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CASE REPORT

Vertical atlantoaxial dislocation

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Abstract An unusual case of vertical atlantoaxial dislocation without medulla oblongata or spinal cord injury is reported. The pathogenic process suggested occipito-axial dislocation. The case was treated surgically with excellent results on mobility and pain.

Key words Cervical spine injury · Atlantoaxial dislocation · Atlantoaxial arthrodesis

Case report

Dislocations of the first two cervical vertebrae are rare and very often fatal. We report a case of vertical atlantoaxial dislocation with no neurological signs and with a favourable outcome.

A 62-year-old man was driving his car with his seat belt fastened when he had a violent head-on collision with a farming machine at the level of the plough. Axial traction responsible for hyperextension of the cervical spine and stretching of the left brachial plexus occurred. On arrival at the emergency unit, he had respiratory distress and blood aspiration due to a facial trauma. The Glasgow score was 3 and a CT scan of the brain showed mild intraventricular bleeding on the right.

Plain radiographs of the cervical spine disclosed isolated C1-C2 dislocation, with a vertical C1-C2 gap exceeding 20 mm (Fig. 1). An emergency arteriogram of the aortic arch showed no abnormalities (Fig. 2).

Immediate external reduction was carried out by applying firm pressure along the axis of the head under fluoroscopy (Fig. 3). Post-reduction stability was assessed during small flexion-extension motions.

Tracheotomy and reconstructive maxillofacial surgery were immediately carried out, allowing secondary spinal stabilisation using a posterior approach in the prone position. Traumatic dura mater tears were identified and treated by biological glue. Posterior atlantoaxial fixation was performed using a combination of atlantoaxial grafting and wiring.

Temporary fixation using an occipital-C4 plate (Howmedica) was performed (Fig. 4) to reduce the need for external immobilisation (cervical brace only). On day 9, an infection secondary to an occipital pressure sore was diagnosed. Microbiological studies of cerebrospinal fluid obtained by lumbar puncture were negative. On day 11, a revision was performed, but the fixation material was left in place.

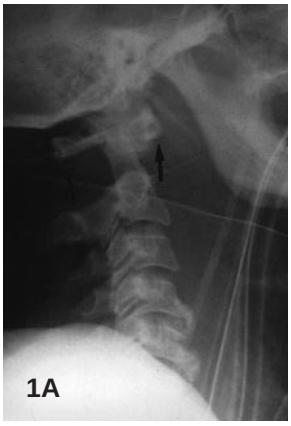
The patient was discharged from the intensive care unit after 3 months. He was still on antibiotic therapy, but had no tracheotomy or evidence of local infection. He had a persistent neurological deficit in the left upper limb, shown by electromyography to be secondary to the brachial plexus stretching. There was no evidence of spinal cord injury.

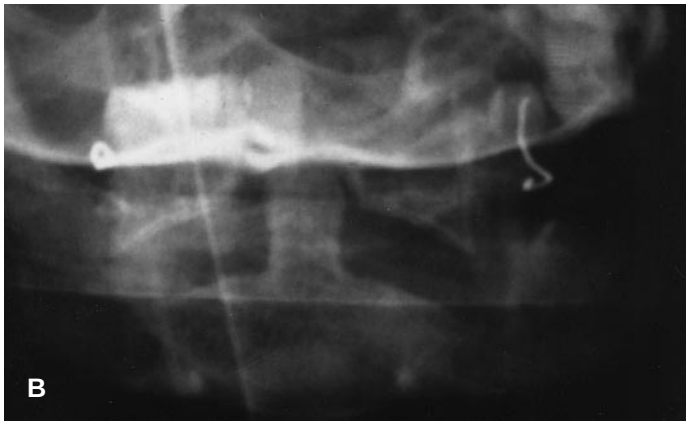
One year later, the occipito-cervical plates were removed. Range of motion in all planes was excellent and the patient was fully pain free. The residual neurological deficit in the left upper limb was very mild (muscle score at 4+).

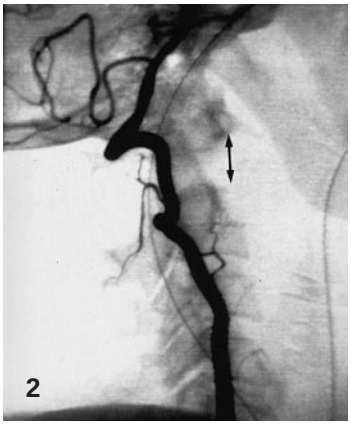
Discussion

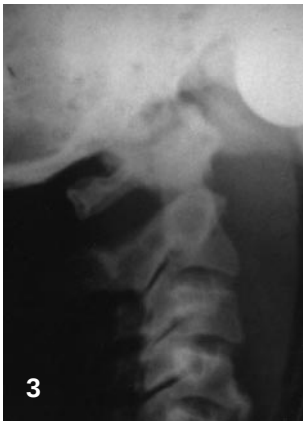
We found no similar case of vertical atlantoaxial dislocation in the literature [2, 10]. Furthermore, our clinical case calls for comments regarding three important points, namely:

