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Original Research Paper

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ACUTE FEBRILE ILLNESS PRESENTING AS DIABETIC KETOACIDOSIS AND DEEP VEIN THROMBOSIS FINALLY DIAGNOSED AS CERVICOTHORACIC SPINAL EPIDURAL ABSCESS: CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT Classically, diagnosis of spinal epidural abscess can be made when the triad of symptoms of backache, fever and neurological deterioration are present. But often, presentations can be varied. In this case, a 50 year old male presented with diabetic ketoacidosis and fever , who did not have the conventional symptoms and was subsequently diagnosed with cervicothoracic spinal epidural abscess measuring 17 cm in length after neurological deterioration and was managed by decompressive laminectomy and antibiotics. Expeditious diagnosis and suitable treatment strategies are the need of the hour for preventing lifelong neurological deficits in the patients.

KEYWORDS : Diagnosis, Laminectomy,spinal epidural abscess

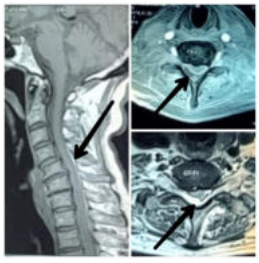
INTRODUCTION:

Spinal epidural abscess(SEA) is a rare diagnosis with an incidence of approximately 0.2–2 cases per 10,000 admissions[1]. Recent literature shows an increase in the incidence- 5.1 cases per 10,000 admissions[2]. Most epidural abscesses are located posteriorly in the thoracic or lumbar spine. Most posterior spinal epidural abscesses are thought to originate from a distant focus such as a skin infection, pharyngitis, or dental abscess[3],[4]. Anterior epidural abscesses are commonly associated with discitis or vertebral osteomyelitis[5],[6]. Methicillin-sensitive Staphylococcus aureus (MSSA) is the most common causative pathogen of SEA, accounting for about two-thirds of cases, followed by other gram-positives, Escherichia coli, etc. Recently, the number of methicillin resistant Staphylococcus aureus (MRSA) has significantly increased [2],[7],[8]. Diabetes mellitus, intravenous drug use are the most relevant risk factors. Others are- long-term systemic corticosteroid therapy, spinal abnormalities such as recent trauma or surgery, epidural anesthesia, treatment around the spinal cord, and systemic bacterial infection from a local infection and injection, local or systemic infections from skin and soft tissue infection, osteomyelitis, UTI, infectious endocarditis, and indwelling vascular access infection, degenerative disk disease, large osteophytes, and chronically hypertrophied facet joints may be the targets of hematogenous bacterial seeding[9],[10],[11]. Presenting complaints of SEA can differ vaguely and often the 'classical triad' of fever , backache and neurological deficits may not be present. The diagnosis is often missed at first presentation, when neurological deficits may be absent.

MATERIALS AND METHODS:

1.1 Clinical history and examination-We present a 50 year old male who presented to a tertiary health care centre in Northwestern region of India, with complaints of fever, shortness of breath, vomiting and abdominal pain. He is a known case of type 2 Diabetes Mellitus, Hypertension and a chronic opium consumer. No neurological deficits were noted at admission. Patient was managed along the guidelines for diabetic ketoacidosis(in view of high blood sugar with metabolic acidosis and ketonuria) and sepsis- Total leucocyte count-16,000/□L, C-reactive protein-135mg/L and temperature-100.1F. No source of infection could be determined. Five days later the patient had complaints of urinary retention, constipation and pitting edema of the left lower limb, along with acute onset paraparesis of bilateral lower limbs. On examination- grade 1 spasticity was noted in bilateral lower limbs, diminution of power in bilateral lower limbs(0/5), and sensory loss bilaterally below T6 level. Deep tendon reflexes were diminished at all levels(+1) and bilateral plantars were mute. Patient was conscious ,alert , responsive and and no evidence of meningeal signs.

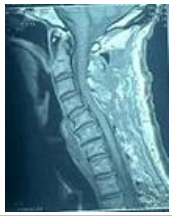
1.2 Radiological investigations-Contrast enhanced Magnetic Resonance Imaging(MRI) showed T1 isointense and T2 hyperintense dorsal epidural collection from C5-T2 level, with mass effect over Spinal cord (**Figure 1**) , dimensions-176mm(CC)x6mm(AP)x16.4mm(TR). Ultrasound Doppler of the left lower limb showed common femoral vein thrombosis and deep vein thrombosis(DVT) of the left lower limb was confirmed.



(Figure 1)

1.3 Management and clinical course-Patient was initiated on Dalteparin and a transfemoral Inferior Vena Cava(IVC) filter was placed. Decompressive Laminectomy was done from C4 to T2 level. Intraoperatively, pale yellow ,thick exudates were noted on the epidural region(**Figure 2**); removal of all visible exudates done under vision. Pus culture showed Coagulase Negative Staphylococcus Species(CONS), sensitive to Coamoxyclav,Clindamycin and Cotrimoxazole. Postoperative MRI showed near resolution of C4-T2 epidural collection with no resultant mass effect(**Figure 3**).

(Figure 2)



(Figure 3)

RESULTS:

Upon follow up at 2 months, the patient had markedly improved in overall health. Neurologically, sensory improvement was noted in the lower limbs , Power in the lower limb had improved to bilaterally and there were no bowel/bladder complaints.

DISCUSSION:

Clinical diagnosis of SEA is an utmost challenge in the absence of classic symptoms. As in this case , the initial presentation of the patient was Diabetic ketoacidosis, which indicates a source of underlying septic focus. Usual fever workups consist of routine blood tests, blood , urine and bronchial culture, cardiac echocardiography. Failure of routine tests to detect the foci can lead to delay in diagnosis. In patients with sepsis with hypotension and septic shock , DVT often complicates the ongoing disease process, as in septic patients, the thrombotic phenomena can occur due to septic emboli, so in the diagnostic phase, it is decisive to keep in mind the differential diagnosis with these atypical presentations[12]. Delay in the diagnosis of SEA results in persistence of neurological deficits even after interventional decompressive strategies[9]. Multiple studies have been undertaken for comparison of medical therapy vs surgical therapy for SEA. Using multivariate analysis, Kim and associates identified risk factors for failure of medical therapy in the treatment of EDA. They specifically identified age >65 years, comorbid diabetes, MRSA infection, and neurological deficit at presentation as significant predictors of medical failure[13]. Surgical decompression remains the mainstay of treatment for spinal epidural abscess. A study by Khanna et al [14] suggested that surgery provides a good outcome when patients' cord symptoms (bowel and bladder dysfunction, paresis, or "plegia") were present for <72 hours, when extent of abscess (the degree of thecal sac compression) is <50 percent, and when patients are <60 years of age. Numerous studies have been done to devise a proper strategy to avoid diagnostic delays, such as- MRI of spine in highly susceptible patients with elevated inflammatory markers[15], to routinely examine for spinal tenderness in septic patients, but none of the approaches have proven to be feasible or cost-effective.

CONCLUSION:

Thorough neurological examination in the SEA susceptible patients, early diagnosis, prompt spinal decompression and comprehensive strategies to search for septic foci in septicaemic patients can surely reduce the diagnostic delays. Until suitable management approaches are devised, the diagnosis of SEA should be kept in mind in patients with acute febrile illness with neurological deterioration.

Consent:

Patient's relatives gave written consent for publication of this case report and publication of radiological and intraoperative images.

Declaration Of Competing Interest:

None

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