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UTI-Associated Lumbar Epidural Abscess with Discitis and Osteomyelitis in a 47-Year-Old Male: Diagnosis and Surgical Intervention

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Abstract

Introduction: Spinal epidural abscess is a serious condition associated with possible catastrophic neurologic sequelae. Acting through a mass effect, an infectious collection threatens spinal cord perfusion by compressing the surrounding vasculature. We discuss the unique presentation of a urinary tract infection-associated lumbar spinal epidural abscess in an otherwise healthy 47-year-old male.

Case Report: A 47-year-old male with no past medical history presented with complaints of worsening low back pain following a trip to India approximately two months prior to his first orthopaedic spine surgery consultation. In the setting of worsening low back pain, failure of trial of nonoperative management, and elevated inflammatory markers, the patient was admitted and found to have urinary tract infection-associated L4-L5 spinal epidural abscess with associated L4-L5 discitis and osteomyelitis. This case report evaluates the clinical history, diagnostic workup, and treatment of urinary tract infection-associated lumbar epidural abscesses.

Conclusion: Epidural abscesses are suppurative collections around the dura with potentially devastating neurologic complications. This report outlines the diagnostic challenges and treatment strategies associated with a urinary tract infection-associated epidural abscess in a middle-aged male with no underlying comorbidities.

Keywords: Spinal epidural abscess; Urinary tract infection; UTI; Lumbar; Spine; Discitis; Osteomyelitis; *E. coli*

Introduction

Spinal epidural abscess (SEA) is a serious condition associated with possible catastrophic neurologic sequelae [1,2]. These infections typically arise in four ways: hematogenous spread (most common), contiguous spread from nearby infection, direct injection, or penetrating trauma. Risk factors include diabetes, history of previous spine surgery, intravenous drug use (IVDU), immunosuppression, chronic lung disease, and renal failure. However, these may be observed in health individuals. The most common pathogen is Staphylococcus aureus, but gram-negative bacteria - *E. coli*, *Pseudomonas aeruginosa*, *Haemophilus influenzae*, and *Klebsiella pneumonia* - have been isolated [3,4].

Case Presentation

A 47-year-old male with no prior medical or surgical history presented to our outpatient orthopaedic spine surgery clinic with one year of insidious lower back pain that had acutely worsened over the previous 5-7 weeks. He travels every other month to India for work. While traveling to India two months prior to presentation, in December, he reported increased back pain and had a magnetic resonance imaging (MRI) lumbar spine completed in India in early January which demonstrated no evidence of infectious collection. He denied any antecedent trauma or penetrating injuries. He did have a severe bout of gastrointestinal (GI) illness with emesis and diarrhea and had to be hospitalized for 24 hours with intravenous (IV) fluid treatment. He also reported contracting a urinary tract infection (UTI) during his stay and was treated with an unspecified oral antibiotic. He recovered from his GI illness and UTI and returned to the United States. He continued to have back pain and presented to our clinic for initial evaluation several weeks after his return. He stated his pain began in the midline lower lumbar spine at the L4-S1 levels without neurologic symptoms in the lower extremities. He had completed one physical therapy (PT) session while in India and had not received any corticosteroid injections. He was taking an unknown muscle relaxer, and Tramadol and Gabapentin were prescribed to him while in India to aid with sleeping. Review of systems was positive for weight loss but otherwise unremarkable. He denied any fevers, night sweats or chills. He denied any bowel or bladder incontinence, urinary tract complaints or diarrhea. MRI films of the lumbar spine from India in January 2020 were reviewed demonstrated mild L4-L5 disc protrusion with bilateral L4-S1 foraminal stenosis.

On physical exam, he was afebrile and hemodynamically stable. His skin was intact with no evidence of ecchymosis or swelling in lower back or bilateral lower extremities. He denied any lumbar tenderness to palpation in the midline or paraspinal muscle region. He had decreased mobility and pain with both flexion (0-45 degrees) and extension (0-10 degrees) and decreased patellar reflexes bilaterally (absent on the right, +1 on the left). He showed bilateral lower extremity 5/5 muscle strength with sensation intact throughout all dermatomes. His feet were warm with +2 dorsalis pedis pulse bilaterally. We recommended PT and possible interventional pain management with repeat MRI in the future if his symptoms did not improve. We gave him a referral for outpatient PT and prescribed anti-inflammatory medications.

