ABSTRACT
Cervical spine injuries are often described as catastrophic events in neurological terms, with very low survival rate. Lesions to C3-C7 segment are the most common (about 80%) followed by segment C1-C2 (20%). The cervical spine injuries are of great importance, both by severity as the neurological implications. It is important to consider that among cervical traumas that do not present neurological damage at the time of the accident, 10% have deficits later, so all cervical trauma should be considered as potential spinal cord traumas, until the evolution of the case shows that definitively there is no spinal cord or nerve root damage. Cases have been reported with both atlantooccipital and atlantoaxial dislocations without neurological deficit, so these lesions went unnoticed in the emergency services. Some of the events to be considered at the time of the accident are suboccipital pain on axial skull pressure and spontaneous stiffness of the patient’s neck. Dysphagia, pain on palpation of the anterior neck and a visible increase of prepharyngeal mass can also be observed, which is why it is important to keep the suspicion of cranio cervical trauma in all patients who have these symptoms and carry out the relevant tests. This paper presents a case of post-traumatic atlantoaxial dislocation, which showed no neurological deficit at the time of the accident, but was later presented, as well as the surgical procedure carried out.

Keywords: Atlantoaxial joint; Joint instability; Dislocation.

INTRODUCTION
Cranio cervical trauma is considered life threatening, due to the devastating neurological damage it causes. Thanks to advances in modern medical systems and the care of patients with trauma, the number of patients who survive these injuries has increased. Universal precautions in the care of the vertebral spine, and better stabilization of the vertebral spine at the moment of transfer to a specialized center, have led to a better outcome.\textsuperscript{1,2} Cervical, occipitocervical, and C1/C2 injuries and their evolution present a confused clinical manifestation that can lead to diagnostic error. These confounding variables include: multiple associated injuries, lack of appropriate imaging studies, and low clinical suspicion due to lack of appropriate imaging studies, and low clinical suspicion due

\textsuperscript{1} Hospital de Especialidades, Centro Médico Nacional de Occidente. Instituto Mexicano del Seguro Social. Guadalajara, Jalisco, Mexico.

Study conducted at the Spine Clinic. Afternoon Shift. Hospital de Especialidades, Centro Médico Nacional de Occidente. IMSS. Guadalajara, Jalisco, Mexico.

Correspondence: Belisario Domínguez No 1000, Colonia Independencia, Guadalajara Jalisco. C.P. 44340. Jefatura Traumatología y Ortopedia. mariocahuexque@gmail.com

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to a lack of specific symptoms. Injuries in 30% of cases may be combined, atlantoaxial and occipitocervical, with high mortality rates. There are reports of patients with cervical injury without neurological deficit, in which delayed diagnosis and initial stabilization lead to a worse prognosis for the patient in the short to medium terms. Once the diagnosis of cervical injury, specifically atlantoaxial, has been established, reduction and early fusion are recommended as definitive treatment, to prevent the development of neurological disturbances, which may be irreversible, even placing the patient’s life at risk. Although there are currently various techniques for the treatment of these injuries, the results depend on the surgical technique that the specialist spinal surgeon is familiar with.

Clinical case

General information: Male patient aged 28 years, born and residing in Guadalajara, Jalisco, a Roman Catholic, educated to high school level, working as a carpenter. Inherited diseases: Denied by the patient. Disease history: Denies chronic-degenerative diseases. No previous surgery.

Patient reports that two months previously (August 2012), prior to admission, he suffered a physical assault causing cranioencephalic and cervical trauma. He was assessed at the Hospital General de Zona, where he was diagnosed with mild cervical sprain. The patient was discharged at the same time, without neurological deficit. The patient reports that after discharge, he began to experience paresthesia of the upper limbs with intermittent onset, followed by a gradual decrease in muscle strength, prompting him to come to this center for assessment.

Patient is conscious and oriented as to time, space and person. The neurological exam presents hypoesthesia of the upper limbs, with a decrease in muscle strength, 4/5 (Daniels scale). Patient has pain on moving the neck and pain on axial compression. Denies dysphagia.

Imaging studies were performed (Figure 1) which showed, in CT image reconstruction, loss of atlantoaxial joint congruence. Figure 2 shows the lateral Radiography and the measurement of C1/C2, the ADI interval, and the Powers index.

The atlantoaxial region is marked and the dissection extends laterally until the atlantoaxial joints on both sides were visualized, as well as the joint facets. The facets of the atlas and axis were visualized directly, and distraction was performed manually, using a cranialhalo, achieving adequate alignment. Transfacet posterior instrumentation of C1/C2 was performed, (Figure 3) without using large screws, and it was seen in the radiography that the screws achieved adequate reduction. (Figure 3)

The normal anatomy between C1 and the odontoid apophysis is restored, as can be seen in the postsurgical control. (Figure 4)

Patient one year after surgery, with adequate evolution and without neurological deficit, stiffness, or pain on movement. (Figures 5 and 6)

DISCUSSION

Rotational lesions of the atlantoaxial joint were described for the first time by Corner, in 1907. These lesions are the result of flexion and rotation, with rupture of the transverse ligament. Atlantoaxial subluxation is rare, and even more so without fracture of the dens (odontoid process) of the axis. As a result, it can sometimes be overlooked.

