Laminectomy for thoracic spinal canal stenosis

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Stenosis of the thoracic spinal canal is a relatively rare disorder with numerous causes. Clinical manifestations include signs and or symptoms consistent with focal thoracic radiculopathy and/or myelopathy. Several surgical approaches for the decompression of the stenotic thoracic canal have been described. Laminectomy is typically reserved for only those cases in which dorsal compression of the neural elements is demonstrated; it is contraindicated when the epidural compression is primarily ventral in location.

KEY WORDS • thoracic spine • spinal canal stenosis • laminectomy

It has long been recognized that congenital and spondylotic narrowing of the cervical and lumbar spinal canal can produce neurological dysfunction. However, the presence of stenosis in the thoracic region with its related clinical manifestations has only been recently appreciated.

The pathogenesis of thoracic SCS is similar to that found in the cervical or lumbar spine. A congenitally narrowed thoracic spinal canal is further compromised by disc degeneration or herniation. This condition can be additionally compounded by degenerative hypertrophy of the posterior elements, calcification of the ligamentum flavum, and the development of ventral epidural osteophytes.

Several different surgical approaches have evolved to help manage a compromised thoracic spinal canal. In this paper the author will address the option of thoracic laminectomy.

THORACIC SPINAL CANAL STENOSIS

Clinical Syndromes

Two distinct clinical syndromes of thoracic SCS have been identified, the most common being associated with a degenerative disorder of the spine. Clinical manifestations include the development of unilateral or bilateral symptoms of pseudoclaudication. Focal radicular pain or paresthesias may also be present. The neurological examination may initially demonstrate normal findings, but as the degree of neural compression progresses, posterior column dysfunction and long tract signs appear. If the disorder is allowed to progress untreated, the patient may develop significant difficulty with gait and bowel/bladder function.

Thoracic SCS secondary to a congenital narrowing of the canal is associated with a more abrupt onset of symptoms. The intrinsically narrowed canal can be easily compromised by a disc herniation that would otherwise be trivial. The typical clinical manifestations of myelopathic signs and symptoms may commence following minor or moderate traumatic injury. Radicular symptoms are rare in congenital SCS.

Differential Diagnosis

Thoracic SCS is a relatively rare disorder. Several other disorders can mimic the clinical signs and symptoms of thoracic SCS and need to be excluded. Among the more common entities resembling the degenerative form of thoracic SCS are tandem canal stenosis (that is, symptomatic cervical and lumbar SCS), diffuse idiopathic skeletal hyperostosis, progressive multiple sclerosis of the spine, and spinal vascular malformations. Tumors, infections, hemangiomas, and spinal cysts should also be considered.

A number of metabolic, rheumatological, and orthopedic disorders can also produce thoracic myelopathy due to an acquired SCS. Among the more easily recognized of these are: achondroplasia, which is characterized by short limbs, a normal trunk, and dysmorphic facial features; and chondro-osteodystrophy (Morquio–Brailsford syndrome), which is associated with joint deformities, a short neck, and thoracic deformities. Although achondroplasia is usually associated with lumbar SCS, it may also produce narrowing of the bone in the thoracic spinal canal.

Hypertrophy of the intracanalicular soft tissue, particularly the ligamentum flavum, may also produce gradual onset myelopathy. Hypertrophic calcification of the ligamentum flavum may be caused by osteofluorosis or may be idiopathic. Acromegaly can cause osteophytosis, disc space narrowing, and hypertrophy of cortical bone resulting in SCS. Other causes of bone compression of the thoracic spinal cord are familial hypophosphatemic vitamin D–refractory rickets, Scheuermann disease, Paget disease, ankylosing spondylitis, and Charcot disease in which syphilis or diabetes has affected the spine.

Neuroimaging Features

Thoracic SCS is best identified by magnetic resonance imaging...
imaging or postmyelographic CT scanning. Plain radiography of the thoracic spine is generally nondiagnostic. As in this study, myelography alone may prove misleading, typically conducted with the patient in the prone position, and generally fails to reveal the posterolateral filling defects that are associated with this disorder. Late in the course of degenerative thoracic SCS, the lateral recesses and posterior midline become obliterated, and myelography may then be diagnostic.

Conventional CT or postmyelographic CT scanning performed at the appropriate level(s) provides a more definitive diagnostic image (Fig. 1). Because of the advancement of CT scanning with the use of spiral imaging technology, multiple spinal levels can now be more easily visualized than in the past. At the level at which stenosis is present, the pedicles are typically widened and short, and there is an associated hypertrophy of the articular processes bilaterally. Magnetic resonance imaging is particularly useful for providing multiplanar images, but it may not demonstrate the bone anatomy as well as CT scanning (Fig. 2).

