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Blunt Traumatic Posterior Cord Syndrome Following a Hangman Fracture: A Case Report

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Learning Point of the Article:

Posterior cord syndrome after blunt cervical trauma is easy to miss unless proprioception, vibration, and two-point discrimination are tested specifically, and a delayed MRI may be needed to confirm the anatomical diagnosis once the acute cord edema has settled.

Abstract

Introduction: Posterior cord syndrome (PCS) sits outside the five incomplete spinal cord injury (SCI) syndromes formally recognized by the International Standards for Neurological Classification of SCI, and carries a reported incidence below 1%. Because routine neurological screening often skips detailed dorsal column testing, PCS is thought to be substantially under-diagnosed, and traumatic PCS in particular has only been described in a handful of published reports, nearly all in the cervical spine.

Case Report: A 24-year-old man sustained a hyperextension injury of the cervical spine in a road traffic accident, presenting with whole-body paresthesia, neck pain, and asymmetric upper-limb weakness. Computed tomography demonstrated a C1 transverse process fracture, a hangman fracture with associated lamina, spinous process, and teardrop fracture components of C2, and a C3 spinous process fracture without subluxation. Magnetic resonance imaging (MRI) showed faint diffuse cord edema and anterior longitudinal ligament disruption at C2-C3. He underwent anterior cervical interbody fusion at C2-C3 with halo immobilization and made a near-complete neurological recovery over 1 year, retaining only mild residual proprioceptive deficits in the hands and feet. Follow-up MRI obtained 10 months after injury anatomically localized the injury to the posterior columns, confirming PCS.

Conclusion: This case shows how traumatic PCS can be overlooked on early imaging and only becomes anatomically apparent on delayed MRI. A structured sensory examination that specifically tests proprioception and vibration, combined with repeat imaging when symptoms persist, is essential for correctly identifying this rare and likely under-recognized injury pattern.

Keywords: Posterior cord syndrome, spinal cord injury, hangman fracture, proprioception, cervical spine trauma, incomplete spinal cord injury.

Introduction

Spinal cord injury (SCI) affects approximately 11,000 people annually in the United States [1,2]. The extent of cord involvement varies widely between patients, and the International Standards for Neurological Classification of SCI (ISNCSCI) formally recognizes five incomplete SCI syndromes: Central cord syndrome, Brown-Sequard syndrome, anterior cord syndrome, conus medullaris syndrome, and cauda equina syndrome [3]. Even syndromes with a well-established clinical picture can present atypically. A recent report described Brown-Sequard syndrome following only minor trauma in a patient with an underlying cervical segmentation anomaly, a reminder that these classification patterns describe a spectrum rather than fixed rules, and that careful modality-by-modality

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sensory and motor testing matters more than the mechanism of injury alone [4].

Posterior cord syndrome (PCS) is not separately classified within the ISNCSCI system, and its reported incidence is below 1%, a figure that probably reflects under-recognition as much as true rarity [1,5,6,7]. Clinically, PCS produces loss of proprioception, vibration sense, two-point discrimination, and deep pressure sensation below the level of injury, occasionally with pain or paresthesia, while motor power, light touch, and pinprick sensation are relatively preserved [2,3,8,9]. Reported causes include posterior spinal artery ischemia, tumor, disc herniation, Vitamin B12 deficiency, and trauma [6,10,11]; iatrogenic injury has also been described, including a case of PCS following electrode lead placement during spinal cord stimulation for chronic pain.[12] Traumatic PCS is the least common of these etiologies and is generally attributed to a hyperextension mechanism [11,13]. Recognizing PCS early also has practical consequences: The sensory ataxia it produces can be mistaken for a non-organic complaint or for a coexisting peripheral neuropathy, which delays rehabilitation planning that specifically targets proprioceptive retraining rather than motor strengthening alone. We report a case of blunt traumatic PCS following a hangman fracture, with the aim of sharpening clinical suspicion for this injury pattern and clarifying the role of delayed imaging in confirming the diagnosis when acute studies are inconclusive.

Case Report

A 24-year-old man with a history of depression and prior appendicectomy, and an otherwise unremarkable family and social history, presented after a road traffic accident. He reported paresthesia involving his entire body, neck pain, and subjective weakness of the right arm. Computed tomography of the cervical spine identified a right C1 transverse process fracture, a hangman fracture of C2 with associated lamina, spinous process, and teardrop fracture components, and a C3 spinous process fracture without subluxation (Fig. 1).

