A rare case of hypoglossal nerve palsy complicating a head injury

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Abstract

Hypoglossal nerve palsy (HNP) may follow fracture of the base of the skull. Of the various aetiologies described, trauma is one of the least frequent. HNP can easily be missed at the time of injury and awareness of this rare complication may assist in early diagnosis. A case of HNP after a head injury associated with the loss of taste sensation on the paralysed side of the tongue and aerocoele is reported.

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Unilateral hypoglossal (XIIth) nerve palsy (HNP) is a rare complication after trauma to the base of the skull in the region of the occipital condyles. This can follow significant decelerative head injuries usually associated with road traffic accidents. There is a difficulty in diagnosing fractures of the base of the skull using conventional radiography.

In the case presented here and other documented cases,¹ although the XIIth nerve palsy was permanent, near normal clinical function returned within one year, suggesting a good prognosis for these cases of HNP.

Case report

A 44 year old driver of a vehicle was involved in a head on collision with another car in December 1994. He was brought to the accident and emergency (A&E) department in a very confused state with a Glasgow coma score of 12/15. He sustained a head injury with a deep laceration in the occipital area with exposure of skull and a small laceration in the right upper eyelid. There was no documentation of bleeding or cerebrospinal fluid leak from the nose or ears. The airway was clear and the vital signs were normal. Apart from swelling and tenderness around his right shoulder there were no associated injuries and no reported neurological deficit. Radiography of the skull showed a long linear fracture of the left occipital bone extending upwards from the base of the skull (fig 1), and also a collection of air in the frontal area (aerocoele) (fig 2). Computed tomography revealed numerous locules of intracranial air, the largest being on the frontal lobes (figs 3 and 4), and also confirmed the presence of a linear fracture extending from the foramen magnum up to the level of the internal occipital protuberance.

The patient was treated conservatively in a neurosurgical unit and eight days later he was transferred back to Grimsby Hospital and noticed to have a slurred speech.

Examination of the oral cavity revealed the presence of wasting with fasciculation of the left side of the tongue with a bite mark on the weak side of the tongue. The patient was able to move the tongue but it deviated to the left side on protruding from the mouth (fig 5). Taste sensation (sweet, sour, and salt) was lacking on the left, wasted, side of the tongue but normal on the opposite side. Protective sensation was normal on both sides of the tongue. The patient did not experience any difficulty with swallowing.

The patient was treated conservatively and within six months of the injury he had regained a near normal speech in spite of the persistent wasting of the left side of his tongue and his

Figure 1 Linear fracture of the left occipital bone.

Figure 2 Aerocoele on lateral view of skull.

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Figure 3 Computed tomography, sagittal scan, showing free air between frontal bones and frontal lobe of brain.

Figure 4 Computed tomography, axial scan, showing locules of intracranial air, mainly on the frontal lobes.

Figure 5 Paralysis of the left side of the tongue with wasting; the tongue deviates to the left (paralysed) side on protruding from the mouth.

taste sensation in the left side of the tongue had not recovered.

Discussion

Unilateral HNP is a rare complication after trauma to the base of the skull in the region of the occipital condyles. Such a complication has been observed after significant decelerative head injuries usually associated with road traffic accidents. This complication can be missed

at the time of injury and may even present up to several months after injury.¹

There are many causes of isolated HNP and associated muscle wasting is a feature of lower motor neurone lesions. Common causes include carotid endarterectomy (usually temporary), tonsillectomy, tooth extraction, adenoid cystic carcinoma of the tongue, metastatic malignant tumours of the middle and posterior cranial fossae, syringomyelia, neck tumours, and surgery. Vertical subluxation of the odontoid process in rheumatoid arthritis has also been reported to cause HNP. Cranial base trauma in the region of the occipital condyles as a result of sudden deceleration head injuries may also result in HNP. In many of these accidents, there may be other significant brain injury and HNP may be bilateral. These bilateral palsies have poorer prognoses for functional rehabilitation compared with the unilateral palsy.¹

When the sole clinical deficit is an isolated unilateral HNP of late onset, the existence of posterior cranial base fracture often goes undetected.

Bilateral HNP is very rare and of the various aetiologies described, trauma is one of the least frequent. The diagnosis can easily be missed in the initial assessment of traumatised patients after severe decelerative injuries in road traffic accidents and subsequently noted after extubation and discharge from the intensive care unit.

