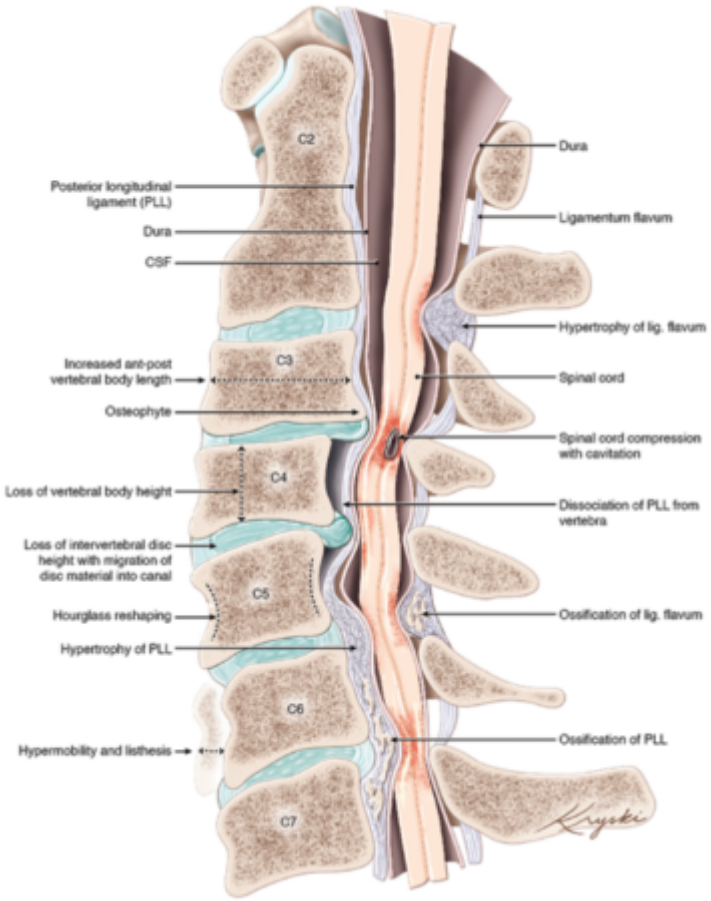
Cervical ossification of the posterior longitudinal ligament

Ossification of the posterior longitudinal ligament (OPLL) has been known as a multifactorial disease [1) 2)].



Epidemiology

Ossification of the posterior longitudinal ligament (OPLL) in the cervical spine and related neurological complications are not uncommon in East Asian countries. The estimated prevalence of cervical OPLL-related hospitalization is 7.7 per 100,000 person-years in Taiwan, and higher incidence rates have been observed in elderly and male patients [3)].

Classification

Cervical ossification of the posterior longitudinal ligament classification.

Etiology

Genetic factors are considered to play an important role in the etiology of OPLL based on nationwide

pedigree surveys, twins surveys, and human leukocyte antigen (HLA) haplotype analysis [4].

The relationship between single-nucleotide polymorphisms (SNPs) in various genes and OPLL has been studied. A case-control association and sib-pair linkage studies have shown that several genes are related to the suscepti- bility to OPLL. These include genes for collagen, type VI, alpha 1 (COL6A1), collagen, type XI, alpha 2 (COL11A2), bone morphogenetic protein 2 (BMP2), ectonucleotide pyrophosphatase/phosphodiesterase 1 (ENPP1), estrogen receptor 1 (ESR1), interleukin-1 beta (IL1B), leptin receptor, and transforming growth factor beta 1 (TGFB1) [5] [6] [7] [8] [9] [10] [11] [12] [13] [14].

Risk Factors

A study of Kang et al. of Severance Hospital and St. Mary's Hospital, was conducted to investigate the course of ossification of the posterior longitudinal ligament (OPLL) progression after laminoplasty (LP) or laminectomy with posterior fixation (PF).

Cervical laminoplasty is now recognized as a standard technique for the treatment of cervical multisegment OPLL; however, PF is beneficial for patients with severe cervical spinal stenosis. In recent years, there has been increasing interest in mechanical stress in OPLL, which is assumed to significantly impact progression.

The progression of OPLL was assessed using midlinesagittal computed tomography images of the cervical spine at various follow-up points. Radiographic parameters including the C2-C7 Cobb angle, C2-C7 range of motion (ROM), and adjacent cranial and caudal segmental ROMs were measured. Postoperative changes and differences between the LP and PF groups in the radiographic parameters were calculated to assess biomechanical stress. Logistic regression analysis was used to analyze the risk factors affecting the progression rate.

