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Case Report

Spontaneous Spinal Epidural and Subdural Empyema Causing Cauda Equina Syndrome: A Rare Case Report and Review of Literature

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Abstract

Background Context: Paraspinal abscesses can spread across the various tissue planes and cause neural compression. It appears that if left untreated, infections can spread from the peri-spinal space to the epidural and subdural or vice versa with catastrophic neurologic sequalae. Cauda equina compression is one of such outcomes. This syndrome presents as low back pain, motor and sensory deficits in the lower extremities, and sphincter dysfunction. Potential septic meningitis via disseminated CSF spread may also occur.

Purpose: To report a case of extensive spine infection involving the facet joints, surrounding soft tissues, extra-dural space as well as the intra-dural compartment causing cauda equina syndrome and to review the literature of this scenario.

Study Design/Setting: Case report and review of the literature.

Methods: We reviewed the literature regarding spinal extra-dural and intra-dural infections causing cauda equina syndrome, in terms of possible aetiology, patho-mechanisms and management options.

Results: A 58-year-old woman, with a 1-week history of fever, back pain and left lower extremity weakness as well saddle anaesthesia and sphincteric dysfunction. She is diabetic and hypertensive and had had left hip ORIF and dental surgery 2 years prior. She also had a history of MRSA septicaemia in the last 2 years.

Her history and exam findings were consistent with cauda equina syndrome. Her MRI scan showed L4/5 septic arthritis, extensive paraspinal as well as epidural abscess with marked canal compromise extending from L4 to S1. The patient underwent a debridement, L3-S1 laminectomies, durotomy and drainage of the abscess. Intra-operative cultures were positive for MSSA.

Conclusions: Spread of infection from the paraspinal to the extradural and intradural space causing cauda equina syndrome is extremely rare. Prompt diagnosis, surgical intervention drainage and antimicrobial therapy in conjunction with infectious disease experts is required for the best possible outcome.

Keywords: Spinal Subdural Empyema; Cauda Equina Syndrome; Magnetic Resonance Imaging Septicaemia

Abbreviation

CRP: C Reactive Protein; CSF: Cerebrospinal Fluid; SSE: Spontaneous Subdural Empyema; MRC: Medical Research Council; MRI: Magnetic Resonance Imaging; MSSA: Methicillin-Sensitive Staph Aureus; MRSA: Methicillin Resistant Staph Aureus

Introduction

The incidence of spinal epidural abscesses, initially rare, has continued to increase in literature. Spinal epidural abscesses are rare, but intra-dural extension of such abscesses is even rarer [1,2]. The classic triad of fever, back pain and neurologic deficit like cauda equina syndrome can be a pointer to an infective aetiology of the spine, but careful evaluation of the MRI specifically looking for the anatomical extent of the infection is necessary for surgical planning and adequate surgical debridement [3]. In patients with predisposing factors, bacteraemia from skin flora like staph aureus accounts for most infection [4-6].

We present below an interesting and rare case of a patient who had lumbo-sacral para-spinal abscess, right L4/5 septic arthritis, epidural abscess with an intradural extension who was referred with cauda equina syndrome. She underwent an emergency decompression as well as debridement with intravenous antibiotic therapy. She made good neurologic recovery and was discharged from the hospital in a stable condition.

Case History

A 58-year-old female, who was referred to the spine service of our hospital with a 1-month history of back pain and fever, and sphincteric dysfunction of 3 days.

She described the pain as dull and constant and worse on movement. She had no immediate trauma, history suggestive of TB or malignancy. She was a known diabetic and hypertensive who was treated for bacteria septicaemia 2 years prior. She also had left hip surgery and tooth extraction in the last 18 months. At presentation, she had MRC power grade 3 in L3,4,5 and S1 myotomes. Knee jerk was normal, but ankle jerk was reduced. She also had saddle hypoaesthesia as well as decreased rectal tone and reduced voluntary anal contraction. There was local midline spine tenderness at maximum at L5/S1.

