Cervical spine stenosis secondary to ossification of the posterior longitudinal ligament

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Ossification of the posterior longitudinal ligament (OPLL) is a well-documented cause of cervical spine stenosis and myelopathy among Japanese patients. Reports of OPLL in North Americans are rare. Choices of diagnostic method and treatment for this entity remain controversial. The authors report the results of management of 20 patients in the United States with symptomatic OPLL of the cervical spine. These represented 10% to 20% of patients operated on over the last 3 years for myelopathy secondary to structural spinal compression. Most of these OPLL patients were Caucasian (60%), male (male:female 4:1), and middle-aged (median age 47.5 years). Six had previously undergone laminectomy or discectomy. Cervical roentgenograms and standard myelography occasionally suggested the diagnosis. Axial computerized tomography (CT) metrizamide myelography with small interslice intervals proved invaluable for diagnosis and operative planning. Magnetic resonance imaging was not necessary for diagnosis. Retrovertebral calcification extended over one to five bodies (mean 2.75). The mass ranged in size from 5 to 16 mm in anteroposterior diameter and reduced the residual canal diameter to a mean (± standard deviation) caliber of 9.42 ± 2.41 mm (mean narrowing ratio 0.44 ± 0.12).

Anterior cervical decompression by medial corpectomy and discectomy with fusion uniformly reduced preoperative myelopathy. Complications were limited to transient neurological deterioration in two patients, recurrent laryngeal nerve palsy in one, and halo device pin site infections in two. At a mean postoperative interval of 15 months, improvement was seen in each category of deficit: extremity weakness, hypesthesia, hypertonia, and urinary dysfunction. All fusions produced solid unions.

It is concluded that OPLL of the cervical spine is an unexpectedly prevalent cause of myelopathy among patients treated in the United States. Thin-section axial CT metrizamide myelography with small interslice intervals is essential for the investigation of patients who may have OPLL. Anterior decompression and stabilization by medial corpectomy, discectomy, removal of the calcified mass, and fusion is a safe and effective method of treatment.

KEY WORDS • posterior longitudinal ligament • cervical myelopathy • ossification • cervical stenosis

Compression of the spinal cord by an ossified posterior longitudinal ligament (OPLL) was originally described in Guys Hospital Reports in 1838. Since 1960, OPLL has been recognized as an important clinical entity among people of Japanese descent, and is frequently called “the Japanese disease.” Recent reports in the Japanese orthopedic literature have emphasized both the prevalence in the Japanese population of about 2.0% and the value of axial computerized tomography (CT) for the diagnosis of OPLL. The choice among surgical procedures, including laminectomy, laminoplasty, anterior fusion, and anterior decompression with fusion, remains highly controversial.

We have recently encountered a number of non-Japanese patients for whom OPLL of the cervical spine was the cause of myelopathy. We reviewed our series to determine the frequency of occurrence of OPLL, to identify the appropriate radiographic method for the diagnosis of OPLL, and to assess the value of anterior decompression and stabilization by medial corpectomy, discectomy, removal of the ligament, and fusion for the treatment of patients with this entity.

Clinical Material and Methods

Patient Population

Between July, 1983, and June, 1986, 19 patients with a diagnosis of OPLL of the cervical spine were treated at our institutions; another patient who was referred to
Clinical Presentation

A steadily progressive cervical myeloradiculopathy was the most common clinical presentation. Weakness or clumsiness of the upper extremity, numbness or paresthesias of the upper extremity, difficulty with walking, leg weakness, and neck pain were the most common symptoms. Leg numbness or paresthesias, urinary incontinence, upper-extremity pain, and impotence were less regularly mentioned (Table 2). The median duration of the interval between the initial occurrence of symptoms and diagnosis was 7.5 months (range 0 to 36 months, mean 12.1 months). All but three patients reported a steadily progressive deterioration at some point in their clinical course; often the period before surgical treatment was marked by an increase in the rate of this progression. Trauma to the neck precipitated acute exacerbations of neurological deficits in six patients and was mentioned as a remote occurrence without definite association with neurological deficit by another five. Six patients had previously undergone surgery on the cervical spine for compressive myelopathy: two had received multiple-level laminectomies, and four had undergone anterior cervical disectomy with or without fusion.

At presentation, the following physical findings were prevalent: limitation of neck motion, upper-extremity sensory loss, myelopathic sensory loss, upper-extremity weakness, lower-extremity hypertonia, Babinski sign, lower-extremity weakness, lower-extremity paresthesias, numbness, and neck pain (Table 2).

