TRAUMATIC ATLANTO-AXIAL SUBLUXATION

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Traumatic atlanto-axial subluxation is a rare injury which may not be revealed on routine radiographs, especially when there is muscle spasm. We report on seven patients with atlanto-axial subluxation as a result of neck injury; only two of them had significant head injuries. Three patients presented with a neurological deficit attributable to the injury, one immediate and two with delayed onset.

Traumatic atlanto-axial instability, occurring in an otherwise healthy patient, has a potential for neurological disaster; early consideration of operative treatment is indicated.

Atlanto-axial subluxation is relatively common in rheumatoid patients with a reported incidence of between 25% and 36% (Conlon, Isdale and Rose 1966; Cabot and Becker 1978). Traumatic atlanto-axial subluxation is relatively rare; few series have been reported (Grogono 1954; Fielding et al. 1974). In the presence of an intact odontoid process the injury carries a serious risk of neurological disaster. We report a series of seven patients.

PATIENTS AND MANAGEMENT

All seven patients had sustained trauma to the upper cervical spine, four in road accidents and two at rugby football. One patient, Case 4, also had a hypoplastic odontoid process which may have contributed to the instability (Spiering and Braakman 1982). Only two of the patients had an associated major head injury. Details are given in Table I.

Initially, the diagnosis was missed in no less than four of the seven patients (Figs 1 and 2), and was made later on standard or flexion/extension radiographs. Three patients either presented with, or subsequently developed, a long tract neurological deficit which was directly attributable to the neck injury.

Three patients, one of whom had a neurological deficit, were treated conservatively by six weeks of skeletal traction in extension followed by two months in a brace. Of these, one patient achieved spontaneous stabilisation, one patient had residual and unchanged atlanto-axial instability and one patient is awaiting operation for fusion. This latter patient presented with quadriplegia and respiratory paralysis but made a full neurological recovery on conservative treatment.

Three patients were treated by operation (Gallie 1939; Brooks and Jenkins 1978). Two of these patients had developed a neurological deficit some time after the initial injury; one (Case 5) within two weeks (Figs 3 and 4), and the other (Case 6) about 13 years after his major injury, when he had a minor fall. After posterior fusion at C1/C2 level, all three patients are stable on flexion/extension views and the two patients with neurological signs have made a virtually full functional recovery.

CASE REPORT

Case 6. A 36-year-old man was involved in a motor vehicle accident in 1973, sustaining a major head injury and other trauma. He was unconscious for four days but then made a full recovery. Despite routine radiographs of his cervical spine, the diagnosis of traumatic atlanto-axial subluxation was missed.

Some 13 years later he had a minor fall and developed left hemiplegia. He was diagnosed as having had a cerebro-vascular accident and was treated accordingly. However, no cranial nerve deficit was found and he was found to be normotensive. There was a gradual spontaneous recovery to MRC Grade 3 power with upper motor neurone signs in both upper and lower limbs on the affected side. The patient complained of persistent occipital headaches and recurrent bouts of neck pain.

Radiographic examination of the cervical spine, including flexion/extension views, showed an atlanto-dens interval of 10 mm and tomography failed to reveal any signs of previous odontoid fracture (Fig. 5). CT scans of the brain and cervical spine revealed no evidence of a previous cerebro-vascular accident and confirmed subluxation (Fig. 6).

A posterior C1/C2 fusion was performed, which provided stability. Neurological recovery eventually gave Grade 4+ to 5 motor power in both arm and leg.
Case 1. Figure 1 – Initial lateral radiograph showing loss of cervical lordosis, but a normal atlanto-dens interval. Figure 2 – Lateral radiograph in flexion taken two weeks after the injury, showing a 8 mm atlanto-dens interval.

Case 5. Figure 3 – Initial flexion radiograph showing an atlanto-dens interval of 8 mm. Figure 4 – Postoperative radiograph showing a Gallie fusion at C1/C2.

Case 6. Figure 5 – Lateral tomogram showing an atlanto-dens interval of 10 mm. Figure 6 – CT scan of the atlanto-dens articulation.
Table 1. Details of seven patients with atlanto-axial subluxation

