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Case Report

Atlantooccipital dislocation in motor vehicle side impact, derivation of the mechanism of injury, and implications for early diagnosis

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Abstract

Numerous reports of atlantooccipital dislocations (AODs) have been described in frontal impacts and vehicle versus pedestrian collisions. Reports of survival after AOD in conjunction with side impacts have infrequently been reported in the literature. The objective of this study is to present a case of an AOD from a side impact vehicle collision, and deduce the mechanism of injury. A clinical and biomechanical reconstruction of the collision was performed to investigate the mechanism of the dislocation. A 51-year-old female was traveling in a four-door sedan and sustained a side impact collision with a compact pickup truck. At the time of extrication, the patient was neurologically intact with a Glasgow Coma Scale score of 15. After admittance to the hospital, the patient developed a decline in respiratory status, right mild hemiparesis, and left sixth-nerve palsy, and magnetic resonance imaging (MRI) and computed tomography (CT) reconstructions indicated a craniocervical dislocation. Surgical fixation was performed and all extra-axial hemorrhaging was evacuated. At discharge, the patient was neurologically intact on the left side, had right mild hemiparesis, left sixth-nerve palsy, and minor dysarthria. Survival rates of AODs have recently been increasing. Morbidity is still more prevalent, however. Due to the variety of symptoms that accompany AODs and the inconsistency of diagnostic imaging techniques, a thorough history of the etiology may lead to increased clinical suspicion of this injury and further raise survival rates.

Key words: Atlantooccipital dislocation, cervical spine injury, side impact collision

INTRODUCTION

Systematic diagnosis of atlantooccipital dislocation (AOD) has been described by Powers *et al.* based on X-rays.[1] Reports of adult AOD have been commonly described in front-end motor vehicle collisions with unbelted occupants and airbag interactions.[2,3] To the knowledge of the authors, only one trauma patient has been reported in the literature who survived AOD during a side impact collision.[4] Reported AODs in the literature suggest that the presence of associated injuries should lead to a high index of suspicion.[5] Cranial nerve deficits are common among such injuries.[1,6,7] We present a rare case of a side impact collision resulting in AOD with related cranial nerve deficits.

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CASE REPORT

Presentation

The patient was a 51-year-old female, and was of 165 cm in stature and 101 kg in total body mass. She was the driver of a four-door sedan stopped at a controlled intersection facing north. As the patient began a left turn into the west bound lane, an east bound half-ton pickup truck entered the intersection and collided with the driver side door of the case patient's vehicle.

The patient was restrained in a three-point seatbelt and the front driver-side airbag did not deploy.

Upon extrication, the patient had a Glasgow Coma Scale (GCS) score of 15, and she was immobilized in a hard cervical collar and transported to a local hospital. From there, she was airlifted to our Adult Level One Trauma Center, with a stabilized GCS score of 15. Twenty-four hours after admission, the patient developed mild dysarthria and bradypnea. After decline in respiratory status, the patient was intubated and immobilized in a halo brace. Diagnostic radiograph, magnetic resonance imaging (MRI), and three-dimensional computed tomography (3D CT) images were obtained. The patient also developed a left cranial nerve VI palsy and quadriparesis at this time.

Radiographic examination

Plain X-ray radiographs indicated an anterior AOD with a Powers Ratio 1.1 [Figure 1][1]. The AOD was confirmed with CT scans [Figure 2], and imaging of the brain revealed a subarachnoid hemorrhage in the posterior fossa. Intraventricular blood was noted in the fourth ventricle and prepontine cisterns. MRI revealed a cervical cord contusion from the occiput to C2, and an epidural hematoma in the tectorial membrane and alar ligaments at the C1–C2 level [Figure 3]. Angiography findings indicated intimal tears of both cervical vertebral arteries and rupture of the left external carotid artery.

Management

The patient was taken to the operating room, general anesthesia was administered, and fiber-optic intubation was performed. The patient was turned to the prone position with the halo vest in place. The posterior portion of the vest was removed and occipitocervical alignment was verified by intraoperative fluoroscopy. The occiput and upper cervical spine were exposed through a standard midline exposure. The atlas was completely dislocated from the occiput, and disruption of all atlantooccipital ligaments and capsule was seen. Rupture of the alar ligaments, tectorial membrane, and inferior portion of the vertical cruciate ligament was also noted at C1–C2 level. The epidural hematoma was evacuated bilaterally from around the spinal cord at the occiput–C2 level. C2–C3 joints were intact. An occiput–C2 fusion [Figure 4] was performed using the Summit (Acromed/Johnson and Johnson, Raynham, MA, USA) system. This included placement of three screws into the occiput, sublaminar wires beneath the arches of the atlas and across the lamina of C2 bilaterally.