Diagnostic Studies

A cervical spine radiographic series and metrizamide myelography followed by axial CT were obtained for each patient. Abnormal findings on plain film roentgenography were: longitudinal retrovertebral opacity consistent with OPLL in six patients, isolated osteophy- tosis suggestive of cervical spondylosis in six, and congenital spinal stenosis in three. Plain spine films were within normal limits in five patients. Metrizamide myelography showed widening of the cord shadow in the anteroposterior projection in association with an anterior epidural defect seen in the lateral projection in 17 patients; among these, there was interruption of the metrizamide column in five patients and complete blockade to passage of metrizamide in one. Axial CT showed an intraspinal hyperdense mass along the posterior aspect of the vertebral body corresponding to the posterior longitudinal ligament in each patient. In cross section the mass was either rectangular, oval, triangular, or pedunculated in shape (Fig. 1). Metrizamide clearly delineated the deformed cord (Fig. 2). Sagittal reconstructions aided estimation of the anteroposterior and superoinferior extents of the mass (Fig. 3). The maximum anteroposterior dimension of the mass ranged from 5 to 16 mm (mean ± standard deviation 7.26 ± 2.40 mm, median 6 mm) and reduced the residual canal at that level to a mean of 9.42 ± 2.41 mm (range 6 to 14 mm, median 9 mm). The mean narrowing ratio (anteroposterior dimension of the OPLL/anteposterior dimension of the entire canal) was 0.44 ± 0.12. Five vertebral levels were involved in four patients, four levels in one, three levels in six, two levels in four, and

### TABLE 1
Profiles of 20 patients with ossified posterior longitudinal ligament (OPLL)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>OPLL Level</th>
<th>OPLL Type</th>
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<tr>
<td>1</td>
<td>39, M</td>
<td>Vietnamese</td>
<td>C3-7</td>
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</tr>
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<td>2</td>
<td>61, M</td>
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<td>C4-6</td>
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</tr>
<tr>
<td>3</td>
<td>33, F</td>
<td>Caucasian</td>
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<td>segmental</td>
</tr>
<tr>
<td>4</td>
<td>37, M</td>
<td>Caucasian</td>
<td>C5-6</td>
<td>segmental</td>
</tr>
<tr>
<td>5</td>
<td>40, M</td>
<td>Caucasian</td>
<td>C4-7</td>
<td>mixed</td>
</tr>
<tr>
<td>6</td>
<td>22, M</td>
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<td>C-4</td>
<td>continuous</td>
</tr>
<tr>
<td>7</td>
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<td>segmental</td>
</tr>
<tr>
<td>8</td>
<td>59, F</td>
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</tr>
<tr>
<td>9</td>
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</tr>
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<td>10</td>
<td>37, M</td>
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<td>C5/6-C5/7</td>
<td>segmental</td>
</tr>
<tr>
<td>11</td>
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<tr>
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<tr>
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</tr>
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<td>Chinese</td>
<td>C3-7</td>
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<tr>
<td>15</td>
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<td>Hispanic</td>
<td>C3-7</td>
<td>continuous</td>
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<tr>
<td>16</td>
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</tr>
<tr>
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### TABLE 2
Clinical presentation in 20 patients in this series

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<th>Symptoms &amp; Signs</th>
<th>No. of Cases</th>
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</thead>
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<td>symptoms</td>
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<td>upper-extremity weakness</td>
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</tr>
<tr>
<td>gait difficulty</td>
<td>17</td>
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<tr>
<td>upper-extremity paresthesias, numbness</td>
<td>16</td>
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<tr>
<td>lower-extremity weakness</td>
<td>16</td>
</tr>
<tr>
<td>neck pain</td>
<td>15</td>
</tr>
<tr>
<td>lower-extremity paresthesias, numbness</td>
<td>10</td>
</tr>
<tr>
<td>sphincter dysfunction</td>
<td>8</td>
</tr>
<tr>
<td>upper-extremity pain</td>
<td>6</td>
</tr>
<tr>
<td>signs</td>
<td></td>
</tr>
<tr>
<td>Babinski sign extensor</td>
<td>18</td>
</tr>
<tr>
<td>lower-extremity hypertonia</td>
<td>16</td>
</tr>
<tr>
<td>upper-extremity weakness</td>
<td>15</td>
</tr>
<tr>
<td>upper-extremity hypertension</td>
<td>14</td>
</tr>
<tr>
<td>lower-extremity weakness</td>
<td>14</td>
</tr>
<tr>
<td>limitation of neck movement</td>
<td>14</td>
</tr>
<tr>
<td>Hoffmann sign</td>
<td>12</td>
</tr>
<tr>
<td>myelopathic sensory loss</td>
<td>12</td>
</tr>
<tr>
<td>upper-extremity sensory loss (radicular)</td>
<td>11</td>
</tr>
</tbody>
</table>
Ossification of posterior longitudinal ligament in the U.S.

