Spinal epidural abscess with myelitis and meningitis caused by *Streptococcus pneumoniae* in a young child

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Background: Spinal epidural abscess (SEA) in children is a rare infectious emergency warranting prompt intervention. Predisposing factors include immunosuppression, spinal procedures, and local site infections such as vertebral osteomyelitis and paraspinal abscess. *Staphylococcus aureus* is the most common isolate.

Design: Case report and literature review.

Findings: A 2.5-year-old boy with tetraparesis was found to have an SEA in the posterior lumbar epidural space with evidence of meningitis and myelitis on MRI spine in the absence of any local or systemic predisposing factors or spinal procedures. *Streptococcus pneumoniae* was isolated from the evacuated pus.

Conclusions: Definitive treatment of SEA is a combination of surgical decompression and iv antibiotics. Timely management limits the extent of neurological deficit.

Keywords: Spinal epidural abscess, Tetraparesis, *Streptococcus pneumoniae*, Myelitis, Meningitis, Paralysis

Introduction

Spinal epidural abscess (SEA) is a collection of pus or inflammatory granulation tissue between the dura mater and the overlying vertebral column. It is an infectious emergency requiring prompt medical and surgical intervention.¹ It is rare in children, with contemporary literature based on case reports and small case series.² *Staphylococcus aureus* is the most frequent etiologic agent, followed by gram-negative bacilli. Primarily, patients with immunosuppression and diabetes mellitus, and those undergoing spinal procedures or surgery are predisposed. It is often associated with infection in the adjacent areas such as vertebral osteomyelitis and paraspinal abscess or systemic complications such as infective endocarditis.³ We report a unique case of a 2½-year-old immunocompetent boy with pneumococcal epidural abscess, complicated by concurrent meningitis and myelitis.

Case report

A 2½-year-old boy presented with complaints of high-grade fever, progressive truncal and lower limb weakness, and bladder and bowel incontinence for 10 days. There was no history of seizures, altered sensorium,

back pain, or features suggestive of cranial nerve deficits. The child was developmentally normal, unimmunized, and his past medical and family history were non-contributory.

At admission he was febrile and had tachycardia, tachypnea, and normal oxygen saturation. Systemic examination revealed a lethargic child with normal cranial nerve functions and fundus. He had Grade 3/5 power in bilateral upper limbs and 0/5 in bilateral lower limbs, with generalized areflexia and hypotonia. He was unable to sit without support. Superficial abdominal and bilateral plantar reflexes were not elicitable. There was hypesthesia below T1 level. While neck rigidity and Kernig sign were elicitable, there was no nystagmus or feature of raised intracranial tension. Blood investigations revealed polymorphic leukocytosis and raised Erythrocyte sedimentation rate (ESR). Urgent MRI cranium and spine revealed an epidural collection measuring $1.2 \times 2 \times 3 \text{ cm}^3$ in the lower lumbar region that was hypointense on T1W, was hyperintense on T2W, and displayed peripheral enhancement in post-contrast images suggestive of epidural abscess (Fig. 1). A similar smaller epidural collection was noted in the lower dorsal region. The entire spinal cord showed altered signal intensity, appearing hypointense on T1W, hyperintense on T2W, and mildly enhanced in post-contrast images with expansion

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Figure 1 Sagittal sections of the spine reveals lower lumbar epidural collection measuring $1.2 \times 2.3 \times 3 \text{ cm}^3$ appearing hypointense on T1- and hyperintense on T2-weighted images with mild post-contrast peripheral enhancement.

Figure 2 Altered signal intensity involving the entire cord with cord expansion on T2-weighted sagittal sections suggestive of myelitis.

suggestive of myelitis (Fig. 2). Note was also made of leptomeningeal enhancement and shaggy enhancement of nerve roots of the cauda equina (Fig. 3). MRI cranium was normal.

In view of lower lumbar epidural collection a lumbar puncture was withheld. Ceftriaxone and vancomycin were administered and an urgent surgical evacuation of the lumbar abscess via laminectomy was performed. The dorsal SEA was managed conservatively. Analysis

Figure 3 Post-contrast sections at the L4 level showing shaggy enhancement of the cauda equina and leptomeningeal enhancement.

of the pus revealed a sugar of 15 mg/dl, protein of 195 mg/dl, and abundant neutrophils. On culture, *Streptococcus pneumoniae* was isolated. Retroviral serology and immunoglobulin profile were normal. During the course of stay, upper limb and truncal muscle weakness recovered completely and no ventilatory or inotropic support was required at any point in time. The lower limb power increased to Grade 3/5 but there was no further improvement. The child was discharged after 4 weeks of IV antibiotics.

Discussion

Epidemiology

SEA is estimated to account for 2.5–3 per 10 000 hospital admissions. It predominantly affects adults especially between the fifth and seventh decades.³ It is rare in the pediatric age group with an incidence of 0.6 per 10 000 hospital admissions. It has been reported in age groups ranging from 14 months to 16.5 years.²

Pathogenesis

Because abscesses are more likely to develop in larger epidural spaces that contain infection-prone fat, they are commoner in posterior than anterior areas and in thoracolumbar than cervical areas.^{2–4} In 50% cases, it results from hematogenous dissemination from a remote focus (skin, soft tissue, and urinary or respiratory tract infections) and by contiguous spread from a neighboring infected structure (vertebral osteomyelitis or psoas abscess) in another 10–30%. Iatrogenic causes such as surgery, lumbar puncture, epidural analgesia,

and nerve blocks are estimated to account for 15% of all cases.³ Infection is acquired either during the invasive procedure or through ascending infection by skin flora, when a device is left *in situ*.⁵ A significant proportion of SEA patients have predisposing factors such as diabetes mellitus, immunosuppressive therapy, cancer, AIDS, or renal failure.³ No source or predisposing illness can be identified in 30–40%, suggesting silent bacteremia seeding the epidural space.^{6,7} It appears that in our patient a similar mechanism was responsible.

