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Traumatic Posterior Atlantoaxial Dislocation without Related Fractures of C1 - C2 in a Patient with Traumatic Brain Injury: Case Report and Literature Review

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Abstract: It is exceptionally rare to have posterior atlanto axial dislocation without an associated atlas or odontoid fracture and very few cases have been reported in literature. We present a case of a 50 year old female pedestrian who was hit by car while crossing a road. She presented with a depressed level of consciousness and the Glasgow coma score of 12/15, pupils were equal and reactive to light and all limbs were moving equally. She had an extensive abrasion on the forehead which extended into scalp. CT scan showed a posterior dislocation of the atlas with respect of axis with no associated fractures of C 1 and C2. She had traumatic subarachnoid hemorrhage in the basal cisterns and multiple brainstem contusions. Closed reduction of the dislocation was done successfully under fluoroscopy guidance. MRI was done post reduction because funds were not available initially and it showed increased signal in the tectorial membrane and transverse and alar ligaments with mild cord oedema at the cervicomedullary junction. However, the patient post reduction began to deteriorate and eventually passed away before definitive treatment of her cervical-spine injury. The case is reported in view of its rarity and to review literature on this uncommon condition.

Keywords: Posterior, Atlantoaxial, Dislocation

1. Introduction

Traumatic upper cervical injuries constitute 20% of acute cervical spine injuries. However, traumatic atlantoaxial dislocation is infrequent and commonly presents as anterior translational dislocation. Posterior atlantoaxial dislocation without odontoid fracture is extremely rare and often results in fatal spinal cord injury after high-velocity trauma. It is commonly detected at post mortem examination [1]. Very few cases have been reported in literature. We here present a rare case of posterior atlantoaxial dislocation without associated fractures in a head injured patient.

2. Case Report

We present a 50 year old female pedestrian who was hit by an automobile while crossing a road and immediately lost consciousness. She presented with depressed level of conscious and was managed as per the Advanced Trauma Life Support protocol. She was hemodynamically stable with mild respiratory distress. She had a Glasgow coma score of 12/15, pupils were equal and reactive to light and had reduced but equal movement in all limbs. Signs of basal skull fracture were present and had forehead abrasion associated with a deep scalp laceration. The rest of the examination was normal.

CT scan showed a posterior dislocation of the atlas with respect to the axis with no associated fractures of C 1 and C2 (*Figures 1 & 2).*

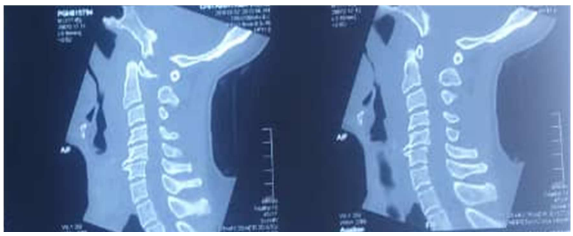

Figure 1. *Shows odontoid peg anterior to anterior C1 arch.*

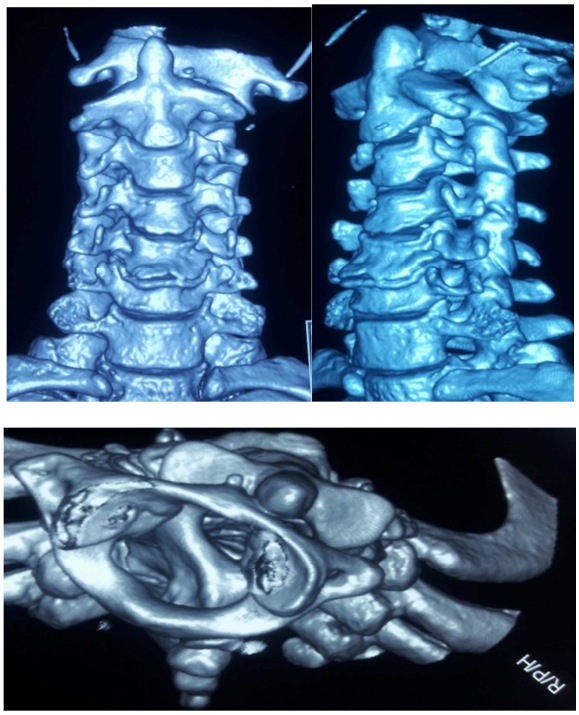

Figure 2. *Top left image – AP 3D CT reconstruction showing intact odontoid process anterior to anterior arch of C1, Top right image- lateral 3D CT reconstruction showing dislocation of the atlanto-axial joint. Bottom image – axial 3D reconstruction showing posterior atlantoaxial dislocation with intact C1 ring.*

She had traumatic subarachnoid hemorrhage in the basal cisterns and multiple contusions in the cerebrum, cerebellum and brain stem (Figure *3*).

