Posttraumatic cervical spondyloptosis at C6–7 with late-onset cord compression: a new clinical entity

Case report

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An unusual case of total spondyloptosis of the cervical spine at the C6–7 level with late-onset cord compression is described in an 8-year-old girl. The patient was treated by anterior decompression and in situ fusion as it was thought hazardous to try an anatomical reduction. The patient’s excellent neurological recovery postoperatively strongly supports the use of this treatment protocol. The authors believe this is the first report of a posttraumatic spondyloptosis of the cervical spine, presenting with a late-onset cord compression. A brief summary of the clinical presentation, the surgical technique, and a review of the relevant literature is presented.

KEY WORDS  ·  cervical spondylolisthesis  ·  spinal cord compression  ·  spinal fusion  ·  decompression surgery

Spondylolisthesis (spondyloptosis) and spondylolysis of the lumbar spine are well-described entities commonly encountered in clinical practice; however, these conditions are rarely seen affecting the cervical spine. Only 11 cases of cervical spondylolisthesis have been reported in the literature and none of these cases had a major neurological deficit. The present case of total spondyloptosis of the cervical spine at the C6–7 level, with late-onset cord compression, is the first of its kind to be reported.

All 10 previously reported cases were treated either conservatively or by interspinous wiring and local posterior fusion. In our patient it was thought to be hazardous to attempt an anatomical reduction, and hence anterior decompression and in situ fusion were carried out.

Case Report

Clinical Presentation

This 8-year-old girl presented to our clinic with a rapidly progressive motor weakness involving both lower limbs. She had been born at home, of a full-term vaginal delivery with breech presentation. The child had an obstetric palsy at birth affecting her left upper limb, for which she was managed by an expectant line of treatment and physiotherapy for 6 months but exhibited no recovery. She achieved each milestone normally, and was a healthy child, save for the fact that she had no strength in her left upper limb.

Six months before presentation at our clinic, the patient developed a sudden onset of weakness of both lower limbs, which rapidly progressed over a period of 2 months to almost complete paraplegia. There was no history of associated mental deterioration, sensory affection, or bladder/bowel incontinence, nor any specific precipitating factor preceding the neurological deterioration.

Examination. On examination, the patient’s cervical spine was found to be freely mobile, with no tenderness or spasm. The rest of the spine was also unremarkable. Neurological examination of the patient showed her higher functions and cranial nerves to be normal and all modalities of sensations to be preserved. Signs suggestive of spinal cord compression, such as weakness, spasticity, extensor plantar reflexes, brisk jerks, and bilateral ankle clonus, were observed in both lower limbs. Muscle strength in the lower limbs was 0 to 1/5 (only a flicker). The left upper limb showed...
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FIG. 1. Preoperative magnetic resonance image of the cervical spine demonstrating the C-6 vertebra situated in front of the C-7 vertebra, with subsequent anterior spinal cord compression.

FIG. 2. Schematic diagram showing preoperative spondyloptosis at the C6-7 level with associated spinal cord compression (left) and the postoperative appearance (right) demonstrating the effect of spinal canal decompression and the location of the strut bone graft.

fig. 3. Postoperative magnetic resonance image of the cervical spine demonstrating the effect of anterior decompression and restoration of the spinal canal.

muscle wasting and hypotonia, its strength being about 1/5. The right upper limb was normal. There was no sphincter disturbance.

Plain roentgenograms and metrizamide contrast studies of the cervical spine showed malalignment of the vertebrae from C-5 to T-1 and elongation of the posterior elements of the C-5 vertebra. A magnetic resonance (MR) image of the cervical spine (Fig. 1) showed total spondyloptosis at the C6-7 level, with the body of the C-6 vertebra lying almost totally in front of the body of the C-7 vertebra and with marked compression of the spinal cord at the C6-7 level. There was no intraspinal abnormality. The MR image also showed a focal area of myelomalacia at the C6-7 level. After reviewing the patient's clinicoradiological status and considering the chronicity of the slip, an anterior decompression and in situ fusion was planned as it was thought to be hazardous to attempt an anatomical reduction.

Operation. The patient was operated on in the supine position under general anesthesia. The cervical spine was exposed by a standard left-sided anterior approach. The cervical decompression was comprised of total corpectomies of C-5, C-6, and C-7 and excision of the C4-5, C5-6, C6-7, and C7-T1 intervertebral discs (Fig. 2). The decompression was extended laterally to visualize the C-5, C-6, C-7, and T-1 roots, especially on the left side, and was followed by an in situ tricortical iliac-crest block bone graft from C-4 to T-1.

Postoperative Course. Since no internal fixation was used to stabilize the bone graft, as a precautionary measure the patient was strictly confined to bed for 8 weeks postoperatively. During this time, she was not allowed to sit up or turn in bed.

Eight days postoperatively, the patient began to show improvement in her neurological status which then progressed rapidly until, by the end of the 6th postoperative week, she had regained useful muscle strength (3 to 4/5) in both lower limbs. During the same period, much to our surprise, her left upper limb, which had been paralyzed since the birth trauma, also showed significant motor recovery, proximally more than distally (proximal from 1/5 to 3/5, distal from 3/5 to 2/5). At the end of 8 weeks, after radiological confirmation of the bone graft stability, the patient was given a soft cervical collar with molded chin support and was allowed to sit up in bed. Mobilization and ambulation were begun gradually.

A postoperative roentgenogram showed the bone graft to be in good position and incorporating well. An MR image obtained 3 months postoperatively (Fig. 3) also showed the bone graft incorporating well and the epidural space re-formed, although there was still persistence of a focal area of myelomalacia at the site of maximum compression. At the time of discharge 3 months after surgery, the patient was able to stand and walk independently with minimal support. The sensations and sphincter control that were unaffected preoperatively remained unaltered.
Discussion

Spondylolysis and spondylolisthesis of the cervical spine are very rare entities. In 1951, Perlman and Hawes were the first to report a case of cervical spondylolisthesis; since then only 10 more cases of cervical spondylolisthesis have been reported in the literature. Interestingly, none of these patients had any major neurological complications, and all were treated either conservatively or by posterior fusion alone. This is the first report of a severe degree of slippage (spondylolysis) occurring in the lower cervical spine and associated with marked neurological deficit.

The etiology in the current case seems to be posttraumatic, subsequent to a significant birth trauma. The definite history of a difficult delivery associated with an obstetric palsy, the elongated posterolateral elements of the dislocated vertebrae (malunited fractures), and the lack of any other associated congenital anomalies strongly support our assumption. The absence of any significant trauma prior to the onset of lower limb weakness, a painless range of neck movements at the time of presentation, and a focal area of myelomalacia demonstrated on MR imaging pointed to a "late onset" or a "delayed" type of extradural spinal cord compression, which was indeed responsible for this most unusual presentation.

All of the cases of cervical spondylolysis and spondylolisthesis reported in the literature were treated either conservatively or by posterior fusion alone. However, the problems in our case were unique, necessitating a different approach. In view of the severe degree of displacement of a long-standing nature, attempting an anatomical reduction was thought to be hazardous. It was therefore decided to perform an anterior decompression and an accompanying in situ fusion to prevent any further damage to the spinal cord. The wide lateral decompression performed during the anterior surgery was also beneficial in terms of root release, as evidenced by the neurological recovery in the left upper limb.

Since the patient's neurological deficit was due largely to an anterior extradural spinal cord compression of a mechanical nature, she had an excellent neurological and functional recovery subsequent to anterior decompression and accompanying in situ fusion. This postoperative outcome strongly supports the value of our treatment protocol.

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