She was febrile at presentation with a temperature of 38° C, pulse rate of 100 and saturation of 100% in room air. Initial lab work at the referring hospital showed hyperglycaemia of 14mmol/L, high anion gap, and metabolic acidosis suggestive of DKA. Her white cell count was $23x10^9$/L, CRP >270mg/L. Blood was drawn for cultures. She was commenced on IV antibiotics and IV fluids with potassium chloride as well blood sugar control regimen. Repeat laboratory work done 24 hours later revealed white cell count of $18.0x10^9$/L, platelets of 473, 000, sodium of 127mmol/L, potassium of 2.8mmol/L. chloride of 87mmol/L and creatinine of 28mmol/L. Blood gas showed metabolic alkalosis with lactate of 1.59mmol. Blood culture remained negative. On the ward, she remained febrile, but hemodynamically stable. She continued to have low back pain with point tenderness, L3 to L5 and focal deficit of strength on hip flexion/extension/knee flexion and ankle dorsiflexion worse on the left. A magnetic resonance imaging (MRI) was done which was reported as left septic arthritis at L4/5, extensive epidural abscess and spinal meningitis as well as paraspinal soft tissue abscesses (Figure 1-3).

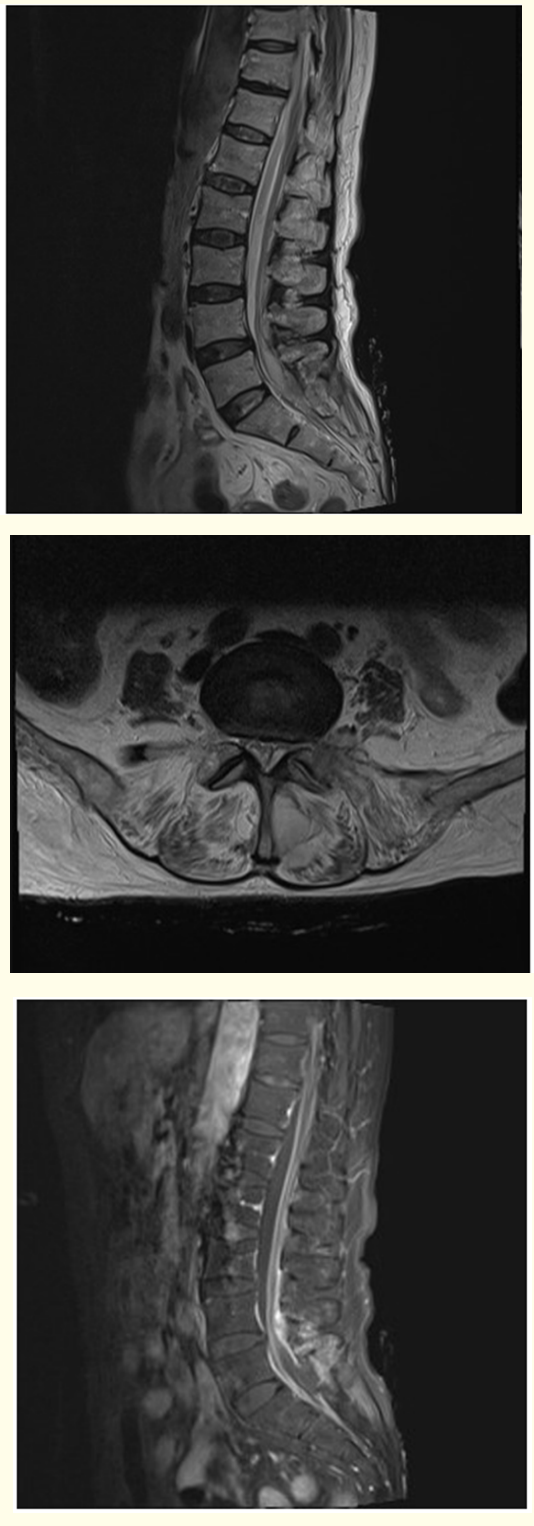

Figure 1-3: Pre op images.

She was booked for emergency surgical debridement and spinal decompression.

In the operating room, she had general anaesthesia and endotracheal intubation. She was positioned prone on an Amsco table, skin was prepped and draped in a standard sterile fashion. A midline incision was made and subperiosteal dissection performed to expose the surgical levels of interest. Pockets of paraspinal abscesses was encountered during the initial dissection. Radical debridement of the involved soft tissues was done. An L4-S1 laminectomy was performed which revealed a collection of epidural abscesses, from L4 to S1 which was drained and washed out using copious warm saline irrigation. However, we noted a continuous egress of purulent material into the surgical field which filled up rapidly when gentle pressure was applied to the thecal sac. A close examination revealed a perforation of about 2mm at the left lateral L5/S1 level. (Figure 4, 5) At this point we extended the laminectomy to L3 and then performed a durotomy cephalad from the perforated point to the middle of L3. Copious amount of purulent material continued to drain. The cauda equina was covered by a thick fibrinous exudate and looked markedly inflamed. (Figure 5) We then threaded a small soft size 6 feeding tube proximal to the top end of the dura incision and irrigated with warm saline until the effluent became clear. A purse string neurilon 4 '0' suture was then placed around the feeding tube and the tube brought out through a separate stab skin incision. The surgical wound was copiously irrigated with warm saline. A separate non- active wound drain was placed and brought out through another stab incision. Particular attention was paid to the fascial closure which was done in a continuous interlocking manner using a prolene '0' suture. A sterile dressing was applied, and she was then turned supine and extubated according to anaesthesia protocol.

