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Cauda equina compression due to a laminar hook: A late complication of posterior instrumentation in scoliosis surgery

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Abstract Whilst neurologic injury following correction of scoliosis with CD-instrumentation is generally known to be an early complication, any late occurrence of cauda compression secondary to employment of a laminar hook-rod construct is exceptional. We report on such a rare case of late occurrence of cauda equina syndrome, when a laminar hook at level L2 became symptomatic causing compression of the cauda equina almost a decade after spine surgery. This case demonstrates that one should not only be

aware of a potential neural injury at intraoperative placement of laminar hooks, but also one is reminded that a laminar hook poses the threat of late neurologic injury years after successful osseous spinal fusion. The surgeon treating patients with scoliosis must be aware of the possible complication described in our patient in addition to those that have already been well documented.

Key words CD instrumentation · Spine-Fix · Scoliosis · Neurologic injury

Introduction

Posterior spinal instrumentation for the correction of deformity was pioneered by Lange [18] and popularized by Harrington with his distraction rod construct in the 1960s [13]. A new posterior instrumentation was developed by Yves Cotrel and Jean Dubousset in 1983 [3], to overcome shortcomings of preceding posterior instrumentation systems. Cortel-Dubousset (CD) instrumentation has gained wide popularity and initiated the development of a great number of similar systems adhering to the CD principles [3, 8, 14, 21, 24 and Synthes 1995, personal communication].

Benefits as well as disadvantages of CD instrumentation are recognized in the literature. Disadvantages include the great number of vertebral implants and ancillary instruments and a permanent rod-implant locking system making instrumentation removal a challenge [10]. Fractures of transverse processes [3], intraoperative lamina fractures [23], dislodgements of bottom hooks on the convex side and an unwanted increase in rotation in uninstrumented segments of the curve have also been noted with use of the CD instrumentation [23].

Whilst neurologic injury is generally known to be an early complication of such instrumentation [1, 3], any late occurrence of cord or cauda compression secondary to CD instrumentation is exceptional. We report on a rare case of late occurrence of cauda equina syndrome secondary to correction of scoliosis with Spine-Fix, a CD-like instrumentation, thereby stressing the importance of considering the possibility of such a complication occurring years after successful surgery.

Whilst neurologic injury is generally known to be an early complication of such instrumentation [1, 3], any late occurrence of cord or cauda compression secondary to CD instrumentation is exceptional. We report on a rare case of late occurrence of cauda equina syndrome secondary to correction of scoliosis with Spine-Fix, a CD-like instrumentation, thereby stressing the importance of considering the possibility of such a complication occurring years after successful surgery.

Case report

We report on a 29-year-old female patient with rapid onset of cauda equina syndrome without preceding trauma, 8 years after scoliosis surgery. In 1989 surgical correction of an idiopathic adolescent scoliosis, type I according to King, had been performed. The preoperative Cobb angle for the instrumented curve was 48°, it was corrected to 33° immediately postoperatively – a correction of 31%. Spinal instrumentation consisted of a rigid posterior interlinked double rod instrumentation with hooks affixed to laminae of

Fig. 1 Radiograph of a 29-year-old woman showing Spine-Fix posterior instrumentation – a derivative of CD instrumentation – from T11 to L3 for correction of idiopathic scoliosis

Fig. 2a, b Myelography demonstrating a tapered column of contrast medium and a filling defect at the level of the laminar hook

Fig. 3 CT scan of the L2 region showing hook position in the spinal canal

T11–L3 (Fig. 1). Spine-Fix, a system bearing great resemblance to the CD system, was used for posterior instrumentation (Synthes 1998, personal communication). Fusion technique included decortication of laminae, facet joint cleaning and the use of autogenic bone strips.

A sudden loss of bladder tone followed by a disabling left-sided leg pain 48 h later were the initial symptoms interrupting an 8-year period of well-being after scoliosis surgery. Anal tone was unaffected. Because initially sensitivity, motor power and tendon reflexes were unaltered, the spine was disregarded and extensive urological examinations were performed. Loss of sensory function was first noted by the patient 7 days after initial symptoms were observed. Hypoesthesia affected the posterior aspect of the left calf and thigh and developed within hours to a full perianal and perigenital anesthesia and analgesia. Weakness of lower limbs with the inability to walk on toes and heels followed loss of sensitivity, with a latency of only 48 h. When neural deficits had developed into a full blown cauda equina syndrome, by the 11th day after on-

set of symptoms, the patient was admitted to hospital. At initial examination at the neurosurgical unit, patellar tendon reflexes were normal and achilles tendon reflexes had vanished. Power in plantar flexion had decreased to MCR grade 4 bilaterally, power in ankle dorsiflexion had decreased to MCR grade 2 on the left and grade 4 on the right, while hip flexors and knee extensors had remained normal. Standing radiographs of the spine were taken and compared to previous spine films available from earlier biannual routine follow-up evaluations. A loss of correction of the instrumented curve by 8° had occurred in between 1989 and 1992; thereafter, evolution of the curve had been without further radiologic deterioration and unchanged Cobb angles of 41° had been recorded since 1992.