Neurological examination followed a standard ISNCSCI protocol, testing motor power, light touch, pinprick, joint position sense, and vibration in each limb. The findings were consistent with an incomplete SCI predominantly affecting the dorsal columns: paresthesia extended from the neck distally to involve joint position sense bilaterally in both upper and lower limbs. Strength testing graded his right arm at antigravity power, his left hand showed only minimal weakness, and both legs were full strength. Because arm weakness was present without corresponding leg weakness, central cord syndrome was also considered in the differential diagnosis at this stage. Cervical magnetic resonance imaging (MRI) showed faint, diffuse cord edema together with disruption of the anterior longitudinal ligament at C2-C3 (Fig. 2), but it did not clearly localize the injury within the substance of the cord itself.

The risks and benefits of operative stabilization with halo immobilization were discussed at length and weighed against a trial of collar immobilization alone. The patient elected to proceed with surgery and gave informed consent for both the operation and the halo orthosis. He underwent anterior cervical interbody fusion with plating at C2-C3, supplemented with a halo orthosis, tolerated the procedure without swallowing difficulty, and began physical therapy and mobilization soon afterward. Because his deficit was primarily sensory rather than motor, his rehabilitation program was weighted toward balance training and proprioceptive re-education alongside standard mobilization, rather than the strength-focused protocol that would typically follow a motor-predominant cord injury.

At his first outpatient review, he had no measurable motor deficit but showed slight impairment of fine finger dexterity. By 3 months postoperatively, mild balance dysfunction persisted alongside improving dysesthesia, and he was ambulating independently without walking aids. At 1 year, his neurological examination was essentially normal apart from minor residual

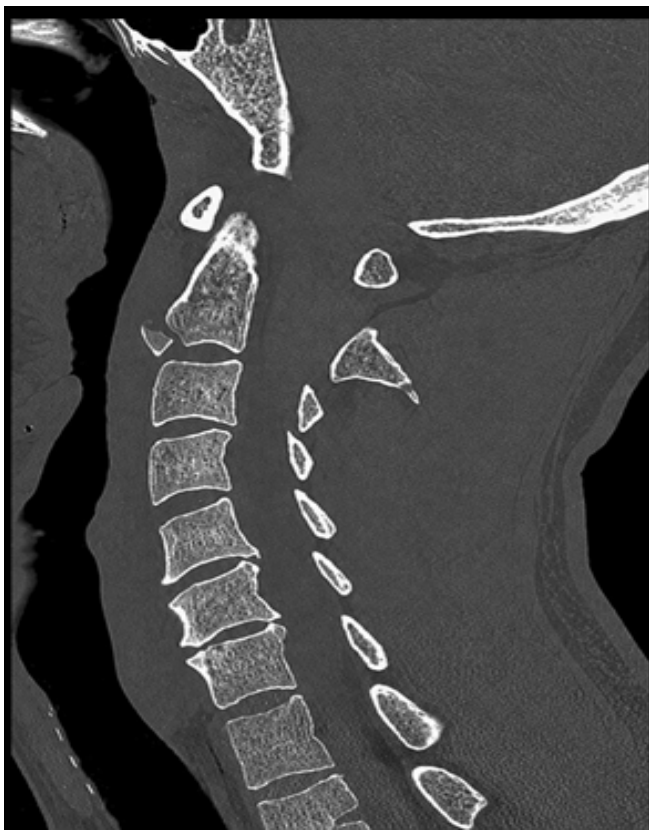

Figure 1: Sagittal computed tomography of the cervical spine demonstrating a right C1 transverse process fracture, a C2 hangman fracture with lamina, spinous process, and teardrop fracture components, and a C3 spinous process fracture without subluxation.

