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CASE REPORT

Recurrent adjacent segment disease and cauda equina syndrome

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Abstract
Purpose A case of cauda equina lesion as a result of recurrent adjacent segment degeneration (ASD) after multiple lumbar fusions is reported. ASD might be a consequence of biomechanical overload or simply a normal degenerative process. The reported clinical relevance of ASD is rather low. We describe an unusual case of cauda equina compression at L1–L2 in a patient who had undergone L2–L4 fusion 8 years previously and 2 decompression-fusion surgeries 16 years before.
Materials and methods A 72-year-old man, who had two previous lumbar fusion–decompression procedures, underwent a third lumbar surgery in December 2000 to treat symptomatic spinal canal stenosis associated with L3–L4 pseudoarthrosis. After a symptom-free period of 8 years, the patient experienced low back pain radiating to both legs while standing, associated with saddle sensory disturbances and incontinence. Physical examination ruled out significant motor deficits. Plain radiographs showed solid fusion from L2 to L4, good spinal alignment, and low-grade L1–L2 retrolisthesis. Stainless steel pedicular instrumentation distorted magnetic resonance imaging, preventing adequate spinal canal evaluation. Electromyography demonstrated signs of cauda equina compression (bilateral L3–S2). CT myelography showed a stop at L1–L2, due to a severe spinal canal stenosis. L1–L2 decompression and fusion were performed.
Results After an uneventful surgery with no complications, the symptoms abated and incontinence recovered.
Conclusions Even if the reported clinical relevance of ASD is very low, fused patients with a constitutional narrow spinal canal are at risk of developing severe neural compression at the level adjacent to the fusion.

Keywords Recurrent disease · Adjacent segment · Cauda equina · Degenerative changes · Narrow spinal canal

Introduction

Several years after posterior instrumented lumbar fusion, radiographic changes showing degeneration of mobile segments may appear homogeneously at different levels cephalad to fusion. This event seems to be determined more by individual characteristics than by fusion, itself [1]. The most common radiographic findings are disc degeneration and hypertrophic facet arthritis, whereas other features, such acquired spondylolisthesis, nucleus pulposus herniation, osteophyte formation, stenosis, and scoliosis, are observed less frequently [2–5]. Adjacent segment degeneration (ASD) has been defined as a recurrence of symptoms associated with the degeneration at the free segment above a fusion after a symptom-free period [5]. The incidence of (symptomatic) ASD is low, ranging from 5.2 to 18% [2]. We report an unusual case of a patient developing cauda equina syndrome due to recurrent ASD, 8 years after his last posterior instrumented spinal fusion.

Case report

In December 2000, a 71-year-old man underwent a third lumbar decompression-fusion procedure to treat symptomatic

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spinal canal stenosis associated with L3–L4 pseudoarthrosis. Sixteen and 15 years before, lumbar decompression with L3–L5 posterior instrumented fusion had been performed elsewhere to treat, respectively, symptomatic L4–L5 stenosis with L5 radiculopathy, and pseudoarthrosis. In December 2000, surgery performed in our center to treat recurrence of symptoms consisted of wide L2–L3 decompression, partial implant removal, interbody fusion to restore lumbar sagittal contour and increase fusion area, bone grafting, and posterior fusion from L2 to L4 using stainless steel pedicle screw instrumentation. In 2009, after a symptom-free period of 8 years, the patient consulted for progressive low back pain radiating to both legs while standing or walking. On this occasion, however, the associated leg pain was accompanied by saddle sensory disturbances, urinary incontinence, and motor weakness. Three months after the onset of symptoms, physical examination ruled out significant lower limb motor deficits. Plain radiographs showed solid lumbar fusion from L2 to L5 with good spinal alignment and L1–L2 retrolisthesis (Fig. 1). Electromyography demonstrated signs of cauda equina compression (bilateral, L3–S2). The patient's stainless steel instrumentation prevented magnetic resonance imaging (MRI) examination of the spinal canal (Fig. 2). However, the computed tomography (CT) myelogram showed a complete stop at L1–L2, due to extradural circumferential compression (Fig. 3). The patient was re-operated for the fourth time. Surgical findings included hypertrophy of posterior elements (joints and ligamentum flavum) as well as a disc prolapse in a constitutionally narrow lumbar canal. L1–L2 decompression and fusion were performed without intraoperative complications (Fig. 4). The postoperative course was uneventful, symptoms abated gradually and incontinence recovered.

