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CASE REPORT

Traumatic posterior atlantoaxial dislocation with associated C1 Jefferson fracture and bilateral vertebral artery occlusion without odontoid process fracture or neurological deficit

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Abstract

Purpose Traumatic atlantoaxial dislocation (AAD) is usually associated with fatal high-velocity road traffic accidents (Xu et al. in Medicine (Baltimore) 94:e1768, 2015). There are few reports of survival following posterior AAD without odontoid fracture (Xu et al. 2015; Zhen et al. in Arch Orthop Trauma Surg 131:681–685, 2011; de Carvalho and Swash in Handb Clin Neurol 119:435–448, 2014).

Method We present a previously undescribed case of posterior AAD associated with a C1 Jefferson fracture but no odontoid fracture and bilateral vertebral artery occlusion without neurological deficit.

Conclusion The presence of bilateral vertebral artery occlusion raised challenges in the surgical management. Survival was only possible due to the presence of robust cerebral collateral circulation.

Keywords Posterior atlantoaxial dislocation · C1 Jefferson fracture · Vertebral artery occlusion

Case report

A 71-year helmeted horse rider was admitted to the emergency department having been struck by a car and thrown to the ground, sustaining an axial compression and hyperextension injury of the spine. There was no loss of consciousness. He was able to stand following the impact, but complained of neck pain and a transitory sensory disturbance affecting the right upper and lower limbs and left upper limb. There was no reported weakness. He was immobilised at the scene with full spinal precautions by paramedics. Apart from mild rheumatoid arthritis, he was previously fit and well.

His primary advanced trauma and life support (ATLS) survey revealed high cervical spine tenderness with no bruising or palpable deformity. Neurological examination was normal. Secondary survey revealed tenderness in the mid thoracic spine and facial abrasions.

A CT trauma study as per the major trauma centre protocol demonstrated a fracture of the anterior arch of C1 with anterior displacement of the intact odontoid process ventral to the ring and associated soft tissue swelling to the upper cervical spine. There were bilateral displaced fractures of the posterior arch of C1 (Fig. 1). There was a T3 compression fracture with no disruption of the posterior elements. There was no significant injury to the head, chest, abdomen or pelvis.

The patient was admitted to the intensive care unit for close monitoring. After 4 h, he deteriorated with upper airway obstruction due to retropharyngeal soft tissue swelling necessitating intubation and ventilation. The next day an MRI/MRA study demonstrated normal craniocervical alignment, which was attributed to spontaneous reduction under general anaesthesia, with no evidence of cord compression. There were signs of cord signal change and significant ligamentous injury associated with bilateral VAO at the level of C2 with retrograde filling of the vertebro-basilar circulation via posterior communicating arteries (Fig. 2).

The following day an instrumented C1–2 stabilisation was undertaken using C1 lateral mass screws, C2 pedicle screws and intraarticular autologous bone grafting. After prone positioning, lateral fluoroscopy confirmed spontaneous reduction in the AAD so that exploration of the vertebral arteries was considered unnecessary.

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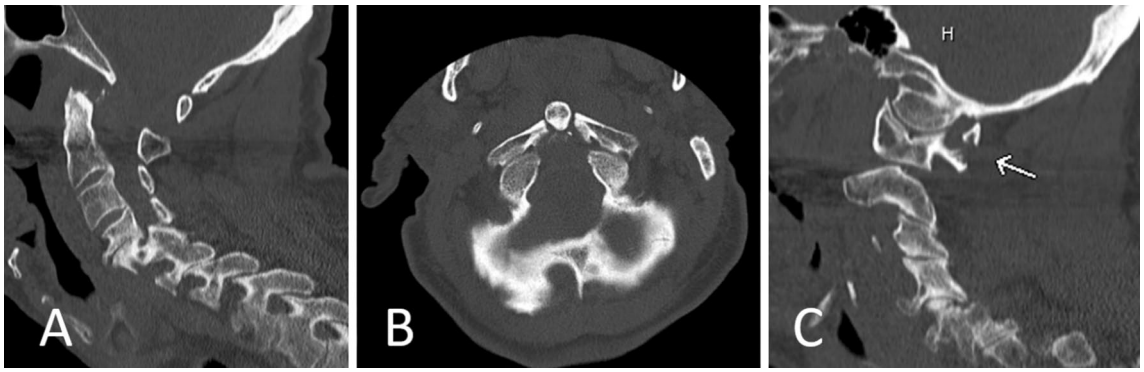

Fig. 1 CT Cervical spine, mid-sagittal (**a**), axial (**b**) and parasagittal (**c**). The intact odontoid process has displaced through the anterior arch of atlas. There is posterior displacement of C1 on C2, consistent with a type IV atlantoaxial subluxation. There is also a fracture to the posterior ring of the atlas