Kang et al. included 14 PF and 36 LP patients, with a mean follow-up period of 28.9±20.8 and 37.6±16.8 months, respectively (P=0.069). After surgical treatment, both groups showed loss of cervical lordosis (9.2±6.9 vs. 5.3±8.2 degrees, P=0.220) and C2-C7 ROM (14.6± 13.5 vs. 13.1±12.2 degrees, P=0.861). The decrease of ROM in the cranial adjacent segment was larger in the LP group than in the PF group (0.7±4.1 vs. 1.4±5.5 degrees, P=0.453). The ROM in the caudal adjacent segment decreased in the LP group but increased in the PF group (-1.4±6.2 vs. 2.6±5.1 degrees, P=0.041). The progression rate was 2.15±1.31 mm/mo in the PF group and 1.53±1.04 mm/mo in the LP group (P=0.041). PF showed an odds ratio of 12.917 for a higher progression rate (95% confidence interval, 1.397-119.443; P=0.024).

The rate of progression of cervical OPLL was significantly higher after PF than after LP [15].

Pathophysiology

The pathologic basis of OPLL is unknown, but there is an increased incidence of ankylosing hyperostosis which suggests a hereditary basis. OPLL begins with hypervascular fibrosis in the PLL which is followed by focal areas of calcification, proliferation of periosteal cartilaginous cells and finally ossification [16].

The process frequently extends into the dura. Eventually active bone marrow production may occur. The process progresses at varying rates among patients, with an average annual growth rate of 0.67mm in the AP direction and 4.1mm longitudinally. [17]. When hypertrophied or ossified, the posterior longitudinal ligament may cause myelopathy (due to direct spinal cord compression or ischemia) and/or radiculopathy (by nerve root compression or stretching). Changes within the spinal cord involve the posterolateral gray matter more than white matter, suggesting an ischemic basis for the neurologic involvement.

Clinical Features

The progression of cervical ossification of the posterior longitudinal ligament (OPLL) can lead to increase in the size of the OPLL mass and aggravation of neurological symptoms.

It is generally clinically silent or gradually progressive but can be acute after even minor trauma [18].

The most characteristic symptom of OPLL is myelopathy resulting from the compression of the spinal cord; other signs and symptoms include sensory dysfunction of the upper and the lower extremities, motor weakness, an in- creased deep tendon reflex, and neurogenic bladder and bowel [19]. [20] [21].

Diagnosis

OPLL is not a two-dimensional (2D) disease, but rather a three-dimensional (3D) disease. Therefore, conventional measurement of parameters using radiography may not be suitable for evaluating OPLL.

3D method of measurement is superior to the conventional method in terms of evaluating the clinical state of symptomatic OPLL patients. Higher 3D OPLL ratio has an adverse effect on the spinal cord [22].

Treatment

Cervical ossification of the posterior longitudinal ligament treatment.

Outcome

see Ossification of the posterior longitudinal ligament outcome.

Case series

Ossification of the posterior longitudinal ligament case series.

Case reports

Yu et al. presented 2 cases of huge beak-type OPLL. Patients underwent minimally invasive anterior decompression without fusion. This method created a space on the ventral side of the OPLL without violating global thoracic spinal stability. Via this space, the OPLL and anterior lateral side of the dural sac can be seen and manipulated directly. Then, total removal of the OPLL was accomplished. No orthosis was needed. In this article, we share our key technique and concepts for treatment of huge thoracic OPLL.

Case 1. 51-year-old female was referred to our hospital with right lower limb radiating pain and paresis. Thoracic OPLL at T6-7 had been identified at our hospital, and conservative treatment had been tried without success. Case 2. This 54-year-old female with a 6-month history of progressive gait disturbance and bilateral lower extremity radiating pain (right>left) was admitted to our institute. She also had hypoesthesia in both lower legs. Her symptoms had been gradually progressing. Computed tomography scans showed massive OPLL at the T9-10 level. Magnetic resonance imaging of the thoracolumbar spine demonstrated ventral bony masses with severe anterior compression of the spinal cord at the same level.

We used this surgical method in 2 patients with a huge beaked-type OPLL in the thoracic level. Complete removal of the OPLL via anterior decompression without instrumented fusion was accomplished. The 1st case had no intraoperative or postoperative complications, and the 2nd case had 1 intraoperative complication (dural tear) and no postoperative complications. There were no residual symptoms of the lower extremities.

This surgical technique allows the surgeon to safely and effectively perform minimally invasive anterior decompression without instrumented fusion via a transthoracic approach for thoracic OPLL. It can be applied at the mid and lower level of the thoracic spine and could become a standard procedure for treatment of huge beak-type thoracic OPLL.

Retraction of this article in 2019 [23].

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