Myelography with water-soluble dye was augmented by CT scanning, revealing a left-sided compression of the cauda by a laminar hook at level L2 (Figs. 2, 3). Surgery was immediately scheduled and the spine prepared from a dorsal approach alongside the implant. Complete removal of the instrumentation was carried out. A laminar hook with a caudad-pointed tip positioned at the con-

cavity of L2 had caused intraspinal cauda compression. Intraoperatively, the spinal implant in its entirety was found to be not disassembled or broken, but mobile. Additional osseous decompression of intact dorsal spinal elements was not indicated, because myelography had detected isolated compression by the implant. Solid posterolateral osseous fusion from T11 to L3 was found when the spine was tested for segmental stability. Bacteriological swabs taken from the wound showed no growth. The macroscopic appearance of the site did not suggest infection; histology described a chronic granulating and fibrous inflammatory process. Postoperative monitoring observed earliest recovery of bladder tone and subsequent bladder control within 24 h. Complete restoration of muscle power within 3 days allowed for a normal gait within less than 1 week; hypoesthesia receded within 1 week. A fortnight after spinal surgery, the patient was discharged, neurologically recovered, with the exception of increased urine retention (150 ml) following voluntary bladder emptying. Bony fusion made external support of the spine unnecessary. The latest antero-posterior standing radiograph of the spine, 12 months following removal of hardware, documents maintenance of previously recorded Cobb angles.

Discussion

Paraplegia following operations for the correction of scoliosis has been reported in approximately 1% of 10,000 recorded operations [6]. Many authors have reported on perioperative neurologic complications [5, 19, 20, 25], especially following the use of sublaminar wiring [7, 15, 25]. Cotrel reported two incidences of neurologic injury in his series of 250 patients using CD instrumentation; paraplegia was detected less than 6 h following surgery, and resolved with implant removal in both instances. Compression of the dural sac by overlapping blades of laminar hooks used at the same level and in the same direction was identified as the sole cause in one patient [3].

Reports of late neurologic complications solely implicating the instrumentation are uncommon [9, 12, 16, 17]. The rare occurrence of late complications leading to neurologic impairment is usually reported in the presence of wire breakage [2] or pseudarthrosis [4, 22]. Beyond that, single cases of late neurologic complications following posterior spinal instrumentation using laminar hooks have been reported, i.e., bone overgrowth leading to paraparesis [9], sciatic nerve compression by hematoma [11], nerve root impingement at the site of lumbar hook insertion [16], neural compression by a caudad migration of an L5 hook through a lamina [12], and cord compression by

a migrated rod after pressure-induced laminar atrophy [17].

In our case the dramatic myelographic obstruction at the time of delayed paraparesis indicates that there was either significant edema of the cauda at this level or actual mechanical dural impingement by the instrumentation. The contribution of edema of the cauda within a canal that is already narrowed by implants residing inside the spinal canal and impinging on neural structures must be considered in determining the pathogenesis of this complication. It is clear that the delayed paraplegia resulted from a progression of spinal ischemic and edema-producing events. However, the question of whether the laminar hook actually moved and displaced into the canal or whether its long-term sublaminar presence exacerbated neural irritation by dural impingement at the time of neurologic deterioration remains unresolved. No pressure atrophy of the affected lamina, nor postmenopausal generalized osteopenia of the spine, nor bone encasement of the hook pressing against the dura were seen. Nor did we suspect a degree of pistoning of the implant that might have enhanced hook migration, because fusion was solid. Etiological uncertainty of this delayed major neurologic deficit remains.

In conclusion, this case demonstrates that one should not only be aware of a potential neural injury during the laminar placement of hooks – especially on the concave side of scoliotic deformities – but one should also keep in mind that a lumbar laminar hook poses a potential threat of neurologic impairment a decade after surgery, despite the presence of osseous spinal fusion. The surgeon treating patients with scoliosis must be aware of the possible complication described in our patient in addition to those that have already been well documented, to prevent delay in early diagnosis and immediate treatment. The possibility of such a complication producing a major neurologic deficit indicates that careful neural surveillance should continue beyond the immediate postoperative period as long as the patient returns for a follow-up examination. However, needless to say, even though the spine surgeon is tempted to search for a mechanical cause of spinal disorders like spinal deformity and spinal implants, other causes of the described symptoms have to be born in mind, i.e., leg weakness due to demyelinating disease or ischemia secondary to aortoiliac disease.

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