the series of Mahadevan et al. [5] the patients were young adults (mean age 31 years) and there was no gross difference between the mean age of PBSI (31.1 years) and SBSI (37 years).

In western countries the majority of people injured in road traffic accidents are car occupants. But in developing countries like India the people vulnerable to road traffic accidents are pedestrians and two wheeler users. Those patients who had head injury due to fall, the majority were injured at construction site. RTA was the cause of BSI in 85.36% (35 cases), fall from height was the cause in 12.19% (5 cases) and assault was the cause in 4% (1 case). In our study 87.5% of PBSI and 84% of SBSI were due to RTA and it was almost similar to other series [5].

When compared to SBSI (mean survival 103.20 hrs) PBSI was associated with significantly shorter survival (41.55 hours). Out of 16 cases of PBSI, 5 cases died within 24 hours (31.25%) and out of 25 cases of SBSI, 4 cases died within 24 hours. PBSI was associated with shorter survival in the literature [5] also.

In our observation antero posterior impact was seen in 10 cases of BSI (4 primary, 6 secondary) and lateral impact was seen in 30 cases of brainstem injury (11 primary and 19 secondary). In one case the impact was on vertex and the patient had PBSI. In the literature the site of impact did not have any bearing to the evolution of PBSI and it was found equally in lateral as well in anteroposterior impacts suggesting that the mechanism of brainstem injury is not merely displacement in these two axes but may be a shear injury. In our study those cases who had lateral impact sustained SBSI due to associated supratentorial hematomas with midline shift.

Out of 41 cases of BSI 26 had fracture skull. Among these 26 cases, 11 cases had PBSI and 15 cases had SBSI. PBSI were associated more with skull base fractures and SBSI cases were associated more with lateral skull fractures (temporal-7, parietal-6). Shukla et al. [5] reported that there was

no association between the site of skull fracture and incidence of brainstem injuries. The association between the site of skull fracture and type of BSI was not available in the literature, except the above one.

All 41 cases of BSI were presented with GCS < 12. Among these, 11 cases were presented with GCS > 8 and 30 cases were presented with GCS ≤ 8. In those 16 cases who had PBSI, 8 cases were admitted with GCS < 5, 3 cases with GCS 6 – 8 and 5 cases had GCS 9 – 12. In those 25 cases who had SBSI 11 were admitted with GCS < 5, 8 cases had GCS 6 – 8 and 6 cases had 9 – 12.

19 cases had GCS of 3 – 5 and among these 8 were PBSI and 11 were SBSI. In this study there is no difference in the level of consciousness (mean GCS) between primary and secondary brainstem injury whereas in the series of Gentry et al. [6] the level of consciousness was most impaired in PBSI.

CT brain revealed brainstem contusion in 15 cases in our study whereas only 2 cases out of 27 examined, revealed hemorrhagic lesion in brainstem in the study of Mahadevan et al. [5] *Zuccarello* stated that cranial CT scan was found to be insensitve and was unable to identify a lesion of less than 0.25 ml volume in the brainstem [7].

Autopsy showed brainstem contusion in 19 cases out of 41 cases of brainstem injury whereas CT scan showed brainstem contusion in 15 cases. In the series of Pathak et al. [8], autopsy of brainstem revealed brainstem contusion in 30 patients, however only 16 patients could show the same on CT scan. The difficultly in visualization is due to the differential co-efficient artefacts inherent to the imaging technique itself [9]. Some authors suggested CT with special zoom cuts for the posterior fossa so that bony artefact is avoided the maximum, however MRI definitely has an edge over CT scan is detecting brainstem lesions [6,8]. MRI is more sensitive in detecting brainstem injury but this modality of investigation was not performed in this study because of economic constraints, lower conscious level and poor haemodynamic state of the patients.

Out of 16 cases of PBSI 8 cases were suspected as secondary brainstem injury before HPE, due to the presence of parenchymal contusion, haematoma (extradural, subdural) and subarachnoid haemorrhage in the CT scan. But in HPE, they had evidences of primary brainstem injury. Gerhard [10] stated that "the histology in generalized pattern of brain injury will also exhibit signs of primary injury in the brainstem and to judge primary and secondary influences changes should be noted in the brain parenchyma also".

In HPE, findings of PBSI such as focal hypoxic neurons, axon retraction balls, focal edema, and findings of SBSI such as gross hemorrhage, necrosis and infarct were noted and correlated with associated parenchymal injury. Evidence of PBSI was seen in 16 cases and evidence of SBSI was seen in 25 cases.

Some suggested looking for neuronophagic cell death, chromatolysis, differentiation, trans-neuronal degeneration, demyelination to differentiate primary and secondary brainstem injury [10]. These changes are better seen with special stains like Sudanblack, Luxol Blue Stains and Neurofilament in Immunohistochemistry. In our study only haematoxylin eosin stain has been used. When compared to other series in the literature the incidence of primary brainstem injury was relatively high in our study [1,5,9].

Conclusion

Most of the brainstem lesions (both primary and secondary) arise from injuries in road accidents. Most of the patients in our study (60.97%) who had brainstem injury were in between 31–60 years of age and 92.68% of the patients who had brainstem injury were males. The study has shown that the lateral impact during trauma has resulted mostly in secondary brainstem lesions. In majority of cases the primary brainstem lesions have been associated with basal skull fractures whereas majority of secondary brainstem lesions were associated with vault fractures. There has been no gross difference in admission Glasgow coma scale score in both primary and secondary brainstem lesions. The patients with primary brainstem injury had shorter survival compared to those with secondary brainstem injury although it was insignificant. Even though MRI with diffusion weighted imaging provides more information about brainstem contusions, CT brain with thin slices can be used as a primary screening modality. In PBSI gross haemorrhagic lesions were seen in dorsal, dorsolateral aspect of midbrain and dorsal aspect of upper pons. In SBSI gross hemorrhagic lesions were seen in the midline and paramedian aspect of tegmentum of midbrain and pons.

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