S. aureus causes two-thirds of the cases of SEA out of which 15% are caused by Methicillin resistant *Staphylococcus aureus* (MRSA).^{2,3,8,9} Other implicated agents include gram-negative bacteria, especially *E. coli* and *Pseudomonas*, coagulase-negative staphylococci such as *Staphylococcus epidermidis*, viridans group streptococci, enterococci, anaerobes, and mycobacterium species.³ *S. pneumoniae* is a rare etiologic agent and isolated from only 1.5% cases with SEA as per one study.^{2,10–12}

Clinical features

Initial symptoms of SEA are non-specific and the classical triad of spinal pain, fever, and neurological deficits is present in only 10–15% of patients at presentation.¹³ The rate of progression of neurological deficit is highly variable, ranging from complete loss of muscle power within a few hours to only slight impairments over several days. Common complications of paresis are pressure ulcers, urinary tract infection, deep-vein thrombosis, and, in patients with cervical abscess, pneumonia.¹⁴ Spread of infection from SEA results in systemic (endocarditis) or local (vertebral osteomyelitis and psoas muscle abscess) complications. There are reports of meningitis and concurrent SEA following epidural space catheterizations by allowing a tract between skin, epidural space, and cerebro-spinal fluid (CSF).¹⁵ However, there is scarcity of literature regarding coexisting meningitis and SEA in the absence of such interventions as noted in our patient. The mechanism of spinal cord damage by SEA remains uncertain. The remarkable neurologic improvement in some patients after decompressive laminectomy suggests direct mechanical compression by the abscess mass.^{4,6,7,16} Spinal cord ischemia due to thrombosis of leptomeningeal vessels or compression of spinal arteries has also been proposed.^{17,18} However, diffuse myelitis, as seen in our patient, has rarely been reported with SEA.

Investigations

Supportive investigative findings include leukocytosis and elevated ESR and C-reactive protein. MRI is the

imaging method of choice because it is less invasive and delineates both longitudinal and paraspinal extension of the abscess (essential for planning surgery) and differentiates between abscess and metastasis.¹⁹ SEA generally extends over three to four vertebrae but, in rare cases, involves the entire spine, resulting in the so-called pan-spinal infection.^{20,21}

Lumbar puncture and CSF studies are less important in the diagnosis of SEA. Both gram staining and culture of CSF have a poor yield, and results of microscopy and biochemical analysis are non-specific. Moreover, as the needle passes through the abscess, there is a risk of spread of infection to subdural and subarachnoid spaces. Secondly, in patients with obstructive hydrocephalus or complete subarachnoid block, removal of CSF might cause downward spinal coning. Thus, it is recommended in only selected cases and when myelography is performed.¹⁹ We refrained from conducting a lumbar puncture in our patient on similar grounds. However, the MRI findings were suggestive of leptomeningeal enhancement.

Treatment

Retrospective studies support surgical drainage together with systemic antibiotics as the treatment of choice. Rapid surgical intervention minimizes not only neurological damage but also spread of infection. Based on the location and extent of the SEA and intraoperative findings, the surgical approaches include laminectomy, hemilaminectomy, interlaminar fenestration, or laminoplasty. Studies done on children with intramedullary tumors have reported that laminoplasty reduced the incidence of subsequent spinal deformity requiring spinal stabilization as compared to laminectomy.²² Pending culture reports, broad-spectrum empirical antibiotic therapy should be provided with coverage against staphylococci. Conservative therapy with antibiotics alone may be tried in patients at high surgical risks or those without neurological deficits.²³

Prognosis

Mortality rates range from 2 to 20%; death is usually due to severe sepsis, particularly in patients with multiple co-morbidities. The final neurological outcome and functional capacity correlate strongly with the severity and duration of neurological deficits prior to surgery and should be assessed at least 1 year after treatment, since patients may continue to regain some neurologic function and benefit from rehabilitation. Irreversible paralysis continues to affect 4–22% of patients.^{3,6,7,16}

Conclusion

This case is unique in multiple aspects. Unlike earlier citations, our patient did not harbor any obvious predisposing factor such as immunosuppression or any local or distant nidus of infection to account for its origin. Secondly, the presence of leptomeningeal enhancement despite the lack of anatomical continuity between the epidural space and subarachnoid region, and in the absence of any event to have created a direct tract between the two, is clearly an unparalleled feature. Thirdly, the presence of pan-myelitis up to the cervical cord is unusual in a lumbar epidural abscess. Lastly, pneumococcus is a rare etiologic organism for SEA. Nevertheless, timely intervention proved helpful in improving the outcome in our patient despite the multiple complications and should be considered by all while treating an epidural abscess.

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