Postoperative course

The patient was positioned in a halo vest and monitored in the intensive care unit. Her quadriparesis partially resolved into a residual mild right-side hemiparesis. The sixth-nerve palsy on the left side did not improve. Significant diplopia was noted initially; however, this gradually resolved over the treatment course. At the time of discharge, the patient was neurologically intact on the left side of her body. The residual right-side hemiparesis persisted, as well as the left sixth-nerve palsy and minor dysarthria.

DISCUSSION

Reconstruction of the case collision

While survival after AOD in a side impact collision has been reported, there is a lack of mechanistic understanding of the mechanism of injury.[4,8] The present study adds to the body of literature and emphasizes the occurrence of AOD caused by vehicle-size mismatch. Investigation of the collision determined that the interior of the sedan door and parts of the truck grille struck the left side of the occupant during impact. The left shoulder and rib cage of the patient were forced transversely toward the midline and downward. During the time of vehicle-to-vehicle contact, the truck hood and upper regions of the grille were above the driver side window sill. Using an exemplar

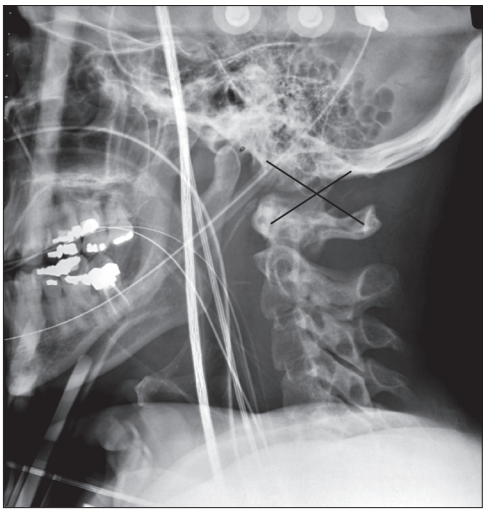

Figure 1: Lateral radiograph of the cervical spine and illustration of the Powers Ratio demonstrating an anterior dislocation of the atlantooccipital joint

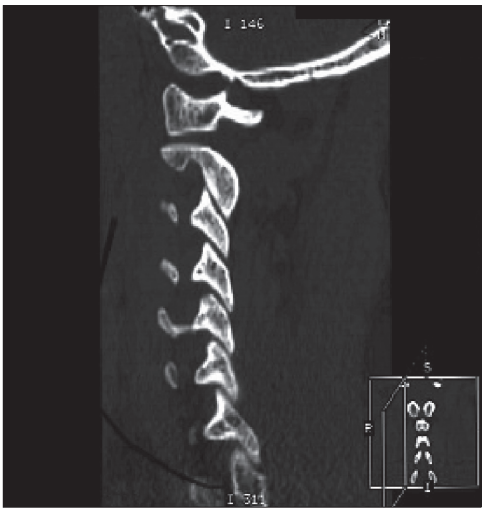

Figure 2: Lateral CT reconstruction showing atlantooccipital dislocations. Note the abnormal width of the atlantooccipital joint and the misalignment of the articulating surfaces

