Case Report

Retropulsion of intervertebral discs associated with traumatic hyperextension of the cervical spine and absence of vertebral fracture: an uncommon mechanism of spinal cord injury

KC Hayes^{*,1,2}, HK Askes^{1,2} and BA Kakulas^{3,4}

¹Department of Physical Medicine and Rehabilitation, University of Western Ontario, Canada; ²Lawson Health Research Institute, St. Joseph's Health Care London, London, Ontario, Canada; ³Department of Neuropathology, Royal Perth Hospital, Perth, WA, Australia; ⁴Australian Neuromuscular Research Institute, Perth, WA, Australia

Study design: Case report of a 68-year-old male who sustained cervical trauma following a bodysurfing accident.

Objective: To describe the pathology of a relatively uncommon mechanism of injury involving extradural cord compression associated with traumatic disc protrusion and herniation, following a cervical hyperextension injury in which there was no vertebral fracture or residual subluxation.

Setting: Department of Neuropathology, Royal Perth Hospital, West Australia.

Method: Postmortem pathology report.

Results: Evidence of multiple ruptures of anterior longitudinal ligament with posterior intervertebral disc herniation and three discrete foci of central cord hemorrhage.

Conclusion: Observations are consistent with cervical extension injury and an injury vector that involves intense axial loading sufficient to cause multiple disc failures, disc herniation and retropulsion leading to extradural disc compression and cord hemorrhage.

Spinal Cord (2002) **40**, 544–547. doi:10.1038/sj.sc.3101344

Keywords: spinal cord injury; intervertebral disc; hyperextension; pathology

Introduction

Hyperextension injuries to the cervical spine constitute 25–50% of all severe traumatic cervical cord injuries.^{1–3} Frequently these involve fracture of the posterior element and/or fracture of the anterior inferior tip of a dislocated vertebra with associated encroachment into the spinal canal. There are, however, an appreciable number of hyperextension injuries leading to tetraplegia, without vertebral fracture or evident dislocation.^{2–7} The mechanism of cord injury in these cases is less clear.

The most commonly invoked explanations for these injuries include impingement of the cord by the anterior bulging of the ligamentum flavum,^{4,6,8} encroachment into a narrowed spinal canal by midline osteophytes,^{5,9} a 'pincer' mechanism involving anterior translation of the vertebra or posterior translation with the posterior-inferior edge of the vertebral body compressing the cord against the superior edge of the

subadjacent lamina,^{4,5,9,10} cord traction² or tensile forces within the cord.⁸ These effects may be additive.⁹

The present report describes a relatively uncommon mechanism of traumatic cervical cord injury following hyperextension without vertebral fracture or residual dislocation. The primary pathology involves multiple sites of failure of the anterior longitudinal ligament, posterior disc protrusion at C₃₋₄, C₄₋₅, C₅₋₆, disc herniation at C₃₋₄ with extradural cord compression, and focal cord hemorrhage at three distinct locations.

Materials and method

The necropsy specimen and clinico-pathological data were obtained from the Western Australian Spinal Cord Injuries Database¹¹ maintained within the Department of Neuropathology, Royal Perth Hospital, West Australia. The pathology reports were derived from post mortem (Drs K Margolius and JR Taylor). In the pathological examination, the vertebral column was removed intact. The block therefore included the whole column with the posterior muscles and the

*Correspondence: KC Hayes, Parkwood Hospital/SJHC, 801 Commissioners Road East, London, Ontario, Canada, N6C 5J1

occipital bone. This method preserved the atlanto–occipital joints and ligaments intact. The vertebral column and the spinal cord were examined together so that the relationships of the damage to each of these structures could be assessed. Specimens were fixed before examination by the intrathecal injection of 500 ml 10% formol–saline, as soon as possible after death. After fixation, post mortem roentgenograms were taken of the spine. Following external examination, the lamina and posterior muscles were removed preserving the facet joints and their capsules, and the extradural space, blood vessels and dura were examined. The vertebral column was later hemisected to display the vertebral bodies, discs, and ligaments.

Case report

A 68-year-old male (Ht: 170 cm; wt: 70 kg), sustained a severe cervical hyperextension injury while body-surfing. He was found unconscious, not moving his limbs and unresponsive, was administered cardiopulmonary resuscitation and transferred to hospital. Computerized tomography revealed widespread brain swelling. He was declared brain dead after 24 h and died shortly after. On admission to the mortuary the presumptive cause of death was ‘neck injury’, based on the context of the accident and the absence of limb movement.

At post mortem, radiological examination of the spine revealed evidence of degenerative disease. No fractures were observed. Macroscopic pathological examination of the brain showed normal dura, prominent sagittal sinus venous stasis and marked cerebral swelling. No other abnormalities were noted. The cause of death was established as ‘respirator effect’.

