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# *A Rare Case of Poncet's Arthritis presenting with Pulmonary Tuberculosis*

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## Abstract

One of the rare presentations of active pulmonary or even extra pulmonary tuberculosis is aseptic polyarthritis with the involvement of multiple large and small joints in the body with no evidence of direct bacillary invasion of the joints; a reactive constellation known as Poncet's disease. The condition is different from tuberculous arthritis which is usually monoarticular and is caused by direct tubercular involvement of the joint. Polyarthritis as a symptom of active tuberculosis can be easily misinterpreted for more common causes of polyarthritis such as rheumatological diseases that present similarly. We describe a case of Poncet's disease in a 70-years old lady whose reactive arthritis overshadowed other clinical symptoms of pulmonary TB resulting in delayed diagnosis and treatment. Anti TB treatment was initiated. Clinical remission occurred after two weeks and the diagnosis of Poncet's arthritis was established concluding that taking a thorough medical history as well as performing relevant examinations and investigations for possible TB especially in endemic areas will help expedite the diagnostic process even in absence of TB symptoms.

**Keywords:** aseptic polyarthritis; poncet's, tuberculosis

## Introduction

The incidence of tuberculosis (TB) has increased exponentially, thus TB remains a major source of morbidity and mortality worldwide [1]. Approximately 10-19 % of extra pulmonary TB involves joints and bones [2]. Almost half of these cases are spinal TB, followed by TB arthritis, TB osteomyelitis and reactive arthritis the latter reactive arthritis is known as Poncet's disease (PD) [3]. PD is a rare syndrome first introduced in 1897 by the Frenchman Antonin Poncet when he described a polyarthritis in an acute stage of TB, which resolved without joint damage. Continuous reports3 on patients with similar characteristics led authors to improve the definition and, in 1978, Bloxham and Addy defined PD as a parainfective arthritis [4], but its existence has been questioned; however, more cases have been reported over the years. PD is characterized by articular impairment in patients diagnosed with tuberculosis, not related to direct invasion by the micro-organism. The exact mechanism underlying polyarthritis is not known, however the induction of cell-mediated immunity and/or autoimmunity are possible postulation. Almost all patients treated with antituberculous drugs have reported resolution of symptoms on therapy. This is an unusual and uncommon condition. The diagnosis needs strong clinical suspicion. This case is reported because of its rarity and in a tuberculosis endemic area of a country like Bangladesh; 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 [5,6].

## Case Presentation

Mrs. X, a 70 years old lady from rural Bangladesh, not known to have diabetes, hypertension, coronary artery disease or bronchial asthma, presented to our hospital with the complaints of progressive pain and swelling in her both elbows, knees, shoulders and small joints of both feet for 10 days without any prior trauma or without accompanying erythema. She reported a considerable decline in her functionality with an inability to carry out even daily routine activities, being restricted to a bed for the last few days. But she had no pain in small hand joints, neck, lower back, buttocks, and sole or over both heels. Pain was constant, persisted throughout the day, more with movement with little inactivity stiffness. She denied any dysuria or bloody

stool preceding the illness. Her vaccination status including BCG vaccination was unknown. There was no family history of TB, rheumatologic disease, or autoimmune disease or any history of contact with anybody with active TB or similar symptoms. He was in a monogamous relationship. On query, she had low grade, evening rise of fever with anorexia, malaise and weight loss of about 6 kg in last 3 months. She also had persistent dry cough and feeling of shortness of breath for the same duration without chest pain, hemoptysis. She was being treated elsewhere with a variety of NSAIDs, steroids which gave her some relief.

Physical examination revealed ill looking, cachectic patient with tachycardia, low grade fever with pulse 102/m, regular, B.P.120/80, temp. 38°C. There was mild pallor but jaundice, cyanosis was absent. There were no nail changes suggestive of clubbing, koilonychia, leukonychia or any vasculitic changes. No lymphadenopathy were found. Joint examination revealed swollen and tender both knee and elbow joints without overlying erythema or raised temperature. There is painful restrictions of both active and passive movement of the affected joints. Patellar tap was present over left knee. Chest examination revealed trachea is shifted to left with reduced chest expansibility over left chest. Percussion note was dull with reduced breath sound and vocal resonance over the same area. There were no added sounds. Examination of other systems revealed no abnormalities.

Laboratory investigation revealed normoocytic normochromic anemia of Hb- 10.6 gm%(MCV 83.5, MCH-27.7), ESR 78 mm in 1st hour, TC-29.15 × 103/cmm(N-88.9%, L-5.5%), TPC-776000/cmm, CRP. 58.5 mg/dl,PBF- normocytic normochromic anemia with neutrophilic leukocytosis and thrombocytosis. Urine R/E revealed trace proteinuria, RBC- 2-3/HPF, pus cell 6-8/HPF with no casts. CRP and S. ferritin was raised with 108.29mg/L(normal <6 mg/L) and 4035 ng/L(45- 150 ng/L) respectively. Renal function tests, RBS, serum uric acid, Blood C/S Urine C/S all were noncontributory. On immunological test, ANA, RA factor, Anti CCP all came negative. Serological investigations for HBV and HCV were negative. Synovial fluid study revealed protein 4.5 gm/dl, glucose 12.50 mg/dl. Cell count showed plenty of neutrophil predominant polymorph. Synovial fluid for gram stain, AFB stain, gene X pert TB, MTB-PCR, culture or crystal was negative but synovial fluid ADA was high:118.19U/L(normal <25 U/L). Her chest x ray was consistent with right sided apical opacity and left sided collapse (Figure 1). Sputum for AFB stain or gene X pert was negative. MT was 4 mm after 72 hours.

