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Letter to Editor

Atypical presentation of tuberculous arthritis of the sternoclavicular joint in a patient on dexamethasone-cyclophosphamide pulse therapy

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Sir,

About 40% of the Indian population is infected with *Mycobacterium tuberculosis* (TB), majority having latent infection. This high prevalence of latent tuberculous infection poses a major threat due to its potency for reactivation to active TB.[1] The risk of TB reactivation increases 2.8–7.7-fold in patients treated with corticosteroids.[2] Here, we report a case of patient with pemphigus vulgaris on dexamethasone-cyclophosphamide pulse (DCP) therapy who developed acute sternocostoclavicular joint swelling diagnosed as tuberculous arthritis.

A 54-year-old male patient with pemphigus vulgaris on treatment with DCP therapy was admitted for the 4th pulse of Phase II. He had tolerated the previous pulses with no complaints. One day after completion of the present pulse therapy, he had an acute chest pain and difficulty in moving the neck. Two days later, he manifested a high-grade fever and swelling over the left upper chest extending to the neck. Examination revealed warm, tender, erythematous, diffuse, nonfluctuant swelling of about 14 cm×6 cm over the left sternoclavicular joint extending to the root of the left neck [Figure 1]. Blood examination revealed raised erythrocyte sedimentation rate (ESR) of 75 mmHg and C-reactive protein of 16.3. The Mantoux test was negative. Chest X-ray and electrocardiography were normal. Soft-tissue ultrasonography showed heterogeneous echogenic particles in the sternoclavicular joint space and inflammation of the surrounding area suggestive of septic arthritis. The patient was started on systemic antibiotics but the symptoms persisted. Further evaluation was done by computerized tomogram of the neck and thorax which showed erosions over the sternal end of the clavicle and sternum at the left sternoclavicular joint with ill-defined fluid collection noted anterior to the joint. Fine-needle aspirate from the swelling revealed granulomatous lesion with necrosis suggestive of TB [Figure 2a and b]. The aspirate was negative in Ziehl–Neelsen and Gram staining. The culture of aspirate did not yield any growth. In view of clinical and investigation findings, TB of the sternoclavicular joint due to reactivation of latent infection was diagnosed and the patient was started on Category 1 antituberculous therapy. The swelling reduced in size within 1 month and completely subsided within 3 months of starting of antituberculous therapy [Figure 3].

Extrapulmonary TB occurs in more than 20% immunocompromised patients.[1] Our patient was on treatment with DCP therapy which is known to cause immunosuppression and reactivation of TB.[3]

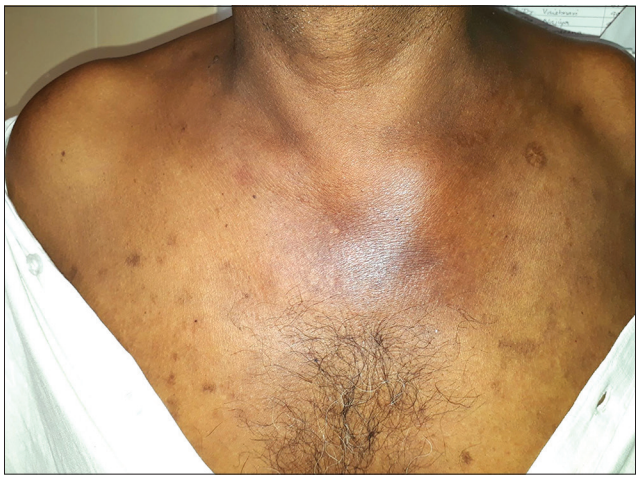

Figure 1: Tender, erythematous swelling over the left sternoclavicular joint extending to the root of the left neck.

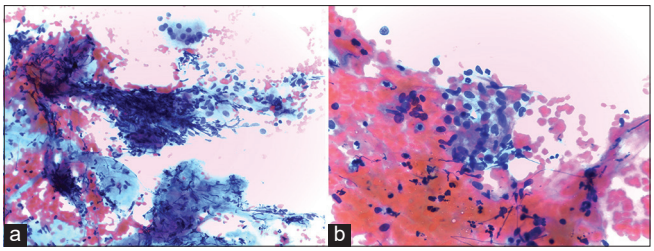

Figure 2: (a) Fine-needle aspirate from the swelling showed multinucleated giant cells and necrosis (Papanicolaou stain, ×100). (b) High-power view of aspirate showing epithelioid histiocytes and multinucleate giant cells (Papanicolaou stain, ×400).