FIG. 1. Preoperative axial computerized tomography scans without metrizamide. These scans from two different patients demonstrate the variability in shape of the ossified mass. Left: The calcified ligament extends from the medial aspect of the vertebral body and the mass appears pedunculated. Right: The calcified mass is seen as an irregularly-shaped osteoma protruding from the entire posterior surface of the vertebral body.

one level in five (median three levels, mean 2.75 ± 1.45 levels) (Table 1). The C-5 level was involved in 17 patients, C-6 in 14, C-4 in 12, C-7 in six, C-3 in five, and C-2 in two. The OPLL mass was segmental in eight patients, continuous in eight, focal in two, and of the mixed type in two.

Treatment
In 17 patients, progressive neurological deficit refractory to bed rest, cervical orthosis, or cervical traction were the indications for surgery. Of the remaining three patients: one was operated on for incomplete recovery from spastic quadriplegia; another, whose trauma-induced quadriplegia resolved completely, requested surgery for intractable neck pain and prophylaxis against recurrent myelopathy; and the third, who had a severe spastic quadriplegia, refused surgery. The goal of the operation was to relieve the compression of the cord by exposing and removing the posterior longitudinal ligament wherever it was hypertrophied or ossified. An anterior approach consisting of medial corpectomy, discectomy, microsurgical removal of the ossified ligament, and interbody fusion was used in each patient.

After endotracheal intubation (often performed by the nasal route in an awake patient) and the induction of general anesthesia, the patient was placed in the supine position. A halo skull fixation ring was attached, and the head was held in neutral position under 5 lb of axial traction. A horizontal neck incision extending from the anteromedial border of the sternocleidomas-

FIG. 2. Preoperative axial computerized tomography scans with metrizamide from two different patients. Metrizamide highlights the cord compression caused by triangular (left) and semicircular (right) ossified masses.
toid muscle to the midline was used if the ossification spanned one or two adjacent disc spaces; an oblique incision extending along the anteromedial border of the sternocleidomastoid muscle was used if the ossification extended over three disc spaces or involved nonadjacent disc spaces or vertebral bodies. Division of the musculus platysma and of the superficial, middle, and deep cervical fascia permitted entrance into the prevertebral space, division of the anterior vertebral aponeurosis, and lateral elevation of the longus colli muscles. The carotid artery sheath was retracted laterally, and the tracheoesophageal complex was withdrawn medially by one, two, or sometimes three sets of Cloward self-retaining retractors.

After the appropriate spinal level was confirmed by intraoperative x-ray film, discectomy was performed wherever an adjacent half of a vertebral body was involved. With the aid of the operating microscope, the posterior margin of the adjacent vertebral body and the width of the ossified ligament were identified. An air drill with a 6-mm cutting stainless steel burr was used to drill a trench 8 to 12 mm in width through the medial portion of the involved vertebral bodies. As the corpectomy proceeded posteriorly, it was widened to ensure access to the lateral margins of the ossified ligament.

After decompression, structural defects resulting from the corpectomy and discectomy were filled with either autogenous or banked fibula or iliac-crest bone graft fashioned and seated to prevent extrusion. The wound was closed in a routine manner without a drain. After recovery from the general anesthetic, the patient was transferred from traction into a halo vest or other orthosis. Postoperative CT scanning was often performed to assess the extent of decompression (Fig. 4). The cervical orthosis remained in place for 3 months or until plain follow-up films showed that the fusion was solid (Fig. 5).

**Results**

**Intraoperative Findings**

The axial extent of the mass necessitated removal of both vertebral bodies and discs in all operative cases (Table 3). Intraoperatively, the posterior longitudinal ligament was solidly calcified into an ivory-hard osteoma in about a third of patients, hardened but malleable in about a third, and of mixed consistency in the remaining third. It was tightly adherent or continuous with the cortex of the posterior aspect of the vertebral body in 14 patients. In three patients, the mass either incorporated dura or had so attenuated the dura that it could not be removed completely without entering the subarachnoid space.