Hence, with the provisional diagnosissss of reactive

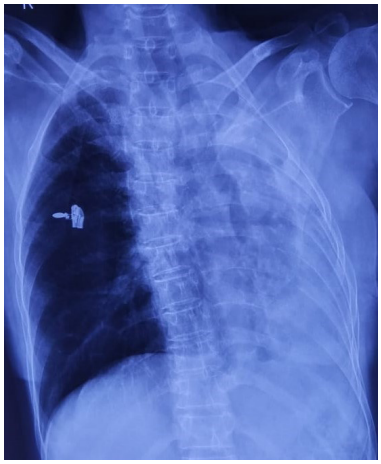

*Figure 1: Chest x ray showing ill defined opacity over right apical region with left sided collapse.*

polyarthritis associated with pulmonary TB, she started on standard anti-tuberculous therapy (ATT) with four-drug regimen (isoniazid, rifampicin, ethambutol, and pyrazinamide) and pyridoxine supplementation. There was alleviation of all the symptoms with no joint swelling and tenderness. She became afebrile and her CRP and ESR came to 36.2 mg/dl and 23 mm in 1st hour respectively on 10th day following ATT. She was discharged with the plan to continue four-drug regimen for initial 2 months followed by two drug regimen((isoniazid, rifampicin) for next 4 months.

## Discussion

Tuberculous rheumatism (Poncet's disease) is a rare, reactive, acute-onset, sterile, non-erosive, inflammatory polyarthritis associated with active extra-articular tuberculosis (TB) [7,8]. It presents as symmetrical involvement of multiple joints without evidence of active infection and resolves without residual joint damage or long-term complications [8,9]. Pulmonary TB was found to be the commonest site of TB infection (43.2%). The sites of extrapulmonary TB infection included lymphatic, renal, bone, skin, and other less common sites. Oligoarthritis was the most common rheumatological presentation (40%) followed by polyarthritis (27.6%) and monoarthritis (24.6%). The most frequently involved joints were the ankles (63%) and knees (59%). The wrists, elbows, interphalangeal joints, metacarpophalangeal joints, shoulders, and metatarsophalangeal joints were affected less frequently. Axial involvement has not yet been reported to date. Although Poncet's disease is considered a reactive arthritis, the clinical presentation of Poncet's disease differs from the classical pattern of reactive arthritis [10]. 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 [11]. The pathogenesis of PD remains to be established; however, it appears to be multifactorial including the activation of CD4+ and CD8+ T cells and hypersensitivity against tuberculosis (TB) proteins [12]. Diagnosis of PD is generally difficult because it starts insidiously, progresses slowly, and has no extra-articular features. The most frequent clinical sign is the change in the range of motions with pain or painless [13].

Poncet's disease often affects young people and is slightly more common in women. Clinical findings include fever, malaise, and polyarthritis of large joints. Diagnosis of PD is generally difficult because it starts insidiously, progresses slowly, and has no extra-articular features. The most frequent clinical sign is the change in the range of motions with pain or painless. The fact that TB septic monoarthritis, in which Mycobacterium tuberculosis can be isolated from the joint, may complicate TB infection is widely known. On the other hand, active TB may also be complicated by a sterile reactive arthritis called PD, which is less recognized and therefore frequently missed.

In contrast to the usual tuberculous arthritis which is monoarticular, infectious and destructive, tuberculous rheumatism (Poncet's disease) is a non-destructive para-infective symmetric polyarthritis occurring in patients with active visceral or disseminated tuberculosis, in which there is neither evidence of bacteriological involvement of joint themselves nor any other known cause of polyarthritis detected.This finding was consistent with the clinical presentation in our case in which the patient reported to us symptoms of painful knee, upper/lower limbs, elbows and shoulder joints. The symptoms resolve completely with anti-tuberculosis therapy. In the present case as well after the initiation of anti-tubercular regimen, the patient reported marked reduction in bone pain, no morning stiffness of joints, increase in appetite, no evening rise in temperature started to reduce.

Considering the incidence of tuberculosis in our country the number of reported cases of Poncet's disease are few thereby confirming that the diagnosis needs strong clinical surmise. Poncet's initial concept of tubercular rheumatism was based on association of polyarthritis with:

1. Active or inactive visceral tuberculosis or
2. Family history of tuberculosis or
3. Presence of a true tubercular joint in any patient before, coincident with or following a polyarthritis of any type

The above criterion was met with many controversies, as it was very broad based and led to diagnostic inaccuracies. The very existence of such an entity was questioned by some.

It largely remains unclear why sterile reactive polyarthritis complicates visceral TB. A vigorous immune response to mycobacteria within joints has been postulated, where mycobacterial antigen-induced activation of T cells then leads to their cross-reactivity with cartilage proteoglycans [7]. This hypersensitivity to mycobacterial antigens may be associated with HLA DR3 and/or HLA DR4 haplotypes [9]. It is interesting to note that anti-cyclic citrullinated peptide (anti-CCP) has high sensitivity and specificity for RA but now its role in active tuberculous arthritis has been reported as well [14]. Anti-CCP is no more specific to RA and frequently seen in patients with active TB [15]. TB arthritis can be confused easily with RA due to the same radiologic features including periarticular osteopenia and marginal erosions [16].

The prognosis in poncet's disease is, however, uniformly good, if the prime cause is treated promptly. In view of the high prevalence of tuberculosis in our country, it is possible that the condition is more common than is reported in the literature.

## Conclusion

Given the debilitating nature of the illness and the ease of cure, tuberculous etiology deserves strong clinical suspicion in the differential diagnosis of polyarthritis of obscure cause or unusual presentation. In conclusion, 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-tuberculosis therapy also furnishes further proof of the diagnosis. Correct identification of this rare complication of TB may avoid delayed initiation of correct treatment.

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