Thoracic spinal canal stenosis

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Hypertrophy of the posterior spinal elements leading to compromise of the spinal canal and its neural elements is a well-recognized pathological entity affecting the lumbar or cervical spine. Such stenosis of the thoracic spine in the absence of a generalized rheumatological, metabolic, or orthopedic disorder, or a history of trauma is generally considered to be rare. Over a 2-year period the authors have treated six cases of thoracic myelopathy associated with thoracic canal stenosis. In four patients the deficits developed gradually and painlessly. The three older patients had a clinical profile characterized by complaints of pseudoclaudication, spastic lower limbs, and evidence of posterior column dysfunction. Two patients were younger adults with low thoracic myelopathy associated with local back pain after minor trauma. Both patients also had congenital narrowing of the thoracic spinal canal.

Oil and metrizamide contrast myelography in the prone position were of limited value in diagnosing this condition; in fact, myelography may be misleading and result in erroneous diagnosis of thoracic disc protrusion, when the principal problem is dorsal and lateral compression from hypertrophied facets. Magnetic resonance imaging and computerized tomography sector scanning were more useful in the diagnosis of this disorder than was myelography. Thoracic canal stenosis may be more common than is currently recognized and account for a portion of the failures in anterior and lateral decompression of thoracic disc herniations.

KEY WORDS spinal canal stenosis thoracic spine myelopathy spondylosis

SYMPTOMATIC compression of the cord and cauda equina are recognized sequelae of acquired or congenital stenosis of the cervical or lumbar portions of the spine, respectively. Reports of myelopathy resulting exclusively from bone and ligament narrowing of the thoracic spinal canal are usually restricted to patients suffering from generalized disorders of bones or joints, such as achondroplasia, osteochondrodystrophy, acromegaly, Scheuermann’s disease, osteofluorosis, or Paget’s disease. We report four cases of the insidious development of thoracic myelopathy due to degenerative hypertrophy of the posterior spinal elements in the absence of known predisposing factors. Two cases in a younger population related to minor trauma are also presented. The radiological features of this disorder are presented as well as review of the limited literature on this topic.

Case Reports

Case 1

This 47-year old man developed paresthesias of the left lower limb, which worsened with ambulation and improved only on sitting. He denied spinal pain, weakness, or sphincter dysfunction. His medical history was remarkable for three left-convexity intracranial meningiomas, all of which had been successfully resected several years earlier. Neurological examination at that time was essentially normal except for mild spasticity of the right lower extremity. Radiological examination to evaluate the spasticity had revealed apophyseal hypoplasia of the lumbar spine with mild stenosis, as determined by computerized tomography (CT) sector scanning. Conservative therapy was recommended because it was thought that the mild spasticity could be explained as a residuum of the intracranial disease. The patient’s complaints gradually worsened, however, and clumsiness of the left leg developed. Reexamination 6 months later revealed increased tone, hyperreflexia, and an extensor plantar response on the left. Pantopaque (iophendylate) myelography in the prone position was interpreted as showing a small anterior defect at T10—T11, consistent with a herniated thoracic disc (Fig. 1). The patient’s symptoms continued to progress.

Examination. Examination showed increased left
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FIG. 1. Prone lateral [left] and anteroposterior [right] Pantopaque myelograms in Case 1. These were interpreted as demonstrating a small thoracic disc protrusion at T10-11. In retrospect, posterior obliteration of the dye column is suggested.

lower limb spasticity, a mid-thigh level of vibratory sensation on the left, and a similar contralateral deficit with respect to pinprick appreciation. Sacral sensation and rectal tone were normal. Repeat myelography was notable only for a persistent small anterior extradural defect and partial block at the T10-11 interspace. A CT scan of the head with contrast enhancement was unremarkable; specifically, it revealed no recurrence of the meningiomas and no hydrocephalus. Sagittal magnetic resonance imaging (MRI) revealed an incidental hemangioma at T-12, but failed to disclose the nature of the lesion at the T10-11 level (Fig. 2 left). Computerized tomography sector scanning at this level (Fig. 2 right) showed narrowing of the anteroposterior dimension of the spinal canal produced by short pedicles and enlarged facets.

