Complete fracture-dislocation of the lower cervical spine associated with significant ligamentous disruption of the upper cervical spine: A case of survival without permanent neurological sequelae

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Case report

A 36-year-old female was referred to the Bedford Orthopaedic Centre (BOC) in Umtata, South Africa following a motor vehicle accident. She was a passenger in a minibus taxi which lost control at high speed and rolled. Precise details of the accident were unavailable and due to the geographical location of the accident there was a delay of several hours before arrival at the BOC. She was helped from the car and taken to the local emergency department complaining of isolated cervical spine pain. At this point the cervical spine was immobilised pending further investigation. There was no history of loss of consciousness or neurological compromise.

On arrival in our accident department she was found to be alert and orientated. Initial trauma evaluation did not suggest major chest, abdominal, pelvic or limb injury. No focal limb neurological deficit was noted. Cervical-spine examination revealed bruising and tenderness over the lower cervical vertebrae in addition to a palpable step deformity of the lower cervical region.

Chest and pelvic radiographs were normal. Cervical spine radiographs revealed complete bilateral fracture dislocation of C6 on C7 (Fig. 1). This was confirmed with sagittal and axial CT scan (Figs. 2 and 3). Urgent MRI scanning was not possible in our unit. The radiograph also showed posterior ligamentous disruption between C1 and C2 with an increased interval between the spinous processes.

Neurology remained unchanged although on further examination she was found to have Medical Research Council (MRC) grade 4-power in the intrinsic muscles of both hands suggesting T1 nerve root compression. She had MRC grade 5-power in all other muscle groups with normal sensation, reflexes and tone. Perianal sensation and anal tone was normal.

Skeletal traction via cranial tongs was applied and reduction of the dislocation was confirmed with serial radiological assessment. Subsequently she was taken to theatre for posterior cervical spine
stabilisation with sublaminar wires and bone grafting (Fig. 4). She was immobilised in a halo vest for 12 weeks. At latest follow up her neurology has completely resolved and she is making good progress.

Discussion

Complete cervical spine fracture dislocations without neurological deficit have seldom been reported in the English language medical literature. Similar injuries of complete dislocation without permanent neurological deficit have been reported in the upper cervical spine, the cervico-thoracic level, the thoracic spine and lumbar spine. In the cervical spine this type of injury has mainly been reported in the lower cervical spine involving...
We did not encounter a previous report of complete dislocation associated with significant ligamentous disruption at another cervical vertebral level.

The goals of treatment of cervical spine injuries include realignment of the spine and maintenance of spinal stability, prevention of further neurological insult and improvement of neurological recovery, and to obtain early functional recovery. Routine cervical-spine immobilisation in all trauma patients is mandatory with early radiological evaluation. Further evaluation of ligamentous, intervertebral disc and osseous structures should be obtained with CT and MRI scanning. The pathological anatomy, particularly looking for associated disc extrusion, must be carefully defined before treatment is determined. If neurological deficit exists high dose methylprednisolone administered within eight hours of the initial injury has been shown to be beneficial in terms of motor recovery.

After initial medical stabilisation and thorough neurological assessment, spinal alignment can be obtained by skeletal traction through spring-loaded Gardner–Wells tongs or a halo ring. Continuous monitoring of neurological status during reduction is essential to prevent iatrogenic injury from overdistraction of an unstable spinal segment which has been reported. Initially 5—10 pounds of traction weight is applied; weight is then added in 5-pound increments. Serial lateral radiographs are obtained as the traction weight is increased, until the spine is realigned. There is no agreement on the safe upper limit of traction weight but most surgeons do not apply more than 40—50 pounds of traction although other authors advocate higher loads. A general guideline is 10 pounds for the head and 5 pounds for each additional level of injury.

Once spinal realignment is obtained with traction and is documented radiographically, traction weight can be reduced. Failure of closed reduction may be due to the presence of a traumatic disk herniation, facet fracture or excessive paracervical muscle spasm. If spinal realignment cannot be obtained by traction, open reduction and stabilisation, usually through a posterior approach, is indicated. If the MRI scan shows an associated herniated disk, an anterior cervical disectomy and instrument fusion should be considered.

In this patient spinal cord damage was avoided as the fracture involved the posterior elements. This allowed anterior slipping of the anterior elements and hence widening of the spinal canal. The mechanism of injury we believe is hyperflexion initially resulting in the upper spinal cord lesion followed by hyperextension associated with axial loading (compression extension injury). Hyperextension and axial loading caused injury to the anterior and posterior longitudinal ligaments with subsequent fracture of the laminae of C5—C7 (‘traumatic laminotomy’). Disruption of the posterior elements created space for the spinal cord to move dorsally and thus avoiding catastrophic neurological injury. This unusual case highlights how a complex spinal trauma problem can be successfully managed in a resource poor area provided basic principles are adhered to.

References