1. Interpretation of the lesion-inducing process
2. Patient status and absence of medulla oblongata or spinal cord neurological signs
3. Surgical strategy









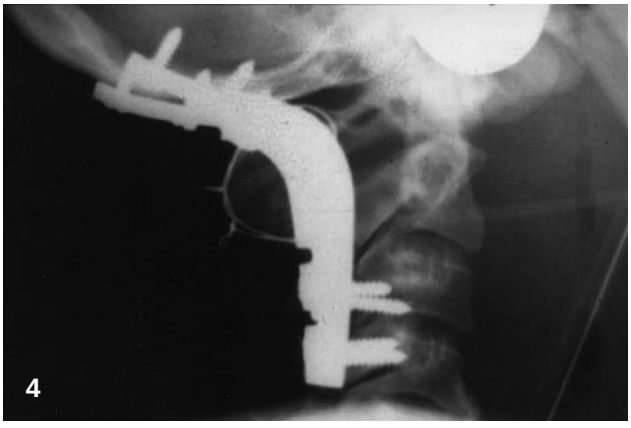


Fig. 1A, B Initial standard radiographs. Lateral (**A**) and frontal (**B**) views

Fig. 2 Vertebral artery condition prior to reduction of dislocation: arteriogram was normal on both sides

Fig. 3 Lateral radiograph following reduction of the dislocation

Fig. 4 Postoperative radiograph, lateral view

Interpretation of the lesion-inducing process

The traumatic mechanism was similar to that responsible for occipito-cervical dislocation, i.e. hyperextension with traction and rotation [3, 5–9] combined with a cranio-facial injury [6, 11]. Stability of the atlanto-occipital and atlantoaxial segments is maintained by a complex ligamentous system [4], including two separate components, namely: a central, intraspinal, occipito-axial component and a peripheral atlanto-occipital component.

Werne [15] demonstrated that resistance to rotational forces is ensured only by occipito-axial factors: occipito-axial dislocation was produced experimentally by severing the membrana tectoria and alar ligament, indicating that these two ligaments act as the “fulcrum” of the occipito-cervical junction.

Injuries to the atlantoaxial segment are generally classified in two groups: transversal instabilities and rotational dislocations.

In a study by Bouchez and Camelot [2], atlantoaxial side-to-side instabilities and dislocations involved either injury of the transverse ligament or an odontoid abnormality. Most of the 23 injuries reported in this study were mild, and half were responsible for neurological abnormalities.

Rotational dislocation involving both lateral masses were observed in patients with an intact transverse ligament. Injury to the transverse ligament resulted in a one-sided dislocation with an increase in the distance between the odontoid process and the anterior aspect of the atlas.

Our patient had a pure vertical dislocation between the atlanto-occipital complex and the axis. No anteroposterior translation indicating transverse ligament disruption was seen on radiographs or during the fluoroscopic anteroposterior stability study. Moreover, there was no radiological evidence of rotational dislocation.

Patient status and absence of medulla oblongata/spinal cord signs

The bradypnoea and severe respiratory distress syndrome observed initially in our patient were the same as those reported in occipito-cervical dislocations [1].

Our case is evidence of the considerable ability of the spinal cord and blood vessels to tolerate pure stretching. In contrast, occipito-cervical dislocation results in compression of the spinal cord by sagittal translation. The severity of the initial distraction raised concern regarding possible vascular injury, especially involving the part of the vertebral artery that runs through the C1 and C2 foramina. However, an arteriogram delineating all the

branches from the aortic arch was normal, attesting to the considerable elasticity of blood vessels.

Surgical strategy

Following reduction of the dislocation, a high degree of stability was obtained in all planes for small motions. The lesion was interpreted as a pure ligamentous injury, similar to a severe cervical sprain [12–14].

The emergency setting led us to choose posterior atlantoaxial arthrodesis, since this allowed strong fixation. In addition, temporary occipital-C4 fixation was performed to improve stability, thus facilitating nursing in the intensive care unit. Early revision after the diagnosis of infection showed that it was possible to maintain the internal plate fixation. After complete fusion was obtained, the plates were removed, thus allowing recovery of an excellent range of motion.

Conclusion

Vertical atlantoaxial dislocation is rare and severe. Immediate reduction can be achieved by axial compression. Posterior stabilisation by C1-C2 wiring and grafting should be considered after reduction is obtained. Temporary occipito-cervical plates are an effective means of providing strong complementary stabilisation.

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