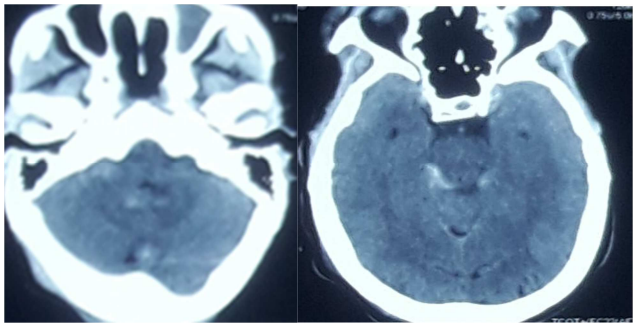

Figure 3. *Left image – multiple contusions – cerebellum and brainstem, right image -subarachnoid haemorrhage in the quadrigerminal cisterns.*

Closed reduction was done successfully under fluoroscopy guidance (Figure 4).

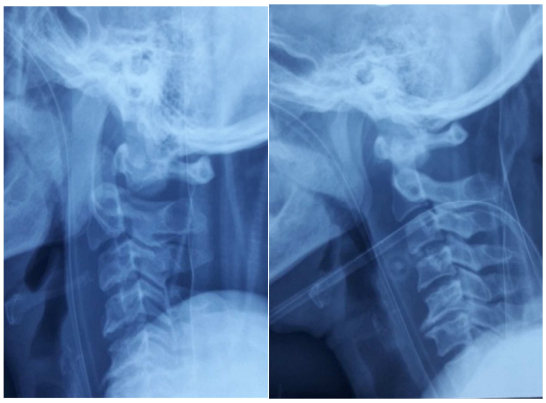

Figure 4. *Left - pre reduction with posterior atlantoaxial dislocation, right - post reduction.*

MRI was done post reduction and showed increased signal in the tectorial membrane and transverse and alar ligaments with mild cord oedema at the cervicomedullary junction (Figure 5).

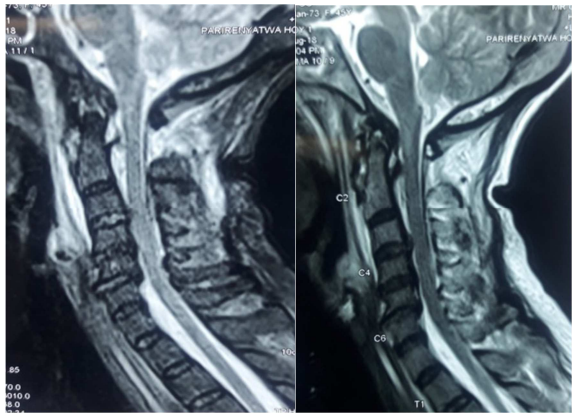

Figure 5. *There was ligamentous disruption with oedema at the tectorial, transverse and apical ligaments at the peg and rupture of the posterior longitudinal ligament. Mild oedema at the cervicomedullary junction. Pre-existing disc degeneration with possibly disc herniation at C3/C4, C4/C5 and C6/C7 levels.*

Unfortunately post reduction patient took a downturn and deteriorated until she died.

3. Discussion

3.1. Anatomy and Biomechanics of the Craniocervical Junction

The craniocervical junction is the most mobile segment of the spine and contributes to about 40°– 47° of motion in the cervical spine. The C0-C1 (occipitoatalantal) articulation is a ball and socket joint and the C1 – C2 is a biconvex joint. The atlantoaxial joint accounts for more than 50% of neck rotation. It also contributes to lateral translation of 3 mm, lateral tilt of about 5°, anteroposterior translation of 3 mm and flexion–extension of 5°–10°. The osseo-ligamentous complex comprising of the atlas, odontoid peg and transverse atlantal ligament is important for stability. The weakest part being the transverse atlantal ligament, so when it ruptures

dislocation is usual anterior. Posterior dislocation without associated fracture is extremely rare and when it occurs it is associated with an odontoid peg and/or anterior arch of atlas fracture [2, 3].

In a 2015 review paper on post traumatic posterior atlantoaxial dislocation without associated fracture of C1 or C2 , investigators found 19 cases in both English and none English literature [4].

3.2. Classification of Atlantoaxial Dislocation

There are four different classification systems for atlantoaxial dislocation. The parameters considered are reducibility, cause, time to diagnosis and direction of dislocation.

According to reducibility AAD can be described as reducible, irreducible on non-reducible. Reducible type can be easily reduced by closed dual direction traction. The irreducible type cannot be reduced by traction and requires surgical release of soft tissue which would be preventing reduction. A non-reducible type is due to bony fusion between C1 & C2 and decompression by odontoid peg resection is necessary [5].

Depending on time from dislocation to definite diagnosis it can be classified as fresh type if diagnosis is made within 3 weeks and old type if it's more 3 weeks old. Time to diagnosis can be prognostic on the success of reduction with the fresh type more likely to be reducible.

