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Clinical Study of Dorsal Ligamentum Flavum Hypertrophy with Compressive Myelopathy and its Management

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Introduction

Ossification of ligamentum flavum (OLF) is a well-known cause of progressive thoracic myelopathy reported in Japanese patients also series are reported from Korea, China, India, Middle East, and Caribbean with sporadic case reports in Caucasians(1). It commonly involves lower thoracic spine (T9–T12) with upper thoracic spine (T1–T4) being the next common site. With the increased use of CT and MRI, ossification or hypertrophy of the ligamentum flavum (OLF) is gradually being recognized as a major cause of acquired thoracic spinal canal stenosis. Spinal cord compression by OLF is more common than by herniated intervertebral discs(3). Posterior decompression by laminectomy is most commonly used. Patients present in variable neurological state. This study aimed at prospective analysis of data of 14 Indian patients who underwent surgical or conservative management for dorsal ligamentum flavum hypertrophy with compressive myelopathy. Correlation of various clinical and magnetic resonance imaging (MRI) parameters and preoperative neurological status and recovery studied.

Patients and Method

Fourteen patients with thoracic myelopathy due to OLF were studied out of which 7 underwent decompressive laminectomy and excision of the ligamentum flavum rest were treated on conservative basis as they didn't give consent for operation. Their MRI delineated a linear or beak like excrescence, uniformly hypo-intense on T1 and T2 weighted images, situated posterior to the thecal sac

Results

There was postoperative neurological deterioration in 5 patients and 9 patients improved in post op period. All the patients were discharged on the 7th postoperative day. On discharge, there was significant improvement in spasticity and tingling sensations of the lower limbs. At follow up, the patients had shown improvement in spasticity and power. However, all of them had residual spasticity so that none of them was able to run briskly.

Summary of Patients with Ossified Ligamentum Flavum Causing Thoracic Compressive Myelopathy

No.	AGE/SEX	CLINICAL	DURATION	RADIOLOGY (MRI)	SURGERY
1	45/F	B/L LL spastic paraparesis, urine incontinence	3 yrs	D10-11 OLF	D10-11 laminectomy with excision of flavum
2	49/f	B/L LL spastic paraparesis, urine incontinence	1yr	D1-2 D2-3 D9-10	D9-10 laminectomy with excision of flavum
3	59/m	B/L LL weakness	6 months	D9-10	D9-10 laminectomy with excision of flavum
4	35/m	Pain and tingling b/l LL with gait imbalance	2yrs	D7-10	Conservatively
5	25/f	Spastic paraparesis	1yr	D7-11	
6	55/m	LBP with b/l radiculopathy with gait ataxia	2yrs	D3-10 Cervical pivd Lumbar pivd	conservatively
7	60/m	LBP with b/l radiculopathy with gait ataxia	1yr	D9-12 L5 over S1 listesis grade 1	Conservatively
8	47/m	Spastic paraparesis	2yrs	D7-10	D 7-10 laminectomy with B/L D7-10TPSRF
9	23/f	Spastic paraparesis	3yrs	D8-11	conservatively
10	38/f	Spastic paraparesis with urine incontinence	2yrs	D9-10	D9-10 laminectomy with excision of flavum
11	41/f	Spastic paraparesis	10yrs	D9-10	conservatively
12	48/m	Spastic paraparesis with urine incontinence	2months	D11	D11 laminectomy with excision of flavum
13	32/f	Spastic paraparesis with urine incontinence	5yrs	D3-4 D7-8 D9-11	D9-11 decompressive laminectomy with excision of flavum
14	53/m	LBP with b/l radiculopathy with gait ataxia	2yrs	D5-6 D9-10	conservatively