The patient returned for follow-up two weeks later because of worsening symptoms despite multiple sessions of PT. He endorsed progressive bilateral lower extremity tightness extending to his thighs and calves, worse on the right and worsening back pain with difficulty sleeping. He denied balance issues but reported weakness in his thighs, worse on the right. He denied any radicular pain, numbness, or tingling in his lower extremities. He denied any fevers, chills, or systemic symptoms. He denied any bowel or bladder incontinence, urinary tract complaints or diarrhea. He now presented with right knee and right groin pain. He was unable to walk more than 100 steps and reported having intermittent low-grade fevers at night. Additionally, the pain pills and muscle relaxers were no longer effective. His physical exam showed bilateral hip pain with flexion and extension of his spine as well as bilateral hip pain with internal and external rotation, worse on the right. As he had not seen a primary care physician regarding his illness, we ordered baseline labs to check for infection or possible malignancy. We also ordered MRI pelvis/bilateral hips to evaluate for possible septic joint or psoas abscesses. We also recommended follow-up with his primary care physician.

The patient presented a week later after completing labs and MRI. He endorsed worsening pain, but his physical exam remained unchanged. His labs showed a normal white blood cell (WBC) 10.6, elevated erythrocyte sedimentation rate (ESR) > 130, and C-reactive protein (CRP) of 6.92. Urinalysis w/ cultures showed 10,000-50,000 colony forming units with gram negative bacilli isolated concerning for *Escherichia coli (E. coli)*. MRI pelvis / bilateral hips showed no fractures, dislocations, osteoarthritis, malignancy, or septic joint. Moderate to severe disc degeneration with L4-L5 vertebral body signal changes were appreciated. On the Coronal STIR sequence, the L4-5 disc had a high signal intensity with increased signal in the surrounding L4 and L5 vertebrae as well, indicating an acute infectious or inflammatory process (Figure 1). We suspected lumbar spine osteomyelitis or discitis and a possible spinal epidural abscess. We recommended the patient be directly admitted to the hospital for further workup and repeat MRI lumbar spine with and without contrast.

Upon hospital admission, he denied any interval changes in his history, and his physical exam was consistent with his most recent clinical visit. The patient was afebrile, and vital signs were stable. Baseline labs showed WBC 8.0 and hemoglobin 10.1. Blood cultures had no growth after 4 weeks. Chest x-ray was unremarkable. Echocardiogram showed normal left ventricular systolic function with an ejection fraction of 55-60% and no evidence of endocarditis. Computerize tomography (CT) abdomen/pelvis demonstrated L4-L5 endplate changes with subchondral lucency (Figure 2). MRI lumbar spine demonstrated an 20mm x 0.9mm epidural collection at L4-L5 concerning for an epidural abscess with associated L4-L5 discitis and vertebral body osteomyelitis (Figures 3 and 4). Orthopaedic spine surgery and infectious disease (ID) were consulted. Due to potential neurologic deterioration in the setting of an epidural abscess, the patient was transferred to the ICU for further monitoring. IV Vancomycin and Ceftriaxone were initiated per ID recommendation.

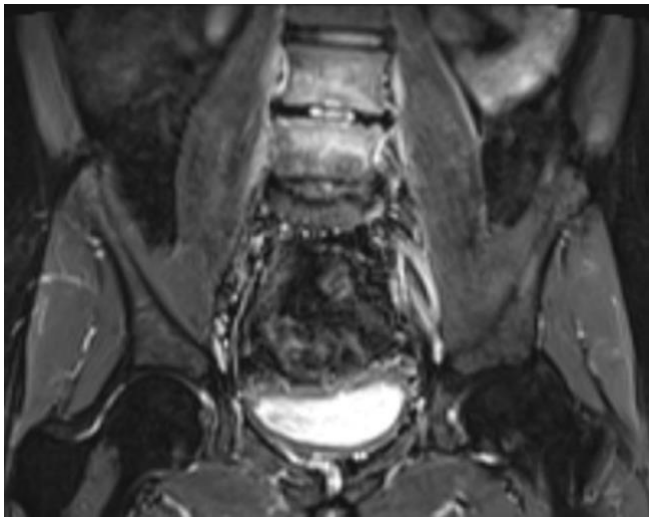

Figure 1: Pre-Operative MRI Pelvis / Bilateral Hips. High intensity signal changes at L4/L5 vertebrae and disc concerning for infectious collection.

