# Traumatic Spinal Cord Injury Without Initial MRI Abnormality: SCIWORA Revisited

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Spinal cord injury without radiographic abnormality (SCIWORA) is a concept first introduced by Lloyd in 1907[1]. In 1982, Pang and Wilberger defined SCIWORA as an acute spinal cord injury including trauma to nerve roots that may result in sensory or motor deficit, or both, with normal findings on plain radiographs and computed tomography (CT) scans[2]. Spinal cord injury without radiographic abnormality is an uncommon syndrome. Ninety percent of described cases occur in the pediatric population[3]. However, a recent study suggests that SCIWORA in adults is underreported[4].

The diagnosis of SCIWORA can be difficult, primarily because it is a rare syndrome and because patients present with a broad array of symptoms. It is most often caused by trauma, such as motor vehicle collision, fall from a height, sports injury, and child abuse[3]. Typically it has a grave prognosis. Treatment is usually nonoperative[5]. Spinal cord injury without radiographic abnormality should be considered in the differential diagnosis of any patient with symptoms of traumatic myelopathy and normal radiographic and CT studies.

Infants and children under eight years are most susceptible to SCIWORA, followed by adults over age 60. Spinal cord injury without radiographic abnormality is exceedingly rare among young adults (16-35 years-old)[2,6].

We present here the unusual case of an 18-year-old male who presented with paraplegia and no evidence of spinal cord injury on initial CT and magnetic resonance imaging (MRI) following a motor vehicle collision. Repeat MRI five days post-injury demonstrated a spinal cord injury corresponding to his neurologic presentation. At eight months post-collision, the patient has recovery of sensation and ambulates independently.

## Case Report

This 18-year-old male forestry climber was the seat-belted driver in a single car accident. He self-extracted from the vehicle but was unable to ambulate. He had not been drinking. On arrival he was awake, alert, and hemodynamically stable with a Glasgow Coma score of 15. Neurological exam revealed bilateral paralysis of the lower extremities and loss of sensation from the level of T4 downwards. His American Spinal Injury Association (ASIA) impairment scale at presentation was C with some variability in reports. Upper extremities were unaffected. His lower extremities were hyperreflexic bilaterally without clonus. No priapism was described but rectal tone was decreased. His spine was tender from T1-T4 and L4-L5.

His chest x-ray and CT revealed bilateral fractures of ribs 1 and 2, bilateral pulmonary contusions, and a left pneumothorax resulting in air accumulation in the upper mediastinum and spinal canal. The CT scan of the spine was normal except for a small amount of air in the ventral epidural space, posterior to the C7 vertebral body (Figure 1), tracking in from the pneumothorax.

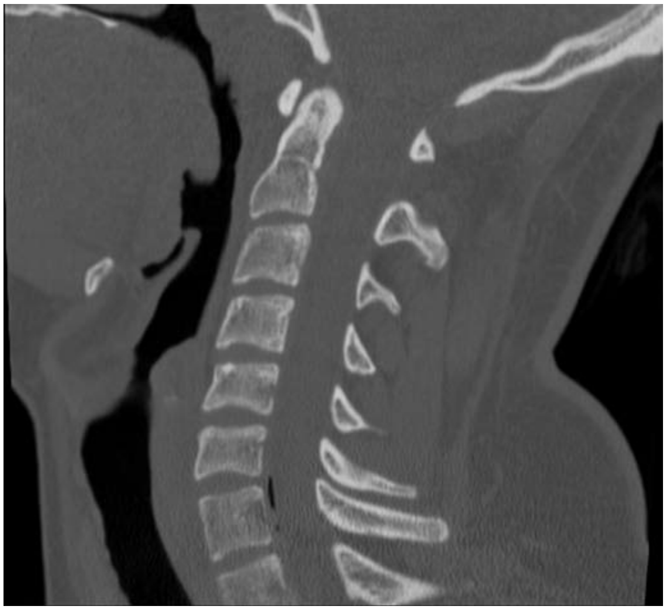

***Figure 1:*** *Sagittal CT reformatted image in bone windows upon presentation demonstrates normal alignment and no fracture. There is a small amount of epidural gas tracking from a pneumothorax (not shown).*

The day of his injury, MRI of the thoracic and cervical spine was performed with sagittal and axial T1, T2, and proton density images using a Siemen's 1.5T Magnetom Avanto. No abnormal signal or deformity was present in the spinal cord. T2 imaging showed a very small epidural hematoma (2.4 cm craniocaudally, 2 mm in width) posterior to the vertebral body of C7, in the epidural space, not compressing the spinal cord, with no

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***Figure 2:*** *Sagittal T2 MRI sequences performed upon presentation reveals a normal cervico-thoracic spinal cord. Note the focal ligamentum flavum prominence at T3-4, and the tiny ventral epidural hematoma at C7.*

associated intrinsic cord signal abnormality (Figure 2). Imaging was otherwise normal, apart from ligamentum flavum thickening at T3-T4 with no effect on the spinal cord.