AAD can also be classified according to the causes which can be congenital, traumatic or pathologic. Trauma is the most common cause of dislocation, it is usually associated with disruption of the atlantoaxial osseoligamentous complex [6].

Classification according to direction of dislocation describe AAD as anterior, posterior or rotational. The most common being anterior dislocation which is often associated with rupture of the transverse atlantal ligament or malformation of the dens. Relatively uncommon is posterior AAD which is often associated fracture of the dens [7].

The case presented here is a fresh traumatic reducible posterior AAD. It uniquely does not have any associated fractures or congenital abnormalities of the odontoid or atlas. It's possible though that the patient might have had pre-existing pathology like connective tissue or an inflammatory disease predisposing her to dislocation.

3.3. Mechanism of Injury of Posterior Atlantoaxial Dislocation

Haralson and Boyd were the first ones to report a case of posterior atlantoaxial dislocation without associated odontoid peg fracture. Their hypothesis for the mechanism of injury was a combination of severe hyperextension and distraction. It has not been shown clinically or experimentally but the fact that 68% of patients have some facial injuries has made many authors to agree with proposed mechanism of injury [8]. Our patient had associated facial injuries which suggest that she might have had some hyper extension during the trauma.

3.4. Clinical Symptoms and Signs

Patients may present with neck pain or stiffness, dysphagia or dyspnoea due to compression oesophagus and trachea respectively. About 53% of patients present with no neurologic deficit and those who have deficits have mild or transient motor weakness. Patients with deficits virtually recover without any residual long term neurologic deficits. The spinal canal sagittal diameter is greatest at the craniocervical junction. According to Steel's rule of thirds at the occipitocervical junction one third of the spinal canal is occupied by the odontoid process, one third by spinal cord and the final third by cerebrospinal fluid. With AAD the spinal cord can displace the surrounding cerebrospinal fluid before it is compressed which explains the large proportion of patients without neurologic deficits [4, 9].

3.5. Diagnosis

Radiographs have a poor sensitivity of detecting craniocervical junction pathology due to overlap of osseous structures. CT scan with multiplanar reconstruction and 3-dimensional reconstruction is always the gold standard in identifying the osseous anatomy and provides excellent visualization of the rare dislocation. MRI is sensitive in detecting the integrity of the critical stabilizing ligaments and the status of the cervical spinal cord [10].

3.6. Treatment

Closed reduction is achieved by using dual direction traction. A horizontal axial force is used to apply traction while a perpendicular force is applied to the neck [6]. We successfully reduced the posterior AAD using dual direction traction. We had no access to neuromonitoring which could make procedure safer given that our patient had a depressed level of consciousness.

For reducible AAD the surgical approach of choice is posterior atlantoaxial fixation. Patients with irreducible or non-reducible AAD will require anterior transoral release or anterior decompression prior to posterior fixation.

3.7. Posterior Approaches

Posterior wiring and clamping techniques can only be used in patients with intact posterior structures. Biomechanical wiring techniques have insufficient anti rotation strength hence need a rigid cervical brace until bony fusion is achieved.

Posterior trans-articular screw fixation, the Magerl technique is used in patients with or without intact posterior elements of C1 and C2. Biomechanically compared with the wiring techniques, it has a more rigid fixation strength and better anti-rotational ability.

Posterior pedicle screw-rod fixation, commonly known as the Goel - Harms technique, provides much greater biomechanical rigidity strength when compared to wiring or trans-articular screw fixation [11].

Posterior Occipital Cervical Fixation has the disadvantage of sacrificing motion at the atlanto occipital joint which may impact negatively on the patient's quality of life. It is however indicated, when there is associated congenital atlanto-occipital fusion, incomplete posterior atlas or atlanto-occipital instability [12].

3.8. Anterior Approaches

Transoral release is an important technique for dealing with irreducible AAD. Soft tissue scarring behind the anterior arch of the atlas may restrict reduction in these cases. Debriding the soft tissue is important to achieve reduction.

Transoral decompression may be necessary if transoral release fails, this may include odontoid or extensive axis resection. Some cases of non-reducible AAD are due to bone fusion of the lateral masses.

After transoral release and reduction, fixation can be done using screw and plate system. The screws are placed in lateral mass of the atlas lateral mass and in axis vertebra along the pedicles. Anterior cervical trans-articular screw is another alternative but it is usually difficult to put a graft into the facet joint [6, 13–15].

4. Conclusion

Managing patients with posterior atlantoaxial dislocation without odontoid fracture who also have concomitant traumatic brain injury presents a unique challenge in low resource settings. Having an MRI done before reduction would provide information on the relationship of osseous and neuro-vascular structures and help guide reduction. Considering that our patient's condition worsened after reduction we strongly recommend the use of neuromonitoring during reduction and operation of patients with craniocervical injury associated traumatic brain injury.

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