CASE REPORT

Transient cervical neurapraxia associated with cervical spine stenosis

F J Andrews

A 43 year old woman presented with a history of a hyper-extension cervical injury resulting in transient quadriplegia. Cervical spine radiography revealed developmental spinal stenosis and magnetic resonance imaging demonstrated underlying spinal cord oedema secondary to contusion, with a herniated disc at C3-C4. The Torg ratio may be used to aid the initial diagnosis of cervical spine stenosis. Indications for operative treatment of these patients are controversial and these patients should receive further expert assessment.

A 43 year old woman was admitted to the emergency department (ED) after an episode of transient quadriplegia. Her partner had accidentally fallen against her and she fell backwards onto uneven ground, her back struck the ground followed by a hyperextension injury to her neck. This was associated with an immediate quadriplegia but with no respiratory compromise, fully resolving after five minutes. Full neurological examination in the ED showed no motor or sensory deficit. A lateral cervical spine radiograph (fig 1) showed cervical spine stenosis; the C7-T1 interface on a Swimmer’s view was unremarkable. Magnetic resonance imaging was performed, which showed diffuse oedema (secondary to contusion) of the entire cervical cord, most marked at C3-C4 with an associated acute central disc prolapse (fig 2). Although the patient had no neurological signs, it was felt that she might be at risk of further neurological injury after further minor neck trauma, and she was referred for neurosurgery. An anterior cervical microdiscectomy with fusion of the C3-C4 vertebra was performed and the patient was discharged home with no neurological deficit.

DISCUSSION

Neurapraxia of the cervical spinal cord with transient quadriplegia was first described as a separate clinical entity in 1986. The syndrome typically presents in young adult contact sport participants who experience an acute neurological episode of cervical cord origin, immediately after a hyperextension or hyperflexion cervical injury. These patients have no fracture or subluxation injury and no loss of cervical spine stability. Both sensory and motor deficits occur, and affect any combination of the extremities. Usually there is complete return of normal neurological function, within minutes of the initial insult. American footballers and rugby players are known to be at risk, especially during high tackles and scrum collapses; some players describe recurrent episodes. The case described in this report is unusual, as the patient was age 43 and not engaging in sporting activity. Hyperextension injuries in older patients with spinal stenosis or degenerative arthritis may instead produce a central cord syndrome affecting particularly the upper limbs, with variable but often incomplete neurological recovery because of central cord necrosis.

Figure 1  Lateral radiograph of the cervical spine shows a developmentally small spinal canal. The Torg ratio at mid C3 is calculated by dividing the anteroposterior (AP) diameter of the spinal canal (X) by the AP diameter of the vertebral body (Y). This ratio is 0.6 at C3. Also seen is a congenital fusion of the spinous processes of C1 and C2.

Figure 2  Magnetic resonance image showing acute disc herniation at C3-C4 associated with spinal cord compression. The increased signal throughout the cervical spinal cord represents oedema secondary to contusion.
Cervical neuapraxia is strongly associated with developmental spinal stenosis. The diameter of the spinal canal between the third and sixth vertebra is most critical because the spinal cord is most mobile here and fills most of the available space compared with the upper cervical vertebrae. The spinal canal sagittal diameter may be measured as a ratio of the spinal canal diameter to the vertebral body width (Torg’s ratio method); this calculation eliminates errors attributable to different tube-target distances when taking the radiograph. A ratio of less than 0.8 defines significant cervical stenosis.

Applying this ratio to a cohort of athletes who returned to their sport after transient neuapraxia, it has been shown that there is a strong and inverse correlation between the risk of recurrence and the Torg ratio. This study also used magnetic resonance imaging to measure the disc-level canal diameter (the shortest distance between the intervertebral disc and the posterior bony elements) and found that this was a more accurate predictor of risk of recurrence.

Hyperextension trauma in particular places patients with cervical spine stenosis at risk of neuapraxia. The mechanism of the transient neurological signs and symptoms is spinal cord compression. Sudden approximation of the posterior inferior aspect of a vertebral body with the superior aspect of the lamina of the next vertebral body below results in a sudden, brief decrease in the anteroposterior diameter of the canal, compressing the cord. This causes transient disruption of cell axonal membrane permeability resulting in reversible depolarisation.

A Torg ratio of less than 0.8 has a high sensitivity for significant cervical spine stenosis but a poor positive predictive value, therefore functional magnetic resonance imaging has been recommended to determine the size of the spinal cord and the functional reserve of the spinal canal. The treatment of such injuries is debated, in view of their transient nature. Studies have shown cervical disc protrusion in 36% of these patients but in those patients successfully returned to contact sports with demonstrable disc herniation on magnetic resonance imaging, half were treated conservatively rather than with surgery.

A study of a large cohort of athletes showed no relation between a developmentally narrowed cervical canal and irreversible injury of the cervical cord. However, progressive neurological deterioration after minor hyperextension injury in patients with cervical spine stenosis is well described, both in adult and paediatric patients. These patients required urgent decompressive surgery to achieve neurological recovery; and some had described previous episodes of transient cervical neuapraxia with full recovery. Other studies have found that patients with a permanent neurological deficit from spinal trauma had significantly narrower sagittal diameters than those who sustained no neurological damage from similar injury.

The Torg ratio may help identify patients at risk for cervical spine cord injuries without fractures or dislocations especially with decreased consciousness; magnetic resonance imaging may be more sensitive but its role in the initial assessment of such patients is unclear. Patients presenting with cervical neuapraxia associated with cervical spine stenosis should be referred for expert assessment.

Funding: none.
Conflict of interest: none.

Authors’ affiliations
F J Andrews, Accident and Emergency Department, St James’s University Hospital, Leeds, UK
Correspondence to: Dr F Andrews, Department of Medicine, UCD, University of Liverpool, Daulby Street, Liverpool L69 3GA, UK; fandrews@liv.ac.uk

Accepted for publication 20 August 2001

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F J Andrews

doi: 10.1136/emj.19.2.172

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