Because of the discrepancy between the patient's clinical findings and the absence of spinal cord injury on the initial scan, a repeat MRI scan was performed five days after the collision. Further sequences included diffusion weighted imaging and gradient refocused echo. The follow-up MRI revealed new abnormal cord signal at the same level of the ligamentum flavum thickening (T3-T4), representing edema and measuring 10 mm in size (Figures 3A, 3B). The diffusion sequence was negative for acute infarction. The gradient refocused echo did not show any blood products (Figure 4). The epidural hematoma seen on the initial MRI had resolved, as had the air seen on CT scan.

Over two weeks, the patient gradually regained motor and sensory function. At discharge, the patient had near normal sensation. He was transferred to a rehabilitation hospital before discharge home. Eight months post-collision, the patient ambulates without assistance. He takes ibuprofen for back pain. Neurological exam reveals that his lower extremities are stiff and somewhat clumsy. He experiences allodynia over his lower extremities and chest. Patellar reflex is 3+ and his Achilles tendon reflex is 4+ with three beats of clonus. He has returned to work part-time as a forestry climber but is unable to drive.

## Discussion

When SCIWORA was originally defined in 1982, MRI scanners were not commonly used. Therefore, the original definition makes no comment about MRI. Pang and Wilberger, the neurosurgeons who coined the term 'SCIWORA', outlined it as a syndrome affecting patients with objective signs of myelopathy due to trauma. In their definition, patients must have normal plain films, tomography, and occasionally myelography of the spine, on admission[2].

In 2004, Pang revised his original definition to include MRI findings. He argued that MRI abnormalities prove the existence of SCIWORA and that MRI can be used to confirm a suspected diagnosis of SCIWORA[7]. However, normal MRI scans do not rule out SCIWORA. One of the major findings of his 2004 study

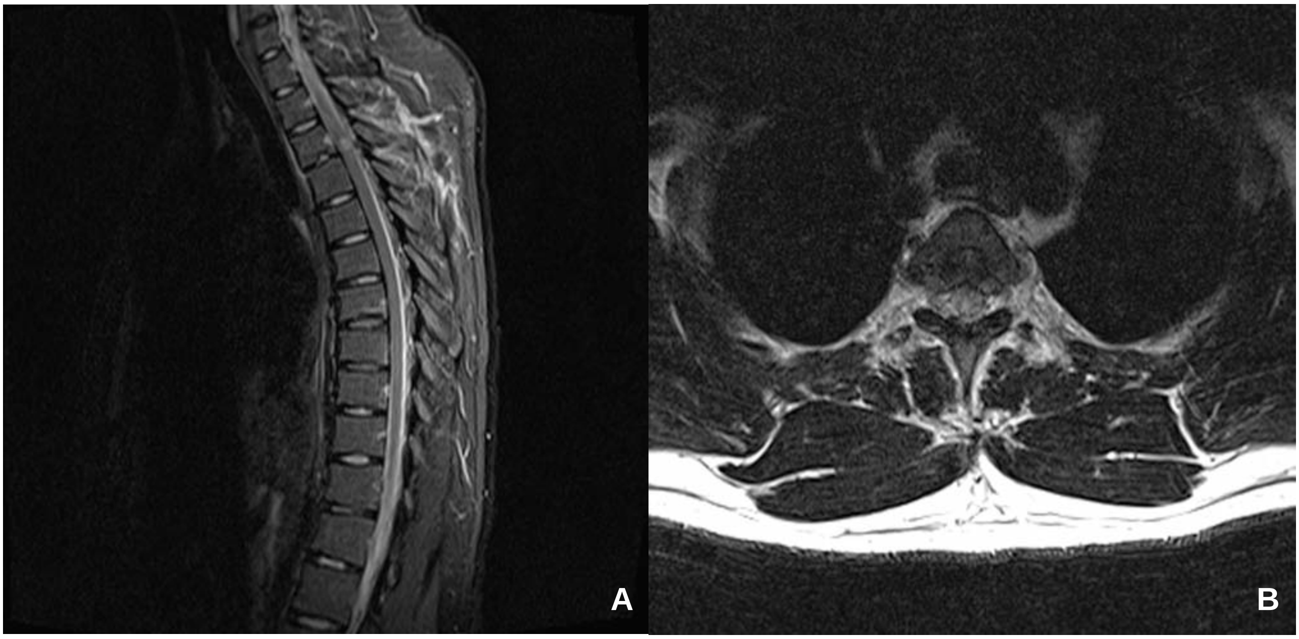


***Figure 3:*** *Sagittal STIR (Figure 3A) and axial T2 MRI (Figure 3B) sequences performed on day 5 reveal a spinal cord abnormality now corresponding to his clinical findings at the T3-4 level.*