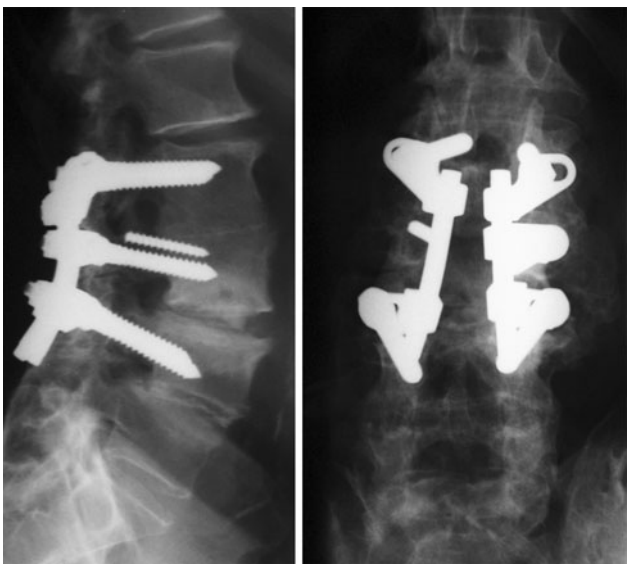

Fig. 1 Preoperative plain films showing solid lumbar spinal fusion from L2 to L4, with good spinal alignment and low-grade L1 retrolisthesis

Discussion

The reported incidence of radiographic degeneration at the unfused segments following lumbar fusion ranges from 8 to 100%, whereas symptomatic ASD is reported less often, with an incidence of 5.2–18.5%. The clinical relevance of ASD is low in the great majority of cases [2, 6–8]. Cauda equina syndrome is a very unusual onset of ASD.

The etiology of ASD is probably multifactorial. Results of exposure-discordant monozygotic and classic twin studies suggest that mechanical factors play a very limited

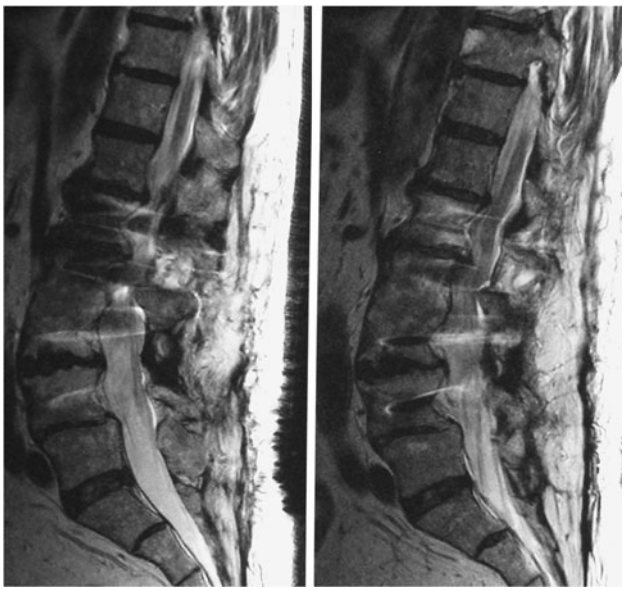

Fig. 2 Lumbar spine MRI images are distorted because of stainless steel pedicular instrumentation. Note grade I L1 retrolisthesis

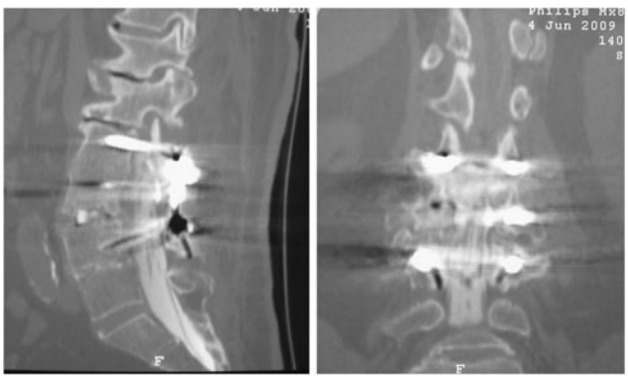

Fig. 3 CT myelogram demonstrates a complete stop in contrast advance at L1–L2 due to a severe spinal canal stenosis

