

# Extensive Spinal Epidural Abscess: Cord Compression with Permanent Neurological Defects

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## Abstract

Spinal epidural abscesses (SEA) are considerably rare and tend to present over two to five vertebral segments. Occasionally, there will be two or more noncontiguous areas of pyogenic collections [1]. Minimal cases have been reported to span the entire vertebral column; a meta-analysis estimates that 1% of all SEA are holospinal [2]. The triad of presenting symptoms includes fever, back pain (often midline), and neurologic defects [1] [2] [3]. Early detection is identified as a critical aspect of improved outcomes. Cases that do not present in this manner or with other masking symptoms can lead to delayed diagnosis, thus delaying treatment. In the event of cord compression, the occurrence of neurologic defects increases. Time from the onset of clinical manifestations to the operating room is crucial in reversing symptoms [2]. This article seeks to review a case of a 65-year-old male that presented to the emergency department (ED) due to a falling second to weakness and thigh pain. On presentation, he was also noted to have rhabdomyolysis causing acute kidney injury (AKI) with tubular necrosis. The patient was admitted to the hospital with a complex history of progressive leg weakness, pain in the lower back, incontinence, and elevated white blood cell count. Days into the admission, a magnetic resonance imaging (MRI) study was performed, which revealed a continuous posterior SEA from C4 to S2 with anterior mass effect causing spinal cord compression. Emergency neurosurgery was scheduled for laminectomies in the cervical, thoracic and lumbar spine to drain the abscess. Evaluation of this complex medical course, surgical approach to drainage of an incessant spinal column abscess, and sustained neurologic defects will be discussed.

## Keywords

Spinal Epidural Abscess, Holospinal Abscess, Cauda Equina, Spinal Compression

## 1. Introduction

Nonspecific clinical features leave room for misdiagnosis upon the first examination. Neurologic deficits are considered the last presenting factor of a typical spinal epidural abscess (SEA) triad: The first being back pain and the second fever, which are thought to present more rapidly [1]. Any development of neurologic changes with the first two symptoms should significantly narrow the differential [4].

Only 0.8% of patients do not have SEA when all three criteria are met, allowing for high clinical diagnostic specificity using the SEA classic triad [5]. Upon suspicion, the provider should obtain an MRI with contrast to rule out a potentially detrimental diagnosis. Despite the clinically relevant symptoms, it is estimated that up to 75% of cases are not diagnosed promptly [5] [6].

Once correctly identified, surgical incision and drainage (I&D) is a mainstay of treatment; however, A neurosurgeon may opt for non-operative management with antibiotics if the disease process is caught early enough. Delayed diagnosis can lead to worsening abscesses, permanent neurologic damage, and can be fatal. In severe cases, such as SEA, that span over the average 3 - 5 vertebral segments [2] [3], I&D is likely a better option than conservative medical management in order to rapidly remove the infection and any spinal compression caused by mass effect. Time is a crucial indicator for determining the treatment course concerning the reversal and preservation of neurologic function.

## 2. Case Presentation

A 65-year-old male presented to the ED after a fall, second to weakness and thigh pain. The patient has a medical history of coronary artery disease (CAD), hyperlipidemia, hypertension (HTN), neuroendocrine tumor with metastasis to the liver, heart failure (ejection fraction of 35%), myocardial infarction (MI), chronic liver mural thrombosis, benign prostate hyperplasia, and morbid obesity.

The history of presenting illness elicited that the patient remained on the floor for approximately five hours before his wife found him. The patient denied loss of consciousness; however, a computer tomography (CT) scan of the head was obtained without contrast. The findings were unremarkable. Lab results demonstrated rhabdomyolysis with a creatine phosphokinase (CPK) of 3041 U/L (reference range 39 - 308 U/L). The patient also had an elevated brain natriuretic peptide (BNP) of 5789 pg/mL (reference range 5 - 125 pg/mL), troponin I of 2868 ng/L (reference range 3 - 78 ng/L), and creatine kinase-MB (CK-MB) of 7.9 mg/mL (reference range 1 - 3.6 ng/mL). The leukocyte count was 18.99 K/uL, the documented temperature on admission was 98F (36.7°C).

The patient was admitted to a medical/surgical (med/surg) unit for rhabdomyolysis. At that time of admission, the musculoskeletal portion of the physical examination only noted no deformities and a full range of motion (ROM). The neurologic examination concluded that the patient was alert and oriented with

no focal deficits, and cranial nerves II-XII were grossly intact. The admitting physician also identified the following problem list; fall secondary to weakness, elevated troponin level with consultation to cardiology, acute on chronic renal failure, chronic CHF, and CAD.

Further assessment on the med/surg floor revealed urinary retention that caused pyelonephritis with Methicillin-resistant *Staphylococcus aureus* (MRSA), encephalopathy developed due to sepsis, and acute kidney injury (AKI) with tubular necrosis secondary to rhabdomyolysis. The patient had a condom catheter placed due to overflow incontinence related to urinary retention and known history of BPH.