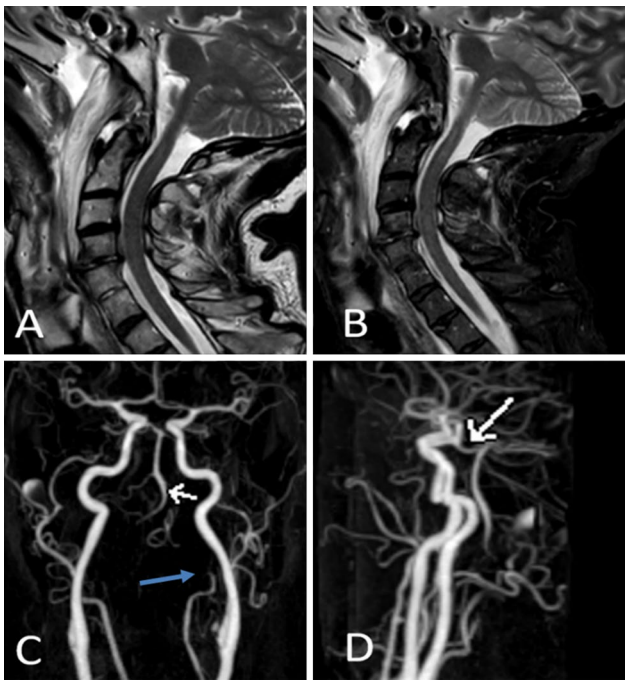

Fig. 2 MRI cervical spine, mid-sagittal T2 weighted (**a**), mid-sagittal short tau inversion recovery (STIR) (**b**), anteroposterior view of 3D MR angiography (**c**), and lateral view of 3D MR angiography (**d**). There has been spontaneous reduction in the atlantoaxial dislocation. There is high signal within the cervical spinal cord as it passes posterior to the odontoid process and through the foramen magnum extending as far as C7, in keeping with contusion. There is no ongoing spinal cord compression. In (**c**) there is bilateral occlusion of the vertebral arteries at the level of C2 (blue arrow) with retrograde filling of the basilar and posterior inferior cerebellar arteries (white arrow). In (**d**) there is good collateral circulation via robust posterior communicating arteries (white arrow)

The patient made an uncomplicated recovery from the operation. Post-operative CT scanning confirmed good craniocervical alignment with satisfactory instrumentation (Fig. 3). He was commenced on 75 mg aspirin for 3 months to reduce the risk of a posterior circulation thromboembolic event. He was mobilised wearing a suboccipito-mental collar.

The patient's post-operative course was complicated by dysphagia, caused by the soft tissue swelling, vagal neuropraxia and muscular deconditioning. This improved with regular speech and language therapy. He suffered two transient episodes of sudden onset dysarthria, with no associated weakness or dysphasia. MRI head showed no evidence of cerebral infarction. A diagnosis of transient ischaemia was made, and he was commenced on 75 mg once a day clopidogrel for 3 months.

Discussion

There are several unusual features of this case including the injury pattern, the associated vascular injury with no neurological deficit and the surgical strategy to restore alignment and stability and avoid thromboembolic events.

Injury pattern

AAD is an uncommon but clinically important condition due to the severity of neurological deficits [1, 2]. The stability of the atlantoaxial complex is provided by the articular processes of C1 and C2, and the osteoligamentous ring that encircles the odontoid process. Conditions that predispose to AAD by weakening these structures include Downs syndrome, rheumatoid arthritis, Paget's disease and other metabolic bone disorders [3].

Traumatic AAD is usually a result of distraction injuries with superimposed hyperflexion or hyperextension. The typical pattern is anterior displacement of the atlas on the intact axis, with or without fracture to the process depending on integrity of the transverse ligament. There are 19 reported cases in the literature of posterior traumatic AAD without