Examination of the vertebral column identified severe disc injuries at C₃₋₄, C₄₋₅, and C₅₋₆, with ruptures of the anterior longitudinal ligament at each level and partial avulsion of each disc from the anterior part of each vertebral end-plate and bleeding into the discs. There was also posterior herniation of the C₃₋₄ disc against the anterior cord. The facet joints at these levels showed hemarthroses. The left C₅₋₆ facet joint showed traumatic detachment of its articular cartilage on both facets and localized damage to the tip of the superior articular process of C₆. Smaller traumatic disc lesions (anterior rim lesions) were present at C₂₋₃ on the left side. There was prevertebral intramuscular hemorrhage and interspinous bruising at the cervico–thoracic junction; this was most notable at the T₂₋₃ interspace. These traumatic changes coexisted with degenerative changes including many anterior vertebral osteophytes but the disc spaces were well preserved and the alignment of the column was normal. There were no obvious fractures or dislocations. These features are illustrated in Figure 1.

Examination of the spinal cord revealed central cord hemorrhage at three distinct sites corresponding to the three injured discs and the upper margins of the

vertebrae below each disc. There were bruises in the dorsal root ganglia of C₂ on both sides.

The mechanism of cord injury appears to have involved discrete foci of antero-posterior compression due to: (a) transiently retropulsed intervertebral discs (with herniation of C₃₋₄ and residual extradural compression) or (b) transiently retroluxed vertebrae (with residual cord compression from the posterior–superior lip of C₅). (Note from Figure 1, however, that the focal hemorrhage at C₄₋₅ was marginally cephalad to the cord compression associated with the C₅ vertebra.) This mechanism may have been compounded by transient infolding of the ligamentum flavum in a pincer-like mechanism.³ Central cord hemorrhage was most evident at the level of the herniated C₃₋₄ disc. The cord pathology appeared localized in the gray matter leaving a circumferential rim of spared white matter, as has been noted previously.^{8,12}

Discussion

While hyperextension injuries constitute a large percentage of all traumatic cervical spine injuries,^{1–3,9} acute traumatic herniation of cervical discs with extradural cord compression and absence of bony abnormality is uncommon.¹³ In 1949, Barnes² reported that there is no tendency for cervical discs to herniate into the spinal canal in traumatic hyperextension injuries. In the series of cases he reported, there was no disc protrusion into the spinal canal in nine patients with hyperextension injuries nor in 12 patients with a fracture and fracture-dislocation of the cervical spine. In subsequent reports, however, this argument has been refuted and several cases have been reported in which cervical disc herniation with cord compression, was associated with traumatic hyperextension injury.^{6,10,13} Many of these cases involved vertebral fractures and/or obvious subluxation,¹³ but others did not.⁶

The mechanism of cord injury following cervical hyperextension, in the absence of vertebral fracture, has been the subject of debate for many years. It has been proposed that the damage was the result of the impingement of the cord from the forward bulging of the ligamentum flavum⁴ or shear loading by transiently subluxed vertebrae.¹ Marar³ noted that impingement by the ligamentum flavum alone was likely insufficient to cause cord damage but in the presence of osteophytes or subluxation may create a pincer mechanism of cord compression.⁸ McMillan and Silver⁵ noted a predisposition in those with narrowed spinal canals associated with spondylosis. Barnes² raised the possibility of a ‘traction’ type injury to the cord, and the notion of cord disruption from longitudinal tensile forces, together with torsional stresses, was introduced by Schneider *et al*.⁸

Cramer and McGowan,¹⁴ reporting on the autopsy findings of a patient who severed the cervical cord by acute protrusion of an intervertebral disc, proposed

Figure 1 (A) Midsagittal section of cervical spine (left side hemisection) showing rupture of anterior longitudinal ligament, disc disruption at C₃₋₄, C₄₋₅, C₅₋₆, including herniation of C₄₋₅, and discrete foci of central cord hemorrhage. (B) C₃₋₄ disc (right hemisection) protrusion causing extradural cord compression. (C) C₃₋₄ disc (left hemisection) herniation causing extradural cord compression and central cord hemorrhage

that when there is sufficient axial loading, there may be 'violent' protrusion of the disc by means of the hydraulic ram-like action of the nucleus pulposus or the rupture of the latter when subjected to sudden intense compressive force. However, Kinoshita⁷ disputed the feasibility of this mechanism noting that 'it was unlikely that the inelastic annulus and posterior longitudinal ligament could stretch sufficiently to allow dorsal displacement of the nuclear material and contusion of the cord without actual rupture'. He noted that there was no evidence to support this mechanism from autopsy studies.

The present case illustrates intervertebral disc (C₃₋₄) herniation, with intact posterior longitudinal ligament, extradural cord compression, and central cord hemorrhage evident in the vicinity of the C₃₋₄ interspace. Cord compression was also evident from the posterior superior edge of C₅. This pathology was associated with multiple ruptures of the anterior longitudinal ligament, indicative of hyperextension injury, and absence of any pathology normally associated with flexion injuries. These observations are therefore consistent with an extension injury and an injury vector that also involves intense axial loading sufficient to cause multiple disc failures, herniation and retro-pulsion leading to extradural disc compression. The possibility that there was concurrent involvement of a

pincer-like mechanism with transient vertebral shear forces, osteophyte encroachment, and/or bulging of the ligamentum flavum, cannot be excluded considering the demonstrated presence of osteophytes and the probable loss of stiffness in the ligamentum flavum by virtue of the patient's advanced age.

