CASE REPORT

Thoraco-lumbar Spinal Cord Injury without Radiological Abnormality in an Adult

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Introduction

Spinal cord injury without radiological abnormality (SCIWORA) has been reported previously in both pediatric and adult patients, the cervical spine being the most commonly affected site¹⁻⁵. The thoracic spine is a biomechanically stable structure supported by costotransverse articulations and the rib cage, which increases axial loading capacity by three to four times. Furthermore, advancing rigidity of the aging spine in adult patients makes thoracic SCIWORA a rare scenario⁶⁻⁹. Although there are a few reports of upper thoracic SCIWORA in adults, we were unable to find any published reports of thoraco-lumbar SCIWORA in an adult⁶⁻⁹.

Case Report

A 21-year-old male student was referred to our hospital 2 days after being injured in a traffic accident. From his history and medical records we ascertained that he had been able to walk immediately after the accident for about 30 min, after which his sensation and motor power in both lower limbs gradually deteriorated, being lost completely within 40 h of injury. His blood pressure was well maintained and he had no medical comorbidities. He had not received methyl prednisolone at the primary level referral hospital.

At the time of admission to our hospital, he had no power in both lower limbs with normal sensation down to the T₁₀ level. There was complete sensory loss below L₂ with a zone of partial preservation from T₁₁ to L₁. There was no perianal sensation associated with loss of anal sphincter tone and bowel-bladder control (American Spinal Injury Association impairment scale-A). He had severe contusions on the fronts of both thighs, the pelvis and lumbar region. His mid-lumbar area was tender but there was no tenderness of the thoracic spine or chest. Radiographs of chest and spine and CT scans were normal. There were no associated injuries to any other part of his body.

An MRI scan performed 24 h after injury at the primary trauma centre showed hyperintense signals at T₁₂-L₁ level in T₂ weighted images but isointense signals in T₁ weighted images, indicating cord edema. There were no signal changes in the vertebrae, intervertebral discs or surrounding ligaments. The MRI also showed subcutaneous hematoma in the lumbar region (Fig. 1). Medical records showed that at the time of MRI his level of neurological impairment was L₁; this had gradually worsened to the T₁₀ level by the time of admission to our institution. Because his neurological level at the time of admission did not correlate with the MRI findings, we ordered a repeat MRI scan 3 days after injury, which showed cord edema extending from the T₈-L₁ level (Fig. 2). The repeat MRI also showed no abnormalities in the vertebrae, intervertebral discs, surrounding ligaments or muscles. Dynamic radiographs done under supervision 1 week after injury did not show any instability (Fig. 3).

During hospitalization, the patient was treated with analgesics to control pain, low molecular weight heparin for deep vein thrombosis prophylaxis, mild laxatives to facilitate bowel rehabilitation and clean intermittent catheterization for bladder rehabilitation. He underwent rehabilitation at our center for 2 months and at final follow-up of 6 months after injury no neurological recovery had occurred.

Discussion

Since the original description of SCIWORA by Pang and Wilberger in 1982¹ and following the advent of MRI, the definition of SCIWORA has been constantly evolving. Pang and Wilberger described it as any spinal cord injury with no abnormalities detected on plain radiographs or CT¹. Two decades after its first publication, Pang reviewed their report and stressed the importance of MRI². In this second article, in order to make the diagnosis less ambiguous, Pang excluded any compressive lesions of the spinal cord and pathological

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Fig. 1 T₂ weighted image 24 h after injury showing hyper-intense signal at the T₁₂-L₁ level. Note the skeletal and soft tissues show no signal changes, nor does the T₈-T₁₁ spinal cord.

abnormalities in extraneural supporting structures detected on MRI from the definition of SCIWORA, labeling them as “non-SCIWORA spinal cord injury”. However, Pang continued to include intraneural lesions such as cord edema and hemorrhage, labeling these as “true-SCIWORA”. Pang concluded that MRI did not invalidate his original acronym because, in a strictly scientific sense, MRI does not involve radiation. Although in recent years there has been a proposal to revise the definition further to specify that “Real-SCIWORA” includes only spinal cord injury (SCI) without any abnormalities, including spinal cord abnormalities, on MRI^{10,11}, this new definition has not yet achieved widespread acceptance.

Although the description of SCIWORA was originally formulated because of its frequent occurrence in pediatric cervical spine trauma, for which there are multiple explanations¹⁻³, it was subsequently found that the syndrome also occurs in adults, its incidence being highest in elderly patients with cervical injuries following hyperextension injuries of spondylotic spines^{4,5}. However, it was not known to occur in the adult thoracic spine until the first report of this phenomenon was published in 1993⁶, approximately 11 years after its first description by Pang and Wilberger¹.

To date, there are only five published reported cases of thoracic SCIWORA in adults. In the first report in 1993, Hirsh

et al. described a patient with SCI at the T₆ level without any fractures but with MRI evidence of posterior ligament injury indicating a traumatic hyper-flexion injury⁶. The second report, in 2002, described two cases. In the first, the SCI was at the T₅ level with a fracture of the T₄ spinous process and fracture dislocation of the costovertebral joints of T₄ and T₅. The other case in the same report had a T₄₋₅ injury with multiple rib fractures and posterior ligament damage identified on MRI. Both cases had evidence of hyper-flexion injuries⁷. The third report was published in 2004 and described a hyperextension injury leading to T₆₋₁₀ cord edema with T₉₋₁₀ cord hemorrhage without any extra-neural findings on radiographs, CT or MRI⁸. The final reported case in 2009 had a SCI at the T₄ level with MRI evidence of contusion in several vertebrae and avulsion of the T₄ nerve root, which was cited as a possible mechanism for spinal cord traction injury⁹.