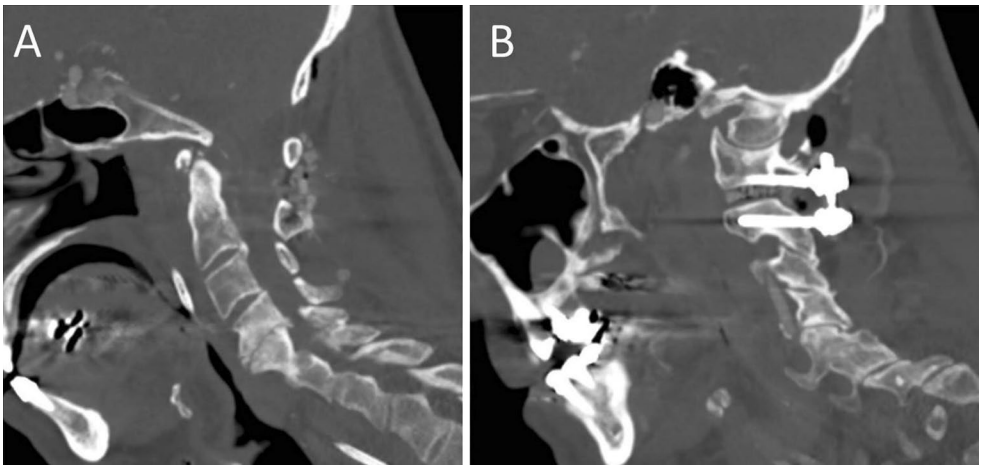

Fig. 3 Post-operative CT cervical spine, mid-sagittal (**a**) and lateral sagittal (**b**). There is reduction in the posterior atlantoaxial dislocation, with lateral mass screws to C1 and pedicle screws to C2. There is autologous bone grafting to the interfacet space

odontoid process fracture [4]. These have typically been the result of distraction hyperextension injuries with associated facial injuries and are not associated with significant neurological deficit. It is postulated that posterior ADD without odontoid fracture may be more common but normally leads to immediate death. In cases that do survive, there is little or no damage to the spinal cord, as the remaining ligamentous structures lock the axis and prevent further displacement, reducing the spinal canal area to approximately 36% [5].

In this case, we speculate the background inflammatory condition predisposed to weakening of the C1 arch and apical and alar ligaments, with relative preservation of the integrity of the odontoid process and transverse ligament. Thus, the craniocervical junction was particularly susceptible to an axial loading and hyperextension injury, generating this atypical form of posterior AAD comprising anterior C1 arch and Jefferson fractures. To our knowledge, this is the first report of this unusual injury pattern.

Associated vascular injury with no neurological deficit

VAO is a rare complication of blunt trauma to the craniocervical junction. It has also been observed following chiropractic manoeuvres [6, 7] and cervical spine surgery. VAO may result in immediate ischaemia and also lead to thromboembolic events causing posterior circulation and spinal cord infarction of varying severity. The prevalence of asymptomatic VAO and clinical sequelae following VAO are not known. Risk factors for ischaemic stroke in the presence of VAO include increasing age and bilateral VAO. Corrective cervical spine surgery potentially decreases the risk by reducing motion across the occluded segments [8].

There is a case report of a patient sustaining a traumatic AAD with associated bilateral VAO and carotid artery injuries [9]. This patient had a type IIA traumatic spondylolisthesis at C2 and C1/2 distraction. The vascular injury was managed by anticoagulation 48 h after surgical treatment. The patient made a reasonable neurological recovery.

In our case the patient remained neurologically intact due to the presence of robust collateral circulation via the posterior communicating arteries. Without this, the injury would have been fatal. Acute post-traumatic VAO presents an ongoing risk of thromboembolic stroke. Antiplatelet treatment and/or anticoagulation following surgery is advocated, but the duration of medical treatment and the role of follow-up vascular imaging are controversial. Our patient has been followed up for a year and remains well with no further episodes of dysarthria.

Surgical strategy

Most cases of posterior AAD have successfully been managed with closed reduction, although it is technically challenging to relocate the odontoid process back into osteoligamentous ring. On-table fluoroscopy, with spinal cord monitoring if the patient is under a general anaesthesia, is recommended. The procedure is terminated if there are any signs of spinal cord compromise due to over-distraction. Of the 19 previously described cases, successful closed reduction was followed by internal fixation in five patients and conservative management in 6. The remaining eight patients were treated by open reduction and internal fixation, including 4 that developed neurological deficits during attempts at closed reduction [4]. Most cases were stabilised posteriorly with wiring, transarticular screw fixation or lateral mass and pedicle screw fixation. Anterior approaches include transoral

odontoidectomy and the anterior retropharyngeal approach. They have the advantage of avoiding prone position and the possibility of further craniocervical displacement [10].

In this case, we initially considered that early operative reduction in the posterior AAD would be associated with a high risk of vertebral reperfusion and distal thromboembolism. Our intended strategy was to isolate and temporarily clip the vertebral arteries before reducing the fracture. Spontaneous reduction in the AAD under general anaesthesia preoperatively made this unnecessary. At operation, the vertebral arteries were insonated with a microvascular Doppler to confirm persisting occlusion. A standard posterior C1–C2 fusion was preferred over an occipito-cervical fixation as there was normal occipital-C1 articulation.

Conclusion

We describe a case of traumatic posterior AAD with associated Jefferson fracture and bilateral VAO without odontoid process fracture or neurological deficit. This variation of AAD has not previously been reported in the literature. We postulate that this injury pattern would usually be fatal and our patient survived due to excellent collateral circulation. Challenges to management include surgical approach and timing, and management of the associated vascular injury.

Compliance with ethical standards

Conflict of interest None of the authors has any potential conflict of interest.

Informed consent Informed consent was obtained from all individual participants in this case report.

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