Case Report

Triple trouble: A case of traumatic cervical spinal cord injury in a patient with ossification of posterior longitudinal ligament and disc prolapse

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1. Introduction

Traumatic spinal cord injuries is a devastating event that often leads to significant long-term neurological morbidity. Every trauma patient is unique in terms of the mechanism of injury, individual anatomical factors, and the complex interactions between the two. Trauma victims may have underlying spinal pathologies that predispose them to certain injuries. One such pathology is the presence of ossification of the posterior longitudinal ligament (OPLL), which leads to alteration of the spine biomechanics. OPLL is very often a subclinical condition which can predispose trauma victims to devastating neurological injuries. Despite the severe complications conferred by this association, there is a lack of awareness about OPLL and its implications in trauma patients. Our aim is to highlight one such case of OPLL with coexisting cervical disc prolapse, which resulted in severe cervical cord injury after trauma.

2. Case presentation

A 45-year-old male presented to our ED with a history of progressive tetraparesis following a road traffic accident that had occurred two weeks prior. He was the restrained driver of a coaster involved in a low-impact, rear-end collision with a light vehicle. The impact resulted in hyperextension and rotatory movement of his neck. Immediately after the trauma he developed neck pain with tetraparesis. There was no history of head injury or loss of consciousness. There were no associated chest or abdominal injuries. In retrospect, the patient admitted that he had been suffering from long-standing neck stiffness and arm pain with associated numbness in his hands. His symptoms had been progressing gradually
over the past few years. He did not seek any medical advice for his symptoms.

Immediately after the accident, cervical spine immobilization was performed using a hard collar, and he was transported to a nearby hospital. The initial CT of his cervical spine did not show any evidence of cervical spine fracture or dislocation. However, segmental ossification of the posterior longitudinal ligament was noted. He underwent spinal decompression surgery by the posterior approach (C3-C6 laminectomy) on the same day. Unfortunately, there was no significant neurological improvement after the surgical intervention, and he was referred to our center for further management.

At presentation his vitals were stable. There were no signs of respiratory compromise. He had increased muscle tone in both the upper and lower extremities with brisk deep tendon reflexes and Babinski’s sign bilaterally. The motor strength was 2/5 and 3/5 in upper and lower limbs, respectively. All sensory modalities were reduced up to the C4 spinal level. He had bladder and bowel incontinence. The patient’s neurological state at admission, as assessed by Frankel functional score was grade C.

His post-operative images were obtained (Fig. 1). CT scan of cervical spine showed the continuous type of OPLL involving the upper cervical vertebrae from C2-C6, which was encroaching upon the bony neural canal (Fig. 1A and B). MRI of the cervical spine showed multilevel posterior and posterolateral disc protrusions from C3-C4 down to C6-C7, causing a significant mass effect on the thecal sac as well as on the descending and exiting nerve roots due to associated OPLL. There was an intramedullary area of altered signal intensity seen involving the cervical cord segment opposite C5-C6 and a smaller one at C3-C4. These areas represented compressive myelopathic changes (Fig. 1C and D).

The patient was reviewed by our trauma team. It was decided to manage his case conservatively. He was referred to the physiatrists for a long-term rehabilitation program. Unfortunately, he did not make any marked clinical improvement on subsequent follow-up visits.

3. Discussion

The posterior longitudinal ligament is present along the posterior surface of the vertebral canal, extending from the clivus to the sacrum. It is composed of thick elastin and collagen fibers, and it functions to stabilize the vertebral bodies and prevent hyperextension of the spine.

OPLL is a condition characterized by abnormal calcification of the posterior longitudinal ligament. This process is postulated to start as fibroblastic hyperplasia with increased collagen fiber deposition. The ligament then undergoes mineralization and ossification and subsequently encroaches into the spinal canal. This may lead to compressive myelopathy.1

A significant proportion of patients with OPLL remain asymptomatic, whereas a small subset develop progressive myelopathy.3 Myelopathy manifests initially as neck pain with numbness and decreased dexterity in the hands. With progressive spinal compression, patients show signs of lower limb involvement.

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Fig. 1. A: Sagittal section of CT cervical spine. Red arrows are used to label the segmental pattern of ossified posterior longitudinal ligament (OPLL), spinal canal, and lamina. B: Axial sections of CT cervical spine showing an ossified mass protruding into the spinal canal. Red arrows are used to label show ossified posterior longitudinal ligament (OPLL) and spinal canal. C: Sagittal section of T2-weighted images of the non-enhanced MRI of the cervical spine with the red arrows demonstrating the intramedullary area of altered signal intensity seen involving the cervical cord segment opposite C3-C4 and a smaller one at C5-C6. D: Sagittal section of T1-weighted images of the non-enhanced MRI of the cervical spine with the yellow arrows demonstrating multiple areas of cervical disc prolapse at C3-C4 down to C6-C7.
Apparently, our patient also had past symptoms suggestive of myelopathy due to progressive cord compression.

OPLL can be diagnosed radiologically. CT is a good imaging modality for delineating the shape and size of an ossified mass. However, MRI is the preferred modality for improved visualization of myelopathic changes in the spinal cord. OPLL is classified according to the radiographic findings into the following types: local, segmental, continuous, and mixed. In the local type there is ossification of the PLL that is exclusively seen at the intervertebral disc spaces; in the segmental type there is ossification at the posterior aspect of each vertebral body; in the continuous type there is ossification over many segments; and in the mixed type there is a combination of any of the above findings.

Patients with mild, non-progressive symptoms are often managed conservatively. But once they develop progressive neurological symptoms or MRI evidence of increasing cord edema, they become candidates for surgical intervention. Two main surgical approaches are used for spinal decompression in such cases: an anterior approach to directly remove the ossified mass; or a posterior approach utilizing techniques such as laminoplasty and laminectomy. In general, patients with segmental OPLL benefit from the anterior approach, whereas those with more extensive OPLL benefit more from the posterior approach.

The presence of OPLL predisposes individuals to severe neurologic deterioration even after mild traumatic injuries. It is now thought that OPLL might be present in a clinically significant number of patients with traumatic cervical spinal cord injury, especially in the subgroup of patients who do not have evidence of bony injury. Two Japanese studies have estimated the incidence of OPLL to be 26%–38% in a population of patients with acute cervical cord injury without any evidence of fracture or dislocation of the spinal column. Unfortunately, the neurological insult acquired as a result of traumatic injuries is often not reversible, even after spinal decompression. This is also reflected in our case.

Our patient had radiological evidence of OPLL with cervical disc prolapse. There is an interesting association between the two pathologies. It is known that the posterior longitudinal ligament is firmly adherent to the annulus fibrosus. In OPLL the pathological changes in posterior longitudinal ligament also invade the annulus fibrosus, thereby causing its weakness and increasing the potential for disc prolapse. The combined effect of spinal canal stenosis due to OPLL and disc prolapse can have severe compressive effects on the spinal cord. This effect can be even more pronounced if there is a superimposed traumatic event, as demonstrated by our case.

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