

INTRODUCTION

The incidence of tuberculosis (TB) affecting the central nervous system (CNS) is 0.5-2% among patients with systemic TB, of which, every 2 in 2000 cases are intramedullary tuberculomas (IMT)[1,2].



Figure 1: bilateral lung showing miliary mottling

CASE STUDY

Here we present a 52-year-old diabetic male with adequate glycemic control, who presented with gradual onset weakness of bilateral lower limbs, progressive and was diagnosed as a case of diabetic neuropathy at an outside hospital. Nerve conduction study done there showed chronic inflammatory demyelinating neuropathy and was treated with intravenous immunoglobulins and steroids for the same. He subsequently developed fever with pancytopenia following which he was transfused with 12 units random donor plasma, 6 units single donor plasma and 1 unit packed red cells. In lieu of suspicion of enteric fever, the patient was given intravenous ceftriaxone. Bone marrow biopsy showed trilineage hematopoiesis. Despite the treatment, the patient was deteriorating clinically. He further developed difficulty in urination, hematuria and bleeding gums. He was referred to our institute for further evaluation and management. A detailed history revealed contact with a pulmonary TB patient a year ago. Nervous system examination showed paraparesis with hyporeflexia of both lower limbs. On admission, haemoglobin was 8.7g/dl, total leukocyte count 2500 cells/ cumm, platelets 44,000 cells. Liver function test showed indirect hyperbilirubinemia. Renal parameters were within normal limits.

The patient was started on intravenous Piperacillin-tazobactam.

Chest X-ray showed miliary tuberculosis pattern (figure 1). Computed tomography thorax Centrilobular nodules in tree in bud pattern in bilateral lung parenchyma . The patient did not have expectoration, hence nasogastric tube aspirate was sent for AFB gene Xpert showed Mycobacterium tuberculosis, without rifampicin resistance. The patient was started on T. Ethambutol 1000mg OD and T. Levoflox 750mg OD(in consultation with pulmonologist) due to deranged liver function tests. MRI spine revealed an intramedullary tuberculoma at the level of D10 vertebral body. (figure 3) Urine AFB was positive. He was diagnosed to have disseminated tuberculosis. Subsequently, the liver function tests improved and he was started on isoniazid, rifampicin and intravenous Amikacin subsequently. A week later, the patient developed symptomatic hyponatremia (sodium-109mg/dl) and was shifted to the ICU. Creatinine increased to 3.8mg/dl. The patient was found to have polymicrobial sepsis, a blood culture showed Candida tropicalis, urine culture grew Klebsiella pneumonia, started on Inj colistin and antifungal. However, the patient had worsening metabolic acidosis and succumbed.

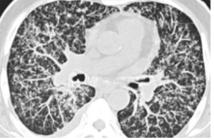


Figure 2: Centrilobular nodules in tree in bud pattern in bilateral lung parenchyma



Figure 3 First arrow: A well-defined intramedullary lesion, with peripheral ring enhancement seen at the level of D10 vertebral body.

Second arrow: showing an ill-defined lesion with peripheral enhancement in the antero inferior aspect of D11 vertebral body.

CONCLUSION.

Pancytopenia is a marker of poor prognosis in military tuberculosis. [3] This could be attributed to hypersplenism, maturation arrest, histocytic hyperplasia and indiscriminate phagocytosis of blood cells by histiocytes in bone marrow or infiltration of the bone marrow by caseating or noncaseating tubercular granulomas. Aminoglycosides are notorious in causing drug-induced nephrotoxicity. Acute kidney injury from aminoglycoside exposure typically manifests after 5-7 days of therapy.[4]Non oliguric renal failure is secondary to loss of renal concentrating ability as a result of distal tubular damage. Imaging of spine at initial clinical presentation is key to early diagnosis and treatment.

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