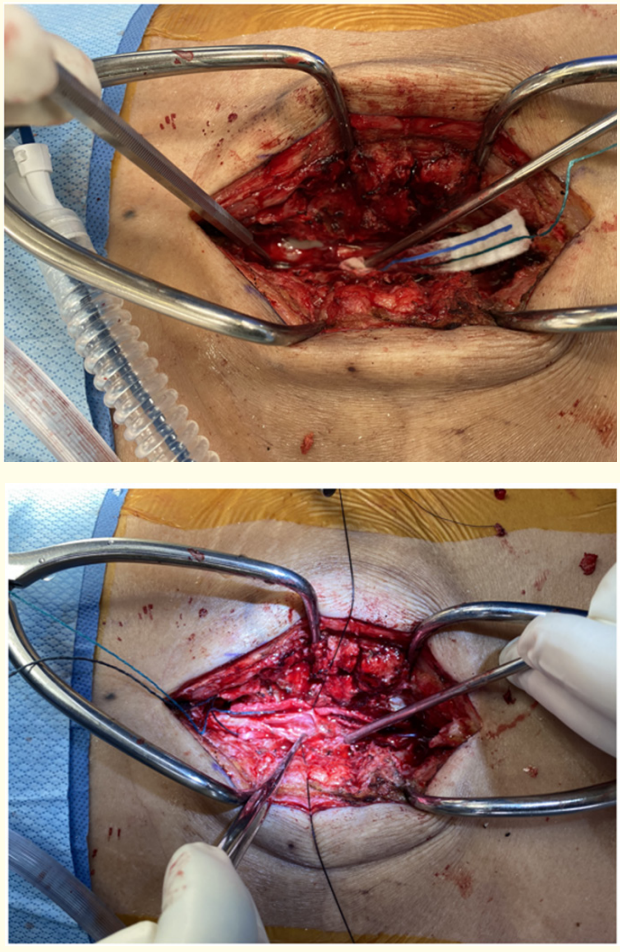

Figure 4 and 5: Intra op Images.

Result

Post operatively, we maintained bedrest with the head of the bed no greater than 10 degrees until her intradural drain was removed. Her intraoperative cultures were positive for MSSA. A surgical infectious disease consultation was obtained, and final recommendations were for her to receive cloxacillin 2 grams IV q.6h. for at least 6 weeks until concurrent radiographic resolution of the infection and normalization of her CRP.

By the10th day post op, she had made significant neurologic recovery and power had improved to 5/5 throughout her L3 to S1 myotomes. The perianal dysesthesia had improved as well. Unfortunately, she was unable to void and lacked sensation of a full bladder. Prior to discharge, she was taught self-urinary catheterization and transferred to spinal cord injury rehabilitation hospital. She was reviewed as an out-patient in 8 weeks with repeat lumbosacral MRI which showed complete resolution of the subdural/epidural abscess figure 4,5.

Discussion

Spontaneous spinal subdural abscess is a rare clinical entity [7] Although previous reports demonstrated obvious aetiology. This index case most likely suggests a haematogenous aetiology. Predisposing factors should be sort in patients suspected of having SSE [2] Our patient had several risk factors- a known diabetic who was treated for bacteria septicaemia 2 years prior, she also had left hip surgery and tooth extraction in the last 18 months [8].

The question however is the pattern of spread of the infection i.e., did the initial intra-dural collection spread to the extradural

space causing septic arthritis and soft tissue abscesses? Or was this an 'anterograde' spread of peri-spinal infection into the epidural and then subdural space? We hypothesize that the later might be the case here for two reasons. Firstly, the extensive peri spinal soft tissue involvement suggests that the infection may have arisen from here. It is most probable that the septic arthritis and pyomyositis preceded the intraspinal infection given the level of soft tissue involvement seen intra-operatively. Also, the acuteness of the neurological symptoms may also have suggested that this coincided with the time of intradural extension.

