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Neurosurgery

BRAIN STEM LESIONS IN EARLY AND LATE DEATHS AFTER CLOSED HEAD INJURY

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KEYWORDS :

AIM OF THE STUDY

This study was conducted to find out whether the pathology of the brain stem lesion are different in Early (<72 hrs) and late deaths(>72 hrs) following closed (Non-missile)head injury.

MATERIALS AND METHODS

Twenty patients with closed (non-missile) head injury were taken for this dtudy-Non of these patients had significant extra cranial injury. Apart form routine neurological examination, mode of injury, radiological findings and the survival time were noted. C.T Scan showed supratentorial lesions which did not require surgical intervention. ICP monitoring was not done. Evoked potentials were also not done as it is well known that vestibulo-ocular reflex is as reliable as evoked potential for evaluating brain stem functions. (Mahapatra et al.1985). Earlier studies showed that 60% if deaths from severe head injury occur in first 3 days) David M.Lyle 1986). Hence, according to survival time these patients were divided into two groups.

Group I - Those died within 72 hrs.(n=13)
Group II – Those died after 72 hrs(n-7)

These patients were autopsied and the brain was removed upto cervicomedullary junction and were preserved in 10% buffered formalin and sent to the pathologist. At autopsy, findings like basal fractures were noted. The pathologist was blind to the clinical features and survival time. The brain were cut after 3 weeks. The brain stem was separated and the supratentorial apportion of brain was cut and the macroscopic lesions were noted. Evidence of uncal herniation was noted. All the brain stems were examined macroscopically for tears and haemorrhages.

Brainstems were sectioned at the following levels.

A. Superior colliculus
B. Inferior colliculus
C. Ponto-mesencephalic junction
D. Mid pons
E. Ponto – medullary junction
F. Medulla

The paraffin blocks were prepared in the usual manner and the sections were stained with,

1. Haematoxylin and Eosin
2. Carbol Thionine for Nissl's substance
3. Luxol Fast Blue B and PAS for myelin and intracellular lipochrome .
4. Chloral hydrate silver stain nerve fibres
5. Holtzer's stain Glial fibres.

Sections were studied with light microscope and findings were noted and analysed. We used Chi square test for statistical analysis.

RESULTS

There was no significant difference in mode of injury and GCS score between Group I Group II. Significant number of patients in Group I had impaired brain stem reflexes and unreactive pupil than Group II and which was found to be statistically significant (Table 2). In autopsy, all the patients of both the groups had associated supratentorial lesion and was similar to the CT finding. And there was no significant difference in basal fracture and uncal herniation between Group I and Group II (Table 3).

The following lesions were observed (Table 4).

1. Coagulative necrosis was seen in collicular and periaqueductal area in 75% of the patients. In Group I the necrosis was more extensive and bilateral in contrast to Group II where they were small and unilateral.
2. Tears were often ventrally placed, seen in pontomedullary junction especially at the junction of pyramid and olive and were extensive in group I. In one patient in Group I there was ventrally placed tear in the midbrain extending from the interpeduncular fossa. In Group II, tears were seen only in

RESULTS

TABLE 2

	Group I	Group II	Stat. Signi.
1. Mode of Injury			
RTA	76.92%	82.70%	P>0.05
Fall	15.39%	0%	
Assault	7.69%	14.28%	
2. GCS Score			p>0.05
3-5	76.92%	71.42%	
6-8	23.08%	28.58%	
3. Pupil			
Non – reacting	84.61%	28.58%	P<0.05
Reacting	15.39%	71.42%	P<0.05
4. Brain Stem Reflexes			
Impaired	100%	71.42%	P<0.05
Present	0%	28.58%	P<0.05

TABLE 3

	Group I	Group II
Supratentorial lesions	**100%**	**100%**
Basal fractures present	**46.15%**	**42.85%**
Uncal herniation		
Present	**15.39%**	**14.28%**
Absent	**38.46%**	**57.14%**
Non observed	**46.15%**	**28.57%**

TABLE 4

HISTOPATHAOLOGICAL	GROUP I	GROUP II
A. COAGULATIVE NECROSIS		
Bilateral Extensive	64.53%	14.28%
Unilateral Small	15.38%	57.14%
Absent	23.08%	28.57%
B. TEARS		
• Ventral Extensive PMJ	23.08%	0%
• Ventral small	0%	14.29%
• Dorsal Small	0%	14.29%
• Absent	76.92%	71.42%
C. HAEMORRHAGES		
• Bilateral Extensive	84.62%	0%
• Unilateral small	7.69%	57.14%
• Midbrain	36.36%	0%
• Absent	7.69%	42.86%
D. STRICH LESION		
Present	38.46%	28.57%

1. 2 Patients of which in one patient teat was small and dorsally placed in rostral brain stemand in the other patient who survived for 15 days there was small tear involving the pyramid and the patient had corresponding hemiplegia. These tears were always accompanied by haemorrhages in the surrounding areas. The more extensive tears were accompanied by basal fractures involving middle and posterior cranial fossae.
2. In group I patients, the haemorrhages were extensive and bilateral. Predominantly in the pons. Subependymal haemorrhages were also more frequent. One third of the patients had haemorrhages in the midbrain. Whereas in group II, the haemorrhages were small., unilateral and none occurred in the midbrain. Some if these haemorrhages were also seen macroscopically while sectioning the formalin fixed specimen
3. Strich lesions were seen predominantly in group I in the reticular formation. In one patient in group II who survived for 2 ½ years. Numerous strich lesions and microglial stars were seen.

