Survival after traumatic ponto-medullary tear

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SUMMARY Two children (aged 10 and 12 years) were admitted unconscious to a neurosurgical department after traffic accidents. Both developed a 6th nerve paralysis on the next day. One patient was able to communicate from the 2nd day and died on the 8th day in an anuric state without major neurological deficit. The second patient remained deeply comatose, tetraplegic, and required intermittent artificial respiration: She died of pneumonia on the 26th day. Neuropathological examination revealed a ponto-medullary rent in each case; additionally there was avulsion of small arteries over the pyramids, haemorrhage and small focal infarcts in the distribution of perforating arteries in the medulla and pons, and abundant retraction balls in longitudinal fibre tracts of the brain stem. The cases show for the first time that traumatic ponto-medullary tears are not always rapidly fatal, and demonstrate that primary focal brain stem trauma may occur in the absence of diffuse trauma of the white substance.

Tears at the ponto-medullary junction as a result of severe cranio-cervical injury are well-recognised,¹⁻⁵ but they are usually associated with almost instantaneous death. Accordingly, it has been argued by Adams and his group⁶⁻⁸ that primary damage to the brain stem as a result of a non-missile head injury does not occur in patients who survive their injury long enough to be transferred to a neurosurgical unit, and there is always diffuse damage to the white matter in such cases. The two cases documented in this paper, however, show that patients with primary traumatic ponto-medullary rents may survive for days or weeks after injury.

Case report

Case 1 This 10-year-old girl was found unconscious fastened in a safety belt after a 100 m crash down a steep slope sitting in a Lada Taiga car. On admission she was unconscious but reacted to pain. Extension spasms, bilateral extensor plantar responses and gaze deviation to the right were observed. She had a haematoma of the occiput and multiple bruises on her body. Radiographs and CT scan of the head were normal. On the cervical spine film there was a suspicion of an atlanto-axial subluxation. On the second day the child was able to communicate and now had paralysis of the 6th cranial nerve on the right side. One

day later she could move all limbs to command. Removal of the endotracheal tube resulted in severe inspiratory stridor and she had to be intubated again. The 6th nerve paralysis improved on the 5th day. The following day she deteriorated and a bilateral pneumothorax was noted and treated. She died in an anuric state the 8th day after injury.

Post mortem examination

Injuries: Bruising of the thorax, occiput and right orbit. Rib fractures from the 1st to the 9th on the right side with pneumothorax; haemothorax on the left (100 ml). Pencil-like extradural haematoma on the clivus from the dorsum sellae to the foramen magnum. No skull fracture, subluxation of the atlas (not investigated properly), so-called shock lungs, bone marrow embolism of the lungs, but no fat embolism.

Neuropathological examination (227-79)

Brain weight 1300 g. There was some subarachnoid haemorrhage over both parieto-occipital regions and in the interpeduncular fossa. There were no cortical contusions or signs of elevated intracranial pressure. There was some discolouration of the ventral surface of the brain stem at the ponto-medullary junction. Sagittal sections through this region demonstrated what appeared to be a deep tear of 3 mm (fig 1). Four sagittal blocks of the entire medulla oblongata with adjacent pons and several blocks of representative areas of the cerebral hemispheres, basal ganglia and upper brain stem were embedded in paraffin wax. Serial sections were made from the four blocks of the ponto-medullary junction and stained with haematoxylin and eosin, cresyl violet, haematoxylin van Gieson, Klüver-Barrera, Bodian, elastica.

The tear at the ponto-medullary sulcus, most pronounced over the left pyramid, was surrounded by a necro-

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Fig 1 Tear at the ponto-medullary junction surrounded by small haemorrhages (arrowhead). Focal infarct in the course of a perforating artery (arrow). Klüver-Barrera.

tic zone which showed reactive changes (fat granular cells, haemosiderin which stained positive with Prussian blue, capillary proliferation) (fig 2). There was some haemorrhage in and around the tear as well as haemosiderin in the parenchyma and in the meninges. A small artery was ruptured in the leptomeninges over the pyramids. The gap was closed by laminated thrombus (fig 3). A thrombosed vein was seen close to the ponto-medullary tear. There were several small necrotic foci in the deep rostral medulla and adjacent pons in the course of perforating arteries. The other striking findings were numerous retraction balls not only adjacent to the tear but also scattered throughout the entire medulla, pons and left cerebral peduncle. They were identified in longitudinal and cross sections and were often surrounded by microglia. They were mainly distributed in vertical fibre bundles (fig 4) (pyramidal tracts, medial lemnisci, inferior cerebellar peduncle, cerebral peduncle). Only occasional retraction balls were identified in horizontal fibre bundles at the caudobasal pes pontis. No retraction balls were noted in the corpus callosum or in the anterior cerebellar peduncles. No Kernohan notch adjacent to the retraction balls in the left cerebral peduncle was seen. The root fibres of the right 6th cranial nerve displayed mild Wallerian degeneration. There was no avulsion of the nerve but its root fibres passed through the necrotic zone around the tear. Central chromatolysis was noted in the dorsal column nuclei and in the 12th nerve nuclei but not in the 6th nerve nuclei. A small focus of necrosis was found in the calcarine cortex on one side. No signs of hypoxia or raised intracranial pressure were present.

