CASO CLÍNICO/CASE REPORT

A Rare Case of Low Back Pain

Um Caso Raro de Dor Lombar

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Abstract

Epidural abscesses are rare but potentially severe suppurative infections of the central nervous system and can cause damage to structures such as the brain or spinal cord, leading to permanent complications or even death. Two distinct varieties of epidural abscess occur spinal epidural abscess and intracranial epidural abscess We report a case of spinal epidural abscess that, on several occasions, was confused with other causes of low back pain, which, consequently, motivated several admissions to the emergency department.

Resumo

Abcessos epidurais são infeções supurativas raras mas potencialmente graves do sistema nervoso central, podendo causar compressão de estruturas como o cérebro ou medula espinhal levando a complicações permanentes, ou mesmo à morte.

Existem duas entidades distintas de abcessos epidurais: abcessos epidurais espinhais e abcessos epidurais intracranianos. Nós reportamos um caso de abcessos epidurais espinhais que, por várias vezes, foi confundida com causas mais comuns de dor lombar o que, consequentemente, motivou várias admissões no serviço de urgência.

Introduction

Epidural abscesses are rare but potentially severe suppurative infections of the central nervous system and can cause damage to structures such as the brain or spinal cord, leading to permanent complications or even death. Two distinct varieties of epidural abscess occur, spinal epidural abscess (SEA) and intracranial epidural abscess (IEA). We report a case of SEA that, on several occasions, was confused with other causes of low back pain, which, consequently, motivated several admissions to the emergency department.

This case describes an extensive SEA that was misdiagnosed on two occasions, which led to 15 days until an accurate diagnosis was made. The symptoms are nonspecific and, for this reason, we should always be suspicious of this entity when faced with a clinic for fever and low back pain.

Though the diagnosis was late and the patient developed signs and symptoms of advanced disease, she evolved well, and completely recovered all deficits.

Case Report

A 54-year-old white female, obese (body mass index 32.6), and diabetic non-insulin-dependent, presented three times to the emergency department (ED) for low back pain.

On the first visit, she complained of mechanical low back pain radiating to the lower limbs, constant, with no relief or worsening factors.

Radiography was performed showing some degenerative changes, and an osteoarticular etiology was assumed. Therefore, the patient was discharged with analgesia.

Ten days later, the patient developed a fever (38.2°C) and the low back pain remained, which motivated a second visit to the emergency department.

A complementary study was carried out which showed leukocytosis (11.360/uL) with neutrophilia (91%), leukocyturia (233/field), nitrituria, and elevated C protein reactive (CRP) (34 mg/dL). The clinic and laboratory findings were interpreted as pyelonephritis and the patient was discharged medicated with cefuroxime.

On the fourth day after the second visit, the patient was still feverish, with low back pain, and started to notice a loss of muscle strength on the left side. She also reported two cutaneous abscesses, one of about 5 cm on the scalp and a smaller one of about 2 cm, on the left side of the neck. She also referred that one bigger abscess of about 10 cm, on the scalp, had drained spontaneously and in large quantities, one week ago.

At the ED, she was hemodynamically stable and feverish (38°C). The neurological exam showed a left hemiparesis grade 4, neck stiffness, and positive Kernig and Brudzinski signs. There were no changes in the assessment of sensitivities, the osteotendinous reflexes had a normal rapid response, and muscle tone was normal. The presence of the abscess in the scalp and neck was observed, with no other abnormal findings.

The complete blood count showed leukocytosis (19.121/uL) with 90% neutrophils, and an increased CRP (34 mg/dL).

A cranial, cervical, and lumbosacral computed tomography (CT) scan, without contrast, was performed. While the cranial CT was normal, the cervical CT showed an apparent epidural hypodense collection between C2-C4 and on lumbar CT, changes of an inflammatory nature / infectious process were observed, images that needed better clarification by magnetic resonance imaging (MRI).

At this point, the main diagnostic hypotheses were (1) occult infection with secondary meningeal affection or (2) empyema. For this reason, and to exclude meningitis, a lumbar puncture was attempted, with drainage of purulent content. Two sets of blood cultures were collected and empirical antibiotics were started with ceftriaxone, vancomycin, and ampicillin. The patient was promptly admitted to the intermediate care unit.

The blood and pus content were positive for methicillin-sensitive *Staphylococcus aureus* (MSSA) and the antibiotic was changed to flucloxacillin. A transthoracic echocardiogram was performed, which showed no signs of bacterial endocarditis.

At this point, the presumptive diagnosis of epidural abscesses was performed and an MRI was required for the entire spine to assess other areas of involvement, which showed a voluminous collection involving the cervical, thoracic and lumbar region, from C0 to S1, which obstructed the circulation of the cerebrospinal fluid, molding the cord in its entire length. Additionally, signs of septic arthritis in L4-L5 were found (**Fig. 1**).

