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Original Research Paper

General Medicine

A CASE OF DISSEMINATED TUBERCULOSIS IN A IMMUNOCOMPETENT PATIENT: TB MENINGITIS, PLEURAL TB, INTESTINAL/PERITONEAL TB

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INTRODUCTION:

Disseminated tuberculosis (TB) is defined as the presence of two or more noncontiguous sites resulting from hematogenous dissemination of Mycobacterium tuberculosis, occurring as a result of progressive primary infection, reactivation of a latent focus with subsequent spread or rarely through iatrogenic origin. Nowadays, the term miliary TB also refers to progressive and widely spread forms of pulmonary TB. It entails a hematogenous spread of the disease to several organ.

Disseminated TB is a life-threatening condition, especially if the diagnosis and treatment are delayed. The diagnosis is difficult because of its nonspecific clinical picture and the paucity of tools available for confirmatory laboratory diagnosis, such as low sensitivity of acid-fast bacilli (AFB) smear, time-consuming cultures, and the inability to easily detect miliary changes in chest X-ray. Miliary pattern on chest radiography is a common finding that has an important role in the early detection of the disease. Nevertheless, approximately 10%–15% of patients have normal chest radiography. Although abnormalities are present, basic hematologic and biochemical tests as well as tuberculin skin test are nonspecific for the diagnosis. Imaging studies are helpful adjunct tools for disseminated TB as they can help determine the involved sites and guide technicians to obtain appropriate specimens for diagnosis. Clinical confirmation of the diagnosis of disseminated TB is usually based on bacteriological or histological evidence. Response to first-line anti-TB drugs is good as evidenced by many reports. This review aims to present a current update on disseminated TB with emphasis on the diagnostic workup of this devastating condition.

Case Report:

A 14years old female patient presented with complains of generalised weakness, low grade fever, weight loss of about 5-6kgs over last 2 month and bilateral lower limb pedal edema, abdominal pain and distension since 18days and dry cough, dyspnea on exertion and chest pain since 6-7days, decreased level of consciousness since 1day. With above complain patient came to civil hospital morbi on 6th October,2022.

On examination, patient was drowsy, not fully oriented and not following verbal command.

On general examination, pallor+, edema+. No icterus, lymphadenopathy, cyanosis, clubbing.

On admission vital, temperature was normal, pulse-140/min, Bp-88/58mmhg, RR-40/min, spo2-78%on room air, RS- left side air entry was decreased and crepitation present over both side, CVS- S1S2+.

On investigation,HB-6.8, WBC-3200, PLATELET-42000, Mp card-negative, PS for MP-negative, ESR-24, creatinine-0.78, s.urea-12, s.billirubin-1.0(direct-0.8/indirect-0.2),SGPT-18,SGOT-20,ALP-42, S.albumin-0.57, S.protein-3.4. S.widal-negative for O and H. HIV/HBsAg/HCV-Non Reactive. Sputum for AFB was negative and CBNAAT-positive.

CXR- left side CP angle blunted, milliary mottling present over both lung zone.

USG CHEST- left sided moderate pleural effusion seen.

USG ABDOMEN- inflamed bowel loops with moderate ascites. No sign of portal hypertension.

Pleural fluid- reddish, without cobweb, TC-3000 (lymphocytes-90%,neutrophils-10%), without malignant cells, sugar- 4, protein-1.35, LDH-1196, ADA-36.71, AFB was Negative & CBNAAT- positive.

Ascitic fluid- turbid, TC- 300 (lymphocytes- 90%, neutrophils-10%), without malignant cells, sugar-38, protein-0.93, albumin-0.13. SAAG ratio - 0.47, ADA-14.32, AFB-negative.

CSF report- hazy, whitish, without cobweb, TC-180(lymphocytes-80%, neutrophils-20%) sugar- 35(matching BLOOD RBS-112), protein-118. ADA-16. AFB was negative & CBNAAT- positive.

DISCUSSION:

Tuberculosis (TB) is a chronic infectious disease caused by M. tuberculosis and may invade all organs but mainly affect the lungs. The clinical presentation of TB is variable with symptoms reflecting the underlying organ involved. It frequently presents as a non-specific constitutional syndrome, with systemic manifestations.

Disseminated TB results from a lymphohematogenous dissemination of Mycobacterium tuberculosis and its atypical clinical presentation often delays the diagnosis. A high index of suspicion and diagnostic persistence are required for diagnosis. The diagnosis is usually confirmed by MT isolation in sputum, body fluids or biopsy specimens, NAA molecular tests and cytohistopathological examination of tissue biopsy specimens. NAA testing should be performed on specimens collected from sites of suspected extra-pulmonary TB due to its specificity >95%. Antibacillary susceptibility tests must be carried out for feasible results.

Although there is a good response to first-line antibacillary therapy, more studies are necessary to clarify the optimum duration of treatment and the unclear role of adjunctive corticosteroid therapy. Prompt TB diagnosis and antibacillary treatment is crucial owing to its associated significant morbidity and mortality.

CONCLUSION:

TB has been well known for centuries, although it is still prevalent in developing and well-developed countries nowadays and it can present unusual presentations. Its natural course can lead to great morbidity and mortality, so it is important to establish prompt diagnosis and treatment to improve its prognosis. Disseminated tuberculosis is a life-threatening condition, with reported mortality of up to 60%. Presentation could be atypical and Disseminated TB could present with normal ESR and with pancytopenia, pleural effusion and ascites in developing nations. Physician should therefore have a high index of suspicion so as to promptly diagnose the condition and offer appropriate treatment.