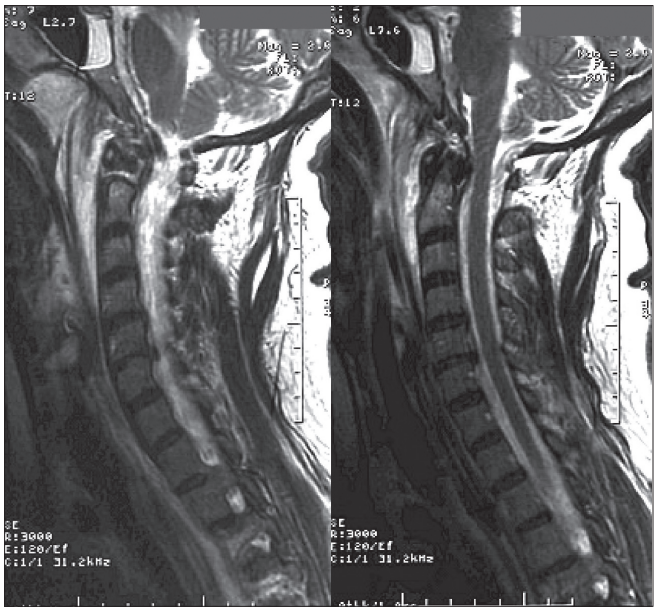

Figure 3: MRI of the spinal cord contusion and epidural hematoma are shown

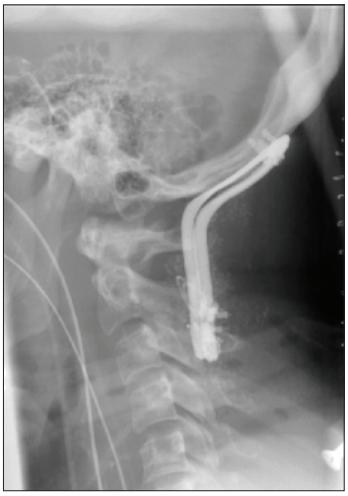

Figure 4: Postoperative image showing the surgical procedure

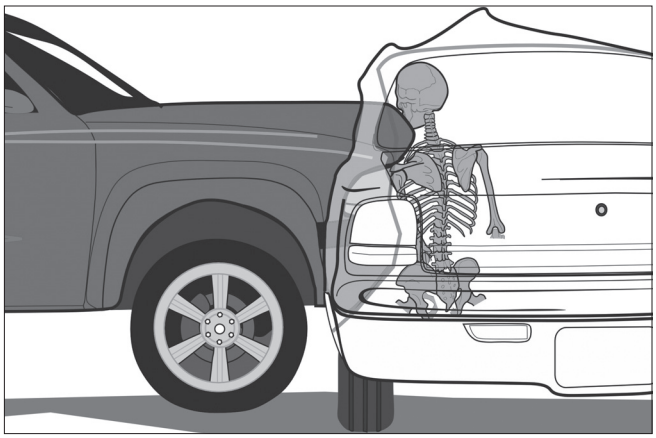

Figure 5: The schematic illustrating the mechanism of the atlantooccipital dislocations in side impact

occupant and vehicle, it was verified that the impacting truck's hood front edge was at the level of the patient's craniocervical junction. Further analysis based on her anthropometry indicated that the level of her skull base and mandible was maintained against the truck hood as the left shoulder and rib cage were fractured and continuously forced transversely and downward. The impact produced a distractive force along the length of the spine and induced tensile forces to the ligaments of the upper cervical spine, resulting in AOD [Figure 5].

Clinical evidence for the injury mechanism

Several reports describe various injuries in combination with AOD. Few specific symptoms have been observed.[7,9-11] Diagnosis is usually based on associated injuries and

Table 1: Associated injuries separated into body region and organs

Injuries	Head and neck	Thorax/ abdomen
Osseous/ligamentous	Subluxation of C1 and C2; complete rupture of the atlantooccipital capsule; complete rupture of tectorial membrane and inferior portion of vertical cruciate ligament; complete rupture of alar, anterior longitudinal, posterior longitudinal, and transverse ligaments	Fractures of left posterior 1–5 and right anterior 1–2 ribs
Vascular	Left conjunctival hemorrhage; left facial laceration (6 cm); contusions to left side of the neck; subarachnoid hemorrhage within posterior fossa; intraventricular blood in fourth ventricle and prepontine cisterns; left external carotid artery tear; bilateral intimal tears of the cervical vertebral arteries; epidural hematoma at C1–C2, anterior and bilateral to the spinal cord; epidural hematoma to tectorial membrane and alar ligaments	Left lung contusion; left side hemothorax; grade 1 liver laceration
Neurological	Left sixth-cranial nerve palsy with resultant diplopia; mild dysarthria; cervical cord contusion with quadriparesis	Residual right mild hemiparesis

a high degree of clinical suspicion.[4,7,10] A cervical spine distraction mechanism resulting in AOD is often associated with facial abrasions and lacerations, mandible fractures, arrhythmia, apnea, asymmetric cranial nerve palsies, loss of consciousness, subarachnoid hemorrhages, and thoracic or abdominal injuries.[1,4,5,7,9-12] Our patient sustained a number of these associated injuries [Table 1]. Limited trauma below the abdomen included a grade 1 liver laceration caused by the seatbelt. Further, evidence of trauma was noted at the upper left side of her thorax, cervical spine, and cranial regions. The initial radiographs showing fractures of the left first five ribs, with hemothorax and concomitant left lung contusion were strong indications that the maximum load of impact was at the upper thoracic region.