The patient was then submitted to laminectomy and flavetomy of C4, T3, T11, and L4, having performed an abundant washing of the epidural space with saline and cefazolin.

Thereafter the patient underwent eight weeks of antibiotic therapy with flucloxacillin. Neurological deficits gradually recovered, culminating in their complete resolution, although she still had some difficulty in mobilizing due to low back pain and also due to some muscular atrophy resulting from prolonged hospitalization. For this

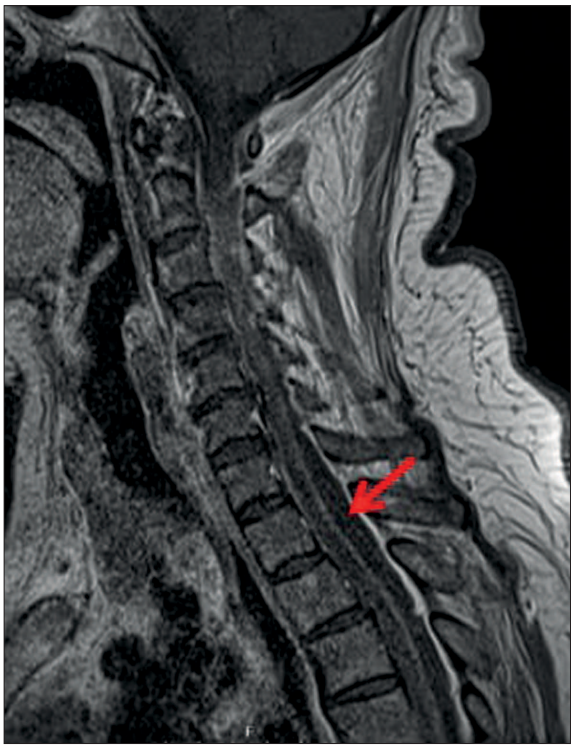

Figure 1. Preoperative (Sagittal section with contrast in the cervical region): voluminous collection, intracanal, posterior epidural. It causes obstruction of CSF circulation anteriorly and posteriorly, molding the spinal cord in its entirety. Red arrow on posterior epidural collection.

reason, the patient was admitted to a long-term care unit where she stayed for two months. Six months after being discharged, an MRI was repeated and showed to be normal (**Fig. 2**).

One year has passed and the patient is completely asymptomatic, autonomous, and without sequelae or residual neurological deficits.

Discussion

SEA is a severe infection of the epidural space that can lead to permanent neurologic deficits.[1]

It is an uncommon diagnosis, with an estimated prevalence of from 1.2 to 3 per 10 000 hospitalized patients, which is increasing in recent years. This increased diagnosis is thought to be attributed to the expanded utilization of invasive spinal procedures, increasing age of the population, and increasing rates of intravenous drug use.[1]

SEA usually occurs in patients over 50 years and males (ratio 1:0.56). One possible explanation for the predominance of the male gender can be found, at least partially, on the risk factors associated with SEA, such as alcohol abuse, use of intravenous drugs, and trauma, which affect men more.[1]

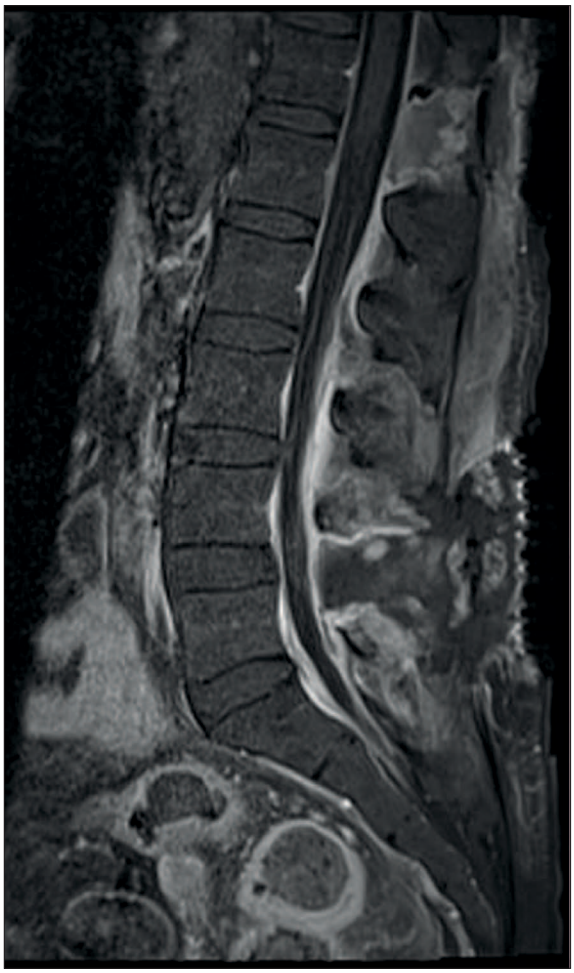

Figure 2. Postoperative (Sagittal section, with contrast, of lumbar region) – Good drainage of the posterior epidural abscess of the dural sac. Artifact in the paravertebral musculature due to recent surgical approach

