Case report

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Thoracic compressive myelopathy due to post-traumatic intradural migration of ossified yellow ligament: Case report

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Abstract: Focal ossification of the ligament flavum (OLF) is a rare cause of thoracic myelopathy. The lower thoracic spine is most frequently affected. It is increasingly being recognized as a cause of myeloradiculopathy with advent of computed tomographic (CT) and magnetic resonance (MR) imaging of spine. We report an instance of ossified ligamentum flavum infiltrating and penetrating segmental dura as a result of trauma causing compressive myelopathy in a middle-aged female.

Keywords: compressive myelopathy, decompression laminectomy, ligament ossification, spinal cord compression, ossified yellow ligament, calcified ligamentum flavum.

INTRODUCTION

With the increasing use of computed tomographic (CT) and magnetic resonance (MR) imaging, ossification of the ligamentum flavum (OLF) is gradually being recognized as a major cause of acquired thoracic spinal canal stenosis after diffuse idiopathic skeletal hyperostosis (DISH) and ossified posterior longitudinal ligament (OPLL) [1, 2, 3]. We report an unusual case of post traumatic intradural extension of ossified yellow ligament.

CASE REPORT

A middle-aged woman presented with history of fall on back six months ago, following which she had developed severe back pain. There was no neurological deficit, and she was advised bed rest and analgesics. She had partial pain relief for next three months, after which she noticed ascending type of paraesthesia in both lower limbs accompanied by progressive stiffness of both lower limbs. She developed increased frequency and urgency of micturition over subsequent two months. Clinical examination revealed hypertonia and hyperreflexia of both lower limbs with extensor plantar response with non-dermatomal numbness and impaired vibration and position sense. She had trunk sensory level corresponding to the lower thoracic compression. Magnetic resonance imaging revealed thickened ligamentum flavum and well circumscribed intradural extramedullary lesion with a linear or beak-like excrescences, uniformly hypodense on T1- and T2-weighted images, situated posterior to the thecal sac in the lower thoracic region at level of tenth dorsal vertebra causing cord compression (Fig 1, 2). CT of thoracic spine revealed irregularly calcified yellow ligament continuous with lamina. Calcified mass was encroaching and compromising the

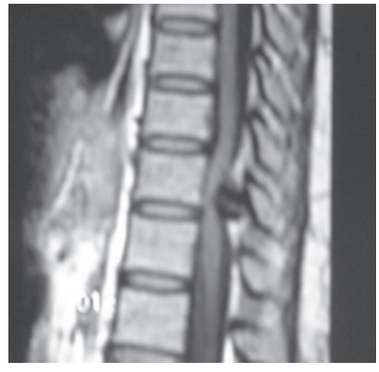

Fig 1: T1-weighted MRI shows uniformly hypo dense lesion situated posterior to the thecal sac in the lower thoracic region at level of tenth dorsal vertebra causing cord compression

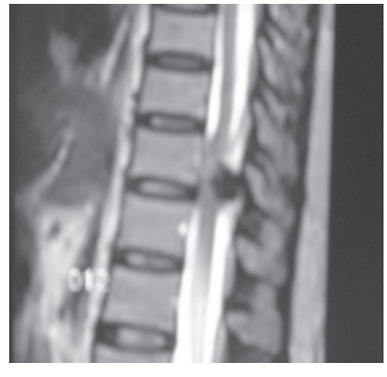

Fig 2: T2 -weighted MRI shows uniformly hypo dense lesion situated posterior to the thecal sac in the lower thoracic region at level of tenth dorsal vertebra causing cord compression.

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spinal canal (Fig 3, 4). There was no radiological evidence of fluorosis.

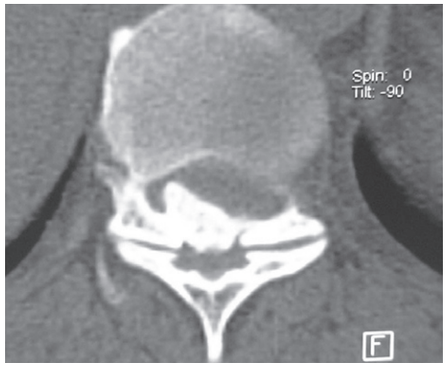

Fig 3: Axial CT of thoracic spine shows irregularly calcified yellow ligament continuous with lamina. Calcified mass was encroaching and compromising the spinal canal

Fig 4: Sagittal CT of thoracic spine shows irregularly calcified yellow ligament continuous with lamina. Calcified mass was encroaching and compromising the spinal canal

Patient underwent decompressive laminectomy with removal of the OLF. The laminae and ossified yellow ligaments formed one mass of bone, which was removed with rongeurs and drills. There was no dura at that level and mass was compressing over the cord substance. Intact dura was definable above and below the level of calcified mass. Patch duroplasty was done using the thoracolumbar fascia. After surgery, the patient demonstrated improvement in motor power. Histological examination of en bloc specimen of ossification of yellow ligament revealed degenerative changes of the elastic fibers in the yellow ligament with adjacent chondrosis, endochondral ossification and lamellar bone formation with foci of dysmorphic calcification and chondro-oseous metaplasia, all characteristics of OLF. Postoperatively patient showed good recovery in motor power and bladder function but she had persistent numbness of both lower limbs at follow-up.