Nursing documentation acknowledged that the patient had complained of generalized weakness and had tried to remove the intravenous line and condom catheter due to confusion. The patient was noted to wax and wane cognitively from hospital day two onward. On hospital day four, the nursing documentation stated that stool stains were observed during the nursing assessment. A neurology consult was completed on day five, which focused on encephalopathy.

After six days of admission, neurology suspected SEA due to worsening leg weakness, cognition, and fecal incontinence and thus ordered a stat MRI. The MRI report confirmed a continuous posterior SEA from C4 to S2 with anterior mass effect causing spinal cord compression. Neurosurgery was consulted immediately. Emergent surgery was performed within hours of the MRI.

The patient was not hemodynamically stable prior to the surgery. Using the American Society of Anesthesiologists classification system, he was deemed ASA 5. Once stable enough to begin the procedure, the neurosurgeon decided to take an approach that required three separate laminectomies, one in each of the cervical, thoracic and lumbar regions, to drain the abscess. Laminectomy provides a dual purpose: first, it decompresses the spinal cord, and second, it creates an entry point to the abscess for debridement.

All three sites produced significant purulent discharge as soon as the lamina overlying the dura had been removed. A culture sample was obtained at that time. Suction with continuous irrigation occurred at each region separately. A red rubber Robinson catheter was threaded through the canal's opening to flush material from cervical to thoracic and thoracic to lumbar, as well as reverse order. The closure occurred after fluid passing through became transparent, and the surgical sites were adequately clean from irrigation.

As a result of being hemodynamically unstable during induction of anesthesia, he remained intubated after surgery with transfer to the intensive care unit (ICU). Abscess cultures came back positive for MRSA. An antibiotic regimen was initiated with intravenous (IV) gentamicin, vancomycin, and zosyn, chosen by infectious disease. Despite the abscess drainage and pharmacological intervention, the patient maintained a systemic MRSA infection for 16 days.

Postoperative day nine, the patient was extubated. Initial exams demonstrated upper and lower extremity weakness; he could not lift his lower extremities off

the bed. A combination of drainage, antibiotic regimen, physical therapy and time aided in a gradual improvement in neurologic function.

At that time, neurosurgery progress notes stated continence is not expected to return; however, some muscular function and sensory sensation may improve with time. At the time of discharge, the patient was noted to have sensation intact with improved movement of upper and lower extremities but more movement in upper extremities. Continence has not returned. Ultimately the patient was transferred to a Long-Term Acute Care (LTAC) hospital facility for rehabilitation.

The MRI report confirmed a continuous posterior SEA from C4 to S2 with anterior mass effect causing spinal cord compression. (**Figure 1**)

#### **Cervical Spine Findings on MRI**

- Upper extremity displayed less weakness and reduced motor function upon physical exam the day of the MRI despite involvement of the brachial plexus nerve roots C5-T1.

#### **Thoracic Spine Findings on MRI**

- Upon first glance in **Figure 2** & **Figure 3**, the epidural abscess appears as the spinal cord in the vertebral column due to the unusual nature of a continuous abscess spanning the entire thoracic spine and beyond.

#### **Lumbar Spine Findings on MRI**

##### **The radiologist also documented:**

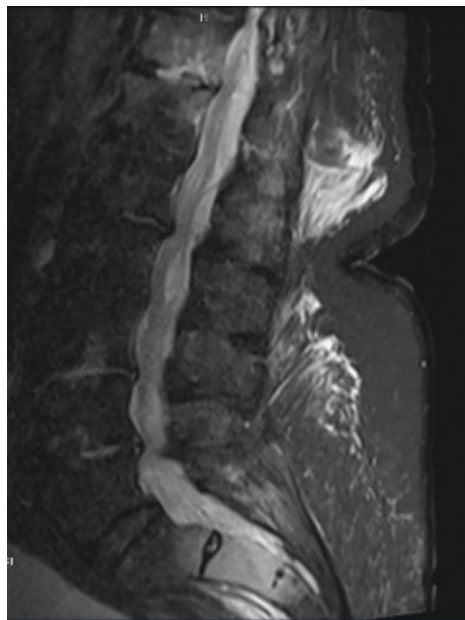
- Rim enhancing 1.4 cm lesion on the inferior aspect of L1 for suspected metastasis
- Severe spinal stenosis of the lumbar region likely worsening mass effect
- Severe osteomyelitis involving the majority of the visualized sacrum



**Figure 1.** MRI cervical spine.



**Figure 2.** MRI thoracic spine.



**Figure 3.** MRI lumbar spine.

### 3. Discussion

Clinically the instance of spinal epidural abscesses has been increasing, which may be correlated to better diagnostic equipment, increased access to the epidural space, and the fact that common risk factors are more prevalent like intravenous drug use [1]. Diabetes, for example, is closely associated with SEA. In 2015, 23.4 million people had been diagnosed with diabetes, compared to only 1.6 million in 1958 [7].