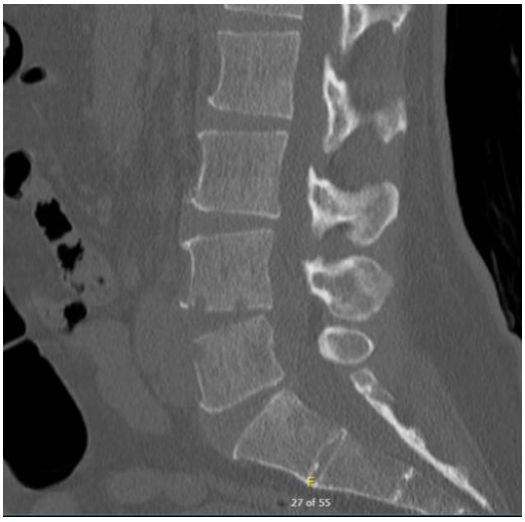

Figure 2: Pre-Operative CT Abdomen/Pelvis Sagittal. L4 inferior endplate and L5 superior endplate changes with subchondral lucency.

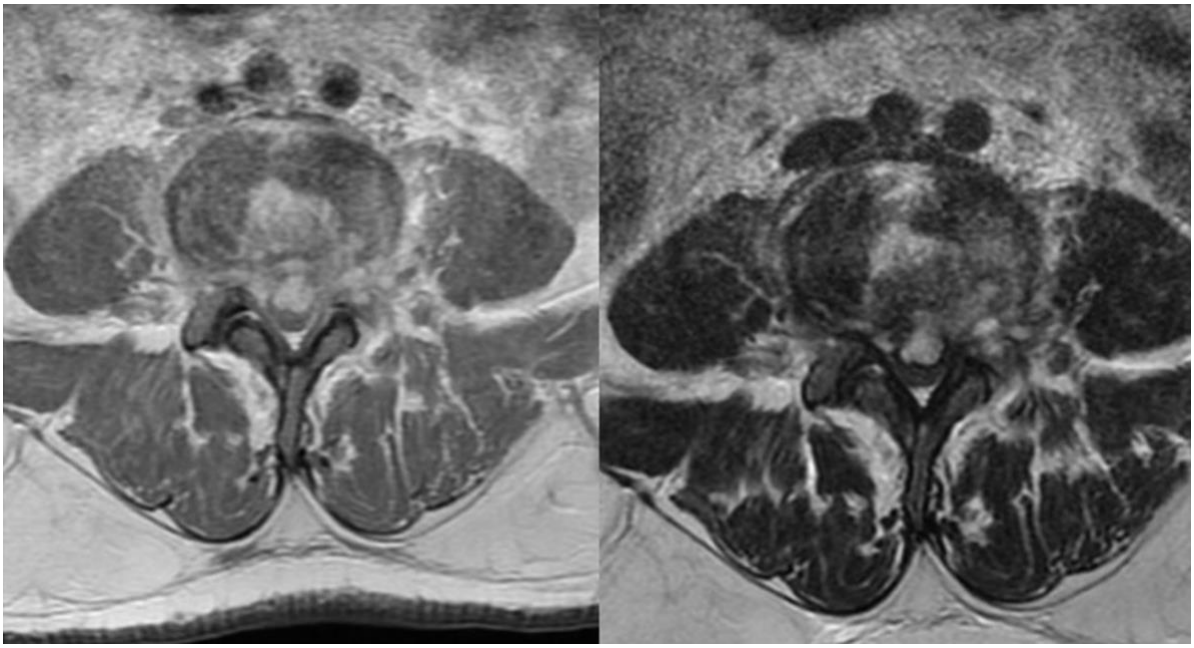

Figure 3: Pre-Operative MRI Lumbar T1 and T2 Axial. L4-L5 epidural abscess with canal stenosis and L4 vertebral body osteomyelitis.

