Epidural abscess related to *Streptococcus mitis* in a 57-year-old immunocompetent patient

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1. Introduction

Spinal epidural abscess, a collection of pus between the thecal sac and surrounding tissue, is a rare condition with an incidence of 2.4 cases per 100 000 persons. Half of the cases are due to a haematogenous spread, and a third because of a discitis spread. *Staphylococcus aureus* is involved in 50–65% of cases. Usually seen in adults older than 60 years, risk factors include immunodeficiency, malignancy and intravenous drug abuse. Adequate timely diagnosis of spinal epidural abscess is crucial because around one-fourth of patients may develop motor deficit or paralysis.

We describe the case of a healthy patient who presented a lumbar spinal epidural abscess related to a dental infection following lumbar torsion.

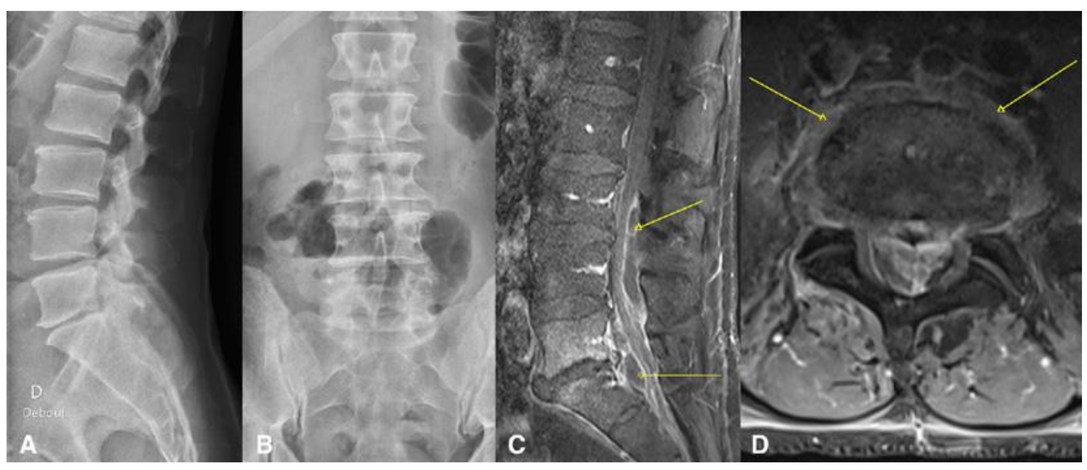

Figure 1. Preoperative lumbar imaging. (A, B) Radiographs showed only slight signs of degenerative disc disease at the L5-S1 level, and no inflammatory signs such as vertebral erosion. Lumbar MRI on T1-gadolinium sagittal sequences (C) and axial views at the admission (D), showing a spinal epidural abscess (arrows) with a severe stenosis, and a peripheral enhancement of the soft tissue around the L5-S1 level (arrows).

2. Case presentation

A 57-year-old male patient, known for risky consumption of alcohol, presented to our emergency department after a 10-day duration of low back pain. He reported a lumbar torsion after a loss of balance on a rough ground 10 days ago, then a persistent and progressive lumbar pain. He reported slight fever a few days before. He mentioned an untreated dental pain of teeth 27 and 28. Clinical assessment found a paravertebral painful muscular contracture of the lumbar spine, and no neurological deficit of the lower extremities.

Laboratory tests revealed a systemic inflammatory syndrome with an ESR of 60 mm/h, a CRP of 111 mg/L and a WBCC of 23 G/L with 91% of segmented neutrophils. Lumbar radiographs showed only degenerative signs, and lumbar MRI found an epidural abscess posteriorly of the thecal sac at the L3-L4 and L4-L5 levels, and anteriorly at the L5-S1 level. There was also a L5-S1 discopathy with a suspected psoas abscess (**Figure 1**).

