Tuberculosis is Protean

Sir,

I read through the recent publication on pulmonary tuberculosis and paraplegia^[1] in the July-September 2013 edition of your journal, and I was excited because I had similar experience of atypical presentations of tuberculosis. Tuberculosis is protean and mimics several other diseases, hence the watch words remain 'a high index of suspicion else it may be missed.' Two cases of tuberculosis; the first was a 7-year-old girl who presented with complaint of fever, cough, diarrhea, difficulty with breathing, and headache for 4-weeks. She had several medications on out-patient without improvement. She was tachypneic, dyspneic with bilateral crepitations; the cardiovascular examination revealed tachycardia, elevated jugular venous pressure displaced cardiac apex-beat; with first, second and third heart sounds and a loud second heart sound. She also had multiple cranial nerve palsies (right oculomotor, abducen nerves); abdominal examination revealed tender hepatomegaly with splenomegaly. She was diagnosed with infective endocarditis with intracranial embolism. Full blood count showed neutrophilia with normocytic normochromic anemia, but blood cultures were negative on three occasions; retroviral screen was also non-reactive. Both Mantoux test and sputum AFB were negative; chest X-ray revealed cardiomegaly with perihaliar opacities while electrocardiogram (ECG) revealed sinus tachycardiac, biventricular hypertrophy, echocardiogram was negative for any vegetation [Figure 1]. She was commenced on genticin and ceftriaxone for 2-weeks but rather deteriorated. She had lumbar puncture; the cerebrospinal fluid analysis revealed elevated protein and normal glucose; cerebrospinal fluid gram stain and AFB were not remarkable while culture was negative; therefore, an aseptic meningitis was entertained, and the thought of disseminated tuberculosis was entertained. She was commenced on anti-tuberculosis, and she became afebrile by the 3rd-week of treatment with progressive resolution symptoms

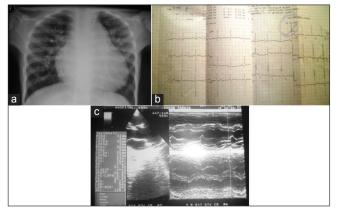


Figure 1: Showing (a) chest-x-ray with cardiomegaly; (b) ECG with biventricular hypertrophy; (c) normal Echocardiography

and was discharged to the directly observed treatment (DOTs) clinic for continuation of supervised treatment.

While the second case was a 14-year-old girl with Potts disease; she presented with back pain, progressive difficulty with walking with subsequent inability to walk within 3-weeks with bladder and fecal incontinence; in her case, there were no preceding complaints of cough or difficulty with breathing. She was paraplegic with kyphosis [Figure 2]. Mantoux test was 12 mm; chest x-ray was normal. She was commenced on anti-tuberculosis therapy.

Tuberculosis still remains a huge health burden in children in developing countries despite presence of effective vaccine; this is further worsened by the ravaging effect of human immunodeficiency virus/acquired immune deficiency syndrome. Diagnosing tuberculosis in children in a resource-limited setting is often difficult; therefore, it is mostly based on high index of suspicion.

Tuberculosis is protean and may mimic several disease entities; however, the clinical features and laboratory results of the first case were typical of a bacterial infection; the diarrhea, weakness initially observed can occur in early stages of tuberculous meningitis, which is easily confused with gastroenteritis, and the heart failure with multiple cranial nerve palsies mimicked a cardiac defect with stroke. Anemia and neutrophilia has been associated with tuberculosis though mostly in adults;^[2,3] however, the exact mechanism of tuberculosis-associated anemia is not completely understood, though like in any chronic infection/ inflammation, a normocytic normochromic anemia (due to impaired erythropoisis) or microcytic hypochromic anemia may occur,^[4] but TB causes more of leukocytosis with lymphocytosis. The observed neutrophilia in the index case could be attributable to the influence of chronic bone marrow stimulation with outpouring of leukocytes including neutrophils, and in extreme cases, left shift with release of immature neutrophils may occur.

As in the case in your publication, my second case had no preceding complaint suggestive of pulmonary disease; therefore, Potts disease may occur without associated chest symptoms.



Figure 2: Showing (a) kyphosis; (b) paraplegic limbs

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