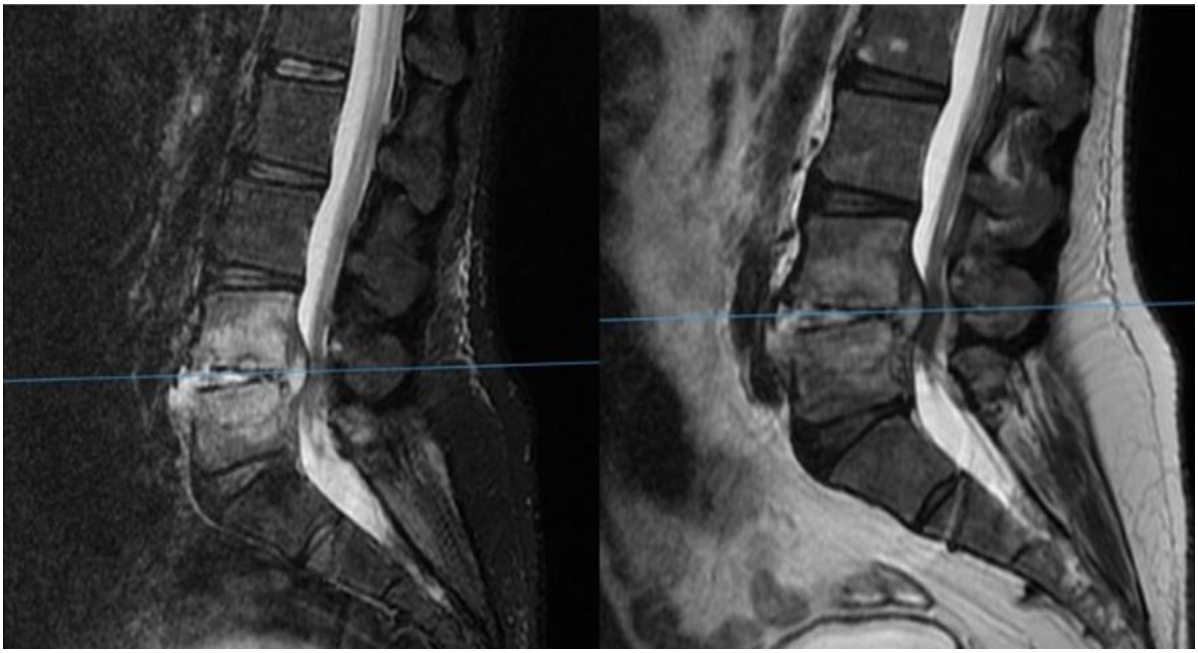

Figure 4: Pre-Operative MRI Lumbar STIR and T2 Sagittal. L4-L5 epidural abscess with canal stenosis and associated vertebral body osteomyelitis and discitis.

In the setting of unremitting lumbar back pain with neurologic symptoms, elevated inflammatory markers, and MRI findings consistent with an L4-L5 epidural abscess with associated discitis and osteomyelitis, the decision was made to perform lumbar spine L4-L5 laminectomies with irrigation and debridement (I&D) of the abscess. In the OR, a 6cm longitudinal incision was made down to L4-L5. No purulent drainage was appreciated with soft tissue dissection, and L4-L5 laminectomy was subsequently performed. There was some edema noted on the outer layer of the dura indicating compressive etiology. We identified the disk space, but there was no purulence appreciated. We used an angled ball tip probe, and we entered a soft, gelatinous, dark brown fluid collection. We sent this in piecemeal fashion to the lab. The wound was copiously irrigated, and a 15-French Blake drain was placed deep to the fascia. The patient tolerated the procedure well with no further complications.

Postoperatively, the patient's hospital course was uncomplicated. He was able to ambulate with less difficulty and minimal pain. He did have mild tingling sensation on the right leg, but this resolved within 2 weeks of surgery. Intraoperative cultures grew *E. coli*. A peripherally inserted central catheter line was placed during the admission, and the patient was discharged with an eight-week course of culture-directed IV ceftriaxone. At 2-week follow-up, he reported decreased lumbar and lower extremity pain, improved walking distance, and minimal dependence on his walker. On exam, his incision was clean, dry, and intact. He was neurovascularly intact with 5/5 motor strength and sensation throughout all dermatomes in the bilateral lower extremities. At six-week follow-up, he endorsed minimal pain medication requirement, no pain with ambulation, and no evidence of recurrent infection. Tuberculosis and Blastomycosis labs were also negative, which was resulted about one month after surgery. His neurological exam was unchanged from his first post-operative visit. At six months post-surgery, he had returned to full activities including hiking without restrictions and minimal discomfort. MRI lumbar spine performed at that time showed normal post-operative changes without evidence of infection.

He had consulted with urology upon discharge and was found to have another recurrent *E. coli* UTI and was placed on oral antibiotics again for 14 days. An outpatient CT abdomen/pelvis with IV contrast showed increased inflammation in the bladder wall without any fistulas or abscess. Four months post-operatively, he underwent an optical urethrotomy due to a urethral stricture found on a previous cystoscopy. Upon last communication, patient is doing well without any voiding complaints.