The fusion involved two vertebral bodies in one patient, three vertebral bodies in 12, four vertebral bodies in four, and five vertebral bodies in two. An iliac-crest graft was used in 11 patients (nine autogenous and two banked bone) and a fibula graft in eight (two autogenous and six banked bone). A total of four disc spaces not adjacent to a resected vertebral body were fused in three patients. In one patient in whom four vertebral bodies were involved, separate operations were used to perform discectomy and semicorpectomies at two different levels.
Ossification of posterior longitudinal ligament in the U.S.

FIG. 4. Postoperative computerized tomography scans. Axial scans (left and center) of the two patients whose preoperative scans are shown in Fig. 2 demonstrate removal of the compressing mass and midline placement of a fibula strut graft. A sagittal reconstruction (right) confirms complete removal of anteriorly compressive elements.

Outcome

The immediate postoperative neurological condition was unchanged or improved in all but two of 19 patients. In one of these, an increase in spastic quadriparesis persisted for 2 days, and in the other increased severity of a Brown-Séquard deficit lasted for 2 weeks. Complications were limited to recurrent laryngeal nerve palsy in one patient, intraoperative hemorrhage from a vertebral body that necessitated postoperative transfusion in one, and infections of halo device pin sites in two, one of whom required a change to a different cervical orthosis.

Follow-Up Results

The 19 patients operated on have been followed for a mean period of 15 months (range 4 to 36 months, median 12 months). There was functional improvement at the most recent follow-up visit in all 17 patients with cervical myelopathy (Table 3). The patient with a hypotonic brachial monoparesis secondary to segmental cord damage also improved, as did the patient with intractable pain. Improvement was uniformly noted in various categories of deficits. Of the 14 patients with upper-extremity weakness preoperatively, all had improved and seven had regained normal arm and hand strength. Similarly, of the 13 who had leg weakness preoperatively, all had improved and six had normal leg strength. Hypertonia and hyperreflexia were decreased in the 15 patients in whom they occurred, but all signs of myelopathy resolved completely in only three patients. The six patients who presented with minor urinary dysfunction were cured of their problem; the single patient who was originally catheter-dependent was left with occasional incontinence. All fusions proceeded to solid union. The spastic quadripariesis of the sole patient who refused surgery did not improve.

Discussion

The posterior longitudinal ligament provides axial support to the spine. From its origin at the basiocciput, it extends along the posterior aspect of vertebral bodies and intervertebral discs and inserts into the sacrum. It has segmental attachments to the disc anuli and adjacent vertebral body cortex, but is usually separated from the cortex of the midvertebral body by a space containing the basivertebral venous plexus. In the cervical region the ligament is normally uniformly broad and flat, but at lower levels it is contracted to the midline behind the vertebral bodies and expanded laterally at the disc level. When hypertrophied and ossified, the ligament encroaches upon the spinal canal. Resultant cord compression or ischemia can produce myelopathy, and nerve root stretching or compression...
Pathophysiology

Although the etiology of OPLL is unknown, the high incidence of diffuse idiopathic spinal hyperostosis (ankylosing hyperostosis) among patients with OPLL suggests a hereditary diathesis of spinal ligament ossification.\textsuperscript{15,23,31,33} The basic pathophysiological processes of OPLL are similar to the heterotopic bone formation in response to mechanical stress in other tissues.\textsuperscript{9} Immunohistochemical studies with anticollagen antibodies have shown that mineralization follows hyperplastic change in the ligament.\textsuperscript{41} Cartilage cells proliferate first in the periosteum of the vertebral body and then in the anulus fibrosus, longitudinal ligament, and dura matter.\textsuperscript{36} The ligament, and in many instances the attached dura, becomes calcified by the process of endochondral ossification.\textsuperscript{39} Mature lamellar bone is eventually formed. The OPLL mass progressively enlarges in thickness, width, and length.\textsuperscript{34} Its rate of growth varies among patients, but the mean annual increase in OPLL size for 13 patients in one study was 4.07 mm in the superoinferior dimension and 0.67 mm in the anteroposterior dimension.\textsuperscript{32} Accretion to the posterior surface of this mass eventually proceeds beyond the normal confines of the posterior longitudinal ligament to compress the spinal cord.\textsuperscript{34} The mass may be round, cuboidal, triangular, or polyoid in shape.\textsuperscript{18}