Operations. Since previous myelograms had suggested a herniated disc, a T10-11 discectomy was performed via a left costotransversectomy approach. The patient obtained no relief from this procedure and his condition continued to deteriorate to the point where he was unable to walk. His sensory loss ascended to a well-defined low thoracic level, and marked spasticity was noted. Repeat myelography now disclosed a complete block to flow at the T10-11 level.

A laminectomy was performed beginning at the T-12 level and extending through T-9. As the decompression was extended superiorly, marked hypertrophy of the posterior elements was noted at the T10-11 vertebral level with severe encroachment on the dural sac. To a lesser extent, a similar situation existed at the T9–10 level. Generous bone decompression was performed from T9–12, and the pathological review of this material was consistent with normal fibrocartilage and bone. Intradural exploration from T9–12 was performed. This procedure revealed no intradural masses, and the cord was of normal contour and size with the exception of the left posterolateral indentation underlying the left T10-11 facet joint.

Postoperative Course. Postoperatively the patient had immediate relief of his leg cramps, dysesthesias, and paresthesias. Decreased spasticity was evident and his strength and ability to walk gradually improved.

FIG. 2. Radiographic studies in Case 1. Left: True sagittal magnetic resonance image of the thoracolumbar area of Case 1. The T10-11 level is denoted by the arrow. There is no identifiable posterior defect, although an incidental hemangioma is present at T-12 (arrow). Right: Computerized tomography sector scan demonstrating a narrowed canal at T10-11 due to hypertrophy of posterior elements. Arrow indicates an incidental retained droplet of Pantopaque.
Case 2

This 31-year-old man presented with complaints of progressive burning paresthesias and numbness of the lower limbs of 4 months' duration, associated with weakness of the left lower extremity. Bowel and bladder symptoms were denied. He suffered from a chronic dense paresis of the right lower limb from polio contracted at the age of 8 months.

Examination. Examination was notable for chronic atrophy and paresis of the right lower limb with absent myotatic reflexes in that limb. Spastic weakness was noted in the left lower limb, more proximally than distally. Bilateral extensor plantar responses and a low thoracic sensory level to pinprick were noted; proprioception and vibratory sensation were severely impaired in the toes and ankles, more so on the left. Routine laboratory studies, including serum levels of calcium, phosphate, and alkaline phosphatase and serology, were unremarkable.

Thoracic spine radiographs were unremarkable. A myelogram (Fig. 3 left) revealed a minimal anterior defect at the T11-12 interspace suggestive of a disc protrusion. A post-metrizamide CT sector scan through this area (Fig. 3 right) failed to confirm a significant anterior defect. It did, however, reveal irregular hypertrophy of the posterior elements producing significant encroachment on the dural sac and cord.

Operation. A decompressive laminectomy at the T11-12 level revealed severe stenosis of the spinal canal due to hypertrophy of the apophyseal joints. The ligamentum flavum, although prominent, was not calcified either grossly or on pathological examination. Intradural exploration was unremarkable. Within 2 months the numbness of the lower limbs had resolved and the patient's strength had improved.

Case 3

This 62-year-old man presented with a 3-year history of numbness of the feet that had markedly worsened over the last year. Weakness and easy fatigability of the lower limbs, exacerbated by standing or walking, had developed over the 4 months prior to admission.

Examination. Examination revealed diminished vibratory appreciation distal to the ankles and decreased position sense of the toes. Brisk tendon reflexes were present at the knees along with clonus at the ankles. Lower-limb tone was spastic, and bilateral extensor plantar responses were present. Serology and serum phosphate and alkaline phosphatase levels were normal. Somatosensory evoked potentials of the posterior tibial nerve were absent. Lumbar and cervical Pantopaque myelography revealed a blockage at the T9-10 vertebral level. Magnetic resonance imaging at this level showed loss of the posterior epidural fat signal on a T1-weighted image (Fig. 4).

