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Case Report

Spinal epidural abscess in the lumbar spine after dental extraction in a 53-year-old man

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ABSTRACT

Spinal epidural abscess is rare in patients following dental extraction. Only seven cases have been described in the literature. We report the first case of an epidural abscess in the lumbar spine following dental extraction, and present a review of the relevant literature. A 53-year-old man presented with low back pain 1 week following dental extraction, and imaging revealed the presence of a lumbar epidural abscess. He underwent surgical drainage by decompressive laminectomy with evacuation of pus and debridement of the infected bone, and he was treated with a prolonged course of intravenous antibiotics. The patient demonstrated no neurologic sequelae at the 6-month follow-up examination. A search of the relevant literature showed that, of the seven epidural abscesses that occurred following dental extractions, five were cervical and two were intracranial. An epidural abscess in the lumbar spine following dental extraction had not been reported. Thus, this is the first report of an epidural abscess in the lumbar spine following dental extraction. It is also the first case of epidural abscess following dental extraction that was determined to be caused by *Streptococcus suis*. Our findings indicate that epidural abscess must be considered as a diagnosis for all patients presenting with intractable low back pain, with or without fever and neurologic impairment, after a recent dental extraction. We conclude that appropriate imaging must be conducted for early diagnosis.

1. Introduction

Low back pain is a common adult complaint that is usually self-limited, with no serious underlying pathology. We present an unusual case of an epidural abscess in the lumbar spine of a 53-year-old man who presented with low back pain following dental extraction. This potentially life-threatening complication was diagnosed based on neurologic deficits, and he was successfully treated with surgical decompression and debridement followed by antibiotic therapy. An extensive search of the literature revealed reports of seven cases of epidural abscesses in the cervical spine or intracranial region that were secondary to dental extraction. To our knowledge, this is the first reported case in the lumbar spine. *Streptococcus suis* (Group R) was cultured from the patient's blood. *Streptococcus* is the second most common cause of spinal abscess.

2. Case report

A 53-year-old man had a history of an appendectomy and a left ureteral stone that was treated with ureterorenoscopy lithotripsy. He was currently employed in the aluminum industry with a work history of more than 30 years. He underwent two tooth extractions under local anesthesia in a local dental clinic about December 31, 2010. There were no significant anesthetic or surgical complications, except for mild swelling of the extraction site for 2 days. However, he developed constant low back pain 1 week following the extractions.

He visited the urology clinic on January 3, 2011. Physical examination showed paraspinal tenderness in the lumbar region. Urinalysis showed 3+ proteinuria. Recurrence of the ureteral stone was suspected, and nonsteroidal anti-inflammatory drugs were prescribed. However, the low back symptoms persisted, and he consulted the nephrology clinic on January 4, 2011. Physical examination revealed paraspinal tenderness. No fever was observed, but laboratory findings included an elevated white blood cell (WBC) count of 11.3×10^9 cells/L (reference range: 5.0×10^9 to 10.0×10^9 cells/L). Abdominal sonography showed a mild fatty liver. A lumbar muscle sprain was suspected, and bed rest and analgesia were advised.

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His low back pain progressively increased. On January 9, 2011, persistent fever developed that was resistant to treatment with antipyretic medication. On January 12, 2011, he sought treatment in our emergency department, presenting high fever, severe low back pain radiating to both lower limbs, urine incontinence, and walking disability. On examination, marked tenderness was detected over the paravertebral region with marked restriction of movement in all planes. Urinary difficulty was reported, and urinary catheterization was performed. Plain lumbar radiographs showed no soft tissue or bony abnormalities.

The patient was admitted to the hospital on January 12, 2011. His body temperature was 39.8 °C, and laboratory results showed a WBC count of 19.7 × 10^9/L with 89.6% neutrophils and 8.91% lymphocytes. His erythrocyte sedimentation rate (ESR) was 39 mm/hour (reference range: 0–20), and his C-reactive protein (CRP) level was 297 mg/L (reference range: 0.0–8.0). Spinal infection was suspected, and betamicin and vancomycin were intravenously (IV) administered.

Because of the severe low back pain, lumbar magnetic resonance imaging (MRI) was not initially performed. We performed a computed tomography (CT) scan of the lumbar region for temporary evaluation, which showed dural compression by an epidural lesion over the L3-4 and the L4-5 discs (Fig. 1). Lumbar MRI was performed under sedation the following day, and the presence of a large spinal epidural abscess (SEA) was confirmed that extended from the L3-4 to the L4-5 disc levels, predominantly on the left side (Fig. 2).

