To present a unique case of a spinal epidural abscess, which was likely secondary to hematogenous spread.

**INTRODUCTION**

Spinal epidural abscesses (SEA) are rare infections that can occur through direct inoculation, extension from nearby tissue, or hematogenous spread. With progression of the abscess along the spinal canal, complications occur through disruption of blood supply, release of inflammatory cytokines, and compression of the spinal cord. Like any infection, septic shock and death can occur without proper treatment.

The classic triad of presenting symptoms is fever, spinal pain, and neurologic deficits, although symptoms may be nonspecific early in the disease process (1, 2). One retrospective study suggests that the incidence of SEA is 5.1 cases per 10,000 hospital admissions. The most common underlying risk factors identified are diabetes and IV drug use. Staphylococcus aureus was found to be the most common causative organism (84%), with 25% of those cases being methicillin-resistant (3).

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**History of Present Illness:**

A previously healthy 14-year-old female presented to the Phoenix Children’s Hospital (PCH) Emergency Department (ED) with complaints of gradually worsening left-sided low back and flank pain, which began 3 days prior to presentation. The pain was 10/10 in severity, radiated to the right flank, and was exacerbated by movement, and improved by nothing. She reported that she had initially visited an outside Emergency Department for her back pain 2 days prior, where she was diagnosed with a urinary tract infection and was prescribed Keflex 500 mg TID. She then returned to the outside hospital 1 day later with worsening pain, now radiating to the left leg. An abdominal CT at that time was performed and found to be unremarkable, and the patient was discharged with a prescription for Flexeril. On the day of presentation to PCH, the patient first went to the Tempe St. Luke’s Hospital ED with worsening pain. A bladder scan was done and showed 2L of urine in the bladder, and she was straight catheterized. Upon discharge from the St. Luke’s ED, the patient collapsed due to lower extremity weakness and was then transferred by EMS to the Phoenix Children’s Hospital ED.

Additional symptoms at the time of presentation to PCH included chills, diaphoresis, abdominal pain, nausea, headache, arthralgias, and myalgias.

Her medical history was negative except for a recent Staphylococcus aureus soft tissue infection in her axilla one month prior, which she reported had been treated with antibiotics which she could not recall. She was unsure if the infection had been Methicillin-resistant. The patient’s social history was pertinent for homelessness with her mother. She denied intravenous drug use, immunocompromised state, or recent travel. Vaccinations were reportedly up to date.

**Physical Exam:**

Vitals: Temperature 40.1ºC (104.2ºF), Heart rate 106 beats/minute, Respiratory rate 20 breaths/minute, Blood pressure 118/73 mmHg, Oxygen saturation 99% on room air

General: Appears uncomfortable and in moderate distress, crying on exam. Glasgow coma scale 15

Cardiovascular: Unremarkable

Pulmonary: Unremarkable

Abdomen: Diffuse abdominal tenderness to light palpation. No guarding or rigidity. No masses felt

Back: Right flank tenderness. Unable to assess range of motion (ROM) or strength due to pain and patient unwilling to move

Extremities: Bilateral (B/L) lower extremity diffuse tenderness to palpation. B/L upper extremities nontender and with full ROM

Neurologic: B/L lower extremity strength 0/5. B/L lower extremity reflexes 0/4 and with decreased tone. B/L upper extremity reflexes 2/4, strength 5/5. Cranial nerves II-XII grossly intact. Sensation intact throughout

**Periment Dignitics:**

CBC (Figure 3) demonstrated leukocytosis with a neutrophilic predominance, and normocytic anemia

CMP, ABR, Lactic acid, Magnesium, Phosphorous, and Creatine kinase were all within normal limits

MRI of the brain and cervical/thoracic/lumbar spine (Figures 1 and 2) showed dorsal loculated spinal epidural collections extending from T3 to T12, with displacement of the spinal cord, and flattening of the cord at T10 and T11. Inflammation of the surrounding ligaments and soft tissues

**Assessment:** Spinal epidural abscess with cord compression and atheria

Hospital course:

She underwent a multilevel thoracic laminoplasty from T4-T12 with epidural evacuation and spinal cord decompression. Abscess cultures were positive for Methicillin-resistant Staphylococcus aureus, and the patient was treated with IV Vancomycin 1250mg every 8 hours and Cefuroxyn 600mg every 6 hours. Blood cultures were consistently negative. The patient had persistent neurogenic bowel and bladder, with bilateral lower extremity paralysis. With rehabilitation, she ultimately regained the ability to use a walker for ambulation.

**DISCUSSION**

A high index of suspicion for spinal epidural abscess (SEA) is necessary for patients presenting with back pain and neurologic deficits. MRI is the best test for early detection. Findings on MRI with high sensitivity for screening for an SEA include paraspinal edema (97% sensitive), and bone marrow or disc edema (65% sensitive). Posot edema is the most specific finding suggestive of an SEA, at 96% specificity (4). Recent studies suggest that ESIR is the best lab test for diagnosing an SEA, with a sensitivity of 100% and a specificity of 67% (5). WBC count may be elevated as well. Immediate surgical management is critical to recovery and improved neurologic outcomes. Failure rates of pure medical management range from 41-65%, leading to delay in surgical treatment (6, 7). Predictors of failure of medical management include diabetes, CRP >115, WBC >12.5, and the presence of bacteremia (6). Differential diagnoses for patients presenting with fever and back pain with or without neurologic deficits include degenerative disc disease, metastatic tumors, osteomyelitis, and meningitis.

Given that inflammation of paraspinal and posot muscles are commonly associated with SEA, look for restriction in range of motion of the back and hips. Tissue texture changes near the level of the abscess may be felt in the paraspinal muscles as bogginess and increased warmth. Indirect techniques such as myofascial release and strain-counterstrain to these areas could help to alleviate discomfort.

**CONCLUSIONS**

- Epidural abscesses require prompt diagnosis and treatment to prevent complications. Because of the lack of specific presenting symptoms, physicians must have a high index of suspicion for patients presenting with fever and neurologic deficits, or with risk factors such as IV drug use or a previous infection.
- Elevated ESIR and an MRI demonstrating paraspinal and posot edema are the best indicators of an underlying spinal epidural abscess that is not otherwise visible.
- Immediate surgical drainage in combination with IV antibiotics with coverage for MRSA is the best way to optimize neurologic recovery.

**REFERENCES**