Operation. A T8-10 decompressive laminectomy was performed revealing a tightly stenotic canal with hypertrophy of the facets. The ligamentum flavum was not calcified. Pathological examination was consistent with cortical bone and connective tissue. The patient's deficits gradually resolved after decompression.
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Case 4
This 79-year-old man had a 6-month history of a progressively worsening "swollen" sensation of his feet, which developed after walking a distance of 10 to 15 feet. This symptom was relieved by sitting. The patient also noted impaired balance on standing. Sphincter disturbances were denied.

Examination. The cranial nerves and upper limbs were found to be unremarkable. The lower limbs displayed mild weakness with spastic tone. Sensory evaluation was notable for markedly diminished vibratory sensation and proprioception distal to the hips. The myotatic reflexes were pathologically brisk at the knees and the Achilles reflexes were equal to the reflexes of the upper limbs. Extensor plantar responses were present bilaterally. Routine laboratory examinations were unremarkable, including serum levels of alkaline phosphatase, calcium, and phosphate and serology. Myelography showed a complete block at the T10-11 disc space. Parasagittal MRI revealed a large low-signal (T2-weighted) posterior mass producing cord compression at the T10-11 level (Fig. 5). The anterior defect appeared less significant on MRI.

Operation. A T10-11 decompressive laminectomy revealed massive hypertrophy of the apophyseal joints, more so on the right, resulting in a very tight stenosis at this level. The ligamentum flavum was not calcified and, although prominent both below and above this level, it was severely thinned at the site of stenosis. As such, the ligamentum flavum did not contribute to the compression. Pathological examination was consistent with fibrocartilage and normal bone. The patient's postoperative course was unremarkable. He noted virtually immediate subjective improvement in sensation, and his ambulation improved with resolution of his complaints of pseudoclaudication.

Comment
The following two cases differ from those described above in that they involve younger patients who developed subacute thoracic myelopathy, and the onset of symptoms was related to traumatic events. Congenital narrowing of the thoracic spinal canal was contributory in both cases.

Case 5
This 30-year-old man had suffered thoracolumbar back pain since lifting a heavy weight 5 years earlier. During the 6 months prior to his most recent hospital admission, he experienced progressively increasing pain, dysesthesias of his left lower limb, numbness of his left foot and ankle, and weakness of the left lower extremity.

Examination. Neurological examination was notable for: percussion tenderness at T-11; a spastic monoparesis of the entire left lower limb; hypesthesia below T-12 on the left; hypalgesia below L-3 on the right; and a left extensor plantar response. Plain roentgenograms of the thoracic and lumbar spine were not remarkable. A lumbar myelogram revealed a circumferential epidural constrictive lesion at T11-12 producing a partial block to the flow of contrast material. A CT scan revealed a narrow spinal canal with a bulging central disc and short thick pedicles and laminae at T11-12 consistent with focal congenital-spondylitic spinal stenosis.

FIG. 4. Parasagittal T1-weighted magnetic resonance image of Case 3 showing posterior obliteration of an epidural fat signal (arrow) by a hypertrophied facet.

FIG. 5. Parasagittal T2-weighted magnetic resonance image of Case 4 showing thoracic cord compression (arrow) principally due to hypertrophy of the posterior elements, although a small anterior defect is also present.
Operation. The patient was treated with a decompressive laminectomy and mesial facetectomy and posterolateral fusion at T11-12. The operative findings were consistent with hypertrophic spinal stenosis. Immediately postoperatively, the patient’s neurological examination was normal with resolution of his preoperative findings. The postoperative course was uncomplicated. The patient was required to wear a polyform clam-shell orthosis for the first 3 months following surgery. At 7 months postoperatively, the patient had returned to full-time work with limitations. His neurological examination has remained normal over a 2-year follow-up period.

Case 6

This 29-year-old laborer was pushing a heavy log when he noted the acute onset of electric paresthesias, originating in the thoracolumbar area and radiating bilaterally into his lower limbs, associated with weakness. These complaints cleared over a 20-minute period. A few days later similar symptoms developed under identical circumstances.