The patient's initial response to the antibiotic treatment was not satisfactory, and surgical drainage was performed on January 15, 2011. The patient underwent a posterior laminectomy of L3-4-5 and the epidural abscess was drained (Fig. 3). A large amount of deep wound pus was removed that yielded no organisms from culturing, presumably because of the prior antibiotic treatment. However, *Streptococcus suis* (Group R) was isolated from blood cultures. No infective focus for metastatic spread or a history of invasive procedures that were likely to have caused the transient bacteremia were identified, other than the recent dental extractions.

The patient received IV antibiotic therapy with betamicin and vancomycin for 2 weeks following the surgical procedure. He was discharged in stable condition on January 28, 2011. He was prescribed 250 mg Fucidin every 8 hours by mouth and 600 mg rifampicin once daily by mouth for 10 days. He made an uneventful recovery, with no significant neurologic sequelae reported at the 1-week and 6-month outpatient follow-up evaluations.

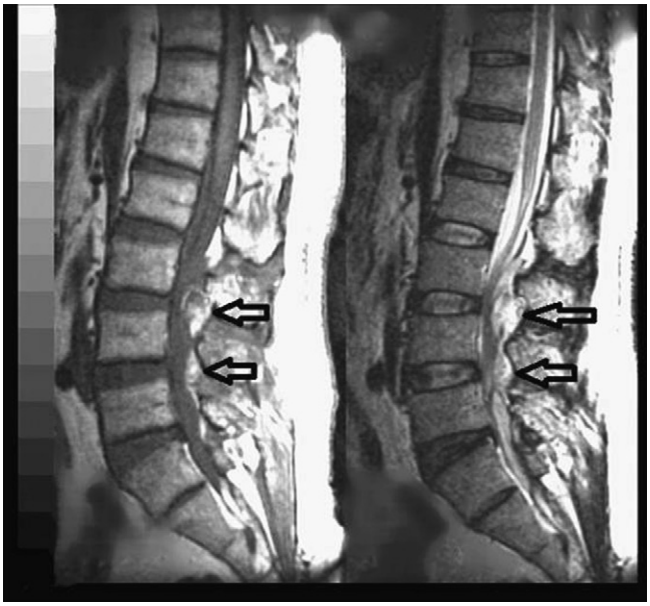

Fig. 2. Sagittal T1 and T2 magnetic resonance imaging of the lumbar spine showing an epidural abscess at the L3-4 and L4-5 disc levels (arrows).

3. Discussion

SEAs are rare, accounting for 0.2–1.2 cases per 10 000 hospital admissions.[1,2] The symptoms associated with an acute spinal abscess develop suddenly within several days or weeks following an infection elsewhere in the body, typically in an immunocompromised patient.[2–4] However, our patient was not immunocompromised, and he developed an SEA, possibly the result of poor oral health status and hygiene.

The clinical presentation of SEAs is variable and frequently nonspecific. The classic triad of back pain, fever, and neurologic deficits has been seen in only 8–37% of patients with SEAs.[1] Fever has been reported in 60–70% of the cases, and spinal tenderness has been reported in up to one-third of the cases.[1,4–9] The rate of disease progression is also variable. Heusner[1] provided a description of the four stages of SEA progression. Stage 1 is characterized by severe back pain, fever, and local tenderness in the area of the spinal column. Stage 2 is characterized by signs of spinal irritation and neck