Discussion

SEA remains a diagnostic challenge with potential for catastrophic, irreversible neurologic damage if not promptly diagnosed. Fever, back pain, and neurologic changes are the "classic triad" but rarely present concurrently (13%) [5]. Therefore, appropriate clinical evaluation, laboratory tests, and imaging are imperative. This workup typically includes a thorough history, neurologic-focused physical exam, baseline labs (including ESR and CRP), urinalysis with culture, blood cultures, X-ray, and MRI with contrast. However, even with targeted workup, studies show delayed diagnosis as often as 75% of the time [6]. For this reason, clinicians must maintain a high index of suspicion even when findings are equivocal.

From our understanding, this is the first reported UTI-associated SEA of the lumbar spine in a middle-aged individual with no known comorbidities. In the setting of dysuria, urine culture-confirmed *E. coli*, and no other suspected infectious source, this patient's SEA is most likely secondary to his UTI. Hematogenous bacterial spread is potentiated by Batson's plexus, a valve-less venous network that extends throughout the epidural space of the vertebral column. These venous channels drain from the bladder and allow bacteria from the urinary tract to disseminate into the lumbar spine [6]. Despite previous oral antibiotic treatment, epidural spread can be attributed to improper antibiotic selection, poor patient compliance, and/or delayed treatment. UTI-associated SEA is atypical in young, immunocompetent patients [7]. While *E. coli*-associated SEA cases have been documented in the literature, these infections are uncommon (2-4% of SEAs) and arise typically in immunocompromised patients with multiple comorbidities – such as diabetes, rheumatoid arthritis, obesity, and Crohn's disease [8-10]. There are reported cases of SEAs in patients with no existing medical conditions, but these abscesses were either localized to the cervical spine or in an elderly patient [8,11]. Despite their rare presentation, UTI-associated SEAs should be considered in middle-aged, healthy patients with lower back pain concerning for infection.

If a patient is not septic or otherwise unstable, IV antibiotic administration should be deferred until culture collection by interventional radiology or in the operating room. Nonsurgical management may be indicated for individuals who are poor surgical candidates or with small abscesses without neurologic symptoms [12]. This patient did not meet either of these criteria. Surgical evacuation and IV antibiotic therapy remain the gold standard for SEA treatment. Timing of surgery - emergent versus urgent I&D - is predicated upon whether evolving neurologic symptoms are present [13]. Epidural evacuation is accomplished through laminectomy to allow the surgeon to gain access to the epidural space. Chaker et al. investigated complications associated with decompressive laminectomy alone versus decompressive laminectomy with posterior instrumented fusion in the treatment of an epidural abscess. Laminectomy with instrumented fusion showed increased rates of reoperation (23.8% vs 12.2%, $p < 0.005$) and post-operative blood transfusions (31.5% vs 14.5%, $p < 0.001$) compared to laminectomy alone [14]. Placing instrumentation in the face of an acute infection can lead to infected hardware as well.

While our patient's postoperative course was uncomplicated, recurrent epidural abscesses is a known sequela of surgery. Risk factors for recurrence include IVDU, surgical site wound infection, and fecal incontinence or retention on admission. IVDU - particularly with contaminated needles - increases the risk for bacteremia and potentiates hematogenous bacterial introduction into the spine. Surgical site wound infections permit direct contiguous spread of bacteria to the underlying epidural space. Fecal incontinence or retention are indicative of advanced spinal cord disease and are theorized to portend a worse prognosis [15]. Recognizing these risk factors and ensuring close follow-up can minimize the risk of reinfection and future operations.

Conclusion

UTI-associated SEAs are suppurative collections around the dura with potentially devastating neurologic complications. This report outlines the diagnostic challenges and treatment strategies associated with a UTI-associated SEA in a middle-aged male with no underlying comorbidities.

Clinical Message

UTI-associated SEAs are atypical infections with potentially devastating outcomes if diagnosis is delayed or missed. Maintaining a high index of suspicion may expedite SEA diagnosis and treatment.

Abbreviations: CRP: C-reactive Protein; CT: Computerized Tomography; ESR: Erythrocyte Sedimentation Rate; GI: Gastrointestinal; I&D: Irrigation and Debridement; ID: Infectious Disease; IV: Intravenous; IVDU: Intravenous Drug Use; MRI: Magnetic Resonance Imaging; SEA: Spinal Epidural Abscess; UTI: Urinary Tract Infection; WBC: White Blood Cell

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