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Case Report

A case of Poncet's disease

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ABSTRACT

Reactive arthritis in tuberculosis (TB) is known as Poncet's disease, a rare aseptic form of arthritis observed in patients with active TB. We report a case of Poncet's disease in a 17-year old girl whose reactive arthritis overshadowed other clinical symptoms of TB resulting in delayed diagnosis and treatment. Anti tubercular treatment (DOTS) was initiated. Clinical remission occurred after two weeks and the diagnosis of Poncet's arthritis was established. Thus, taking a thorough medical history as well as performing relevant examinations and investigations for possible TB will help expedite the diagnostic process.

Keywords: Reactive arthritis, Tuberculosis, Poncet's disease

INTRODUCTION

Tuberculosis (TB) is a major public health concern. It remains the leading cause of death attributed to infectious diseases. Atypical presentation of TB can also be a contributing factor for the incidence of TB to remain high. Associated rheumatologic diseases such as reactive arthritis may overshadow typical clinical features of TB. Tuberculous rheumatism, also known as Poncet's disease is a rare syndrome described by the Frenchman A. Poncet in 1897.[1] It is characterized by articular impairment in patients diagnosed with tuberculosis, not related to direct invasion by the micro-organism, but to an immune reaction to the tuberculo-protein, constituting a reactive arthritis. This case is reported because of its rarity and in a tuberculosis endemic country like India, one should keep this possibility in mind in patients with polyarthritis, as early recognition of this complication is of major importance to avoid delayed initiation of appropriate treatment.

CASE DESCRIPTION

A 17-year-old girl presented to us with complaints of low grade fever, swelling and joint pains of both upper and lower limbs for 2 weeks. There was no history of photosensitivity, malar rash, oral ulceration, back pain, rash over the body, diarrhoea, or burning micturition. On examination, pallor was present and there was involvement of both wrist joints, both shoulder joints, and both ankle joints in form of pain, tenderness and local swelling (Figure 1) and there was no lymphadenopathy or erythema nodosum. Investigations revealed TLC-6900 cells/cmm (N-71,L-24,E-01,M-04), Haemoglobin of 10.2 gm% and microcytic hypochromic picture on peripheral smear. Aerobic throat swab culture was negative. An ESR of 81 mmHg per 1st hour and Mantoux test was strongly positive (20 x 12 mm). Rheumatoid factor, anti-nuclear antibody, anti-CCP antibodies, c-ANCA and p-ANCA were negative. USG of Whole Abdomen reveals B/L polycystic ovaries. Serum CRP (3 mg/L), LDH (204.71 U/L), Uric

Acid (6.12 mg%) and ACE(33.9 U/L),LFT(Total Bilirubin-0.3 mg%, Direct bilirubin-0.2 mg%,SGOT-20 U/L,SGPT – 9 U/L, Alkaline Phosphatase – 82 U/L), FBS-72 mg%. PPBS- 86 mg%, Serum Sodium-138 mmol/L, serum potassium-4.3 mmol/L, serum urea-14 mg/dl, creatinine-0.5 mg/dl, all levels were within normal limits. Urine routine examination was normal. Chest X-ray (Figure 2) revealed nodular opacities bilaterally in mid-zones. CECT chest revealed necrotic cervical, hilar and mediastinal lymphadenopathy. X-rays of the involved joints showed periarticular soft tissue swelling (Figure 3), and there were no changes of active tuberculosis. We gave the patient anti-tubercular therapy. On follow-up she became afebrile, and joint pains and joint swelling reduced after 2 weeks of treatment. After 6 weeks of treatment the patient completely became free of joint pain.

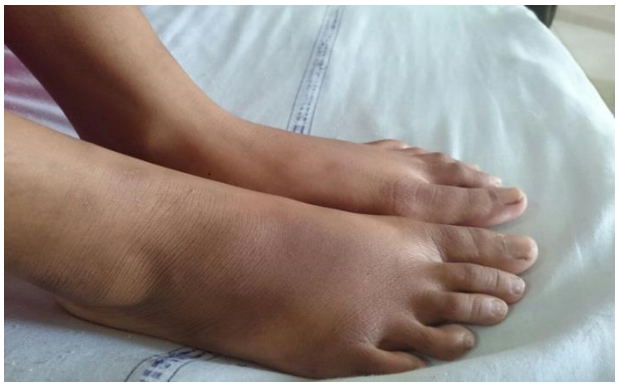

Figure 1: Photograph of both feet showing diffuse swelling of both ankle joints, subtalar joints and metatarsophalangeal joints.