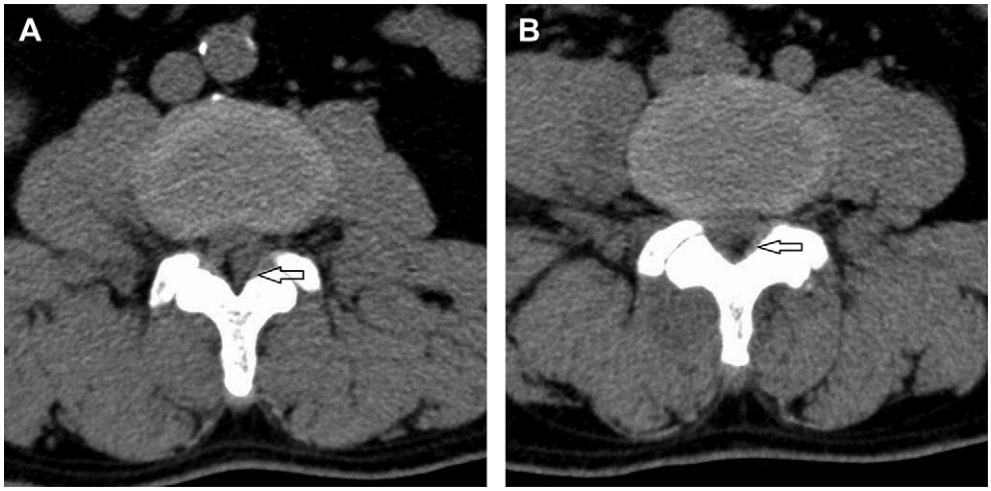

Fig. 1. Computed tomography scan showing dural compression by an epidural lesion over (A) L3-4 (arrow) and (B) L4-5 (arrow).

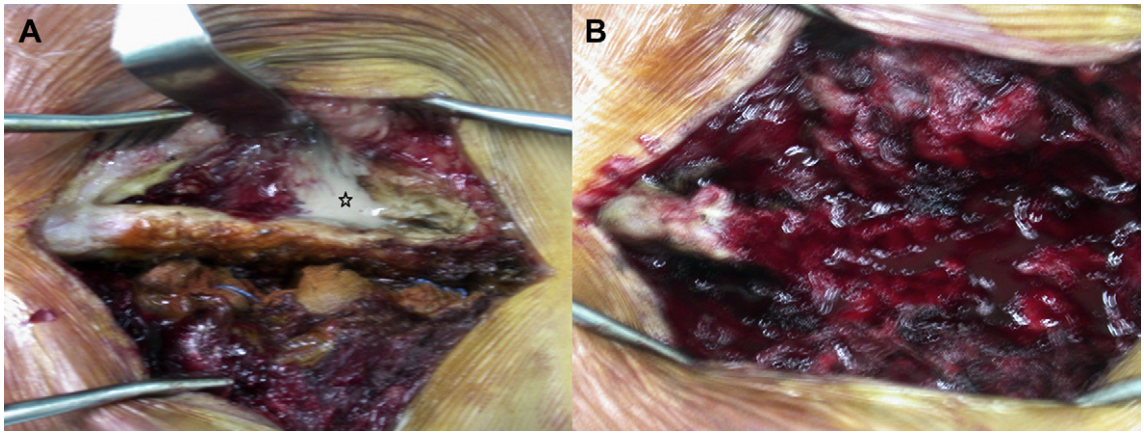

Fig. 3. (A) A massive amount of pus (asterisk) was noted during decompressive laminectomy; (B) posterior laminectomy of L3-4-5 with debridement of the infected bone and soft tissue was performed.

stiffness. Stage 3 is characterized by weakness of the voluntary musculature and fecal or urinary incontinence, with or without sensory deficits. Stage 4 is complete paralysis. Patients typically first present in either Stage 1 or 2, but are often not definitively diagnosed until either Stage 3 or 4 is manifested.[1] To avoid irreversible neurologic damage, a prompt diagnosis is necessary because early treatment is an important determinant of patient outcome. In the current patient, only the low back pain and the paravertebral tenderness were initially observed during the urology and nephrology clinic examinations, with no fever or neurologic deficits reported. Eight days later, the disease had progressed to stage 3 before the diagnosis was confirmed based on the MRI results. Therefore, the absence of fever and neurologic symptoms in an individual with low back pain does not exclude a diagnosis of an epidural abscess following a recent invasive surgery, including dental extraction.

Low back pain, with or without sciatica, is a common symptom that is usually self-limited, with no serious underlying pathology, and subsides in 80–90% of patients within 6 weeks.[10] However, in our patient, the progressively increasing low back pain with marked radiation to the lower limbs, paravertebral tenderness, ambulatory difficulty, and fever prompted us to perform the CT and MRI imaging and the further laboratory analysis.

