Case Report

Pan Spinal Epidural Abscess in a Young Healthy Male: A Case Report

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Pan spinal epidural abscess is rare in a young healthy adult and may lead to permanent neurological deficits and meningitis if not treated promptly. A case is described of a pan spinal epidural abscess, from C1 to S1, in a young, healthy adult without risk factors that presented with ascending back pain and no fever. The abscess was diagnosed with MRI and treated with hemilaminotomies, surgical drainage, and antibiotics with a satisfactory outcome. The literature is reviewed herein.

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1. Introduction

Spinal epidural abscess (SEA) is a rare condition with potentially devastating consequences. Many SEA patients have a direct source of infection such as epidural injections or spinal injury [1–7]. Conditions that are usually associated with SEA include diabetes mellitus, cancer, AIDS, IV drug abuse, alcoholism, steroid treatment, chronic renal failure, herpes zoster, and reflex sympathetic dystrophy [1–3, 8, 9]. SEA occurs commonly in short segments of the spinal cord. Although multiple or solitary abscesses with cervicolumbar extension have been described previously [10, 11], Pan spinal epidural abscess (C1-S1) in a young healthy adult with no predisposing factors has never been described in the literature with Medline and Pubmed.

2. Case Report

A 23-year-old male manual laborer presented with sudden onset low back pain. He was diagnosed with lumbar muscle spasm and treated with an NSAID by his family doctor. The pain progressively worsened and ascended. Two weeks later, he developed severe worsening of his low back pain with radiating left anterior thigh pain. He visited the emergency room twice for severe pain and was diagnosed with an L3-L4 disc herniation and given Naproxen and Oxycodone/acetaminophen for analgesia. On the day of his admission, the patient was awoken by excruciating back and neck pain refractory to analgesics. He presented to the emergency room where a lumbar CT scan was performed and revealed questionable epidural and paraspinal abscesses. The patient was then transferred to our hospital.

The patient denied fever, headache, sore throat, cough, dysuria, illicit drug use, or any past medical history. He recalled a tick bite to his left anterior thigh one month earlier, with no apparent skin reaction.

On physical exam, he was a 5’11”, 95 kg, well-educated male in mild distress. Temperature was 36.5°C, with a blood pressure 140/77 mmHg, heart rate 110/min, and a respiratory rate of 24/min. There was no palpable superficial adenopathy. He had slight neck stiffness and moderate cervical tenderness. There was diffuse tenderness along the thoracic and lumbar spine and L3 paraspinal muscle tenderness. Neurologically, the patient had a normal level of consciousness and mental status with a mild decrease of left hip flexor muscle strength and no sensory deficit. There was 1.0 × 1.5 cm pigmented area on the left anterior thigh that was nontender and without other signs of inflammation. WBC was 37,900 with 35 bands and both ESR and CRP were elevated. An MRI of the lumbar spine demonstrated an epidural abscess extending from T1-S1 (Figure 1) with a collection in the paraspinal muscles to the left of L3. The patient refused additional imaging.
The patient was taken directly to the operating room from the emergency department, where he underwent a T12-L1 partial hemilaminotomy on the right side. A large amount of liquid pus drained from the epidural space which was gently irrigated followed by placement of a Jackson-Pratt drain. Simultaneously, the L3 paraspinal abscess was debrided and irrigated. There was no connection between the paraspinal abscess and the epidural abscess that could be identified at the time of surgery. Subsequently, the patient was admitted to the surgical ICU, with antibiotic and steroid therapy. By post-op day 1, the patient’s lower back pain significantly decreased and his left hip flexor muscle strength returned to normal. However, he still complained of neck pain and mild stiffness. An MRI of the cervical and thoracic spine was performed which showed a C1-T12 epidural abscess (Figures 2 and 3). A C6-C7 right hemilaminotomy was performed and the abscess drained. The patient’s neck and low back pain subsided after surgery. Each JP drain (T12-L1 and C6-7) was removed on post-op day 2. The cultures from the cervical, lumbar, and L3 paraspinal specimens all grew β-hemolytic group A Streptococci. The AFB and fungal cultures were negative. Multiple blood and urine cultures were negative. An MRI of the head was normal. Chest, abdominal, and pelvic CT scans did not show any other collections. An HIV test was negative. Vancomycin had been given initially. Then, the antibiotic therapy was tailored based on culture and sensitivity. On hospital day 4, the patient was discharged home with an additional 6 weeks of antibiotic therapy. A CBC, ESR, and MRI (Figures 4 and 5) were normal on follow-up at day 9. The patient returned to normal life with complete recovery and no neurological deficits.