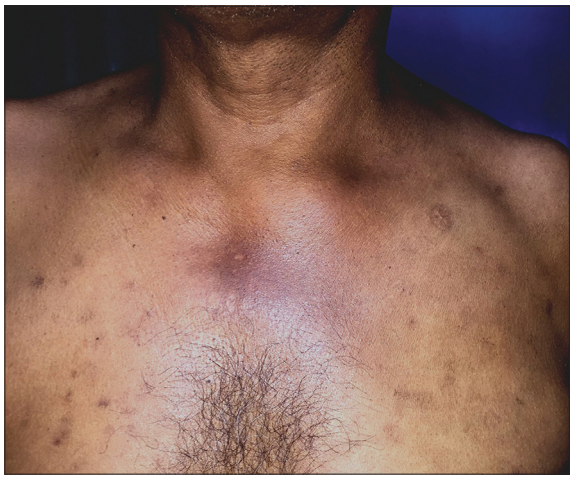

Figure 3: Subsiding swelling after 1 month of starting treatment.

Bone and joint infection accounts for 1%–3% of patients with TB, the most common site being long bones. Sternoclavicular joint infection is a very rare form of osteoarticular TB reported in only 1%–2% of all cases of peripheral tubercular arthritis.[4] Osteoarticular TB is usually a result of hematogenous spread from a fresh or reactivated pulmonary focus. However, blood supply to the sternoclavicular joint is from a periarticular arcade of blood vessels that originate from the internal mammary artery, making hematogenous spread from the lung unlikely. Contiguous spread from an apical pulmonary tuberculous focus to the sternoclavicular joint is feasible.[5] Our patient had the initial presentation as sternoclavicular joint infection with no evidence of TB elsewhere. Sternoclavicular joint TB is usually insidious in onset. However, our patient had acute symptoms such as severe pain, tenderness, swelling, and fever mimicking septic arthritis.

Acute suppurative presentations, though described in lymph nodes, thyroid, and breast, are very rare with osteoarticular TB. Acute exudative presentation may be due to hypersensitivity response to an already existing untreated or partially treated tubercular lesion. Superficial bones and joints such as phalanges, metacarpals, clavicle, sternum, and shoulder joint can be affected in this manner.[5]

Dhillon *et al.* have suggested dual course for sternoclavicular TB; a more aggressive one leading to a painful joint associated with constitutional symptoms or a slowly progressive, relatively painless swelling without constitutional symptoms due to differences in the virulence of the organisms and host resistance.[6] Our case seems to follow the first pattern.

The negative Mantoux test in our patient may be due to immunosuppression by high dose of corticosteroids and cyclophosphamide. Plain radiograph is not considered as a good imaging tool as this region of the chest is poorly visualized due to confluence of structures.[4] The chest X-ray in our patient was found to be normal without depicting any bony abnormality. Soft-tissue ultrasonography showed features suggestive of septic arthritis. Elevated ESR and persistence of symptoms in spite of systemic antibiotic therapy prompted us to evaluate further with better imaging methods.

Computerized tomogram of the neck and thorax delineated the findings of arthritis of sternoclavicular joints. Due to financial constraints, magnetic resonance imaging could not be done in our patient.

The imaging methods could not clearly differentiate septic and a tubercular etiology, and therefore, tissue diagnosis was imperative and fine needle aspiration performed showed histological findings suggestive of TB.

Osteoarticular TB is a paucibacillary disease, and therefore, microbiological tests may be negative as seen in our case.[5]

Lack of awareness and atypical presentation has made extrapulmonary TB a difficult to diagnose condition.[7] Although reactivation of latent tuberculous infection is a known complication of DCP, the same presenting

as sternoclavicular joint arthritis is not reported in the literature. Moreover, the atypical acute presentation of tuberculous arthritis mimicking septic arthritis might lead to a delay in diagnosis, and treatment unless a high degree of suspicion of TB is maintained, especially in an immunocompromised patient. Since the detection of tubercle bacillus is very difficult in microscopic examination and by culture, histological findings of granulomatous lesion with necrosis warrant prompt treatment with antituberculous therapy.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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