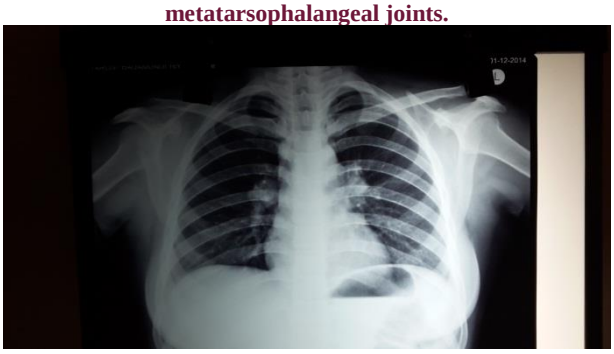

Figure 2: Chest X-ray showing nodular opacities bilaterally in mid-zones.

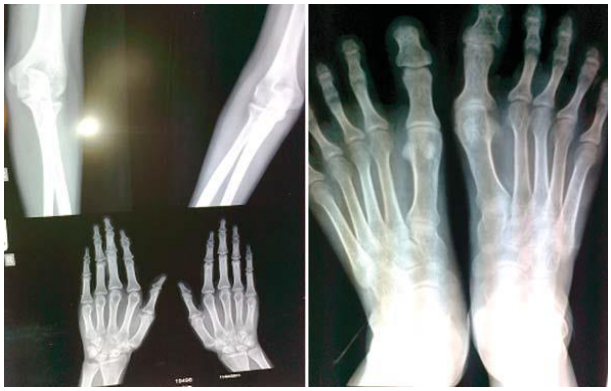

Figure 3: X-rays of the involved joints showing periarticular soft tissue swelling.

DISCUSSION

Tuberculosis is a very prevalent disease in developing countries. Approximately 10% to 11% of the extrapulmonary tuberculosis cases affect bones and joints, corresponding to 1% to 3% of all cases of tuberculosis.The potential of tuberculosis, even when subclinical, to trigger reactive conditions should be remembered. That possibility becomes increasingly important as the careless use of corticosteroids, immune suppressants or biologicals can trigger the reactivation or dissemination of the disease. Active tuberculosis may be complicated by reactive arthritis known as Poncet's disease. It is widely known that tubercular septic monoarthritis, in which M. tuberculosis may be isolated from the joint, may complicate tuberculous infection; that active TB may be complicated by a sterile reactive arthritis is less known and therefore often missed. Poncet's disease is used to indicate an aseptic polyarthritis, presumably a reactive arthritis, developing in the presence of active TB elsewhere.[2] Although Poncet's disease is considered a reactive arthritis, the clinical presentation of Poncet's disease differs from the classical pattern of reactive arthritis.[3,4] In contrast to reactive arthritis, the onset of symptoms in Poncet's disease before the start of arthritis is much longer than just a few weeks, whereas resolution of arthritis upon starting of adequate anti-tuberculous therapy is mostly within a few weeks. Chronic arthritis has never been reported in Poncet's disease. Furthermore, Poncet's disease is generally – except in two reports[5,6] – not associated with sacroiliitis. The etiopathogenesis of that disease is still very controversial. The more accepted hypothesis include molecular mimicry and thermal shock proteins.[7] Thermal shock proteins represent a group of proteins produced by all species in face of varied stimuli, such as heat, radiation, viral infection, and cytokines. Several pathogens, including mycobacteria, have antigens homologous to proteins, suggesting autoimmunity. Bacillary antigens share amino acid sequences with cartilage proteoglycans, producing cross reactivity, mediated by T lymphocytes, and lesion in host cells. There must be genetic predisposition, because the HLA-DR3 and HLA-DR4 genotypes show an exacerbated T-lymphocyte-mediated response to mycobacterial antigens, and can represent the expression of an immune response that also occurs with tuberculin.[8,9] In Poncet's disease, the oligo or polyarticular impairment is more frequent than the monoarticular impairment, similarly to other reactive arthritis, involving mainly the large joints,such as knees, ankles, and hips, often accompanied by articular effusion. There is no microbiological evidence of the mycobacterium invasion in the affected joint,[10] In our patient, the serological tests for autoimmunity are negative, and the tuberculin test, as well as acute phase proteins, are altered.[11] It has been hypothesised that after infection, as a result of systemic immunisation, sensitised CD4 cells together with bacterial antigens migrate to the joints and cause arthritis.

CONCLUSION

The differential diagnosis of patients at risk for TB presenting with arthritis should definitely include Poncet's disease. The diagnosis of Poncet's disease remains clinical and is established on excluding other potential causes of arthritis in a patient with active tuberculosis. The complete resolution of arthritis of Poncet's disease on anti-tubercular therapy also provides further proof of the diagnosis.

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Ethical approval: Not required

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