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CASE REPORT

Osteochondral loose body: an unusual cause of lumbar spinal stenosis

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Abstract Lumbar spinal stenosis is most often a degenerate condition observed in the older population. We describe the case of a lady with typical claudicant symptoms with an unusual cause of stenosis identified at the time of her decompressive surgery. On review of the literature this is only the second case of osteochondral loose body as a cause for lumbar spinal stenosis and thus remains a rare phenomenon.

Keywords Lumbar spinal stenosis · Osteochondral loose body · Spinal claudication

Background

Central lumbar spinal stenosis leads to a variable syndrome of back and leg pain due to thecal sac compression. Males are more commonly affected than females and this is most often a degenerate phenomenon seen in the older population. In this subset of individuals, spinal canal narrowing is commonly due to facet joint hypertrophy, buckling of the ligamentum flavum, degenerative spondylolisthesis or a combination of these pathologies.

Composed of cartilage alone or cartilage and bone, intra-articular loose bodies result from any process that leads to disruption of the articular surface. They derive nutrition from synovial fluid and contain any of the cells of bone or cartilage. The surface cells form more cartilaginous layers, so enlarging the body over time. Deeper cells receive less nutrition resulting in cell death and calcification [1]. Such loose bodies tend to be found in articulations such as the knee or hip joint. There are two reports in the literature of loose bodies in the cervical spine, one asymptomatic [2] and the other a cause for cervical myelopathy [3]. To our knowledge, this is the third reported finding of a loose body in the lumbar spine. The first case reported is that similar to our own with a single-level symptomatic stenosis [4]. The second case is one of multiple loose bodies associated with a symptomatic lytic spondylolisthesis [5].

Case presentation

We introduce a 64-year-old female who presented with classic symptoms of spinal claudication. Her claudication distance was variable coming on between a couple of dozen and a couple of hundred yards leading to pain radiating over the buttocks down the backs of the legs. This was ruining her quality of life, preventing her from going shopping and threatening to affect her independence. She would sit to relieve the pain and the time taken for the leg pain to subside varied between a few minutes and any time up to an hour. Back pain was a minor component for her. She did not describe any sinister features or symptoms suggestive of cauda equina syndrome.

Her past medical history was largely unremarkable, she had no problems with her peripheral vasculature, she was not diabetic, epileptic or asthmatic and had no history of strokes or heart attacks. She was hypertensive, but this was well controlled by medication.

On examination she walked with a normal reciprocal gait and could walk both on heels and on tiptoes. She demonstrated an exacerbation of her pain when she tried to stand fully upright. She had no elicitable reflexes in the

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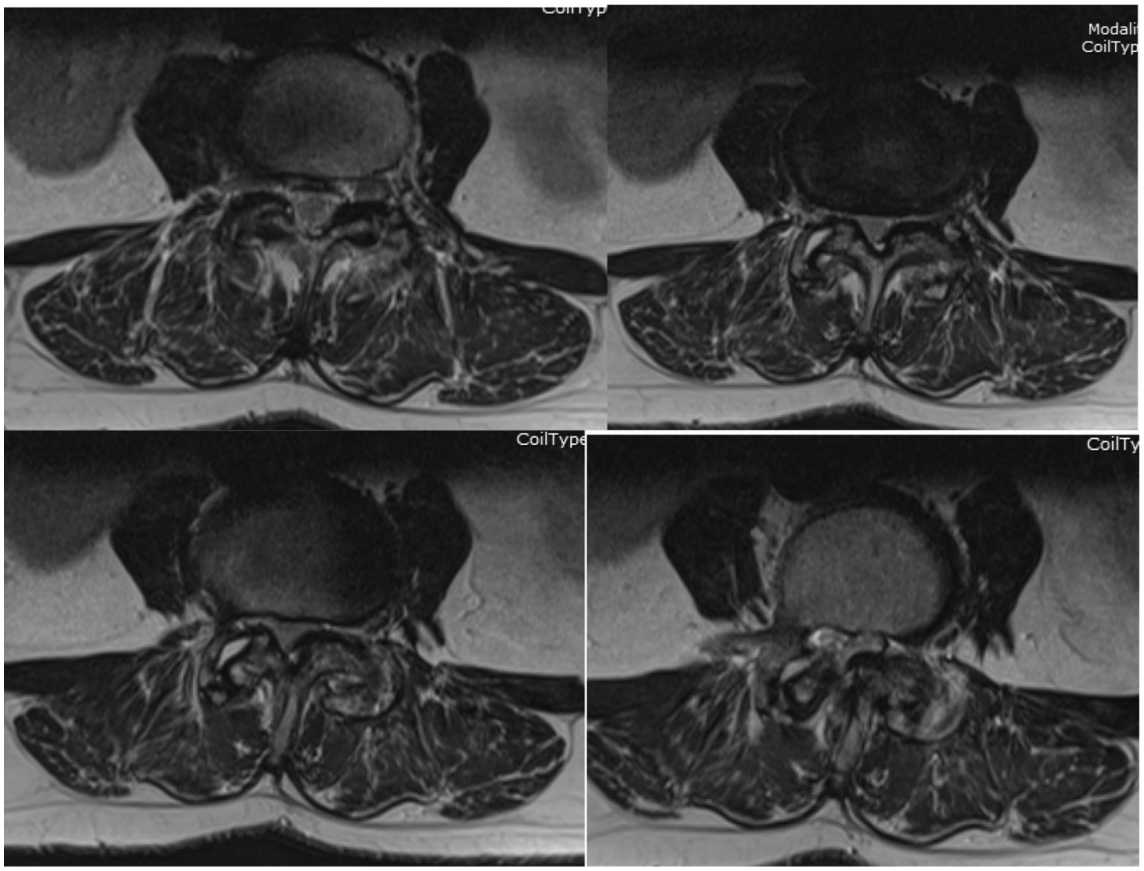