In a meta-analysis of 915 patients, the most common risk factor was diabetes mellitus, followed by trauma, intravenous drug abuse, and alcoholism. Epidural anesthesia or analgesia had been performed only in 5.5% of the patients with SEA. Skin abscesses and furuncles were the most common source of infection.[1]

The association of SEA and diabetes mellitus may be explained by the reduced immunocompetence of these patients.[2]

In the case we report, the patient was immunocompetent and had no trauma nor surgery history, despite being a non-insulin-dependent diabetic with poor metabolic control. Moreover, the patient had an infected sebaceous cyst that she devalued and only reported at the time of her third ED admission. Therefore, the treatment has been made belatedly, which may have contributed to a disseminated infection that ended with the development of SEA.

According to Hlavin *et al*, the combination of spinal cord compression and vascular damage with resultant hypoxia represents the pathogenic basis of SEA.[1]

The most common location for SEA is the thoracic epidural space,[1,] which is involved in 31%-63% of cases, followed by the lumbar spine 21%-44%, and finally by the cervical spine 14%-26%.[3,4,5-7] The thoracolumbar segment of the vertebral column has a greater extension of the epidural space which may explain a preferential localization of SEA for this region. Another factor that may contribute is the developed extradural venous plexus in that region.[8]

In some cases, no source can be identified but, according to recent reviews, in 71%-78% of cases it is, and the infecting organism can be found in the skin, urinary tract, lower respiratory tract, and site of invasive spinal procedures.[9]

In the case we report, the source was an infected skin abscess which, as mentioned earlier, is the most common source of infection of SEA. The infection occurred both by contiguity (since the patient had abscesses in the cervical region) and by hematogenous route since the agent isolated in pus and blood was *Staphylococcus aureus*, which is also the most commonly isolated agent in SEA.[1] The extension to practically the entire spine as well as bone involvement can be explained by the late diagnosis of this pathology, allowing its evolution.

SEA is a "painful, febrile spinal syndrome" as it was described by Hancock[10], that may occur with radiating root pain followed by limb weakness or with a nonspecific clinic, with fever being absent and with normal hematological indices.[8]

Heusner *et al*[11] published his description of four stages of SEA that continue to be being referred to in recent literature.

He described a first stage in which the patient would feel spinal ache, followed by a root pain (with nuchal rigidity/neck stiffness) that characterizes the second stage. A weakness of voluntary muscles and sphincters and sensory abnormalities only appear in the third stage, which precedes the fourth, where paralysis occurs.[11,12] The clinical use of these stages may allow accurate diagnosis before the appearance of irreversible neurological damage.

According to this classification, we can conclude that our patient was in stage 4 of SEA, reflecting an advanced stage of the disease facilitated, once again, by its late diagnosis.

Thereby, pain is the most frequent symptom and virtually occurs in all patients at some time during their illness.[13] Almost two-thirds of the patients develop a fever.[1]

Initially, our patient had complaints of low back pain of mechanical characteristics and, on the second admission to the ED, she had fever associated with pain but also urinary changes suggestive of infection, with no reference to skin abscesses. This caused the complaints to be interpreted in the context of osteoarticular pathology and urinary tract infection, respectively, which, again, delayed the diagnosis.

When clinical signs and symptoms raise the suspicion of SEA, a whole-spine MRI is mandatory to exclude multisegmental involvement.[12] MRI scan may also show concomitant discitis or osteomyelitis adjacent to the abscess.[8]

The lumbar puncture in our patient was performed to exclude meningitis and/or involvement of the central nervous system (CNS). According to the characteristics of the drained liquid and by the findings in the MRI, that have been made afterward, we believe that the collected material would be of an epidural abscess. Despite this, the procedure occurred without complications and allowed the isolation of *S. aureus*, which was useful *a posteriori*.

Urgent surgical decompression in combination with long-term antibiotic treatment is generally considered the treatment of choice for extensive SEA.

The antibiotic therapy should be started as soon as the diagnosis is made, and should be continued intravenously for up to four weeks and orally for a total of eight weeks, unless osteomyelitis is present and, in that case, many months may be necessary.[8,13]

In an initial phase, empirical antibiotic therapy was administered in meningeal doses, as a CNS infection was a possible diagnosis. At the moment of the isolation of a specific agent, the patient was started on flucloxacillin, which she completed for eight weeks.

The evaluation of the indication for decompressive surgical intervention should always urgently be considered since neurological improvement is unlikely if the duration of paresis exceeds 24-36 hours.[14] The therapeutic method of choice is a laminectomy.[1]

Finally, it is important to remind that about 50% of patients are initially misdiagnosed at the time of presentation,[15] and that was exactly what happened with our patient. This may be explained not only by the low incidence of this entity but also by the non-specific symptoms, which combined can make an early recognition very difficult. ■

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