The hypoglossal nerve emerges from the medulla oblongata as a series of rootlets between the pyramid and olive, passes laterally and superior to the vertebral artery leaving the cranial cavity through the hypoglossal canal of the occipital bone superior to the occipital condyle. The hypoglossal nerve innervates all the ipsilateral intrinsic and extrinsic muscles of the tongue except the palatoglossus.² In trauma, the forces involved in high speed deceleration head injuries can cause fracture of the occipital condyle, thereby injuring the hypoglossal nerve. In the case reported here, a fracture line was demonstrated running from the foramen magnum on computed tomography and is thought to have caused damage to the hypoglossal nerve, although the fracture line was not seen running through the hypoglossal canal. Posterior cranial base fractures are extremely difficult to demonstrate on plain radiographs and computed tomography is essential to confirm the diagnosis.²

The XIIth nerve may have also been injured indirectly at the time of impact. Severe neck hyperextension at the time of injury can cause traction and elongation of the nerve (produced by subluxation of the atlantoaxial articulation or occipital condyle trauma) but this mechanism of injury is unlikely because the patient did not have any symptoms suggestive of neck injury.

To explain the late onset of some HNP it is thought that the healing process and subsequent callus formation may play a part in obliterating the hypoglossal canal.

Bilateral HNP causes dysphagia, dysarthria, and a non-mobile tongue. No tongue movements can be made on attempted protrusion.

In the case presented by Paley and Wood,² sensation of the tongue was found to be normal and the taste sensation was found to be unaltered, although the tongue was not mobile. Swallowing was found to be difficult and videofluoroscopy demonstrated abnormality of the oral phase of swallowing due to lack of tongue function. The patient could only manage fluids and semisolids with the aid of gravity. Electromyographic studies may be useful in showing evidence of bilateral denervation of the tongue compatible with bilateral axonotmesis of XIIth nerve.³

In our case, only one half of the tongue was paralysed and this patient was able to use his tongue. He could move and protrude his tongue and although his tongue tended to be pushed to the paralysed side (by the normal side of the tongue), he did not have any problem with swallowing, although his speech was slurred. Examination of the tongue also revealed a rare finding of loss of taste sensation to sweet, salt, and sour on the paralysed side of the tongue after injury.

ANATOMICAL AND PHYSIOLOGICAL BACKGROUND

The sweet taste is localised principally on the anterior surface and tip of the tongue, the salty and sour taste on the lateral side of the tongue, and bitter taste on the circumvallate papillas on the posterior surface of the tongue. Taste impulses from the anterior two thirds of the tongue pass first into the lingual branch of the maxillary nerve, which is the third division of the trigeminal nerve. Impulses then pass through the chorda tympani into the facial nerve and then to the tractus solitarius in the brain stem.⁴

The chorda tympani is given off from the facial nerve as it passes vertically downwards at the back of the tympanum, about a quarter of an inch before its exit from the stylomastoid foramen. It passes forwards through the cavity of the tympanum and emerges from that cavity through a foramen (canal of Hunguier) and then descends between the two pterygoid muscles and meets the gustatory (or lingual) nerve at an acute angle.

In our patient, there was no facial nerve palsy and so the facial nerve itself was intact. The paralysed half of the tongue had not lost the protective sensation and so to explain the loss of taste sensation the patient most probably had damaged the chorda tympani nerve after it left the facial nerve and before joining the lingual nerve.

Another interesting feature was the presence of an aerocoele; the entry of air into the cranial

cavity usually occurs in association with cerebrospinal fluid rhinorrhea and indicates a tear of the dura (usually basal) and a fracture involving the paranasal sinuses (frontal, ethmoid, or spheroid). This complication occurs particularly if the patient blows his nose.^{5 6}

This unusual combination of cranial nerve palsies was not reported in the case reports reviewed in this paper. This complication did not cause any major concern to the patient because taste sensation was intact elsewhere in the tongue. Both tongue paralysis and the loss of taste sensation have not resolved on follow up and indicate permanent nerve damage. In spite of that, functional recovery was satisfactory by compensatory mechanisms.

This combination of cranial nerve palsies represents a rare complication of fracture to the base of the skull, which can easily be missed at the initial presentation of these patients to A&E departments when the efforts of trauma teams are, quite rightly, focused on the more serious aspects of injury.

In this case, the presence of slurred speech was the first indication to examine the oral cavity and the tongue in particular. In head injured patients, examination of the cranial nerves should not be overlooked once the initial trauma management has been carried out and awareness of such cranial nerve palsies would facilitate early diagnosis.

There is no specific treatment for XIIth nerve palsy and the lesion usually improves spontaneously by compensatory mechanisms.

Decompression of the injured cranial nerves has been suggested, but it seems that there is no indication for this. If the nerve injury does not recover, it is likely that the mechanisms of injury have been a combination of traction and crushing which will not be helped by decompression and patients often make good functional recovery.⁷

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