The patient had emergency surgery, using a posterior approach, with a decompressive laminectomy in a right cross-over shape from L3 to S1 without any stabilization. Collections were taken for microbiological studies (**Figure 2**). He received intravenous amoxicillin-clavulanic acid 2.2 g 3x daily until the microbiological results were available. Abscess cultures revealed a multisensitive *Streptococcus mitis* in all positive samples (5/7), similarly to blood cultures. A transoral echocardiography found no endocarditis. Empiric antibiotic treatment was replaced by penicillin-G 5M of units 4x daily for 10 days.

Lumbar pain decreased and the patient presented no fever during his hospital stay. CRP decreased from 256 mg/L at postoperative day 3 to 47 at the last hospitalization day, as well as the WBCC (4.7 G/L). The patient was discharged from the hospital after 11 days. The antibiotic treatment was finally changed to intravenous ceftriaxone 2 g daily for the remaining 4 weeks in order to achieve a 6-week duration of antibiotics. The infected teeth were treated by surgical avulsion. At a 6-week follow-up, the patient was asymptomatic, and the CRP was 6 mg/L, with satisfactory wound healing. After 1 year, a lumbar MRI showed the same collapse of the L5-S1 but no sign of persistent or recurrent infection (**Figure 3**).

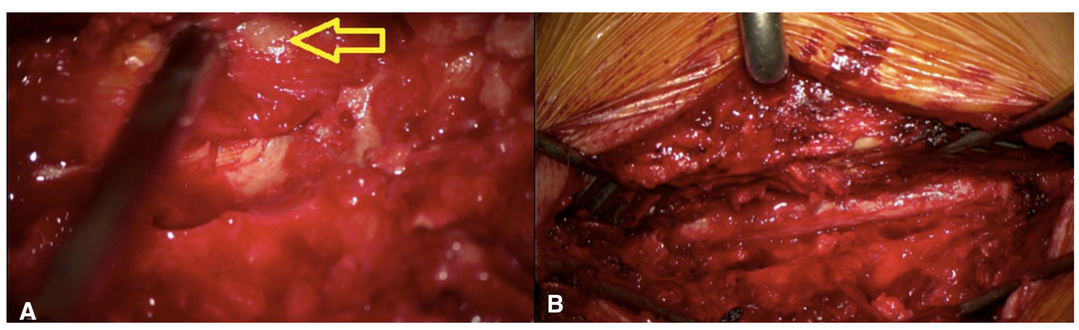

Figure 2. Intraoperative images of the spinal epidural abscess (arrow) after decompressive laminectomy (A). The abscess was immediately washed out for samples, and the dura mater was free of infectious material and was not affected (B).