The WBC count, ESR, and the CRP level are useful indices of infection for the diagnosis and management of SEAs. Generally, we first examine the WBC differential count to exclude spinal infection. However, patients with SEAs may be normothermic with unremarkable WBC counts.[11] Leukocytosis is seen in only 60–78% of patients with SEAs.[1,4,6,7,9,12] Neither the total WBC count nor the percentage of neutrophils are sensitive enough to be indicators of SEA.[1,13] ESR is a more sensitive screening tool than the WBC count,[1] and most patients with SEAs have an elevated ESR. Indeed, Reihsaus et al[6] reported elevated ESR in 110 of 117 patients (94%). The CRP level, another useful screening tool, also rises rapidly with the onset of inflammation.[1,13,14] Accordingly, the recent surgical history, the WBC differential count, and the ESR or the CRP level should be evaluated in patients presenting with persistent low-back pain. Such assessment may increase the likelihood of early diagnosis and treatment of an SEA, thereby reducing neurologic complications.

The CRP level is also a useful measure of the response to treatment because it returns to reference levels more quickly compared with the ESR.[1,8,13,14] In this case, broad-spectrum antibiotics were administered IV until the CRP level was below 30 mg/L (reference range: 0.0–8.0).

Lumbar MRI is highly sensitive, highly specific, and can accurately delineate the extent and location of a spinal abscess. On T2-weighted images, the hyperintense signal of the epidural mass can be differentiated from the non-enhanced thecal sac and neural elements.[1] We initially performed lumbar CT scan for our patient because his low back pain was too severe for the MRI procedure without sedation. An epidural lesion was observed using CT, but we were unable to confirm the type of lesion involved. CT currently has a more limited role in the diagnosis of SEAs because it cannot distinguish SEAs from other lesions that compress the thecal sac.[1] Therefore, the first choice in the diagnosis of an epidural abscess should be MRI if there is no contraindication for the procedure.[15,16]

Abscesses may form in the epidural space by three basic pathophysiological mechanisms: (1) hematogenous spread, (2) direct extension from an infected contiguous structure, and (3) iatrogenic inoculation.[1] The skin and soft tissue represent the most common primary source of infection, accounting for 15–25% of reported cases.[1,6,7] Abscesses formed by this route are generally localized in the posterior epidural space,[1] which is consistent with the SEA in our patient. Our patient had undergone two dental extractions 2 weeks before our initial examination, but had never undergone an invasive spinal procedure. He also had not received conservative chiropractic therapy, which had been previously reported for one SEA patient.[17] We were unable to identify any potential sources of infection, other than the dental extractions. The patient's history suggested a pyogenic infection caused by hematogenous spread.

General anesthesia given during dental extraction procedures is a risk factor for bacteremia, and is known to increase the prevalence and duration of bacteremia.[18] However, for this patient, dental extraction was performed under local anesthesia, which generally lowers the risk of bacteremia.

To our knowledge, this is the eighth reported case of an epidural abscess resulting from dental extraction and the first describing an SEA in the lumbar spine. All previous cases involved the cervical spine or the intracranial region.[3,15,19–22] The most common causative organism, *Staphylococcus aureus*, was present in up to 70% of SEA patients.[1,6] *Streptococcus* ssp. are the second most common, accounting for approximately 7% of the cases.[1,6] In our patient, *S suis* (Group R) was isolated from the blood culture, but was not isolated from the abscess itself, probably because the blood was collected prior to the antibiotic treatment that preceded the surgical intervention.

More than 70 microbial species have been isolated from oral mucosa, saliva, denture surfaces, and dental plaque. Oral streptococci represent over 80% of the mouth microflora.[23] In our case study, the blood culture was collected before the antibiotic treatment was initiated. *S suis* was isolated from the blood sample, and

the transient bacteremia was highly suspected to have originated from infection in the oral cavity. No infective focus for metastatic spread or a history of invasive procedures that were likely to have caused the transient bacteremia were identified, other than the recent dental extractions.

Epidural abscesses constitute a medical emergency, as rapid, irreversible damage to the spinal cord can occur.[21] Despite advances in imaging techniques and surgical treatments, SEA remains a challenging problem because early diagnosis is often difficult and treatment is therefore frequently delayed.[12] Ideally, the diagnosis should be made before neurologic symptoms develop. Treatment involves prompt surgical drainage by decompressive laminectomy with the evacuation of the pus and the debridement of the infected bone, followed by parenteral antibiotic treatment.[3] A delay in drainage may result in permanent damage of neurologic function.

Prognosis is related to the degree of neurological impairment and the duration of the symptoms before treatment.[3,4] The lumbar epidural abscess in our patient was identified after neurologic symptoms developed, but the outcome was good, with no neurologic sequelae observed during follow-up examinations.

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