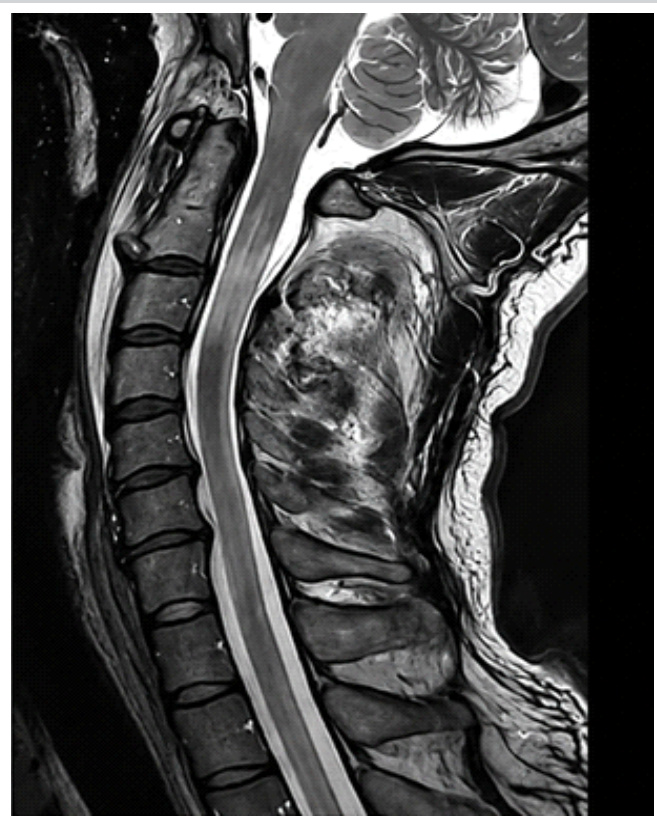

Figure2: Initial post-traumatic cervical spine magnetic resonance imaging. Sagittal view show faint, diffuse spinal cord edema, and disruption of the anterior longitudinal ligament at C2-C3.

proprioceptive loss in the hands and feet; this residual numbness did not interfere with his daily activities, and nerve conduction studies performed at that visit were normal. He continued to describe some anxiety about the residual weakness, though objectively his function had returned close to baseline. A follow-up cervical MRI without contrast, obtained 10 months after the injury, clearly delineated the injury to the posterior columns (Fig. 3a and b), anatomically confirming a posterior SCI and establishing the diagnosis of PCS in retrospect.

Discussion

Traumatic PCS remains uncommon in the published literature. Most reported cases of PCS are atraumatic in origin [5,8,11], and among the small number of traumatic cases, we identified, all but one involved the cervical spine [2,5,9,13,14,15]. Hyperextension is the mechanism most often implicated [1,13,15], and both hangman fractures and teardrop fractures of the axis, as seen in our patient, are recognized hyperextension injuries [1,7]. Only two prior reports specifically discuss surgical management of traumatic PCS [13,15], which makes the operative course and 1-year outcome described here a useful addition to a thin evidence base.

Belen and Weingarden described a 41-year-old man injured in a motorcycle collision who presented with paresthesia and bilateral upper- and lower-limb weakness [15]. His examination differed from ours in that weakness was bilateral rather than confined to one side, pinprick sensation was lost below C5, and rectal tone was reduced, although proprioception was similarly absent distally, with milder loss proximally at the shoulders. A cervical myelogram in that patient showed stenosis, and he underwent C4-C7 laminectomy; by 3 months, strength had recovered to 4/5 in all limbs, though proprioception and vibration remained absent in the right leg and only partially returned on the left. The authors attributed the upper-limb weakness to a coexisting central cord component, a combination that appears to be common after hyperextension injury.

Steidl reported a construction worker struck on the head by a falling wooden block, who developed tingling in the neck and shoulders followed by severe bilateral hypersensitivity in the C3-C4 dermatomes, without any radiographic abnormality [9]. His symptoms resolved spontaneously within 3 weeks. Because imaging was normal, Steidl proposed that the posterior gray matter is particularly vulnerable to contusion, and that transient perivascular bleeding causing limb paralysis lasting up to 4 h could occur through excessive anteroposterior motion of the cord rather than direct bony injury. The same author suggested that PCS results from cord compression by inward buckling of the ligamentum flavum and ventral osteophytes, a proposed mechanism distinct from that of central cord

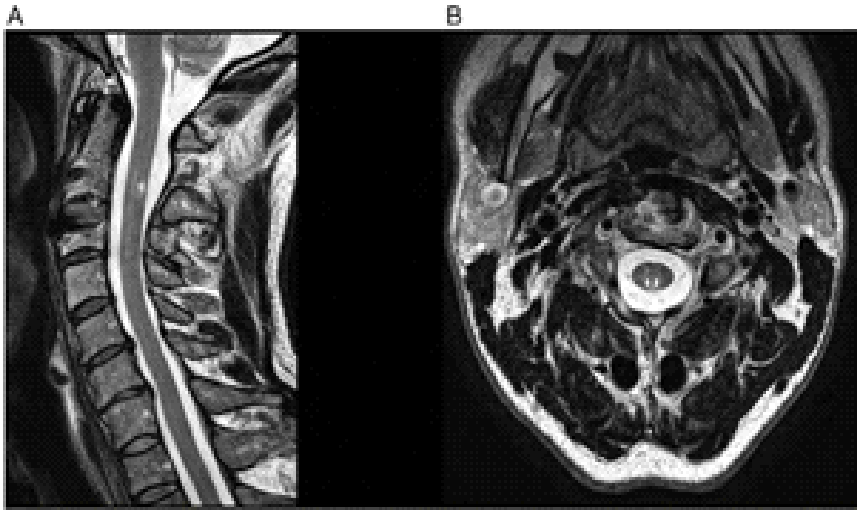

Figure 3: Follow-up cervical spine magnetic resonance imaging obtained at 1 year. Sagittal (a) and axial C3-level (b) images clearly demonstrate a spinal cord injury confined to the posterior columns, anatomically confirming posterior cord syndrome.

syndrome [8].

