Ossification of the Posterior Longitudinal Ligament

Tom E. Reinsel and Gunnar B. J. Andersson

*AJNR Am J Neuroradiol* 1992, 13 (4) 1068-1070

http://www.ajnr.org/content/13/4/1068.citation

This information is current as of October 27, 2023.
Commentary

Ossification of the Posterior Longitudinal Ligament

Tom E. Reinsel and Gunnar B. J. Andersson

Ossification of the posterior longitudinal ligament (OPLL) is most often diagnosed on lateral plain films, but is frequently overlooked. Tomography and (CT) computed tomography scanning are much more sensitive and remain the “gold standard.” Recent years have seen application of magnetic resonance (MR) imaging in OPLL (1, 2). In this issue of the AJNR, MR imaging of OPLL is discussed by Otake et al (1). Their study is the largest in the literature concerning MR imaging and OPLL. The study shows that MR can be used in the diagnosis of OPLL. The T1- and T2-weighted sagittal images allowed diagnosis in only 32%-44% of patients, and usually only in patients with thick lesions. Axial imaging was more sensitive. Proton-density images in both planes were superior and are recommended by the authors. Since plain films, tomograms, and CT are more sensitive, the usefulness of MR in the diagnosis of OPLL is questionable. Its main use seems to be in the assessment of associated cord compression.

Although infrequent outside Japan, OPLL should always be kept in mind when neck films are reviewed. Cervical radiculopathy and myelopathy secondary to ossification of OPLL is rare. First described by Key (4) in 1838, it was not until 1960 that OPLL was truly recognized following Tsukimoto’s careful autopsy description (5). Tsuyama and colleagues (6-8) have subsequently added significantly to our understanding of the etiology, natural history, and treatment.

While there have been reports on non-Asian populations (9), OPLL is primarily an eastern Asiatic disease and has been called “Japanese disease” because of its relative rarity outside Japan (10, 11). Radiographic studies suggest an incidence of 2.0% in Asians and 0.16% in non-Asians (6), although the incidence in non-Asians was as high as 1.7% in one report (12). The true incidence is probably much higher, since OPLL is often asymptomatic and early changes from OPLL are often inapparent on lateral radiographs (13). Ossification usually occurs at the C5, C4, and C6 levels and the average number of vertebral levels involved is 3.1. The highest incidence is in the sixth decade (6).

The cause of OPLL has been debated since Tsukimoto’s autopsy descriptions (5). Fluoride intoxication, diabetes mellitus, growth-hormone imbalance, disk protrusion, recurrent minor trauma, abnormal calcium metabolism, and infection have all been suggested (1, 2, 6-21). A high association has been noted with various hyperostotic spinal changes such as diffuse idiopathic skeletal hyperostosis (DISH), ligamentum flavum ossification, and ankylosing spondylitis (22).

Because of the relative rarity of OPLL in non-Asian populations, a genetic predisposition has been postulated. Not until recently, however, has this been conclusively demonstrated (23, 24). Teryama et al (23) argued that OPLL is most likely an autosomal dominant disorder. Sakow et al (24) have demonstrated the association of specific human leukocyte antigen (HLA) haplotypes with OPLL and have cast doubt on the autosomal dominant inheritance theory, since both haplotypes associated with OPLL were necessary for OPLL to occur. They acknowledged, however, that multiple factors beside genetics may contribute to the manifestation of the disease (24).

OPLL is radiographically classified into four types based on the sagittal plane appearance: 1) segmental (37%), 2) continuous (27%), 3) mixed (29%), and 4) circumscribed (8%) (6). CT has allowed further classification based on the transverse plane appearance of OPLL into three major groups: 1) mushroom (62%), 2) square (19%), and 3) hill (19%) (25, 26). The prognostic value of the various classification groups has not been defined. However, it has been noted that the continuous or mixed types constrict the spinal cord more severely (6).
Most patients with OPLL are asymptomatic with mild complaints such as neck pain and paresthesias (8). Over a 5-year period, progression is usually slow, which explains the marked canal compromise sometimes seen with a paucity of symptoms. After 5 years, only 18% of patients deteriorated clinically, while 27% improved and 55% remained unchanged (27). In approximately 21% of patients, acute deterioration occurs after a mild traumatic episode (8). Cord involvement, i.e., spastic gait and finger clumsiness, has been identified in 10%-15% of patients. In 20% of patients, disability secondary to OPLL affects their activities of daily living.

Conservative options for treatment are similar to treatment options for other types of neck and radicular symptoms; viz, nonsteroidal anti-inflammatory drugs, traction, halo brace, bed rest, and halter traction. These measures often relieve the acute irritation but not the myelopathy. Myelopathic patients should be considered for surgery. The Japanese Orthopaedic Association has established criteria for surgery that include an assessment of activities of daily living (upper and lower extremity function), sensory exam, and bladder function (27). Once a decision for surgery has been made, a choice between anterior and posterior approaches must be made. This somewhat controversial choice involves either anterior decompression and fusion, laminectomy, or laminoplasty. The anterior approach is occasionally unsafe, as the dura may become ossified and adherent to the OPLL mass (29). Laminectomy had been the procedure of choice until follow-up studies revealed that the ossified mass continues to grow after surgery. This progression was somewhat surprising, but is probably partially explained by the instability created by the laminectomy. Malalignment and susceptibility to neck trauma are other complications of laminectomy. Because of these complications, a laminoplasty procedure was developed by Hirabayashi (30-33). This procedure involves widening the spinal canal by hinging open the neural arch, trimming it, and leaving it lightly open. This technique is also not without complications, which include a "re-closure" of the hinged lamina, transient muscle paraparesis, and severe neck pain—presumably from tethering of the nerve roots (28). Although no controlled prospective studies exist, good results are obtained in roughly 60% of patients regardless of the type of procedure.

References

3. Deleted in proof.