<table>
<thead>
<tr>
<th>Case number</th>
<th>Age in years</th>
<th>Sex</th>
<th>Cause of injury</th>
<th>Clinical presentation</th>
<th>Standard radiographs</th>
<th>ADI* in flexion (mm)</th>
<th>Management</th>
<th>Follow-up and results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>23</td>
<td>M</td>
<td>Hyperflexion</td>
<td>Pain Tender in upper cervical region</td>
<td>Loss of lordosis ? flake off lateral mass of atlas (Fig. 1)</td>
<td>8 (Fig. 2)</td>
<td>Initially missed, cervical collar for 2 weeks</td>
<td>Gallie fusion</td>
</tr>
<tr>
<td>2</td>
<td>28</td>
<td>M</td>
<td>Hyperflexion</td>
<td>Pain Sensation of instability “wobbly”</td>
<td>No abnormality</td>
<td>7</td>
<td>Halo-body cast for 3 months SOMI* brace for one month</td>
<td>Gallie fusion</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>F</td>
<td>Motor accident</td>
<td>Pain Femoral fracture and abdominal trauma</td>
<td>No abnormality</td>
<td>7</td>
<td>Halo traction for 2 months SOMI* brace for 2 months</td>
<td>Gallie fusion (Fig. 4)</td>
</tr>
<tr>
<td>4</td>
<td>30</td>
<td>M</td>
<td>Motor accident</td>
<td>Pain Tender in upper cervical region</td>
<td>? Os odontoideum</td>
<td>8</td>
<td>Halo traction for 6 weeks Cervical collar for 6 weeks</td>
<td>Gallie fusion (Fig. 4)</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>M</td>
<td>Motor accident</td>
<td>Head injury Fractured femur Neck injury missed Progressive hemiparesis MRC Grade 3</td>
<td>No abnormality</td>
<td>8 (Fig. 3)</td>
<td>Gallie fusion (Fig. 4)</td>
<td>Gallie fusion</td>
</tr>
<tr>
<td>6</td>
<td>36</td>
<td>M</td>
<td>Motor accident</td>
<td>Head injury Neck injury missed (L) hemiparesis (see text)</td>
<td>Atlanto-axial subluxation</td>
<td>10 (Figs. 5 and 6)</td>
<td>Atlanto-axial subluxation</td>
<td>Atlanto-axial subluxation</td>
</tr>
<tr>
<td>7</td>
<td>36</td>
<td>M</td>
<td>Motor accident</td>
<td>Blunt chest injury Quadriaparesis Paralysis of diaphragm Grunting respiration</td>
<td>No abnormality</td>
<td>10</td>
<td>Skeletal traction for 6 weeks SOMI* brace</td>
<td>Gallie fusion (Fig. 4)</td>
</tr>
</tbody>
</table>

* ADI, atlanto-dens interval (normal is 3mm)
† SOMI, sterno-occipital-manibular

DISCUSSION

A missed odontoid fracture is a well known pitfall in the management of a patient with a head injury. It has, however, been noted that traumatic atlanto-axial subluxation, with an intact odontoid process, is potentially a much more serious injury (Fielding et al. 1974; Steel 1968). This diagnosis can, however, be easily missed, especially when there is marked spasm of the paravertebral muscles.

This was a significant finding in our series, with a late diagnosis being made in four out of seven patients. An additional factor is that in the management of trauma, the initial lateral radiographs are taken with the patient supine and the neck in slight extension. This may serve to “reduce” the atlanto-axial subluxation. Furthermore, the radiographic sign of an increase in the shadow of the anterior prevertebral soft tissues was not noticed in any of our patients.

Atlanto-axial subluxation, with or without myelopathy, has been described in association with various congenital anomalies at the atlanto-axial articulation (McRae 1953; Greenberg, Scoville and Davey 1968; Spiering and Braakman 1982), but none of our patients had any suggestive radiographic features. Two of our patients were children aged six years and seven years. Cattell and Filtzor (1965) have noted that 20% of normal children under eight years of age may have an atlanto-dens interval (ADI) of 3 mm or more during forward flexion, but they did not specify an upper limit of normality. We feel that the ADIs of 7 mm and 8 mm in our two patients were definitely abnormal and attributable to the injury sustained.

In our series, atlanto-axial subluxation was not necessarily associated with a severe head injury (only two of seven patients), though this was found in all eleven patients reported by Fielding et al. (1974). Two of our patients were rugby players; this again emphasises the association between contact sports and the risk of significant neck injury.

The choice of treatment remains controversial. The only large series of patients with atlanto-axial subluxation have been those with rheumatoid arthritis (Conlon et al. 1966; Cabot and Becker 1978). In these patients the indications for surgery have included neurological complications, severe verteobasilar insufficiency, and persistent pain unresponsive to conservative management (Crelin, Maccabe and Hamilton 1970; Conaty and
Mongan 1981). Some authors feel that the presence and degree of atlanto-axial subluxation is not, per se, an indication for surgical intervention (Crelin et al. 1970; Fielding, Hawkins and Ratzan 1976), but this approach must be questioned for active patients without rheumatoid arthritis who have traumatic subluxation.

The incidence of myelopathy should logically relate to the space available for the cord (SAC) within the spinal canal, when it has been reduced by the subluxated odontoid process. McRae (1953) reported six patients with chronic atlanto-axial subluxation due to various causes; three of these patients had associated myelopathy. The ADI averaged 11.5 mm in his series and the SAC averaged 15 mm, but he found no correlation between the severity of subluxation and the presence of neurological deficit.

One of our patients presented with a major neurological deficit and two subsequently developed one as a result of traumatic atlanto-axial subluxation. This complication has rarely been reported (McRae 1953; Dunbar and Ray 1961) but is of major importance. Our experience shows that otherwise healthy patients are more at risk of developing disastrous neurological complications, and that a more aggressive protocol for treatment is warranted.

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REFERENCES