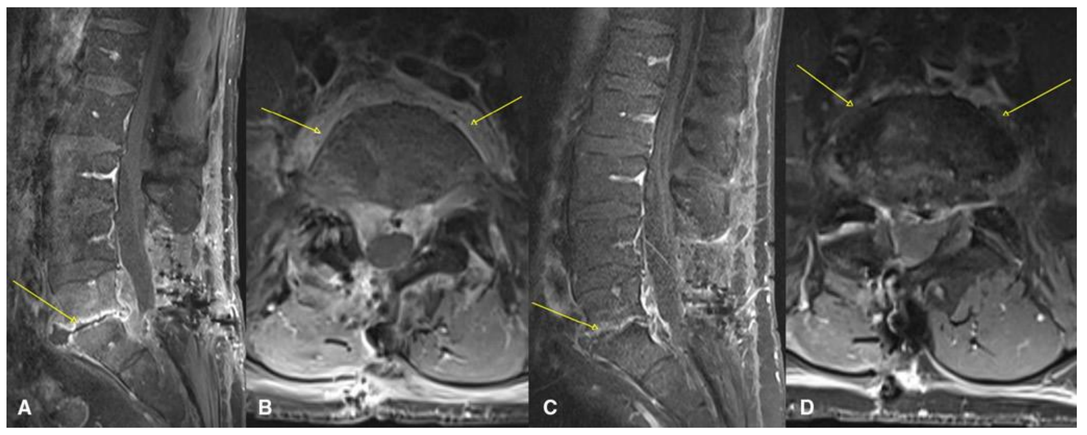

Figure 3. Postoperative lumbar MRI on T1-weighted gadolinium sequences. (A) At 6-week follow-up, demonstrating resolved infection and complete collapse of the L5-S1 disc (arrow). This collapses with peripheral inflammatory pannus (arrows). (B) Confirmed the spondylodiscitis diagnosis. (C) At 1-year follow-up, there was no sign of persistent or recurrent abscess, and the collapse of L5-S1 disc was stable. There was also no evidence of spondylolisthesis related to secondary instability. The peripheral inflammatory pannus of the soft tissues around the L5-S1 level resolved (D).

3. Discussion

Spinal epidural abscess related to *S. mitis* was rarely reported in the literature, with only 6.8% cases caused by *Streptococcus species*. *S. mitis* can be found in oral biofilms and occasionally may cause systemic infections. Its association with endocarditis and dental infections is well known. Transoesophageal echocardiography is mandatory to rule out endocarditis. Other cases of skeletal infections due to *S. mitis* were reported in the literature. The most likely joint infected by *S. mitis* is the knee. Cariati *et al* described a thoracic spondylodiscitis in a patient with chronic sinusitis. Feder and Gruson reported a case of glenohumeral infection, Nomura *et al* reported an osteomyelitis of a lower extremity bone in a child. Yusuf and colleagues and Cinar *et al* published cases of pelvic ring infections. Finally, another case of spondylodiscitis related to *S. mitis* was reported by Prior-Español. Cone and colleagues described 4 cases of endocarditis leading to spondylodiscitis, with one related to *S. mitis*. Only a few cases of spinal epidural abscess due to *S. mitis* have been published. Martin and Lee described a case about a C4-C5 anterior abscess in a 57-year-old haemophilic man. They emphasized the need for adequate microbiological diagnosis by sampling during the surgical procedure. Byrd and Nemeth described the case of a 57-year-old man with poor dentition and chronic alcohol abuse who presented a cauda equina syndrome related to an epidural abscess and a septic endocarditis with bacteraemia related to *S. mitis*. All patients showed recovery after drainage and antibiotics.

Most of the authors recommend early decompressive surgery for epidural abscesses in case of occurrence of neurological deficit. Here, the role of surgery is crucial to protect neurological function and to prevent irreversible severe deficits. The surgery helped to define the pathogen and the most appropriate antibiotic treatment.

In the current case, the huge volume of the epidural masses and the extension in the spinal canal with severe stenosis, despite the absence of neurological deficit, encouraged us to perform a decompressive laminectomy. Ju *et al* recommend early surgical treatment for patients with severe spinal stenosis to prevent from motor deficit. Most probably, our patient would have worsened without surgery. This was emphasized by the review of Tuchman and colleagues who conclude in favour of urgent surgical decompression for patients able to undergo surgery before an unpredictable progression in the disease may lead to a neurological impairment.

Studies reporting a link between vertebral blunt trauma and spinal epidural abscess formation are rare. Baker *et al* reported 12/39 cases of blunt trauma such as heavy lifting or a fall. Heusner reported 4/20 cases of spinal epidural abscess related to trauma. Finally, Hulme and colleagues described only 1 case among 10, and the literature review from Reihsaus *et al* reported trauma in 25–34.7% of cases of spinal epidural abscess, including direct inoculation by spinal puncture, without mention of torsion trauma reported. In the present case, we think that, considering a pain-free period before the lumbar torsion of the patient, an insidious onset of L5-S1 spondylodiscitis was related to *S. mitis* spreading from dental caries infecting tissues damaged during the torsion, leading to abscess formation. The final collapse of the L5-S1 disc on lumbar MRI at the 6-week follow-up favours this hypothesis (**Figure 3**).