Distinguishing PCS from central cord syndrome, and more broadly from other incomplete SCI patterns, is not always straightforward at initial assessment, since arm-predominant weakness can arise in either condition and both are common consequences of hyperextension injury. A recently published case from the Journal of Orthopedic Case Reports illustrates this overlap well: A 63-year-old man with C5-C6 bilateral facet dislocation presented with quadriparesis heavier in the upper than the lower limbs, a pattern classically ascribed to central cord syndrome, and was managed successfully with staged posterior and anterior stabilization after closed reduction [16]. Our patient's asymmetric arm weakness raised the same differential early on, and it was only the specific pattern of sensory loss, together with the appearance of the follow-up MRI, that ultimately pointed to a posterior rather than a central lesion. The anatomical basis for this distinction is worth stating plainly: Central cord syndrome reflects damage concentrated in the central gray matter and medially situated corticospinal fibers serving the upper limb, whereas PCS reflects damage confined to the dorsal white matter columns carrying proprioceptive and vibratory input, with motor pathways relatively spared. Both patterns can coexist after a single hyperextension event, and where they overlap, as in the Belen and Weingarden case discussed above, the resulting picture may not fit cleanly into either textbook category. Incomplete SCI syndromes can also emerge from unexpected mechanisms, as in the Brown-Sequard case described above, which is a further reminder that the physical examination, and not the mechanism of injury on its own, should drive the diagnosis [4].

Other rare SCI patterns make the same point: Clinical findings and imaging findings do not always evolve at the same pace. White cord syndrome, a reperfusion injury first described after decompressive surgery for chronic cord compression, produces new post-operative deficits accompanied by delayed T2 hyperintensity on MRI that can be mistaken for a fresh structural injury rather than recognized as a consequence of sudden reperfusion to chronically ischemic tissue [17]. Anterior spinal cord syndrome from fibrocartilaginous embolism can similarly mimic transverse myelitis on early imaging, with a vascular pattern only becoming apparent once inflammatory and demyelinating causes have been excluded and the clinical course is reviewed in full [18]. In each of these entities, as in the case reported here, imaging obtained too early in the clinical course can under-represent, or simply fail to localize, the true anatomical extent of injury, and a period of watchful reassessment with repeat imaging is often what finally establishes the diagnosis. This is a practical argument for building a planned follow-up MRI into the management pathway of any patient with a clinically incomplete cord syndrome and an acute study that does not fully explain the examination findings, rather than treating a normal or non-specific early scan as reassurance that no structural injury is present.

PCS is a rare subset of SCI that can be missed entirely if vibration and proprioception are not specifically tested during a standard ISNCSCI examination [2,6,18]. In our patient, MRI performed in the acute phase failed to delineate the injury clearly within the substance of the cord, so PCS could easily have gone unrecognized at that stage. Patients with PCS frequently exhibit a positive Romberg sign, which is sometimes misattributed to neurosyphilis [2,3,5], and because PCS sits outside the formal ISNCSCI syndrome classification, it is arguably even more prone to being overlooked than the five recognized syndromes [1,3,6]. What sets our case apart is that we were able to confirm the site of injury anatomically to the posterior columns on a follow-up MRI obtained a year after the initial presentation, an anatomical correlation that most of the prior reports we reviewed did not achieve.

Conclusion

This report describes PCS diagnosed after blunt trauma to the axis, with near-complete resolution of symptoms following cervical fusion at C2-C3. A thorough neurological examination, with deliberate attention to proprioception and vibration rather than pinprick and light touch alone, is central to recognizing PCS, since routine screening will otherwise miss it. Where the acute MRI is inconclusive, as it was here, we suggest that a sub-acute or delayed study, obtained once the initial cord edema has settled, can help confirm the anatomical diagnosis and guide counseling about long-term recovery. For spine surgeons managing patients with predominantly sensory deficits after cervical trauma, this case supports setting expectations around a slower, proprioception-focused recovery trajectory rather than assuming that a normal early motor examination and an unremarkable acute scan rule out a clinically significant cord injury.

Clinical Message

Arm-predominant weakness after cervical hyperextension trauma is not always central cord syndrome. When proprioceptive and vibratory loss is disproportionate to motor findings, clinicians should specifically examine the dorsal columns and consider PCS, repeating MRI later in the clinical course if the initial study does not fully explain the findings.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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