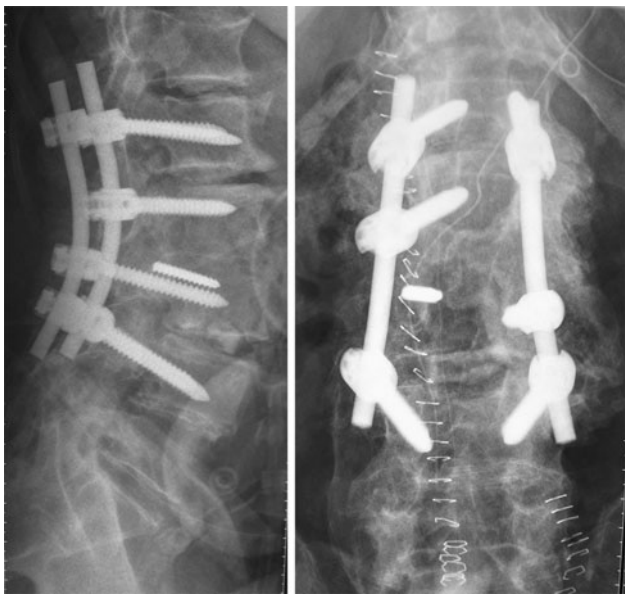

Fig. 4 Postoperative plain radiographs after spinal canal decompression and fusion extension from L1 to L4

role in segmental spinal degeneration, while constitutional factors and heredity may have a dominant role, explaining 74% of the variance in the adult population studied to date [9]. In vitro studies have found altered biomechanics in the mobile segments adjacent to fusion, including increased mobility and increased intradiscal pressure [10, 11]. These studies constitute the basic-science body of evidence supporting the idea that ASD is due to the mechanical overload as a consequence of fusion. However, the in vivo situation is probably more complex, involving muscles, external loads, and a combination of forces. Axelson et al. [12] analyzed the motion pattern of juxtafused L4–L5 in six patients with lumbosacral fusion by roentgen stereophotogrammetric study and found that transformation of preoperative mobility in the lumbosacral segment to the adjacent segment was not a general phenomenon.

The results of some radiographic studies suggest that the fused patients have a greater prevalence of degenerative changes than the non-fused population. Comparing case series in non-randomized trials certainly entails a selection bias. Patients needing an operation because of segmental degeneration may have a higher risk of further degeneration than the population not needing surgery. Unfortunately, the prevalence of degenerative changes in patients for whom fusion has been prescribed, but not performed, is uncertain. A recent prospective randomized study [8] comparing the effect of fusion with natural history in isthmic spondylolisthesis suggests that laminectomy and fusion accelerate degeneration of the adjacent segment. However, the prevalence of severe ASD is low and the effect on clinical outcome limited.

Our patient had a constitutional narrow lumbar canal prone to develop symptomatic compression of neural structures. He was first operated at the age of 56 to treat further narrowing of the lumbar canal as a consequence of "natural or spontaneous" segmental degeneration unassociated with previous fusion. Subsequently, he underwent surgery twice, in 2000 and 2009, to treat symptomatic spinal canal stenosis at the level adjacent to the fusion area. Is ASD in our patient purely mechanical and linked to lumbar fusion overload or does it reflect the natural history of constitutional lumbar canal stenosis? It is likely that both genetics and mechanics play a role. Previous studies have shown that genetically determined natural progression of degeneration might be modified to some degree by environmental factors [9]. ASD is likely a consequence of normal degenerative progression influenced by altered biomechanics related to fusion [1, 2, 4, 6, 13–16]. Fusion length, stiffness of instrumentation, posterior interbody fusion, and sagittal balance could be important parameters linked to ASD [2–4, 6].

Cauda equina compression is an extremely rare clinical onset of ASD. We found only one previous report in the literature, by Okuda in 2004, describing two patients with neurologic deterioration 2 years after posterior instrumented lumbar fusion. Both patients were surgically treated with laminotomy without fusion because no instability was detected. The authors suggested that coexistence of laminar horizontalization and asymmetry in the facet joint at the adjacent segment is a major risk factor for the development of neurologic complications after spinal fusion [13].

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Conflict of interest None of the authors has any potential conflict of interest.

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