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Short report

Traumatic transection of the brainstem

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SUMMARY A case of nearly complete transection of the lower brainstem following skull fracture with detailed histological study is presented.

Brainstem lesions are a well recognised cause of coma and early death following severe head injury[1-3] but massive lower brainstem laceration resulting from closed head injury is highly unusual.[4 5] We report here one such case which in addition had some unexpected histological features.

Case report

An 8-year-old boy was in good health until his injury. While playing on the roof of a local technical college, he tried to jump on to a lower roof and fell through a closed glass sky-light on to the floor of a laboratory workshop 11 feet below. He may have landed on some machinery, for when he was found over half an hour later by relatives, he was lying on his right side close to a work bench. He was deeply unconscious on arrival at hospital; radiograph showed an occipital skull fracture. At first there was slight clinical improvement: his pupils were equal and reacted to light, his breathing was normal, and he made strong extensor responses to stimulation of his limbs although he showed no spontaneous movement. However, on transfer to the National Hospital he started to bleed profusely from his nose and mouth. This bleeding was thought to be arterial and to originate from the nasopharynx; all measures to stop it were unsuccessful. The boy's condition deteriorated rapidly and he died approximately 7 hours after his injury.

At post-mortem examination, a few abrasions and bruises on both elbows were noted. There was a small occipital scalp laceration through which blood had trickled and the right side of the neck and face were swollen and bruised. Internal injury was entirely confined to the head. There was a closed skull fracture, extending from the midline posteriorly and continuing on the right side towards the skull base. At the back the fracture was comminuted and involved the posterior parts of the parietal bones and the squamous part of the occipital bone. The main fracture line continued forwards along the right temporal bone, turning medially at the base, and was seen to reach the medial end of the petrous bone. Some epidural haemorrhage was present mainly on the right side, but subdural haemorrhage was much more extensive and bilateral. There were large areas of contusion of both frontal lobes, mainly on their orbital surfaces, and over both temporal poles, and this was more extensive on the left side than the right. The posterior temporal lobes and the lateral and inferior surfaces of the occipital lobes were also contused, but here the damage was more severe on the right. The inferior and lateral surfaces of the right cerebellar hemisphere also showed contusion. On sectioning the fixed brain, in addition to the extensive contusions there was haemorrhage in the left temporal pole, discoloration of the right pulvinar and some blood in the lateral ventricles and fourth (but not third) ventricle. The aqueduct was slit-like but there was no other evidence of midbrain distortion. In the lower pons there was a laceration (fig 1a) situated mainly in the tegmentum. It was an irregular tear, mostly on the right side, but reaching and slightly crossing the midline and branching both ventrally and dorsally towards the ventricle. At the ponto-medullary junction the tear almost reached the left surface effecting a nearly complete transection of the brainstem. The brainstem above and below the tear appeared macroscopically normal.

Histological examination revealed one microscopic focus of haemorrhagic infarction in the right medial occipital cortex and another in the right pulvinar. In view of the particular interest of this case numerous step serial sections were examined from blocks taken from the lower brainstem region surrounding and including the complex pontomedullary tear which was everywhere marked by small haemorrhages in its walls. Seen in its maximal extent at the ponto-medullary junction the tear cut a jagged path across the ventral half of the tegmentum, and impinged posteriorly upon the olives. On the right side irregular spurs projected from the main line ventrally, roughly perpendicular to the fibres of the middle cerebellar peduncle, and dorsally towards the angle of the fourth ventricle. Further down the tear ran in an oblique plane,

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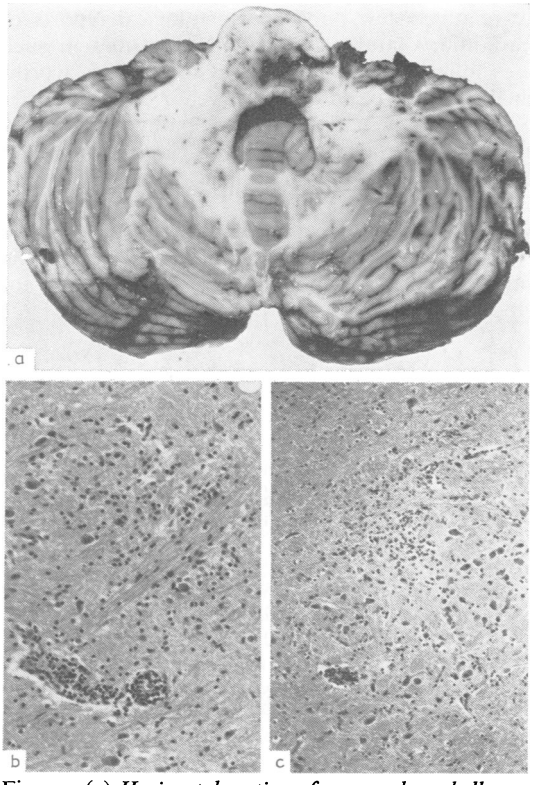