The intrinsic stability of the atlantoaxial complex is provided by the dens apophysis, which binds to the ventral osteoligamentous ring, formed by the anterior arch of the atlas, and dorsally by the transverse ligament.

The pattern of injury is consistent with a mechanism of high-degree hyperextension.

The diagnosis is generally difficult; the clinical manifestations are usually rare, such as suboccipital pain on axial pressure on the skull, injuries and around 20% of lesions of the cervical spine.
and on moving the skull the patient spontaneously stiffens the neck, contracting the sternocleidomastoid and trapezius muscles, with dysphagia and pain on anterior palpation of the neck,5,8, torticollis, and occipital neuralgia. Symptoms of verteobasilar insufficiency may sometimes be present. Where there is suspicion, imaging studies, plain AP and lateral radiography should be carried out, with their respective measurements, and even more importantly, computed axial tomography with 3D reconstruction.1,3,5,9,13

Early diagnosis is essential, as a longer period between the lesion and its reduction is correlated with higher rates of recurrence and failure of the reduction through nonsurgical techniques.14 We believe that simple radiographic measurements (ADI and Powers Index) of the cervical spine can help in the interpretation of anomalies in segment C1/C2, as in this case, where both measurements showed values compatible with C1/C2 dissociation, as well as helping the team of the emergency unit avoid overlooking these potentially devastating entities.

The craniocervical joint is an anatomically complex functional structure that represents the transition zone between the skull and the spinal cord, enabling extension, flexion and lateral rotation of the head. In particular, the atlantoaxial segment has unique characteristics compared with the lower cervical portion, due to its ample range of rotation.1,2

There are magnetic resonance imaging testing that suggest that interruption of the alar ligament is the mechanism by which a rotatory subluxation is produced.15,16 The alar ligaments are the main structures responsible for the stability of rotation, as they limit the rotation to 45°.5,14

Fielding and Hawkins10 describe 4 types of atlantoaxial rotation with rotation of the transversal ligament. (Table 1) Our patient is classified as having a type IV lesion, in which the atlas can dislocate bilaterally forwards or backwards, with concomitant narrowing of the vertebral canal.

Dislocation in C1-C2 is a dynamic process that is generally irreducible and cannot be reduced by cervical traction. The treatment of this type of chronic and reducible dislocation is a challenge for surgeons, although improvements in surgical techniques have brought some encouraging results over the years. The main procedures reported in the literature are posterior occipitocervical/C1-C2 fusion and instrumentation, and transoral odontoidectomy. Recent improvements in the emergency diagnosis and therapeutic management have shown a higher rate of patient survival.

Fielding and Hawkins10 describe 4 types of atlantoaxial luxation with rupture of the transverse ligament; (Table 1) this case presented a type IV lesion, therefore it was decided on reduction with transfacet screws of C2 to C1, that even without using longer screws, has achieved adequate reduction, and good results after one year of follow-up. In these cases, occipitocervical fusion is generally considered as a form of neuroprotection, and should be performed as soon as possible.1,5,7

Although there have been few published cases, the treatment by this type of pathology is fixation with transfacet screws, a technique that provides stability and adequate anatomical reduction.1,5,7,9,10,15

FINAL CONSIDERATIONS

The diagnosis of posttraumatic atlantoaxial luxation should be suspected in the presence of sprain and neck pain. However, atlantoaxial luxation can occur in the absence of any clinical symptoms, therefore this diagnosis should be systematically considered and ruled out in any patient with multiple traumas. The radiographic findings of the cervical spine, in AP and lateral views, can hinder the interpretation of luxation C1/C2, therefore the routine use of CT scan of the cervical spine should be considered in all patients with cervical cranial trauma. When instability of the C1/C2 joint is diagnosed, timely reduction and fusion is recommended as definitive treatment.

All authors declare no potential conflict of interest concerning this article.

REFERENCES


Table 1. Classification of Atlantoaxial Luxation by Fielding and Hawkins.6

<table>
<thead>
<tr>
<th>Type</th>
<th>Transverse Ligament</th>
<th>Atlantodental interval</th>
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<tbody>
<tr>
<td>Type I</td>
<td>None</td>
<td>&lt;3mm</td>
</tr>
<tr>
<td>Type II</td>
<td>Slight</td>
<td>3-5mm</td>
</tr>
<tr>
<td>Type III</td>
<td>Deficiency of the Transverse and Alar ligaments</td>
<td>&gt;5mm</td>
</tr>
<tr>
<td>Type IV</td>
<td>Deficiency of the Transverse and Alar Ligaments</td>
<td>Posterior change of the Atlas</td>
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Figures 5 and 6. Evolution 1 year after surgery.