

Disseminated tuberculosis with splenic involvement in an immunocompetent host: a rare entity

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Received November 25, 2015. Accepted December 2, 2015

Abstract

Disseminated tuberculosis with liver and spleen involvement in an immunocompetent host is a rare entity today, not frequently diagnosed and reported. It is an unusual but possibly fatal form of tuberculosis observed largely in immunocompromised patients. Here, we report a case of a 22-year old immunocompetent woman who presented with multiple nodular opacities on chest X-ray. After investigations, she was diagnosed as a case of disseminated tuberculosis involving lungs, liver, abdominal lymph nodes, and spleen and responded well to antitubercular therapy.

KEY WORDS: Disseminated tuberculosis, immunocompetent host, splenic tuberculosis

Introduction

Tuberculosis (TB) infection is an important public health problem. *Mycobacterium tuberculosis* infection can involve many organs. The lung is the most common lesion site, but extrapulmonary involvement is not uncommon. Besides the usual radiological presentation of pulmonary TB, the infection can imitate several illnesses with fluctuating image presentations.

Disseminated TB refers to the simultaneous participation of not less than two nonadjacent organs or sites of the body or involvement of the blood or bone marrow by tuberculous process.^[1] It is a very unusual form of TB, and, in literature reviews, its frequency is reported to be 2.8% of all TB infections.^[2] It usually occurs in the presence of immunocompromising conditions such as advanced age, cancer, organ transplantation, immunosuppressive and cytotoxic therapy (including biologic agents antitumor necrosis factor), malnutrition, alcoholism, corticosteroids, poorly controlled

diabetes, silicosis, end-stage renal disease, and most importantly HIV/AIDS. Although rare in the immunocompetent population, it is important to recognize that certain genetic defects may predispose immunocompetent individuals to disseminated TB such as abnormalities in the production or metabolism of interferon-gamma and interleukin-12, which are essential for granuloma formation and protective immunity to *M. tuberculosis*. Unfortunately, quantitative or qualitative tests for these cytokines are not widely available in clinical practice.^[3]

Case Report

A 22-year old immunocompetent woman presented with dry cough, fever, breathlessness (MMRC GRADE-II), loss of appetite, and weight loss since one-and-a-half month. Patient was referred to our hospital from a peripheral health center as a suspected case of malignancy. There was no history of antitubercular therapy intake in the past. No history of any comorbid illness or contact with TB patient was found. History of biomass fuel exposure of 10 years was noted.

Patient was thin built with BMI of 18.2 kg/m². Vitals were normal. Oro-dental hygiene was satisfactory. Physical examination revealed swelling over left supraclavicular area. Auscultatory findings suggested bilateral crepts all over the lung fields. Per abdomen, liver and spleen were palpable. Chest X-ray posteroanterior (PA) view [Figure 1] showed bilateral nodular opacities of variable sizes. Routine serum biochemistry and complete blood count was within normal

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DOI: 10.5455/ijmsph.2016.25112015259

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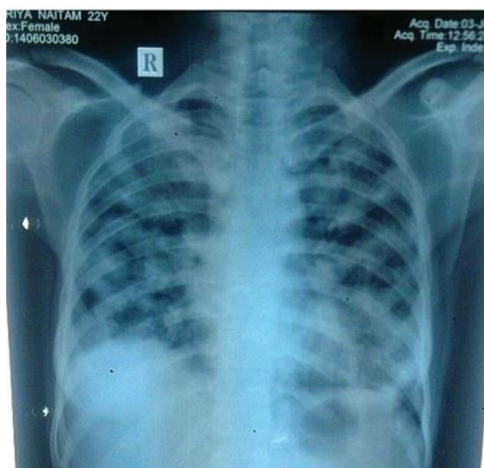


Figure 1: Chest X-ray PA view on admission showing diffuse nodular opacities of variable sizes.

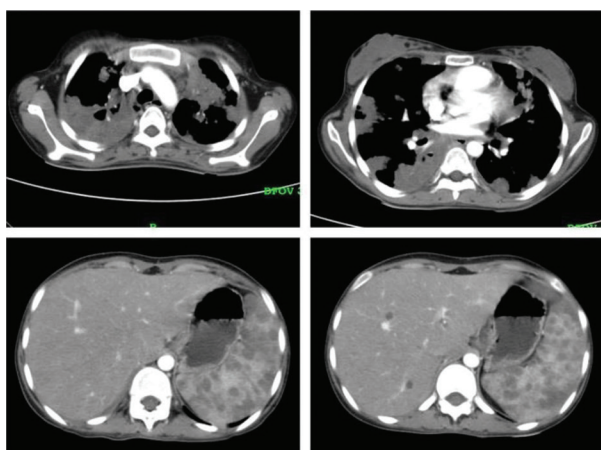


Figure 2: CT scan images on admission.

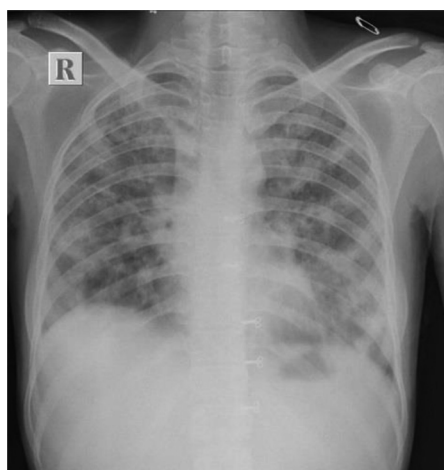


Figure 3: Chest X-ray after 2 months of antitubercular therapy.

limits except for anemia (Hb: 8.1 gm%). High resolution computed tomography thorax [Figure 2] showed multiple fluffy alveolar opacities in both the lungs with hepatic and splenic granulomas with mediastinal and intraabdominal lymphadenopathy. Ultrasonography of abdomen and pelvis showed hepatomegaly with few hypoechoic lesions, heterogeneous echotexture of spleen with multiple hypoechoic lesions within, and multiple small intra-abdominal lymphadenopathies.