Iatrogenic causes of SEA include all types of invasive procedures, such as surgery, lumbar puncture, epidural anesthesia, epidural analgesia, and nerve blocks, and are estimated to be responsible for 15% of cases. [5] Bacteremia was identified as the inciting source about 26% of the time in a recent case series [8]. Patients with indwelling catheters like central venous catheters or urinary catheters are at an increased risk of SEA as they are the most common sources of bacteremia [9].

According to a meta-analysis of international literature that included 915 cases from 1954-1997, the incidence of SEA was 2 cases per 10,000 admissions [6]. However, according to a more recent case series looking at 101 cases from 2004 to 2014 at a large academic hospital, the incidence was 5.1 per 10,000 admissions [8].

Regarding delayed diagnosis, clinicians need to have SEA high on the differential when the triad appears, regardless of what other symptoms may present, and act as a mask. Timely diagnosis may create an opportunity for medical management avoiding surgery, and prevent permanent neurologic defects [3] [10]. However, most cases will require surgical intervention [1].

The presenting patient had all the classic signs, which was clearer when reviewing this case retrospectively. The patient had called his primary care physician, who utilized the same electronic medical record (EMR) two consecutive days prior to presenting to the emergency room with complaints of back pain and lower extremity weakness. However, at the time of the presentation, distracting diagnoses and symptoms were leading to a significant delay in diagnosis. It is reasonable to extrapolate that the patient may not have adequately communicated the weakness in his lower extremities.

Once the MRI was complete, confirmation of an abscess that extended the entire length of the vertebral column required immediate care. Surgery was critical for this patient's survival, as evidenced by worsening white blood count, mentation, muscle weakness, and bladder and bowel incontinence.

The techniques described in the case presentation follow surgical standards, including decompression, debridement, and biopsy [1]. The surgical approach minimized exposed spinal segments for an abscess spanning most of the vertebral column. Three separate incisions allowed the lamina of one to two vertebrae of each spine region. The surgeon used a Robinson catheter to irrigate the remaining unexposed segments. This technique reduced the tissue trauma of surgery, which was a concern due to the patient not being hemodynamically stable.

Despite abscess drainage and combination antibiotic therapy with intravenous (IV) gentamicin, vancomycin, and zosyn, the patient continued to have systemic sepsis. The average time to clear systemic sepsis caused by MRSA is 5 - 14 days [11]. Postoperative day 16, infectious disease documented that the infection had been cleared. The patient was subsequently discharged to a long-term acute care hospital for rehabilitation needs. Before discharge, the physical exam documented improved strength and sensation in both the upper and lower extremi-

ties. Upon discharge, the patient continued to be incontinent of bowel and bladder, as expected by neurosurgery.

#### 4. Conclusions

Sensitivity screening for ruling SEA in regarding differential, in the presence of the classic triad of midline back pain, fever, and neurological defects is as high as 99.2% [5]. However, all three symptoms may not be identifiable upon initial presentation. The classic triad may not be evident in some patients due to other presenting factors. Providers must consider SEA as a part of the differential, and have a low threshold for neuroimaging in those patients with red flag symptoms or risk factors for developing SEA when they have any suspicious presentations.

BPH appeared to be causing urine retention in this particular patient, resulting in overflow incontinence. Cognitive changes were attributed to encephalopathy from sepsis. Despite a leukocyte count of 18.99 K/uL, the patient was afebrile. It is noted that SEA patients do not always present with all three symptoms of the classic triad [1] [3] [6]. This particular patient presented with midline back pain and neurologic defects. Lower extremity weakness and pain were present on hospital day one. Although it is more common for a patient to present with midline back pain and fever, SEA should have been considered sooner. The median diagnostic delay of SEA is 12 days, with an estimated 60% of the diagnostic errors having the potential for severe harm or death [12].

Surgical intervention appears to have been integral to this patient's survival, considering the rapid deterioration of cognitive function, muscle strength, sensation, and continence, as well as how hemodynamically unstable the induction of anesthesia. The posterior column had significant purulent materials creating an anterior mass effect causing cauda equina symptoms qualifying for emergent surgery. Decompression and debridement provide immediate relief versus attempting medical management alone [6].

Neurological recovery is likely to be achieved if the surgery occurs within 24 - 48 hours after the neurologic deficit onset. Limited data is available in the literature about neurological recovery after the surgery [6]. Irreversible neurologic defects are commonly expected [13]. Multiple factors, including age, health status, comorbidities, and time to diagnosis, play a role in recovery. [6] Thus, timely treatment initiation is crucial in any patient experiencing SEA.

#### Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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