Autopsy studies frequently show an anteriorly concave, very thin, crescent-shaped cord.\textsuperscript{13,24} Extensive damage to gray matter and long tracts is seen but the most severe destruction occurs in the central gray matter and neighboring ventral fibers of the posterior columns. Necrosis, cavitation, and venous stasis result in the gray matter, and demyelination, axon loss, and status spongiosus in the white matter.\textsuperscript{24} These cord regions are fed by the terminal supply of the central artery after it arises from the anterior spinal artery.\textsuperscript{24} The myelomalacia and the degenerative changes found in the adjacent arterioles and veins suggest that partial restriction of spinal cord circulation is important to the pathophysiology of OPLL.\textsuperscript{13} In addition, nerve roots are often atrophied and show evidence of demyelination and axon loss; damage is most severe where the roots exit the cord and where they penetrate the dura.\textsuperscript{13}

Only a minority of patients who have OPLL are symptomatic.\textsuperscript{26} Even patients with very large masses that produce deep indentations into the ventral cord may be free of noticeable deficits. This may reflect the ability of the spinal cord to adapt, without loss of function, to gradually applied pressure.\textsuperscript{8,38} As was evident in five of our patients, even minor trauma to a severely compressed cord can lead to acute and severe deterioration of function.\textsuperscript{29} However, in the absence of trauma, the neurological course is usually one of progressive deterioration.\textsuperscript{27,38}

The other important determinant in the development of neurological symptoms is residual canal size. Although the narrowing ratio (anteroposterior diameter of the OPLL/anteroposterior diameter of the entire canal) has been used as an index for predicting injury to the cord,\textsuperscript{14,48} the absolute residual canal diameter should prove to be more important.\textsuperscript{32} Enlargement of an OPLL is more likely to produce symptomatic cord compression in a canal already compromised by congenital stenosis, spondylosis, or hypertrophy of the ligamentum flavum than in a canal of normal dimension.\textsuperscript{8} In our series, the residual diameter critical to the development of symptoms was 9 to 10 mm.

**Clinical Presentation and Management**

The large number of recent reports in the Japanese literature on symptomatic OPLL of the spine has been attributed to a particularly high prevalence of the disease among the Japanese and to the improvement in diagnosis of spinal disorders by the widespread use of CT. A Japanese Orthopedic Association cooperative study\textsuperscript{38} estimated that the prevalence of OPLL evident in the plain cervical spine films of nonhospitalized adult patients with spinal problems was 2.0%. Cervical
spine tomography demonstrated OPLL in 3.5% of 698 Japanese volunteers without symptomatic spinal disease.\textsuperscript{17,26} The prevalence increased with age: by the age of 60 years, over 10% of the population had OPLL. In a review of plain cervical spine films, a 2.04% (143 of 6994 cases) prevalence of cervical OPLL was found among Japanese patients; for Korean patients the prevalence was 0.95%, for North Americans 0.12%, and for Germans 0.10%.\textsuperscript{17} This study and the extremely low number of non-Japanese patients among reported cases suggested that OPLL was almost exclusively limited to Mongolian Asians. In a more recent United States study, however, a higher prevalence was found among Caucasians: seven cases of OPLL in 1000 x-ray spine films (431 cervical, 107 thoracic, and 462 lumbar).\textsuperscript{5} The 20 patients in our 3-year study represented between 10% and 20% of the patients operated on for myelopathy secondary to structural spinal compression during the same interval at these institutions. This and the United States study cited above suggest that the prevalence of both symptomatic and asymptomatic OPLL among people in this country is higher than is generally appreciated.

The demographic, clinical, and pathophysiological characteristics of the patients in our series are similar to those in reported series: male predominance; onset of symptoms in middle age; more frequent involvement of mid-cervical vertebrae; involvement of an average of three vertebral levels; and relative frequency of occurrence of segmental, continuous, mixed, and, rarely, localized types.\textsuperscript{12,24} The majority of patients in this and other series also presented with a slowly progressive myelopathy. Although neck pain and arm radicular sensory loss were common, the most prevalent feature at presentation was a spastic quadriplegia.\textsuperscript{11,12} When graded according to extent of lower-extremity dysfunction, at the time of presentation about one-third of our patients were severely impaired (Grades IIIB, IIC, or IV; see Table 3), one-third were moderately impaired (Grades II or IIIA), and one-third were only mildly impaired (Grades 0 or I).