Examination. Neurological examination was entirely normal except for symmetrical hyperactive myotatic reflexes in the lower limbs and unsustained ankle clonus. Plain roentgenograms of the thoracic and lumbar spine were unremarkable. Myelography revealed a circumferential epidural constrictive lesion extending from T-10 to T-12 producing a partial block to the flow of metrizamide. A CT scan demonstrated a narrow spinal canal at the T10–12 level with short wide pedicles and thickened laminae and central bulging intervertebral discs at T10–11 and T11–12.

Operation. Decompressive laminectomy and mesial facetectomy of T10–12, left transpedicular discectomy at T11–12, and posterolateral fusion of T10–12 were performed 3 months after onset of symptoms. Postoperatively, the patient was neurologically normal and his recovery uncomplicated. He was required to wear a polyform clam-shell orthosis when carrying weight during the first 3 postoperative months. He returned to light work at 6 months and has continued to do well during a 10-month postsurgical follow-up period.

Discussion

Reports of anterior bone ridges associated with cervical myelopathy date back to Key’s accounts in 1838. In 1957, Payne and Spillane pointed out the importance of congenital narrowing of the cervical spine in producing myelopathy. Spondylitic cervical myelopathy is now a well-known entity. Although first noted by Goldthwait in 1911, spondylitic lumbar canal stenosis and its characteristic symptom complex of radicular pseudoclaudication due to compromise of the cauda equina was first regarded as a true clinical disorder by Verbiest, who reported the spinal dimensions, later amplified upon by Wilson, et al., and Pennal and Schatzker.

Descriptions of myelopathy secondary to stenosis of the thoracic spine are distinctly unusual except in the setting of generalized skeletal disorders. Achondroplasia frequently produces stenosis of the entire spinal canal (generally worse in the lumbar region), and resultant thoracic myelopathy has been reported. Similarly, metabolic disorders such as osteochondrodystrophy (Morquio-Brailford’s disease), acromegaly, osteofluorosis, and familial hypophosphatemic vitamin D-refractory rickets may produce similar sequelae. Myelopathy reported in Scheuermann’s disease is associated with a gibbus kyphosis and underlying degeneration of anterior spinal elements. Bone overgrowth from Paget’s disease and ankylosis have also been noted to result in thoracic spinal cord dysfunction.

Our patients had no clinical, radiographic, or hematological stigmata of such disorders. Serology and serum levels of alkaline phosphatase, calcium, and phosphate were uniformly normal. There were no occurrences of antecedent trauma in the older patients; however, onset of local back pain during exertion was found in two of the younger patients.

Thoracic canal stenosis has been considered a rare entity. The role of hypertrophied ligamentum flavum in contributing to discogenic thoracic myelopathy was suggested by Love and Schorn and further discussed by Carson, et al. Osseous hypertrophy of the posterior elements, however, was not a feature of their cases. Govoni described a case of a woman aged in her late 60’s with a 3-month history of progressive thoracic myelopathy characterized by “dragging her left foot” and a “numb, heavy feeling.” Neurological examination was consistent with a mid-thoracic myelopathy. Plain x-ray films revealed a mild kyphoscoliosis. A myelogram showed a complete block to flow at the T9–10 level. At operation, the laminae of T-9 exhibited “severe thickening which acted as a signet ring, resulting in spinal stenosis.” Intradural exploration was apparently not performed, and there was no objective evidence that the posterior elements were truly hypertrophied. The patient subsequently improved.

Hypertrophy of the ligamentum flavum and pedicles, producing symptomatic spinal cord compression, was reported by Kodama, et al., in seven patients. Initial unilateral or bilateral sensory disturbances gradually progressed to weakness, followed by sphincter dysfunction. Hypertrophy of the facets was not a noted feature in this report. Marzluff, et al., described four cases of the spinal cord compression as a consequence of articular process hypertrophy of the thoracic spine. Common features of their series included symmetrical sensory disturbances and weakness; however, sphincteric dysfunction was unusual. They also concluded that this disorder was likely to be more common than was previously recognized. Notable, however, was the lack of postural exacerbation in the patients.