Discussion

The ligamentum flavum is a yellowish elastic ligament extending form second cervical vertebra to the S1 segment of sacrum consisting of 80% elastin[5]. The ligament is in the dorsal portion of the spinal canal, proximal insertion of ligamentum flavum is the ventral part of cranial lamina extending to the dorsal part of caudal lamina and extending to the capsules of facet joints and the posterior aspects of the neural foramina and is separated from the dura mater by epidural fat(9). LF is divided into two parts as a capsular portion and an inter-laminar portion. The mechanism of hypertrophy and progression of ossification is limited to the ligamentum flavum and neighboring spinal bony arch is spared[7]. The pathogenesis of ossified ligaments is not very clear. The various factors leading to ossification of LF include trauma, mechanical stress, diffuse idiopathic skeletal hyperostosis, ankylosing spondylitis, hemochromatosis, flourosis, growth factors (BMPs, VEGF9, TGF, Cartilage derived morphogenetic protein-1) and disorder of calcium and phosphorus[20]. Clinical manifestations and imaging tests are the two mainstays in the diagnosis. Local thoracic pain and slowly progressing spastic paraparesis are the distinctive features. Posterior cord syndrome includes loss of vibration and proprioception, develops in the early stage of disease process due to compression of the spinal cord from the posterior side. As the ossification progresses, the lateral corticospinal tracts begin to compress and then spastic paraparesis develops. Further extension leads to compression lateral spinothalamic tracts causing loss of sensation[24].

The most common clinical manifestation at the time of the diagnosis is loss of functional gait.

Radicular pain and loss of pain and temperature sensation are rare. Bladder disturbance is seen in late stages[21].

The role of radiologic studies is important in determining the etiology of myelopathy. CT remains the investigation of choice to demonstrate the characteristic contours and density of calcific changes (V shaped hyper-density). Spinal MRI shows hypertrophy of ligamentum flavum as dorso-lateral low signal mass appearance on both T1 & T2 weighted images. T2 sagittal MRI is most useful in examining the whole spine and discovering multiple OLFs[26]. Posterior decompression with laminoplasty or laminectomy combined with lateral fusion is the mainstay treatment. For thoracic ossified ligamentum flavum fenestration or complete laminectomy are usually performed. The ossified ligament should be removed carefully taking care of ossified dura matter which usually concurs[5].

This ossification of LF was reported to occur in up to 20% of Japanese individuals >65 years old and many reports viewed it as a normal feature of the ageing Asian spine[12]. The thickest OLF was located predominantly in the lower third of the thoracic spine (T9 to T12). This region is prone to degenerative processes due to the high tensile force present in the posterior column with associated kyphosis in the lower thoracic spine[15]. OLF occurred most frequently at the thoraco-lumbar junction due to unique orientation of the zygapophyseal joints that contributed to increased rotatory instability and micro motion[19]. The ligament hypertrophies and calcifies before it ossifies. High expression of transforming growth factor beta-1 (TGF-β1) by fibroblasts was found in the ossified matrix within ossified ligaments and in chondrocytes within cartilaginous areas. TGF-β1 might have played a role in chondroid metaplasia and ectopic ossification in OLF[17].

On MRI DORSAL SPINE it appears as a linear or beak-like excrescence, uniformly hypointense 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. Also detects high-signal intramedullary cord intensity within the spinal cord on T2-weighted images that might indicate poor prognosis[18]. Early and prompt surgical intervention with appropriate rehabilitation plays key role in improving the functional outcome of myelopathy caused by OLF[23]. The prognosis after decompressive surgery can be good if intramedullary hyper intensities are absent on preoperative T2-weighted MR images. Posterior decompressive laminectomy with or without medial fascetectomy and removal of the ossified ligament are the most common surgical procedures performed in patients with dorsal ligamentum flavum hypertrophy with compressive myelopathy[14]. Laminoplasty with preservation of the posterior element after decompression can also be performed because of late neurological deterioration due to the recurrence of OLF at the same site or increased kyphotic deformity of the spine observed after laminectomy alone. Also there is no fusion after more than 1-level decompression[6].

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