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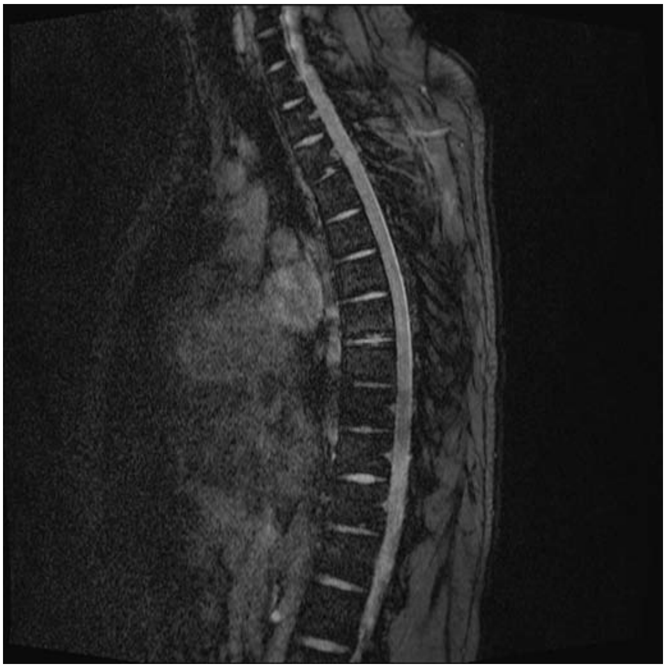

***Figure 4:*** *Gradient echo sequence shows there is no hemorrhage associated with the cord contusion at T3-T4 (shown in Figures 3A, 3B).*

is the utility of MRI in the prediction of a patient's outcome after SCIWORA.

In the original study by Pang and Wilberger, they found that the most important prognostic factor in patients with SCIWORA was their neurological status at admission. Twelve of the 14 children (85.7%) who presented with complete or severe cord syndromes either died or were left completely disabled[2]. However, more recently, Pang published that patients with SCIWORA and normal MRI scans tend to have excellent prognoses for recovery[7]. In fact, he concluded that the best predictor of outcome in SCIWORA is the result of the MRI scan, and not the patient's neurological status at presentation. This corresponds to the outcome that our patient experienced.

In 2008, Yucesoy and Yuksel argued that the term 'SCIWORA' had become ambiguous. They declared that since MRI had become a commonly-used tool for investigating spinal injuries, patients should not be given the diagnosis of SCIWORA unless their MRI at presentation is normal as well. They stated that plain radiography and CT are neither sensitive nor specific enough to detect spinal cord injury, while MRI can detect these lesions. They claimed that cases labelled as SCIWORA but without normal MRI should be rejected, and thus that the true incidence of SCIWORA is much lower than reported. Furthermore, they suggested abolishing the term 'SCIWORA' and redefining the syndrome as 'Spinal Cord Injury Without Neuroimaging Abnormality'[8]. As early as 1999, Gupta et al suggested that the term SCIWORA was likely to become redundant[9].

Regardless of whether one accepts or rejects the original definition of SCIWORA, we feel the patient described here is unique and constitutes a case of SCIWORA, with no evidence of spinal cord injury on radiographs, CT scans, or MRI at presentation. Our patient was 18-years-old, placing him in the age group in which SCIWORA is the least likely (16 years to middle age)[2,6]. Additionally, the thoracic spinal column is the segment that is least likely to be injured in SCIWORA, especially between T1-T6[7]. This makes this patient's injury at T3-T4 even more unusual.

In terms of the mechanism of injury in our patient, we speculate that the ligamentum flavum thickening and resultant canal narrowing shown at T3-T4 predisposed the spinal cord to injury. With the trauma of the motor vehicle accident, the patient may have transiently compressed his spinal cord at this level. It took several days for this injury to manifest on MRI. Therefore, although his initial imaging showed normal intrinsic cord signal, his follow-up T2 and STIR MRI images revealed a spinal cord lesion. This allowed us to rule out conversion disorder as the cause of his paralysis. This MRI finding underscores the importance of performing short interval follow-up MRI studies to assess for delayed presentation of pathology in cases of SCIWORA.

In conclusion, our patient is a young adult who suffered paraplegia following a motor vehicle collision and was given the diagnosis of SCIWORA. He had normal MRI scans at presentation but repeat MRI scan five days later showed a spinal cord lesion corresponding to his clinical deficits. Recovery was nearly complete by eight months later. His case stands out from the literature in terms of his age, the spinal level of his injury, the results of his MRI scan, and his favourable recovery.

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