CASE REPORT

Unsuspected reason for sciatica in Bertolotti’s syndrome

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Patients with Bertolotti’s syndrome have characteristic lumbosacral anomalies and often have severe sciatica. We describe a patient with this syndrome in whom standard decompression of the affected nerve root failed, but endoscopic lumbosacral extraforaminal decompression relieved the symptoms. We suggest that the intractable sciatica in this syndrome could arise from impingement of the nerve root extraforaminally by compression caused by the enlarged transverse process.

Bertolotti’s syndrome is of unknown aetiology and is characterised by anomalous enlargement of the transverse process of the most caudal lumbar vertebra, frequently combined with a transitional vertebra. These anomalies articulate or fuse with the sacrum or ilium and are associated with low back pain. Secondary degeneration of the proximally adjacent disc also causes sciatica. Surgical treatment often fails, and unrelenting sciatica may persist.

We describe a patient with this syndrome and discuss the surgical treatment.

Case report

A 46-year-old woman presented with a six-month history of deteriorating low back pain and right-sided sciatica making walking difficult. The pain radiated from the right buttock to the ankle and foot where it was most severe. On a visual analogue scale (VAS) for pain, at 0 to 100, the pain was 88. The Japanese Orthopaedic Association score1 for low back pain (JOA score) was 10/29. Kemp’s test was positive, and at 40° straight leg raising caused radiation of pain into the right buttock. There was grade 4/5 power in the right extensor hallucis longus. Leg reflexes were normal bilaterally, but she had hypoaesthesia in the right foot. Plain radiographs showed 11 thoracic and six lumbar vertebrae (Fig. 1). CT scans showed an enlarged right-sided transverse process at L6 (Fig. 2). MRI scans showed moderate compression of the dural sac on the right side at L5-6 (Fig. 3), and three-dimensional MRI showed mild compression of the right L6 spinal nerve.

Dynamic radiographs showed no instability. An attempted selective radiculogram of the L6 nerve was unsuccessful as the needle did not reach the spinal nerve.

In August 2007 we performed micro-endoscopic decompression of the spinal canal at L6-S1, but the procedure provided little relief of pain. A further attempted selective radiculogram of the L6 nerve was successful and showed entrapment of the right spinal nerve in the extraforaminal zone (Fig. 4). Administration of lidocaine to the right L6 nerve produced complete but transient relief of pain. We concluded that the pain was due to nerve root impingement caused by the extraforaminal stenosis.

In February 2008 we used the METRx system (Medtronic Sofamor Danek, Memphis,
Tennessee) to perform an extraforaminal decompression of right L6. With the patient in the prone position, a 2 cm long skin incision was made 4 cm to the right of the mid-line, tissue layers were sequentially dilated and a tubular retractor was introduced. The lower part of the enlarged transverse process, the lateral wall of the body of L6 and the upper part of the sacral ala were removed with a burr. This revealed the L6 nerve root. Post-operative CT images showed the extent of the decompression (Fig. 5). This operation relieved both the low back pain and the sciatica. At the final follow-up, 30 months after surgery, the VAS had decreased to 10 points and the JOA score had increased from 10 to 25. The strength in the right extensor hallucis longus recovered fully, and she was able to walk well and returned to work.

Discussion
In 1917 Bertolotti\(^2\) described anomalous enlargement of the transverse process of the most caudal lumbar vertebra, which could articulate or fuse with the sacrum or ilium and be associated with low back pain. Cases are often associated with transitional vertebrae and there may be six lumbar vertebrae. These changes are associated with disc degeneration or instability at the adjacent proximal level. It is estimated this syndrome accounts for 4.6%\(^3\) to 7%\(^4\) of cases of low back pain in adults, and for more than 11% of patients with low back pain who are under 30 years old.\(^3\)

There are several treatment options for the syndrome. Marks and Thulbourne\(^5\) showed that eight of ten patients who received steroid and local anaesthetic injections into the anomalous lumbosacral articulations experienced immediate relief of severe low back pain, but five relapsed after a mean of 8.2 days (1 day to 12 weeks). At long-term follow-up three patients continued to have partial relief, but only one remained pain-free.

Two surgical series have been reported. Jönsson, Strömqvist and Egund\(^6\) observed that resection of the anomalous process in cases in which a local anaesthetic injection had relieved the symptoms produced total (n = 7) or significant (n = 2) pain relief in nine of 11 patients. Santavirta et al\(^7\) found that posterolateral fusion of the transitional segment (n = 8) and resection of a unilateral anomaly (n = 8) produced comparable results, after a mean
follow-up of nine years, ten reported improvement in low back pain, but the outcome for sciatica was unsatisfactory. This was the first study to report the results for sciatica. In our case, the first surgical procedure (spinal decompres-
sion) also failed to relieve pain. We had not established extraforaminal impingement of the L6 nerve root as the precise cause of the pain because we were unable to perform a successful selective radiculogram until the first spinal canal surgery had failed. Three-dimensional MRI of the spinal nerve was less helpful than the selective radiculogram (Fig. 4). We emphasise the importance of obtaining a selective radiculogram if other procedures leave the diagno-
sis uncertain.

Extraforaminal decompression greatly relieved the patient’s pain, and weakness of the right extensor hallucis longus recovered fully, probably because the presence of 11 thoracic and six lumbar vertebrae meant that the L6 nerve root was functionally L5 (Fig. 1). To our knowledge, this is the first study to demonstrate lumbosacral extraforaminal involvement in Bertolotti’s syndrome. The authors of an earlier study of transverse process resection7 did not describe impingement of the spinal nerve in this zone which was necessary in our patient. The articulation between the transverse process and the sacrum itself did not cause impingement (Figs 4 and 5).

Lumbosacral extraforaminal stenosis can be caused by disc herniation and by several other conditions, including impingement by the transverse process, the Far-out syndrome, a lumbosacral ligament, vertebral body and sacral ala,8-10 and a spur from the L5 vertebral body.9,11

The surgical options for this condition include fenestration, posterolateral fusion and transforaminal interbody fusion, but the anatomical features in the region of a lumbosacral extraforaminal lesion make all of these procedures difficult. The sacral ala, the iliac crest and thick muscles hinder access. We agree with a previous report that microendoscopic decompression is particularly useful in this location.11

In conclusion, surgeons should be aware that sciatica in Bertolotti’s syndrome may arise from impingement of the spinal nerve in the extraforaminal zone, and that microendoscopic surgery may successfully relieve the compression and the symptoms.

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References


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