**Indications for Surgery**

One indication for a thoracic laminectomy in a patient in whom neuroimaging has demonstrated evidence of SCS is the presence of a significant static or progressive myelopathy. The stenosis should be either concentric or secondary to hypertrophy on the posterior elements. A laminectomy should not be the primary approach when SCS results from a significant ventral epidural osteophyte or herniated disc, as these lesions are more effectively and safely managed by a posterolateral (transpedicular, transfacial, or costotransversectomy) or an anterior approach. However, either one of the posterolateral approaches can be extended to include a laminectomy if this will facilitate the decompressive procedure.

**Surgical Technique**

A thoracic laminectomy for SCS is typically performed after induction of general anesthesia with the patient in the prone position. Alternatively, for patients who are morbidly obese, have severe respiratory dysfunction, or are in late-term pregnancy, a lateral decubitus position may be used. For patients in whom the upper thoracic spine is affected, the sitting position is another option. Regardless of the surgical position selected, appropriate antibiotic and steroid agents are administered intravenously prior to induction of anesthesia. During induction, care is taken to avoid inducing hypotension, as this may further compromise blood flow to the spinal cord in the stenotic thoracic canal.

With the patient in the prone position, attention is required for positioning of the head to avoid excessive cervical extension or pressure on the orbits. The arms are abducted and brought rostrally, with care taken to protect the ulnar nerve at the elbow. If the patient’s shoulders are not easily mobilized, the arms are placed at the side; however, this causes the surgeon to be at a greater distance from the operative field. The patient’s thoracic spine should be positioned in as neutral a position as possible. Excessive flexion of the thoracic spine may accentuate a minor encroachment from a ventral epidural lesion and result in a postoperative neurological deficit. The abdomen is allowed to hang as freely as possible to prevent epidural venous engorgement.

The use of SSEP monitoring during the surgery remains controversial. By providing “after the fact” information, this technology may be not be effective in preventing a neurological deficit. Whether SSEP monitoring is used does not change the fact that during the decompression of the thoracic spinal canal, careful and precise surgical techniques are required. In many cases, SSEP monitoring is used merely because of regional medicolegal concerns.

The appropriate location of the midline incision can be determined by palpation of the spinous processes, using the 12th rib as a landmark. If this technique is difficult to perform, a preoperative lateral radiograph can be obtained in which a spinal needle is inserted in the paraspinal region. If the patient is positioned on a radiolucent table, an anteroposterior radiograph will provide adequate information.

Following the usual sterile preparation of the operative field, a midline incision is made and the paraspinal muscles are reflected bilaterally. If electrocautery is used to dissect the paraspinal muscles, care should be taken to avoid placing the tip of the instrument in between the thoracic laminae and violating the dura. A low threshold
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should exist for obtaining a second localization x-ray film. Depending on the level of the surgical exposure, either the sacrum, the 12th rib, or the occiput can be used as a landmark for localization of the appropriate spinal levels.

After a satisfactory exposure and localization have been achieved, the spinning processes of the vertebral levels to be decompressed are removed. A high-speed drill can be used to thin down the laminae prior to their removal by using a narrow (1–2-mm) rongeur. The surgeon should begin the initial decompression at the lower lamina, move in a rostral direction, and focus as much as possible on the less stenotic regions of the field. Although care should be taken to avoid disrupting the adjacent facet joints, their removal in the thoracic region will not have as much of a destabilizing influence on the spine as it would in the cervical or lumbar regions.

When a satisfactory decompression of the stenotic canal has been achieved, meticulous hemostasis is performed out and the wound is closed. The insertion of an epidural drain may reduce the risk of postoperative hematoma formation. The drain can be removed after 1 to 2 days. Antbiotic and steroid treatments are continued for 24 hours following surgery.

Complications

In the properly selected patient, complications following thoracic laminectomy are rare. When a new neurological deficit is present postoperatively, it is typically found in the patient in whom a significant ventral epidural encroachment was observed as part of the patient’s preoperative stenosis. As mentioned previously, surgery via a posterolateral or anterior thoracic approach is more appropriate in these patients.

Other complications that occur following thoracic laminectomy include the usual low incidence of wound infection, cerebrospinal fluid leakage, epidural hematoma formation, and deep venous thrombosis that are associated with other spinal surgeries. The occurrence of iatrogenic spinal instability following thoracic laminectomy is rare.

CONCLUSIONS

Several different surgical techniques are available for the management of symptomatic thoracic SCS. Laminectomy is typically reserved for those patients in whom there is a concentric narrowing of the canal or a predominantly posterior component to their stenosis. In the properly selected patient, laminectomy is associated with a relatively low risk and provides patients with a long-term prognosis for improvement or resolution of their symptoms.

References


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