Figure (*a*) *Horizontal section of pons and cerebellum showing the massive tear from the right side and contusion of the adjacent cerebellum.* (*b*) *and* (*c*) *Micrographs of the pontine tegmentum to show perivascular mononuclear cuffing with neighbouring microglial nodules.* (*Haematoxylin and eosin;* (*b*) × *100,* (*c*) × *70.*)

involving even the upper part of the medullary pyramids where small tears and haemorrhages were found. Tiny subependymal and laterally situated tegmental haemorrhages were also present in the pons and medulla, mainly on the right side. In the vicinity of the tear and for several hundred microns above and below it there were several small vessels with conspicuous cuffs of mononuclear cells: these were particularly evident in the right superior vestibular nucleus, pontine tegmentum and medial aspect of the middle cerebellar peduncle. In this same region a few microglial nodules were also found quite close to a perivascular inflammatory cuff (fig 1b,c). Although silver stains failed to give convincing evidence of axon retraction balls, abnormally thickened and contorted axons were present in both central tegmental tracts and the right facial nucleus. In addition, a number of acutely degenerate neurones with shrunken eosinophilic cytoplasm were present in the right superior vestibular nucleus and the right superior olive.

Discussion

Our initial reaction to such a massive tear was somewhat cautious. However, microscopic haemorrhages along the margins of the tear clearly rule out post-mortem artefact. The question arises whether the subependymal and lateral tegmental haemorrhages seen in our case were part of the primary lesion or a secondary result of raised intracranial pressure. Brainstem haemorrhages frequently occur with raised intracranial pressure but, unlike our case, are usually in the midline involving the base as well as the tegmentum. Furthermore in our case, the necropsy appearances did not indicate significant brain swelling and there were no internal herniae. The study of Adams and Graham[6] is relevant in this regard. In patients monitored for ventricular fluid pressure, they found lesions which they thought characteristic of significantly raised intracranial pressure: viz pressure necrosis of the parahippocampal or cingulate gyri, infarction of the medial occipital cortex, or infarction of the cerebellar tonsils. In our case there was only one microscopic focus of infarction in the medial occipital cortex on the right side, which was near to the contused inferior and lateral occipital cortex.

The occipital scalp laceration and right sided soft tissue bruising, the nature and extent of the bony injuries, particularly the severe comminuted occipital fracture, and the fronto-orbital contusions (more severe on the left) which can be ascribed to a "contre-coup", are suggestive of a right-sided occipital impact. In Crompton's[2] series of 32 cases with traumatic primary brainstem lesions by far the most frequent site of impact was occipital (39%) and middle fossa fracture was present in 70%. Tomlinson[1] also remarks that laceration of the pons and medulla may be found where there is severe bony damage in the vicinity of the foramen magnum. Furthermore Lindenberg[5] in mentioning the rare instances of tears in the lower brainstem, especially at the ponto-medullary junction, notes an association with fractures of the skull base, particularly the clivus. On retrospective review of the skull radiographs it was thought that a clival fracture had most likely occurred (Professor G du Boulay). As to the mechanism of the pontine laceration this must remain speculative, although from the above comments regarding a right occipital impact and considering the right lateral and inferior cerebellar hemisphere contusions, it would be possible to envisage a ventro-caudal movement of the cerebellum and brainstem against the occipital bone. The pontine tear runs near the cerebellar contusions. If the pons continued to move after the cerebellum became stationary a sheering force might produce a laceration. However

this situation is rather different from that described by Lindenberg and Freytag[4] in their series of 21 cases of sudden death following traumatic hyperextension of the head. These tears were situated at the pontomedullary junction involving both pyramids and adjacent parts of the ventral medulla with haemorrhages extending towards the pontine tegmentum. In all these immediately fatal cases the head was forcefully retroflected but in half of them no skull fracture had occurred. In our case on the contrary, it appears from the lesions that the head was hyperflexed rather than extended and this might explain the more dorsal situation of the tear.

The most interesting findings resulting from the microscopical analysis of the region of the pontomedullary junction are the perivascular collections of mononuclear cells and the closely situated microglial nodules. These were particularly unexpected findings in view of the dearth of similar reports in the literature. However, many published cases had survived rather longer than our patient and so one might speculate that such perivascular cuffings could be a short-lived transient reaction. On the other hand, it might be argued that these inflammatory foci have a different aetiology from the traumatic lesions and perhaps predate the accident. This would be a surprising coincidence for the boy was apparently entirely well up until his accident; moreover perivascular cuffs are entirely confined to the region of the laceration. Microglial nodules have been demonstrated as early as 15 hours after head injury by Oppenheimer,[7] however patients dying within 12 hours of injury were specifically excluded from his series. The presence of microglial nodules in our patient surviving for only 7 hours after injury may appear less surprising when one realises that there are relatively few published histological reports of head injury fatal within 12 hours. Often in such cases injuries are so massive as to preclude worthwhile study or else death intervenes before transfer to a centre where detailed neuropathological study might be undertaken. Clearly this case demonstrates the need for further efforts in this area.

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