Embedded glass was found in the left facial laceration. A left conjunctival hemorrhage was noted in addition to substantial swelling and bruising on the left side of the patient's face, neck, and shoulder. Angiographic findings of the left external carotid artery tears indicated a substantial amount of force, applied to the cervical spine, during impact.

As indicated by MRI, complete rupture of the inferior vertical cruciate ligament, alar ligaments, tectorial membrane, and the entire atlantooccipital ligament complex further localized the maximum load of impact to the high cervical spine. A considerable amount of force is required to rupture the vertical cruciate ligament and the tectorial membrane which suggested a subluxation at the C1–C2 level.[13] This was a strong indication that majority of the force was directed to the upper cervical spine.

Biomechanical studies of cervical spine ligament tensile strength have revealed that the ligaments of the OC–C1 junction require a high force for complete rupture.[13,14] Thus, the resultant AOD and rupture of the ligament complex in this patient substantiated the proposed distraction mechanism. Upon comparison, cervical spine radiographs did not reveal any apparent differences from other AODs caused by frontal impact. Thus, it can be concluded that the maximum load of impact was focused at the atlantooccipital junction of our patient.

Analysis of brainstem and cranial nerve deficits in atlantooccipital dislocations

Asymmetric motor deficits are often caused by direct spinal cord injury or compression of the brainstem through damage to the surrounding bony or vascular structures.[6] Delayed onset, with rapid progression, of neurologic deficit indicates that symptoms are due to compression and not direct injury. Based on the time of onset of specific neurological deficits in our patient, diagnostic angiography was used to investigate the surrounding vasculature. Intimal tears of both vertebral arteries (consequent to dissection) provided one plausible explanation for the dysarthria, bradypnea, and quadriparesis that occurred in a delayed fashion.[15-18] Compression of cranial nerve X in the same region was associated with the bradypnea.[16-18] The cranial nerve VI palsy was not immediately explained by the angiographic findings.

Analysis of the cervical MR scans revealing the anterior and bilateral epidural hematoma and the spinal cord contusion provides the basis for quadriparesis and a further explanation for the decline in respiratory status. Compression and stretching of the spinal cord was notably influenced by displacement of the occipital condyles from the atlas, which resulted in the spinal cord contusion. The epidural hematoma found in the tectorial and alar ligaments along with the ruptured atlantooccipital ligament complex and capsule probably allowed swelling in the region to further compress the brain stem and cranial nerves.[9,11,12,19]

Damage to vertebral and carotid arteries, and vascular structures within the ligament complexes probably resulted in the subarachnoid hemorrhage in the posterior fossa, and the intraventricular blood in the fourth ventricle and prepontine cisterns.[12,19] It is likely that blood in these regions created pressure on the nucleus of the sixth-cranial nerve, and the nuclei of the hypoglossal and vagus nerves.[19,20] The resultant abducens nerve palsy and dysarthria may be explained by these observations.

Additional support for our hypothesis is provided by the related symptoms of occipital condyle fractures. In these injuries, Collet–Sicard syndrome is commonly found which includes deficit of cranial nerves IX–XII, and in AOD, some variation of the Collet–Sicard syndrome can be expected.[20] Consequently, if the dislocation is not recognized early, damage caused by AOD could result in compression of the lower cranial nerves and brain stem with delayed onset of neurological deficits.[1,7,12]

We suggest immediate careful radiological evaluation of the atlantooccipital junction whenever lower cranial nerve deficits are observed in the context of a side impact collision. We also suggest that even when patients are neurologically intact at the scene, a side impact collision of this nature probably merits a careful consideration to cervical stabilization and urgent radiological evaluation for potential AOD. The suggested mechanism of injury should assist in the biomechanical understanding of these injuries. Examination of the distribution pattern of associated injuries as noted in this case is an additional reliable way to indicate AOD in trauma patients.

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