Fig. 1 Axial T2-weighted MRI sequences through L3/4 disc space (cranial to caudal)

lower limbs and no long tract signs. There were no nerve root tension signs.

An MRI scan of her lower back demonstrated multilevel degenerate change with a grade 1 degenerate spondylolisthesis at L3/4 causing effacement of the cerebrospinal fluid from the cauda equina at this level. Axial slices through this level, mid-sagittal and left para sagittal images are included below (Figs. 1, 2).

Management options were discussed with the patient who decided to proceed with lumbar decompressive surgery. At the time of operation she was positioned prone on Oswestry cushions and a standard lumbar decompression performed. During exposure the L3 spinous process was found to be mobile and a large loose osteochondral fragment (Fig. 3), the size of a thumbnail, was discovered lying in the left side of the spinal canal beneath the lamina.

On the first postoperative morning this lady was neurologically intact, independently mobilising and was discharged home later the same day without complication. At follow-up in the outpatient clinic 3 months later, she reported no complications and was delighted as her leg pain remained resolved.

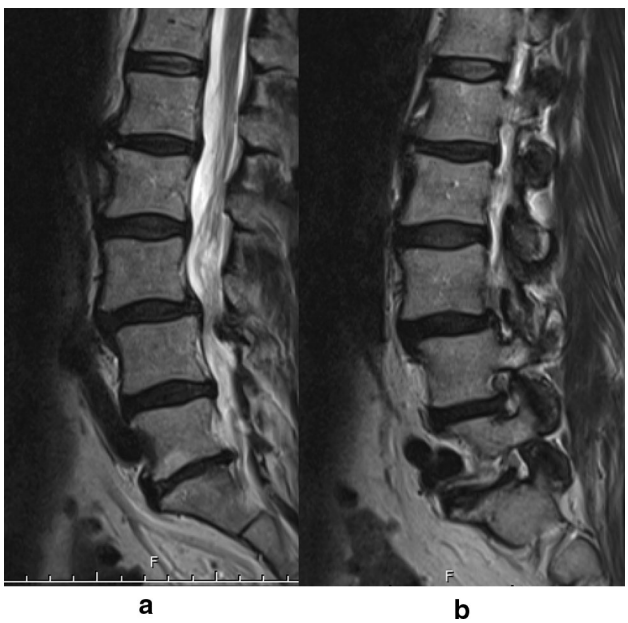

Fig. 2 Sagittal T2-weighted MRI images of lumbar spine. **a** Mid-sagittal, **b** left para sagittal

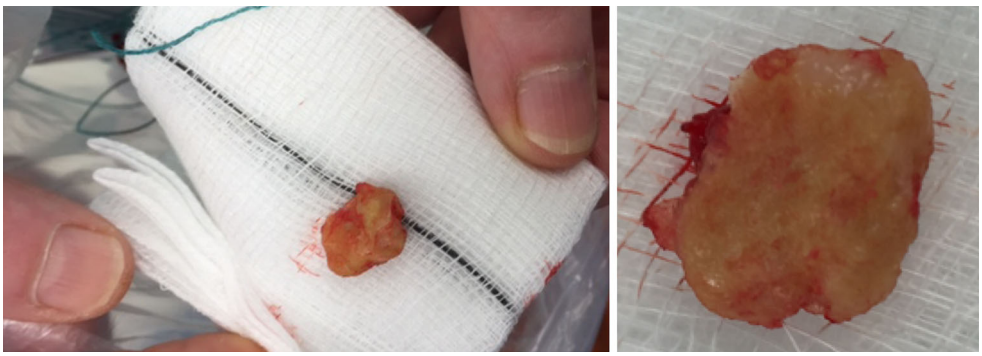

Fig. 3 Photograph of osteochondral loose body

Discussion

We describe the second case in the literature of a single loose body as a cause of lumbar spinal stenosis. The relatively short onset of leg symptoms on a longstanding history of lumbar back pain alongside the immediate relief of leg pain postoperatively correlates with the loose body indeed being the causative factor for her spinal claudication as no other compressive areas were found intraoperatively. As in the 'Spinolith' described by Tambe et al. in 2002 [4] the origin of this loose body remains uncertain however, given the grade 1 degenerate listhesis, we postulate that facet joint hypertrophy has been the original source of the osteochondral fragment. There is, of course, a possibility that origin has been from the mobile spinal process; however, as this was intact at its base and mobile outwith the spinal canal only, we think this to be unlikely.

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Compliance with ethical standards

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

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