Xu presented four cases of thoracic myelopathy due to degenerative hypertrophy of the posterior spinal elements. Complaints were characteristic of “numb-
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ness," weakness, and bladder disturbance. Myelograms revealed complete blockage at the levels of stenosis. An additional case of such stenosis at the thoracolumbar junction was also presented. The patients' complaints worsened when erect, and this feature of pseudoclaudication was a prominent point of the report. In 1982, Assmann and Besel reported the case of a 66-year-old man with pseudoclaudication due to thoracic myelopathy caused by hypertrophy of the posterior elements. Paresthesias and dysesthesias in the lower limbs progressed and were associated with severe cramps. He became unable to walk. Right leg weakness and a T-12 sensory level were present. An extensor plantar response was present on the right. Spinal tenderness was absent. Cervical and lumbar myelography revealed a complete block from T-10 to T-12. Surgical exploration demonstrated hypertrophy of the posterior bone elements producing an "hour-glass" compression of the cord. The patient succumbed to sepsis before benefit from the operation could be reliably assessed.

We believe that this is the first report defining the CT, MRI, and myelographic appearance of this disorder, along with photographic documentation of cord compression. In our patients, both CT and MRI gave a more accurate representation of the pathology than either oil or water-soluble myelography. Indeed, myelography gave incomplete or misleading information in certain patients, falsely suggesting a causative thoracic disc protrusion when in fact the compression was posterior.

One of our patients (Case 1) first underwent costotransversectomy for discectomy based on the results of two myelograms that suggested disc protrusion. In another patient MRI was misleading because the sagittal study was negative. Since the compression is largely from the posterolateral bone elements, thoracic stenosis is best seen on either parasagittal or axial reconstructions. Computerized tomography sector scanning provided the best evidence of thoracic canal stenosis in the cases where this study was obtained. Because of the limited ability of myelography to define thoracic stenosis, it may well be that some patients who have failed treatment for thoracic disc herniation by anterior or lateral discectomy may suffer from thoracic canal stenosis. Computerized tomography sector scanning of the affected area should prove definitive in patients where this diagnosis is suspected. It is, of course, possible that a patient might have both thoracic stenosis and compression from a small anterior disc protrusion. Our first patient (Case 1) may be an example of this. In such individuals careful analysis of CT, MRI, and myelographic images may be required to select the most appropriate operative approach.

Our older patients were similar to those described by Xu in that symptoms were exacerbated by an erect posture. Extension of the thoracic spine when standing may further compromise the pathologically narrowed canal, causing worsening of symptoms on the basis of either venous obstruction or direct compression of the cord itself. The photographically documented indentation of the cord in our first case lends credence to the latter scenario. Unlike the series of Marzluff, et al., pseudoclaudication was a prominent feature in our older group as well as in several of the other reports cited above. Unlike Xu's series, all our patients and those of Marzluff, et al., had little sphincteric dysfunction; this is likely attributable to earlier intervention in the latter two series.

Common to all these reports is localization of stenotic elements to the lower thoracic spine. In their analysis of human spine kinematics, White and Panjabi showed that thoracic spine mobility, particularly flexion-extension movements, is greatest in these lower segments. It is quite possible that the same processes that promote degenerative canal stenosis of the cervical and lumbar spine are contributory to the pathogenesis of thoracic canal stenosis. Thus, this condition may evolve in a patient with a congenitally narrow canal and abnormal mobility of lower thoracic segments.

Conclusions

Older patients with complaints of pseudoclaudication and painless thoracic myelopathy without recognized predisposing bone or joint disorders may suffer from cord compression due to degenerative overgrowth of the posterior spinal elements. Congenital narrowing of the thoracic canal may potentiate minor injury in younger patients and lead to neurological symptoms and signs. Loss of sphincter function and pain and temperature appreciation tend to occur late in the course of the disorder. Myelography may be nondiagnostic or misleading. Computerized tomography sector scanning of the thoracic spine and MRI are the diagnostic tests of choice. Posterior decompression is recommended.

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