The following possible diagnoses should be considered in patients with this clinical presentation: structural spinal disease; tumor or abscess of the spine or cord; vascular insufficiency or malformation; or myelitis, multiple sclerosis, motor neuron disease, or other inherited or degenerative myelopathic disorders. The first category includes congenital or traumatic spinal deformity, congenital canal stenosis, cervical spondylolisthesis, disc herniation, ankylosing spondylitis, diffuse idiopathic spinal hyperostosis, ossification of the liga-
mentum flavum, and OPLL. Since its introduction a decade ago, CT has proved to be the diagnostic study of choice for patients with structural spine disease.\textsuperscript{15} Numerous studies have shown it to be particularly valuable in the assessment of OPLL.\textsuperscript{11,18,40} In many instances, including 14 of our patients, OPLL is indistinct or even undetectable on plain films but is well defined by CT.\textsuperscript{7,10} In addition, transverse CT metrizamide myelographic images permit an analysis of the degree of encroachment on the spinal canal and compression of the cord that is superior to that obtained from conventional tomography or myelography.\textsuperscript{4,16} In our clinics, CT scans are obtained whenever cervical myelography is indicated. In all cases of cervical spine stenosis, CT is considered essential in order to rule out or diagnose OPLL. Even when this technique is used, however, significant cord compression by a segment of OPLL can be missed if the scan is limited to the level of the disc space. To ensure that an adequate study is obtained, scans are taken at 3-mm intervals throughout each vertebral body. Otherwise, OPLL of the retrocorporal focal type may be missed entirely and OPLL of the continuous type may be misdiagnosed as a spondylotic bar confined to the level of the disc space.

A trial of conservative therapy consisting of bed rest and cervical traction is indicated for all patients except those with a rapidly developing myelopathy.\textsuperscript{38} Persistence or progression of a neurological deficit, radiographic evidence of risk of further cord damage, and intractable pain are indications for surgery.\textsuperscript{14} In the 1960's and 1970's, posterior decompression was the preferred treatment.\textsuperscript{2,30} The benefit of laminectomy, however, is limited by the following factors. First, the OPLL remains and will continue to enlarge\textsuperscript{34,38} such that disruption of circulation and even cord compression may persist.\textsuperscript{14} Second, some authors\textsuperscript{25} recommend removal of two to three laminae and their corresponding medial facets both above and below the OPLL mass; therefore, extensive bone removal required by this approach carries the risk of instability, deformity, loss of osseous cord protection, and delayed neurological deterioration.\textsuperscript{6,20} Third, because the nerve roots remain stretched around the ventral mass, radiculopathy often persists.\textsuperscript{3,5} Dissatisfaction with the results of laminectomy led some Japanese authors to advocate various methods of laminoplasty or a combination of laminectomy with anterior cervical fusion.\textsuperscript{7,36} Anterior cervical discectomy combined with fusion has also been used as the sole treatment.\textsuperscript{36} Unless instability contributes substantially to cord injury, or the OPLL mass is of the bridging type exclusively limited to the level of the disc space, cord compression will persist, especially when underlying developmental stenosis is present. Similarly, corpectomy and discectomy with release but not removal of the OPLL mass may permit reattachment and regrowth; one group using this technique reported incomplete decompression in seven of 15 cases.\textsuperscript{12}

Anterior cervical decompression by corpectomy, discectomy, and removal of the OPLL mass followed by interbody fusion is the most direct approach to the problem.\textsuperscript{1,12} In six of our patients and in patients reported in other series, this approach has proven successful when others have failed. With proper technique, the mass can be removed in its entirety without injuring the cord. Regrowth is precluded, lateral and posterior
structural elements remain intact, and nerve-root compromise is relieved by the combination of removal of the mass and foraminotomies. The anterior approach does require fusion and, if more than a single vertebral body and disc pair are removed, external fixation in a halo or other orthosis for 8 to 12 weeks. In all our patients treated by this approach, grafting produced a solid union.

In this and other series, anterior decompression and fusion led to neurological improvement in most or all patients. Although recovery occurred for each type of deficit encountered, completely normal function was regained by fewer than half of the patients. This irreversible injury may have been prevented if proper treatment had been effected earlier in the clinical course. More widespread awareness of the pathologic entity, its accurate depiction by thin-section CT metrizamide myelography with a narrow interslice interval, and prompt treatment of OPLL by anterior corpectomy, discectomy, removal of the mass, and interbody fusion may decrease the number of patients who suffer irreversible damage.

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References

Ossification of posterior longitudinal ligament in the U.S.


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