Autopsy and Histopathological Analysis of 28 Cases of Traumatic Brainstem Haemorrhages

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ABSTRACT

Background: Brainstem hemorrhage was first described by Cheyne in 1812 in a pathological study of patients presenting with lethargy and coma. In 1877 Bode reviewed the findings of 67 patients reported in the literature (1). In the United States, the incidence of closed head injury is estimated to be approximately 200 cases per 100,000 persons per year. The financial burden of head injuries in the United States is estimated to be $75-100 billion annually (4).

Materials and Methods: This cross-sectional study, the brainstem was studied by both gross and histopathological examinations.

Results: During a period between between July 1, 2010 and June 31, 2011, a total of 71 head injury cases were autopsied and brainstem hemorrhage was seen in 28 of them.

Discussion: Out of the 28 cases, most of the hemorrhages occurred in the midbrain (68%) and least in medulla oblongata (11%), while four cases had pontomedullary junction tearing and one of them presented presented cervicomedullary junction tearing.

Conclusion: Hemorrhages are usually the only evidence of injury to the brainstem in those dying immediately or within a few hours. Failure to realize this has led to underestimation of the frequency of brainstem hemorrhage after injury, and, indeed, to the frequency of brainstem lesions of all kinds.

Keywords: Autopsy, Brainstem Hemorrhage, Head Injury.
was separated with an axial cut. The whole brain was taken out from calvarium and the brainstem was analyzed. The brain stem was cut in eight levels according to anatomical standards: two cuts in midbrain, two cuts in pons, and four cuts in medulla.

Consent: We took consent from the first degree relatives after explaining the methods and the goals of the study.

RESULTS

During the period between July 1, 2010 and June 31, 2011, a total of 71 head injury cases were autopsied and brainstem hemorrhage was observed in 28 cases. The maximum number of cases belonged to the age group of 35 to 40 years. In terms of gender, 55 out of the 71 patients were male and the rest 16 were female.

<table>
<thead>
<tr>
<th>Total number of head injury cases</th>
<th>Total number of brainstem Haemorrhages seen</th>
<th>Haemorrhages in midbrain</th>
<th>Haemorrhages in pons</th>
<th>Haemorrhages in medulla oblongata</th>
</tr>
</thead>
<tbody>
<tr>
<td>71 cases</td>
<td>28 cases</td>
<td>19 cases (68%)</td>
<td>6 cases (21%)</td>
<td>3 cases (11%)</td>
</tr>
</tbody>
</table>

DISCUSSION

Out of the 28 cases, the most hemorrhages occurred in the midbrain (68%) and the least in medulla oblongata (11%), while four cases had pontomedullary junction tearing and one of them had cervicomedullary junction tearing.

On histopathological examination, the tegmentum of the midbrain, tegmentum and basis pontis, and medulla oblongata were almost completely devoid of neurons and replaced by a mass of fatty macrophages and proliferating astrocytes.

Meyer CA et al. reported that the most frequent site of hemorrhage, in 31 (69%) of the 45 patients, was the ventral rostral midbrain adjacent to the interpeduncular cistern (7). Ropper and Miller proposed three mechanisms causing traumatic brainstem hemorrhage. Brainstem hemorrhage can be caused by primary lesion resulting from either rotational forces or transient acceleration-deceleration forces that cause contusion against the tentorium or secondary to the brainstem compression (8).

Jellinger (1967) reported that only 43.5% of 415 fatal head injuries showed evidence of brainstem lesions, but extensive histology was done only in the 38 cases dying after prolonged coma. Tandon and Kristiansen (1966) reported a group of 37 fatal cases with brainstem symptomatology in whom no structural damage was seen at necropsy, yet histological studies had not been apparently done. In cases with brain-stem symptomatology, Zoltan (1966) found no lesions at that level and was prepared to seek the origin of the symptoms in cortical and subcortical damage. By contrast, in a careful histological study, Matsuoka, Sakaki, and Okamoto (1967) found haemorrhagic lesions in the brainstem of 22 out of 25 cases while Mayer (1967) found them in all his 25 cases dying within an hour of injury. Mayer plotted primary brainstem haemorrhages only from such a group (9).

In a study by Gunji and colleagues, the most involved site was known to be medulla and the common reported lesions were respectively pontomedullary tearing, medullary contusion, and cervicomedullary tearing (10).

In Ohshima’s study (1998), the most involved site was pontomedullary junction. Moreover, Kondo ,et al. (1995) reported similar findings (11). In addition, Simpson et al. (1989) reported the most involved site to be pontomedullary lesions (12).

CONCLUSION

Haemorrhages are usually the only evidence of injury to the brain-stem in those dying immediately or within a few hours. Failure to realize this has led to underestimation of the frequency of brain-stem
haemorrhage after injury, and, indeed, to the
frequency of brain-stem lesions of all kinds.

Intracranial hemorrhage is an important
cause of acute neurologic dysfunction and
accurate early diagnosis of cortical versus
brainstem hemorrhage, with initiation of
appropriate therapy, may help minimize
morbidity.

Competing Interests: The authors declare
that they have no competing interests. Authors
have read and approved the final manuscript.

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