Several possible causes of thoracic SCIWORA are proposed in published reports:

1. Hyper-flexion injury, as in seat belt injuries, leading to direct damage to the spinal cord via its distraction and also because of intersegmental instability attributable to the elasticity of the vertebral column in children².
2. Hyper-extension causing shortening of the cord and leading to its thickening. Compounding this, bulging of interlaminar ligaments into the spinal canal causes narrowing of the canal. According to the Taylor-Blackwood mechanism, this makes the cord susceptible to injury by reducible retrolisthesis of vertebra during hyperextension^{2,12}.

Fig. 2 T₂ weighted image 3 days after injury showing extension of cord edema from the T₈ to L₁ level. Note the subcutaneous hematoma in the lumbar region indicating a powerful force around the lower back and pelvis.

Fig. 3 Dynamic radiograph showing no signs of any bony injury or instability.

3. Ischemic injury due to watershed vascular supply of the cord and hyper-flexion causing spasm or damage to the anterior spinal artery or artery of Adamkiewicz; the latter is particularly important for the lower thoracic spinal cord^{7,13–15}.
4. Direct trauma as occurs in vehicular accidents². It is also postulated that direct trauma to the spine that is insufficient to cause skeletal damage could set up shock-wave-like oscillations in the cord that result in the cord slapping against the bony spinal canal, causing slapping damage^{5,11}.
5. Avulsion or traction along the spinal root as can occur during stretching of the sciatic nerve or any thoracic nerve root, leading to traction injury to the spinal cord^{9,16}.

Proposed Mechanism of Injury in the Present Case

Because the patient did not have any clinical signs of ligamentous laxity, hyper-flexion injury due to elasticity of spinal column is unlikely at his age. However, we propose that the large hematoma in his lower back confirms his statement that he was struck in the lower back from behind by a moving car while standing, causing extension of his lower back and pelvis. At the same time, because the patient was facing a stationary car in front of him, he received contusions on the fronts of both thighs, indicating violent hyperextension of his hips leading to traction on the femoral nerves. These traction forces thus were transferred proximally via the relatively mobile and elastic cauda equina to be expressed in the relatively more fixed

segments proximal to the conus. Two more findings support our contention. First, absence of any tenderness at the T₈–L₁ level rules out any forces acting at that level to cause injury and second, most reported cases of thoracic SCIWORA have evidence of rib cage injury indicating an injury severe enough to counter the protective rigidity of the rib cage, thus causing a hyperflexion injury to the spinal cord^{7,9}. There was no evidence for this in our case. Samsani *et al.* described a similar mechanism in a 17-year-old boy with thoracic SCIWORA at the T₁₀ level following a traffic accident¹⁶. This mechanism also directs our attention to possible traction injury to the spinal cord due to sudden violent hyperextension despite absence of tethering.

Extension of cord edema from the T₁₂–L₁ to the T₈ level and deterioration in his neurological signs and symptoms may be attributable to either of the following two factors. Firstly, secondary ischemic injury to the spinal cord is known to cause worsening neurological manifestations¹⁴. However, ischemic injury most commonly leads to anterior spinal artery syndrome, which is characterized by loss of motor power, pain and temperature with preservation of light touch, vibration and proprioception^{17,18}. Because our patient had complete loss of sensation below the level of the injury, ischemic injury is a remote possibility. Secondly, occult instability causing recurrent spinal injury is another possible cause for our patient's worsening neurological manifestations, although the latter proposition is contentious^{19,20}.

The main predictor of long-term neurological outcome in patients with SCIWORA is the neurological status at admission². Our patient presented to us 2 days after injury, by which time he had neurologically complete paraplegia. The fact that his paraplegia was neurologically complete probably explains why no recovery had occurred even six months after the injury. We did not consider giving him methyl prednisolone as he presented to us well after the recommended duration for this therapy of within 8 h of injury²¹.

The present case is unique because all the reported cases of SCIWORA in adults have involved only the thoracic spine, whereas our patient is the first reported case of SCIWORA at the thoraco-lumbar junction in an adult.

Four of the five reported cases of thoracic SCIWORA involved the upper thoracic spine. This might be due to: (i) its watershed zone of vascular supply; and (ii) distraction forces have maximum impact on the upper thoracic cord because of the distribution of the exiting nerve roots and dentate ligaments². Our patient's SCI began at the thoraco-lumbar junction. At this level, the spinal cord has reasonable mobility and space to escape injury compared to the upper thoracic spinal cord. However, our patient still developed an injury; this difference can only be explained by traction injury via the femoral nerves.

Though SCIWORA is common in the cervical spines of children, its uncommon presentation in adults at the thoraco-lumbar junction should be kept in mind. This case directs our attention to a rare scenario of "true SCIWORA" at the thoraco-lumbar junction with worsening neurological deficits in an adult patient.

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