DISCUSSION

Clinical features

Rosenblum et.al. in 1981, found that 21 of 23 autopsied patients with primary brain stem lesions showed impaired oculocephalic reflexes and 19 of 23 had unreactive pupils. They compared these clinical findings with multimodality evoked potentials and they concluded that the oculocephalic reflexes and unreactive pupils were the clinical equivalents of the evoked potentials. In our series also all the patients (100%) of Group I had impaired oculocephalic reflexes and 85% of the patients had unreactive pupil. Hence, these reflexes are useful to evaluate the brain stem damage following head injury. There was no significant difference in mode of injury, age, sex and Glasgow come scale score (GCS score) between Group I and II. Pattern of basal fractures and incidence of uncal herniation and associated supra tentorial lesions were found to be similar in both the groups.

Haemorrhages

Haemorrhages in the pons may be caused by shearing injury to the perforating branches of brain stem. Crompton in 1971, studied 100 autospy cases and found ischaemic infarction in the lateral midbrain tegmentum and unilateral haemorrhages. The similarities which we found in comparison with Crompton series (1971) were the presence of sub-ependymal haemorrhages and extensive haemorrhages in the basis pontis and tegmentum, whereas in our with bilateral collicular necrosis In Group II patients, haemorrhages were in midline involving tegmentum and basis pontis suggestive of secondary brain stem haemorrhages.

Tandon in 1964 noted petechial haemorrhages in patients who survived for short time and longest survival was noted with small single or multiple haemorrhages. In our study, multiple, extensive, bilateral haemorrhages were seen in Group I patients and unilateral, small haemorrhages in Group II.

There is difference in location of primary and secondary brain stem haemorrhages. Tomlinoson in 1970 noted some overlap between the distribution of primary and secondary brain stem lesions. It is very difficult to differentiate, these two types of lesions if they coexist. In our series also few patients showed overlapping lessions.

Necrosis

A knowledge of spectrum of pathological lesions occurring in severe head injury provides us a perspective for devising and evaluating therapy. Rosenblum et al. in 1981 studied 35 autospy cases and found 80% incidence of collicular necrosis which is similar to our study. And in group I patients collicular necrosis was bilateral, extensive and involved periaqueductal region also. Whereas in Group II, collicular necrosis was often small and unilateral. Bilateral extensive collicular necrosis was seen in many mechanisms proposed by Tomlinson (1970), Lindenberg (1964) and forces, injury of vessels with subsequent thrombosis leading to infraction and downward displacement of brain stem by traumatic forces at the time of impact leading to its brushing against the tentorium. We know of no way yet to distinguish various mechanism of midbrain injury using histo – pathological techniques.

Brain stem Tears

Lindenberg in 1970 described a characteristic lesion in the pyramid at pontomedullary junction in traumatic hyperextension of head. He described that the tears were common in rostral medulla. This is possibly due to the anatomical peculiarity that pia and adjacent sub-arachnoid tissues are fused, forming a tough sleeve around the caudal medulla and cervical cord. This tough sleeve protects the peripheral tissues of medulla against abnormal distortion or stretching. Therefore if any tensile stress occurs in the ventral medulla, it will become effective more likely in its less protected rostral portion particularly pyramid.

The tears in the rostral pyramid can occur at the time of impact and also during postmortem if the brain is forcefully removed from the cranium. However these artifacts can be differentiated from antemortem tears by the absence of haemorrhages in the deeper pontine tissues. In artifactitious tears, there may be only some haemorrhages along their margins (Lindenberg, 1970)

Adams in 1982 recorded 2 patients with pontomedullary rets who survived 8 days and 26 respectively after being involved in RTA. Both had clinical features suggestive of primary damage in the brain stem. In our series also, one patient had tears in rostral pons and corresponding contralateral hemiplegia. He survived for 15 days after head injury. It shows that the pontomedullary tears may not always produce instantaneous death.

Diffuse axonal injury

Evidences of diffuse axonal injuries like axonal retraction balls,. Axonal edema and microglial stars were seen predominantly in group I patients. Axonal retraction balls were more commonly seen in reticular formation. These lesions were less extensive and infrequent in Group II patients, though there was no significant difference between both the groups. In one patient in Group II, who survived for 2 ½ years, apart from numerous axonal retraction ball and microglial stars there was areas of demyelination also seen.

CONCLUSION

1. Oculocephalic and pupillary reflexes are useful to evaluate the patient with brain stem damage.
2. Common changes we observed in brain stem are
   a) Collicular and periaqueductal necrosis
   b) Haemorrhages in sub- ependymal, tegmental and basispontis region of pons.
   c) Ponto- medullary tears which were seen mainly in patients who had basal fractures.
3. Extensive, bilateral brain stem damage seen in patients who died early was sustained at the time of impact.
4. An assessment of brain stem damage shortly after arrival at hospital. May provide a perspective for divising and evaluating therapy.
5. Vigorous treatment might be of benefit only to those patients with small brain stem lesions, in whom other secondary effects could be controlled.

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