Case 2 This 12-year-old girl was knocked down by a car while she was crossing the road. The driver did not notice her and his speed has not been recorded. In the ambulance the girl had a respiratory arrest but was successfully resuscitated. After intubation and shock therapy she was brought to the neurosurgical department. On admission she was unconscious, did not respond to pain and required assisted respiration. The pupils were small, and equal, and reacted to light. The corneal reflexes and all tendon reflexes were absent. There was no plantar response.

Fig 2 Necrotic zone adjacent to the tear displaying capillary hyperplasia. H&E $\times 60$.

Fig 3 Ruptured meningeal artery on the surface of the pyramid. The gap is closed by a thrombus. Rupture of elastica interna marked by arrows. Elastica $\times 120$.

Haematomas were seen on the forehead and on the right hip.

Radiographs and CT scan of the head and percutaneous carotid angiography showed no abnormality. The cervical spine film displayed minimal derangement at C 2 level. On the second day a left 6th nerve paralysis was noted and the level of consciousness improved slightly. During the following days the plantar response became extensor on both sides and nystagmus appeared. After two weeks the patient regained spontaneous respiration. The level of consciousness remained at some reaction to pain. The left 6th nerve paralysis and the abolished corneal reflexes remained as

Fig 4 Axonal swelling and retraction balls confined to vertical fibre tracts of pons. This area is about 2 cm distant to the tear. There is no tissue necrosis. Bodian $\times 100$.

well. She died with severe pneumonia the 26th day after the accident.

Post mortem examination

There were haematomas over the frontal bones and right hip. No skull fracture was seen. The cause of death was severe necrotising pneumonia.

Neuropathological examination (23-80)

There were no intracranial haematomas or cortical contusions but there was bilateral pressure necrosis in the parahippocampal gyri. An extensive tear at the ponto-medullary junction separated the entire pyramids and olives from the pons (fig 5a). The points of emergence of both 6th nerves were rostral to the tear (fig 5b). A sagittal section through the ponto-medullary junction showed that the tear approached the floor of the 4th ventricle up to half a centimetre on the right side. Representative regions of the cerebrum, the basal ganglia, the upper brain stem and three blocks of the whole ponto-medullary area in sagittal plane were embedded in paraffin wax. Serial sections were stained as in our first case. The deep tear was surrounded by a necrotic zone (fig 6) within which there were established reactive changes as shown by the presence of large astrocytes, lipid phagocytes, phagocytes containing haemosiderin, and capillary hyperplasia. The rostral pyramids and olives displayed coagulative necrosis. Apart from the tear, several necrotic and haemorrhagic foci were seen in the tegmentum pontis in the course of the perforating arteries including the left 6th nerve nucleus. In addition necrotic foci were found in the lateral medulla which had a contrary distribution from rostralbasal to dorsocaudal including parts of the dorsal column nuclei. A ruptured artery was observed in the leptomeninges over the pyramids covered by a laminated thrombus. Haemorrhagic infarction was noted in a part of the choroid plexus in the caudal 4th ventricle (fig 7).

Numerous retraction balls occurred in the surrounding

Fig 5a Deep ponto-medullary tear in mid-sagittal section. b: Basal view of the ponto-medullary rent. The edges of the tear are swollen, the emergence of the 6th nerves is situated rostral to the tear.

Fig 6 Ponto-medullary rent surrounded by a broad necrotic zone which approaches the floor of the 4th ventricle. Klüver-Barrera.

of the tear but also in other parts of the medulla and pons namely in the pyramids, the inferior cerebellar peduncles (fig 8), the medial lemnisci and vertical fibre tracts of pons except the superior cerebellar peduncles. A few retraction balls were seen in the corpus callosum. The 6th nerve root passed through the necrotic zone beside the tear. Its extrapontine portion on the left side displayed Wallerian degeneration as well as some root fibres of the 12th nerve. Central chromatolysis was present in the dorsal column and 12th nerve nuclei.

Extensive infarction was present in the boundary cortex zones of both hemispheres, the calcarine cortex and the Purkinje layer of the cerebellar cortex but the Sommer sector was not damaged. Pressure necrosis in the parahippocampal gyri and a tiny infarct in the 3rd nerve nucleus were attributed to tentorial herniation.

Discussion

1 THE PONTO-MEDULLARY TEAR

The tear was more impressive in the second case, but in both cases there were unequivocal signs of reactive changes in the form of astrocytic, microglial and capillary hyperplasia, and the presence of haemosiderin within the tears. Some artificial enlargement of the tear at autopsy cannot be excluded, but organisation was also noted at the tip of the deep tear in the second case. The distribution and appearance of the lesions, as well as the fact that similar tears have been reported by others, favour the view that they were primary traumatic and not merely an infarct split open during removal of the brain. Similar lesions have been described by several authors.¹⁻⁵ Death usually occurred immediately after the trauma, but one of Hardman's cases survived three hours. Our cases seem to be the only ones which survived this kind of lesion for a longer period. In our second case early death was prevented by successful resuscitation and assisted ventilation but the extensive hypoxic changes show that a critical situation had occurred. In the first case the tear was small and patient might have survived without major neurological deficits but died of extracranial causes. The tear was found by chance when the emergence of the 6th nerves was inspected thoroughly because of the clinical dysfunction of this nerve. Such small lesions can be easily overlooked in routine brain cutting.

