

Case Report

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Spinal Epidural Abscess in Neonate: A Case Report

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Abstract

Incidence of the spinal epidural abscess is rare in neonates [1,2]. Despite no associated specific signs and symptoms, this condition usually commonly presented with neurological deficits. **Case Report:** A case of the spinal epidural abscess is reported, who initially present with nonspecific signs and symptoms and reduced bilateral lower limb movement for months. Magnetic resonance imaging was done for this case, and the results showed T4 to T8 spondylodiscitis with paravertebral enhancing collection causing cord compression. Antituberculosis treatment was started for this patient for a duration of 6 weeks. The patient underwent spinal decompression with drainage. **Conclusion:** Surgical intervention is generally warranted in cases involving spinal instability, worsening neurological symptoms, and inadequate response to pharmacological treatment.

Keywords: Paediatric, Spinal Epidural Abscess, Spinal Decompression.

INTRODUCTION

Primary spinal epidural abscess in neonatal and infant population is rare, and most cases are associated with a previous history of lumbar puncture or epidural anaesthesia [3]. Children under two years old and those more than 12 years old are more common to have spinal epidural abscess [2]. Patients usually present with signs of cord compression as signs and symptoms of spinal epidural abscess are nonspecific. Diagnosis before neurological manifestation is inherently tricky and requires a high level of clinical suspicion.

CASE REPORT

A case of a 10-month-old boy presented with fever and cough for a week. The patient was born via spontaneous vaginal delivery with no antenatal complications. Laboratory investigations were normal, and the patient was treated for pneumonia with antibiotic therapy for one week. However, after two months patient came back with shortness of breath and neurological deficits. Patient unable to lift up chest on prone position and sit without support with the presence of abnormal movement of lower limb (clonus). Additionally, the patient also unable to roll from prone to supine position. Physical examination was normal except reduced power over bilateral lower limbs from the level of L2 to S1 (2/5). Laboratory investigations showed hemoglobin was 11.3 mg/100 ml, white blood cell count was 16.4 with 44% polymorphonuclear leukocytes, sedimentation rate was 83 mm/h, and C-reactive protein was 1.2 mg/l. Blood culture and urine analysis were normal, and coagulation tests were normal. Immunodeficiency screening was normal, and tuberculosis workout was negative. Radiography of the chest and spine was normal except for an interscapular region soft tissue mass in the lateral view of the thoracic spine. Magnetic resonance imaging (MRI) demonstrated T4 to T8 spondylodiscitis with rim enhancing multiloculated paravertebral collection measuring 1cm x 3.2cm x 2.6cm (AP x width x CC) at prevertebral and 0.6cm x 0.5cm x 3.2cm (AP x Width x Cc) at epidural space. It caused significant compression to the spinal cord at the T4 to T8 level, measuring 0.4cm in AP diameter. (Figure 1 & 2). The patient was started on antituberculosis treatment with four drug regime: rifampicin, isoniazide, pyrazinamide and ethambutol for six weeks. Magnetic resonance imaging (MRI) was repeated, and the result showed an extension of paravertebral collection from T3 to T9 vertebral level with cord compression and cord edema. Further reduction in vertebral body height of T5 and T7 with complete collapsed at T6 vertebrae, posterior displacement of the upper vertebrae, and worsening of the kyphotic deformity. The patient underwent spinal decompression and drainage with the application of body cast. The patient completed six weeks of antibiotics in the hospital and then was discharged home. Upon follow up after 12 months, the patient showed progressive recovery in neurology on bilateral lower limbs with no neurological deficits.

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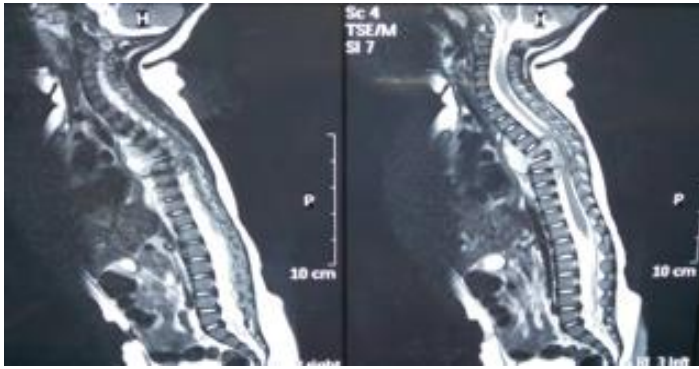


Figure 1: Sagittal view of MRI Spine T2 weighted.

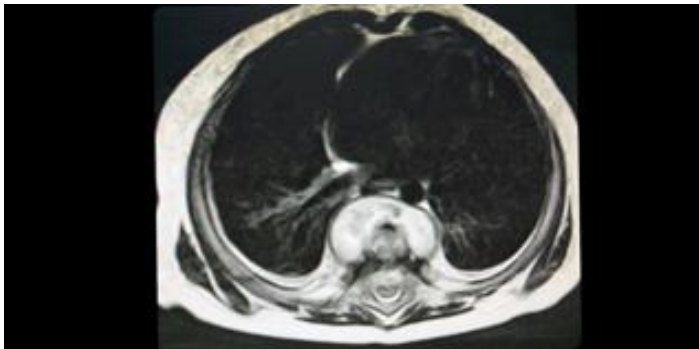


Figure 2: Axial view of MRI Spine at level of T7.

DISCUSSION

Fewer than 90 cases of spinal epidural abscess were reported in children [4]. There are only a few reports of suppurative spinal epidural abscess in neonates. In the absence of any history of spinal instrumentation, the spinal epidural abscess is very rare in the neonate, and this causes a delay in detecting spinal epidural abscess [2]. Its usual main route is through hematogenous spread from primary site [1,2]. Co-morbidities such as diabetes mellitus, chronic renal disease, immunocompromised patient, alcoholism, malignancies, intravenous drug abuse, recent spinal surgery, and spinal trauma increase the risk of spinal epidural abscess [5].

Most of the spinal epidural infections caused by hematogenous spread of the causative organisms, often from a distant site of infection [1]. In 12 to 50% of patients, there is no prior source of infection can be identified [5,6]. *Staphylococcus aureus* is the primary causative organism and is suggested that in infants, breast milk is a source of *Staphylococcus* [7]. Abscesses are most commonly located dorsally in the upper to midthoracic or the lower lumbar regions [6,7]. Spinal epidural abscess is diagnosed based on the clinical history and radiological finding [8]. Patients present with neurological deficits in association with leukocytosis and elevated sedimentation rate suggestive of spinal epidural abscess. Blood culture can sometimes be positive. Magnetic resonance imaging (MRI) is the best use for evaluating the extent and location of an abscess. on T1-weighted images is useful in showing spinal cord displacement and compression. It is reported that treatment with the use of antibiotic therapy alone for the eradication of the abscess is successful, and this leads to controversy in the treatment of spinal dural abscess as others state that surgical decompression plays an important role [9-11]. The purpose of therapy is to eradicate the infection so that patients will have good recovery from neurological deficits and minimal spinal residual deformity. Early diagnosis is important, so paralysis can be prevented [6]. Postoperative antibiotics are given intravenously up to six weeks if no concomitant osteomyelitis is present. Oral antibiotics can be continued for several

weeks after intravenous antibiotics have been discontinued if it is indicated. Good outcomes are shown in those patients with a slow course of the disease, and surgery is done before the development of neurologic deficits [5].

CONCLUSION

Surgical intervention is generally warranted in cases involving spinal instability, worsening neurological symptoms as well as poor response to pharmacological treatment.

Conflicts of interest

The authors declare no conflicts of interest.

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None.

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