Sputum smear stained by Ziehl–Neelsen stain was 2+ positive for acid-fast bacilli (AFB). ultrasonography (USG)-guided fine-needle aspiration cytology of lymph node showed sparse nonspecific inflammatory cells in proteinaceous background. A final diagnosis of disseminated TB was made based on abovementioned investigations, and patient was started on category-I antitubercular drugs. Gradually, patient improved symptomatically, sputum conversion occurred within a month, and radiograph repeated after 2 months [Figure 3] showed good radiological improvement. Follow-up USG abdomen also showed regression of liver and spleen lesions.

Discussion

Disseminated TB is characterized by a wide dissemination into the human body by the tiny size of the lesions (1–5 mm) in the lungs and possibly in other organs of the body, mostly liver, spleen, lymph nodes, pleura, pericardium, meninges, and bone marrow.^[1] It is an unusual but possibly fatal form of TB observed mostly in immune-compromised patients. It is, however, not commonly reported in immunocompetent hosts.

The clinical manifestation of disseminated TB can be acute, subacute, or chronic. Acute disease is rare and may occur in advanced HIV/AIDS or other immunocompromised states.^[3] It is usually severe, including multiorgan system failure, septic shock, and acute respiratory distress syndrome (ARDS).^[4] Hence, miliary TB should always be considered in patients with ARDS of unknown causative factor, particularly if risk factors are present. The subacute or chronic manifestations of miliary TB are more usual than acute disease, and patients may exhibit failure to thrive, fever of unknown origin, night sweats, or dysfunction of one or more organ systems.

The most common laboratory abnormalities include anemia, leukopenia, thrombocytopenia, and lymphopenia. Other laboratory abnormalities may include elevated erythrocyte sedimentation rate and C-reactive protein, hyponatremia, hypercalcemia, and sterile pyuria.^[5] Advanced age (> 60 years), lymphopenia, thrombocytopenia, pancytopenia, hypoalbuminemia, elevated transaminase levels, and delayed treatment have been identified as independent predictors of mortality.^[6]

The classic chest radiograph appearance is a faded, reticulonodular infiltrate distributed fairly consistently throughout the lungs. Other chest radiograph abnormalities include pleural effusion, hilar/mediastinal adenopathy, interstitial or alveolar infiltrates, or cavities. Chest computed tomography (CT) scan is a more sensitive test for evaluation.

Acid-fast microscopy and culture of body fluids, tissue, or drainage from an infected focus establishes the diagnosis,

especially if organisms or caseating granulomas are observed. Fiberoptic bronchoscopy is usually warranted if AFB are not noticed at multiple sites (sputum, gastric aspirate, pleural fluid, ascites, urine, etc.) and there is evidence of pulmonary involvement on chest radiography.^[4]

The tuberculin skin test (PPD) can be a supportive diagnostic tool if positive, but anergy is seen more commonly among patients with disseminated TB (up to 68% of cases) than those with pulmonary or isolated extrapulmonary involvement. PPD conversion may often occur following treatment.

The approach to antimicrobial therapy for treatment of disseminated TB is the same as for pulmonary TB. Early empirical treatment for possible but not yet definitive disseminated TB increases the likelihood of survival and should never be withheld while test results are pending.

Conclusion

Disseminated TB is a potentially lethal form of TB arising from hematogenous dissemination of *M. tuberculosis* bacilli. It mostly manifests in immunosuppressed patients but can also affect immunocompetent adults. Diagnosis is often difficult owing to variable clinical presentations, poorly sensitive smears, and diverse radiologic findings. Although positive chest radiographic findings or a positive tuberculin skin test may sustain the diagnosis, negative results, however, do not eliminate extrapulmonary TB. A high index of clinical suspicion is required and antimycobacterial therapy should be given immediately to avert an otherwise lethal consequence.

References

1. Lessnau KD, Cunha BA, de Luise C. *Miliary Tuberculosis*. Medscape, 2012. Available at: <http://emedicine.medscape.com/article/221777-overview> (last accessed on August 3, 2015).
2. Charfi MR, Dougui MH, Louzir B, Mestiri A, Zbiba M, Belalgia MS, et al. [Disseminated tuberculosis in non-immunocompromised host: three case reports]. *Rev Med Interne* 1998;19(12):917–20.
3. Sharma SK, Mohan A, Sharma A. Challenges in the diagnosis and treatment of miliary tuberculosis. *Indian J Med Res* 2012; 135(5):703–30.
4. Seif F, Armitage K, Petrozzi M. Unusual presentation of a common disease: disseminated tuberculosis in an immunocompetent patient. *Am J Med* 2010;123(9):e5–7.
5. Maartens G, Willcox PA, Benatar SR. Miliary tuberculosis: rapid diagnosis, hematologic abnormalities, and outcome in 109 treated adults. *Am J Med* 1990;89(3):291–6.
6. Hunt BJ, Andrews V, Pettingale KW. The significance of pancytopenia in miliary tuberculosis. *Postgrad Med J* 1987;63(743):801–4.

How to cite this article: Ghewade BD, Sharma T, Chaudhari S, Cladius S. Disseminated tuberculosis with splenic involvement in an immunocompetent host: a rare entity. *Int J Med Sci Public Health* 2016;5:1731-1733

Source of Support: Nil, **Conflict of Interest:** None declared.