The cord pathology associated with cervical hyperextension, in the absence of vertebral fracture or subluxation, has previously been characterized as: (a) hematomyelia or hemorrhagic necrosis of the gray or white matter,^{8,10} or (b) white matter edema and loss of large diameter axons, without significant demyelination, primarily in the lateral funiculi^{6,15} and often extending over several segments.¹⁰ Central cord syndrome, with its clinical presentation of disproportionate weakness of upper limbs compared to lower limbs, bladder dysfunction and variable sensory loss,^{6,8} is frequently associated with these injuries⁸ and there remains some dispute as to whether this is a consequence of destruction of the central gray matter or is due to neuropathology in the corticospinal tracts in the dorsolateral columns.^{6,15} In the present case, the most evident cord pathology consisted of three discrete focal points of hemorrhage within the central gray matter located at levels corresponding to the three disrupted discs. This suggests three discrete points of compression, as distinct from a single focal point of

compression, with associated cephalad and caudad extensions of pathology, and would therefore tend to rule out the longitudinal compression mechanism,¹⁰ or 'traction'² or tensile force mechanisms,⁸ that have been previously proposed.

In summary, this report describes an unusual case of extradural cord compression associated with traumatic disc protrusion and herniation, following a cervical hyperextension injury in which there was no vertebral fracture or residual subluxation. The cord pathology, evident as discrete foci of hemorrhage at sites corresponding to disc disruption, is suggestive of either contusion as a result of transient disc retropulsion (coupled with compression from the herniated C₃₋₄ disc), or possibly a pincer-like process involving transiently retroluxed vertebrae. The cord pathology is not consistent with the notion of a central longitudinal compression or tensile force disruption of the central gray matter.

Acknowledgements

This work was supported by the Sir George Bedbrook Award of the International Spinal Research Trust and a grant from The University of Western Ontario. Dr Hayes was a recipient of the Robin K Gray Research Fellowship at the University of Western Australia.

References

- 1 Forsythe HF. Extension injuries of the cervical spine. *J Bone Joint Surg Am* 1964; **46A**: 1792.
- 2 Barnes R. Paraplegia in cervical spine injuries. *J Bone Joint Surg Br* 1948; **30B**: 234–244.
- 3 Marar BC. Hyperextension injuries of the cervical spine. The pathogenesis of damage to the spinal cord. *J Bone Joint Surg Am* 1974; **56A**: 1655–1662.
- 4 Taylor AR. The mechanism of injury to the spinal cord in the neck without damage to the vertebral column. *J Bone Joint Surg Br* 1951; **33B**: 543.
- 5 McMillan BS, Silver JR. Extension injuries of the cervical spine resulting in tetraplegia. *Injury* 1987; **18**: 224–233.
- 6 Martin D et al. MRI-pathological correlations in acute traumatic central cord syndrome: Case report. *Neuroradiology* 1992; **34**: 262–266.
- 7 Kinoshita H. Pathology of cervical intervertebral disc injuries. *Paraplegia* 1993; **31**: 553–559.
- 8 Schneider RC, Cherry G, Pantek H. The syndrome of acute central cervical spinal cord injury with special reference to mechanisms involved in hyper-extension injuries of cervical spine. *J Neurosurgery* 1954; **11**: 546–577.
- 9 White AA, Panjabi MM. *Clinical Biomechanics of the Spine*. Philadelphia: JB Lippincott, 1978; pp 158–162.
- 10 Jellinger K. Neuropathology of cord injuries. In: Vinken PJ and Bruyn GW (in collaboration with Braakman R) (eds). *Injuries of the Spine and Spinal Cord*. Part I. Amsterdam: Elsevier North-Holland, 1976, pp 43–119.
- 11 Woods A, Gaekwad UH, Kakulas BA, Smith ER. Establishment of a clinicopathological database for traumatic human spinal injury. *Paraplegia* 1991; **29**: 296–313.
- 12 Hayes KC, Kakulas BA. Neuropathology of human spinal cord injury sustained in sports-related activities. *J Neurotrauma* 1997; **14**: 235–248.
- 13 Davis SJ et al. Cervical spine hyperextension injuries: MR findings. *Radiology* 1991; **180**: 245–251.
- 14 Cramer F, McGowan FJ. The role of nucleus pulposus in the pathogenesis of so-called 'recoil' injuries of the spinal cord. *Surg Gynec Obstet* 1944; **79**: 516–521.
- 15 Quencer RMN et al. Acute traumatic central cord syndrome: MRI pathological correlations. *Neuroradiology* 1992; **34**: 85–94.