DISCUSSION

Most reports of thoracic myelopathy originated from Japan. One of the largest series was described by Miyasaka et al[4]. Most common symptomatic level was T10 to T11 (64%), OLF most commonly occurs at T9 to T12. Lower dorsal region appears to be particularly prone to degenerative processes due to high tensile force present in the posterior column and altered mechanical stresses at this area leading to the development of OLF. Unique orientation of the zygapophyseal joints in lower thoracic spine and thoracolumbar junction contributes to increased rotatory instability and micromotion[4, 5, 6, 7].

Otani et al[7] postulated a localized mechanical etiology when they noted that 88% of symptomatic thoracic disc herniations were associated with OLF at the same level. Collagen hyperplasia and hypertrophy in response to localized stress, results in thicker ligament and higher collagen content with deposition of both calcium pyrophosphate dehydrate and calcium hydroxyapatite in the ligament. The ligament hypertrophies and calcifies before it ossifies. The ossification process starts at the base of the ligament with endochondral ossification of the hypertrophied, vascularised fibro cartilaginous tissue, ossification is usually found on the capsular side of the ligament in front of the facet joints. It expands on the outer surface of the ligament anteriorly towards the spinal cord, gradually giving rise to 2 paramedian nodules within the spinal canal that compress the posterolateral portion of the spinal cord. This lateral or capsular growth rarely impinges on an exiting segmental nerve root, resulting in foramina stenosis and radiculopathy. Both masses are usually connected by a film of elastic fibers which subsequently also ossifies on its epidural surface and occupies the spinal canal in the form of a central nodular mass. This further contributes to the cord compression as it thickens and extends in a caudocranial direction along the posterior part of the spinal canal in a linear fashion as a "beak-like" outgrowth[5,6,7]. High expression of transforming growth factor beta-1 (TGF-â1) by fibroblasts may play a role in chondral metaplasia and ectopic ossification in OLF[8]. OLF can significantly contribute to a significant spatial reduction of the thoracic spinal canal, resulting in slowly progressive paraparesis or acute paraplegia after an episode of trauma[9,10]. Yamaura et al[9] found that apoptosis in acute spinal injury induced both secondary

degeneration at the site of injury, and chronic demyelination of tracts away from the site of injury. Persistence of residual spasticity at follow-up due to irreversible change within the cord is caused by significant thecal compression[9,11,12].

OLF may present as 2 distinct syndromes[13]: the first involves chronic spinal cord compression over a long period of time and presents with unsteady gait, difficulty with balance and climbing stairs, with or without unilateral/bilateral neurogenic claudication. In the second syndrome, OLF may present as acute myelopathy after minor trauma. There is a sudden compromise in an asymptomatic, but narrowed, spinal canal by haematoma and edema, with or without bony/ soft tissue impingement secondary to trauma. Differential diagnosis including high meningomyelocele, haemangioma, arteiovenous malformation, neurosyphilis, multiple sclerosis, spinal tumor and infection, must be excluded.

Spiral CT with multi-axial reconstruction, although it is best combined with intrathecal myelogram. Thickened ligamentum flava are often found with degenerative disease and spinal stenosis at multiple levels. With the advent of MR imaging, more cases of OLF are being detected. The T2-weighted sagittal image of MR imaging is the modality of choice for screening the longitudinal extent of OLF, with increased diagnostic accuracy when combined with computed tomographic scan. This appears as a linear or beak-like excrescence, uniformly hypo dense on T1 and T2-weighted images, situated posterior to the thecal sac. T2 weighted MR imaging is particularly useful in showing the degree of spinal cord injury and the longitudinal extent and multiple sites of spinal cord compression. It also detects high-signal intramedullary intensity within the spinal cord on T2 weighted images that may indicate poor prognosis. It is useful to combine CT and MR imaging: the former diagnoses the etiology of the compression, while the latter indicates the compression level and cord changes[2,3].

Posterior decompressive laminectomy (with or without medial faceectomy) and removal of enlarged ossified yellow ligament are the most common surgical procedures performed in patients with compressive thoracic myelopathy due to OLF[11,14,15].

Trivedi et al[16] suggested that the spinal cord in the OLF-related thoracic narrow spinal canal, being ischemic and edematous due to prolonged cord compression was more prone to slightest cord damage. Long-standing ossified ligament adheres to the dura and its removal risks major dural disruption. Occasionally, thick OLF had to be excised with the adherent outer layer of the dura.

We postulate that our patient had developed penetration of segmental dura by overlying ossified ligamentum flavum after the episode of trauma and callous formation occurred on injured ossified yellow ligament which incorporated the penetrated dura. Therefore it formed a calcified mass compressing the cord substance like an intradural extramedullary lesion.

OLF should be considered in the differential diagnosis of posttraumatic compressive myelopathy. Furthermore, it is advised that computed tomography scanning and magnetic resonance imaging be combined to provide an accurate diagnosis and proper preoperative evaluation of the bony changes, spinal cord, and compression of the spinal cord. The magnetic resonance imaging and CT appearance of the spine in such cases is characteristic. Decompressive laminectomy and excision of the OYL is the commonly performed surgical procedure. A rapid neurological improvement follows decompression. The persistent spasticity and residual numbness in certain patients is due to irreversible changes within the cord, which are attributed to significant thecal compression and delay between the onset of initial symptoms/signs and surgical decompression. OLF can significantly contribute to a spatial reduction of the thoracic spinal canal, resulting in gradually progressive paraparesis after trauma to the back.

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