In their report, Ramos., *et al.* postulated that the septic emboli could be seeded through the epidural and subdural spaces independently because their patient developed SSE without a known existing dural defect. They noted that their patient also developed a psoas abscess and had evidence of septic emboli on brain MRI [9].

While the classic triad of fever, back pain, and symptoms of spinal cord/cauda equina compression, may be pathognomonic according to Frasier., *et al*. the extent of paraspinal involvement and intradural extension requires a very careful study of the imaging and meticulous intra-operative evaluation. In our case, we couldn't pre-operatively ascertain intradural extension until we had drained the paraspinal and extradural abscesses. It was at the point of irrigation of the extradural space that the continuous egress of pus was noticed and found to be emanating from the intra-dural space.

From an imaging standpoint, the imaging study of choice is a contrast-enhanced MRI which shows the spinal cord and extent of compression, bony and ligamentous tissues, and intervertebral discs. Unfortunately, because of paucity of published cases on imaging characteristics of SSEs and lack of robust imaging descriptions, the diagnosis may be difficult to differentiate from epidural infection, as was the situation with our patient.

When combined with neurological features and imaging, haematological indicators are useful for diagnosis and for monitoring response to therapy. Leukocyte count, ESR, and CRP which were markedly elevated in our patient, are useful but usually not sensitive indicators of spine infections [10].

The consensus in the literature suggests that surgical management and antibiotic therapy as guided by culture results are the mainstay of treatment in a patient with neurologic deficits [11] The extent of surgical debridement and the use of intra-dural drains have not been clearly addressed in contemporary spine literature since current treatment regimen is guided by anecdotal evidence. We did a radical soft tissue debridement in this case to reduce the bacterial burden and improve the efficacy of antibiotics. A size 6 tube was threaded into the subdural space and left after thorough irrigation because we weren't certain of the adequacy of the subdural washout. This contrasts with most studies where primary water-tight dural closure is advocated.

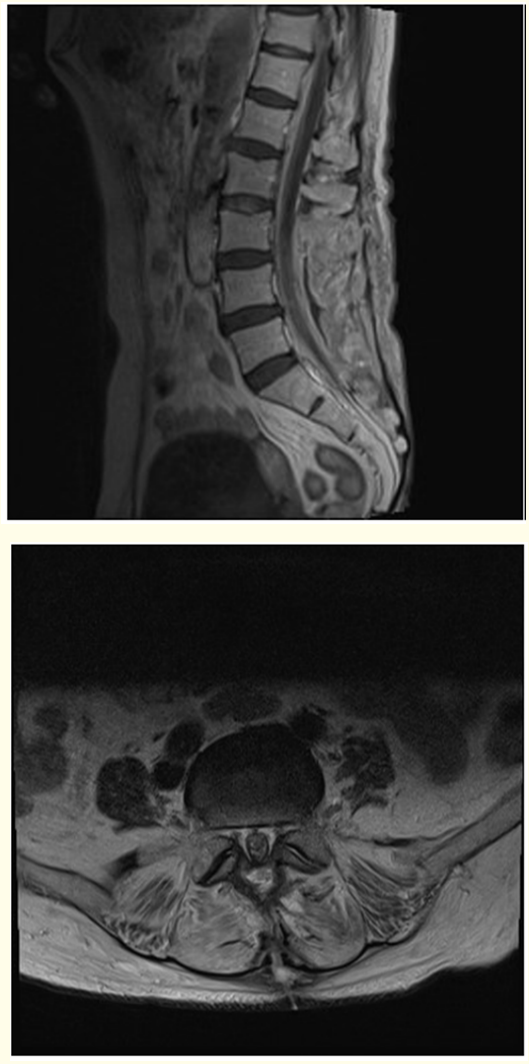

Figure 6 and 7: Post op images.

Conclusion

Spinal epidural and subdural empyema causing cauda equina syndrome is a very rare clinical entity which if not properly treated can be associated with high morbidity and mortality.

Careful, prompt, clinical and radiological evaluation to properly define the extent of the spinal and paraspinal involvement is required for good clinical outcome. Thorough surgical debridement as well as culture-guided antibiotic therapy is mandatory for proper treatment.

Declaration of Interest

The authors have no conflicts of interest to declare

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