3. Discussion

The etiology of this patient’s SEA is unknown. An epidural hematoma caused by epidural analgesic injections or trauma (injury or surgery) may later become the focus of bacterial colonization by hematogenous dissemination from a cutaneous or mucosal source, or infection can occur from direct seeding from the invasive procedure itself [5–7]. Epidural abscess may also result from the direct spread of infection into the epidural space from a source adjacent to the spine such as spondylodiscitis, paraspinal abscess, or vertebral osteomyelitis [7, 12]. Systemic illness, such as diabetes mellitus, immune deficiency states, such as AIDS, or
or cancer, renal failure, IV drug abuse, and steroid therapy contribute to this process [1–8]. SEA in young healthy patients without any risk factors is extremely rare. Pan spinal epidural abscess (C3–S1) was reported previously in an infant with a congenital spinal malformation [12]. In our patient, there was no significant focal infection. The only potential infectious source we could identify was the left anterior thigh tick bite without any reported signs of inflammation which occurred one month earlier.

The valveless, low pressure vertebral venous system is a possible route for infectious metastasis [13] and the fatty tissue in the epidural space makes the epidural abscess easily expandable. Staphylococcus aureas is the most common micro-organism found in an epidural abscess. Streptococcus pyogenes species (β-hemolytic group A) is uncommon [8, 9]. It produces streptokinases, deoxyribonucleases, and hyaluronidase, which hydrolyze the ground substance of connective tissue and aid in the spread of infection.

SEA is a rare condition that can be easily misdiagnosed. There is no specific clinical manifestation. The most common symptoms include back pain, fever, and neurological deficits. Leukocytosis may or may not be present. However, in severe ascending back pain, even without fever or major neurological compromise, SEA must be suspected. SEA
may present as meningitis with fever, neck stiffness, and neurological deficit [5, 8, 9].

Imaging studies play a key role in diagnosing SEA. Although CT myelogram yields high specificity, MRI is considered the first line imaging modality since it is non-invasive [1, 2, 9, 14, 15]. Tung et al. classified epidural inflammation into phlegmon and abscess based on contrast-enhanced MRI images [14]. Phlegmonous tissue is characterized by homogenous contrast enhancement that consists of vascularized granular tissue with microabscesses but little or no drainable pus. In comparison, a peripheral pattern of contrast enhancement is consistent with a frank abscess containing a liquefied, purulent core. Clinically, surgically treated patients with pustular abscesses had better outcomes than patients with granular tissue or granular tissue plus pus [4]. Even with a delayed diagnosis of 3 weeks and a long segment abscess (extending through the whole spine), as seen in our patient, there was an excellent outcome without any complications. The length of the abscess and degree of neural compression did not negatively impact the outcome of our patient. Incomplete recovery of neurological deficit is seen in 40% of cases due to delayed diagnosis of SEA [5, 8], therefore early diagnosis and treatment is crucial for decreasing mortality and morbidity [15]. In the past two decades, the mortality rate has been improving due to improved antibiotic therapy and advanced neuroimaging [2]. Early decompressive laminectomy with antibiotic treatment is the gold standard for SEA [7–9]. However, caution should be used in pediatric patients given that multiple segment laminectomies in a child can cause spinal malformation with growth [6]. If an MRI shows a peripheral pattern of enhancement consistent with liquefied pus, hemilaminotomies with drainage may be adequate, as it proved to be with our patient. Kindler et al. reported that epidural needle drainage with antibiotic therapy yielded satisfactory results [2]. Du Pen et al. reported using antibiotics only for epidural abscess with good results [4]. However, continued vigilance is required with medical therapy alone given the consequences of progressive spinal cord compression.

4. Conclusion

We have reported a rare case of pan spinal epidural abscess in a young, healthy adult. SEA should be considered in a patient with severe ascending back pain even in the absence of risk factors, fever, or major neurologic deficit. MRI is the diagnostic method of choice, and early diagnosis, adequate surgical decompression, and antibiotic therapy can result in an excellent outcome.

References