All authors agree that cervical hyperextension is the most important traumatogenic mechanism. Patscheider² and Wuermeling and Strick³ also stress the role of axial tension forces leading even to extension

Fig 7 Haemorrhagic infarct (contusion?) of the choroid plexus in the lower 4th ventricle. The adjacent cerebellar cortex is also damaged. Cresyl violet $\times 22.5$.

Fig 8 Numerous retraction balls in the inferior cerebellar peduncle. Bodian $\times 100$.

fractures of the skull base and reported corresponding experiments on dead bodies. During axial tension on the brain stem avulsion always occurs at the ponto-medullary junction. This fact corresponds with the experience that tears can occur in this region after forceful removal of the brain from the cranium.⁴ In animal experiments Gennarelli and co-workers⁹ reported avulsion of the brain stem at this level after extremely high angular acceleration of the head in the sagittal plane. It is very likely that in our cases cervical hyperextension occurred too; the equivocal changes at the craniocervical junction and the haematoma on the forehead and occiput point to extensive movements in the sagittal plane. The extradural haematoma on the clivus in the first case could be due to avulsion of the apical ligamentum of the dens but this region was not investigated properly at necropsy.

2 VASCULAR LESIONS

In both cases ruptured arteries were found in the meninges facing the pyramids, surrounded by haemorrhages. Additionally focal infarcts and haemorrhages occurred in the distribution of perforating arteries in the medulla and the pons. These lesions are due to shearing of perforating arteries which result from the differential motility of brain stem and vessels. In cervical hyperextension the basilar and vertebral arteries lag behind in relation to the brain stem as they run in a bigger radius curve. An unusual focus of haemorrhagic infarction was observed in the choroid plexus of the 4th ventricle in our second case. These changes could be attributed to a vascular lesion but more likely result from contusion of the choroid plexus itself being squeezed

between cerebellum and medulla in severe hyperextension. This kind of lesion to our knowledge has not been reported previously.

3 RUPTURE OF NERVE FIBRES

Numerous retraction balls occurred in both cases in the medulla and pons predominantly in vertical fibre tracts. Axonal swelling can be observed in focal infarcts but their arrangement is different from disseminated retraction balls as sequel of shearing or rupture of nerve fibres. This type of brain damage in head injury was first described by Strich¹⁰ and later expanded by Adams and co-workers.⁶⁻⁸ Retraction balls in head injuries are observed predominantly in the white matter of the hemispheres, the corpus callosum, the internal capsule and the anterior cerebellar peduncles. Their occurrence in the long fibre tracts of the medulla and pons in the present cases is to be explained by the particular trauma mechanism of hyperextension and axial distention of the brain stem.

4 CLINICO-PATHOLOGICAL CORRELATION

The 6th nerve paralysis observed in both cases seems to be an important clinical sign pointing to this type of lesion. It occurred on the second day after the accident in both patients and thus excluded avulsion of the nerve. Because the ocular signs clinically are watched very carefully in cases of cranio-cerebral trauma, it is unlikely that the 6th nerve paralysis was overlooked on the first day. The cause of the nerve lesion could not be proved but might be the course of the intrapontine nerve root through the necrotic zone surrounding the tear. There was no central chromatolysis in the 6th nerve nuclei but

there was a small infarct in the nucleus of the affected side of our second case. Schneider and Johnson¹¹ reported traumatic 6th nerve paresis due to cervical hyperextension accompanied by fractures of the cervical spine and attributed nerve damage to shearing forces at the dural passage. They did not say if the 6th nerve paralysis appeared immediately after injury or somewhat later. Extension spasm and gaze deviation in the first case was probably due to shearing of nerve fibres in the pyramidal tracts and left cerebral peduncle. In the second case the deep tear entirely disconnected the pyramids from the pons and thus led to a flaccid tetraparesis. Extensor plantar responses and some reactive movements in the legs developed later. Nystagmus might have been caused by shearing of the inferior cerebellar peduncles, while the abolition of the corneal reflexes can be explained by the disconnection of the descending trigeminal root. Central chromatolysis of the 12th nerve nuclei in both cases and Wallerian degeneration of the 12th nerve root fibres in the second case suggest that paresis of the tongue might occur clinically. The respiratory centre of the medulla oblongata is situated in the reticular formation medial and dorsal to the rostral apex of the lower olives.¹² This region lies close to the caudal edge of the deep tear in the second case and is in accord with the recorded respiratory arrest and insufficient respiration although spontaneous respiration was regained later. Forensically it is of great importance to appreciate that even big lesions of the ponto-medullary junction need not be immediately fatal.

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