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Case Report

Poncet's disease (tuberculous rheumatism) in a Nigerian male: a frequently overlooked diagnosis

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ABSTRACT

Poncet's disease is an uncommon syndrome and a reactive polyarthritis associated with active tuberculosis with no evidence of mycobacterial infection of the joint. The condition is different from tuberculous arthritis which is usually monoarticular and caused by direct tubercular involvement of the joint. There has been a paucity of case reports on Poncet's disease in Nigeria despite the high incidence of tuberculosis. We report a case of a 45-year male who presented with inflammatory polyarthritis involving the knees, wrists, ankles, small joints of the hands bilaterally of four weeks duration. This was preceded by three week's history of productive cough. Chest radiograph and sputum Gene Xpert revealed features consistent with tuberculosis. Synovial fluid from knee joint effusion showed no evidence of mycobacterium tuberculosis. He was diagnosed to have Poncet's disease based on the clinical findings and investigation results. He was commenced on antituberculous medications with complete resolution of symptoms after two months. Poncet's disease is a rare manifestation of tuberculosis and should be considered a differential in any case of active tuberculosis presenting with arthritis. The correct and prompt identification of this condition by clinicians is important with the aim of instituting the appropriate therapy.

Keywords: Poncet's disease, Polyarthritis, Tuberculous rheumatism

INTRODUCTION

In sub-Saharan Africa, tuberculosis (TB) is a significant public health problem and a cause of morbidity and mortality. Skeletal TB accounts for about 10-35% of extra-pulmonary TB and 2% of all types of TB.[1] Skeletal TB presents as TB osteomyelitis, TB arthritis, Spinal TB, and reactive arthritis known as Poncet's disease.[1,2]

Poncet's disease (PD), which was first described by Antonin Poncet in 1897, is a rare disorder. It is a reactive polyarthritis associated with acute tuberculosis with no evidence of mycobacterial infection of the joint.[3-5] There has been a paucity of case report on PD in Nigeria despite the high incidence of tuberculosis.

The underlying mechanism of PD is not fully known but probable postulation includes induction of cell-mediated immunity and/or autoimmunity.[6] It is a form of reactive arthritis that develops from an immune reaction to the tuberculous protein.[7,8] Treatment involves the use of antituberculous drugs with complete symptom resolution.[9] We report a case of a 45-year-old man with PD in association with acute tuberculosis of the lungs.

CASE REPORT

A 45-year old male presented with a four-week history of inflammatory joint pains involving the knees, wrists, ankles, and small joint of the hands bilaterally. The patient had difficulty ambulating due to the gradual worsening of joint pains in the preceding weeks. There

was morning joint stiffness lasting over thirty minutes. The patient also reported a history of cough productive of mucoid sputum three weeks before the onset of joint pains. He had a low-grade fever, weight loss, loss of appetite, and night sweats. There was no history of chest pain, conjunctivitis, bladder, or bowel symptoms. There was no history of alopecia, proximal myopathy, cardiac, or renal symptoms. There was no family history of rheumatic or autoimmune diseases.

Examination findings revealed a temperature of 37.2°C, heart rate of 89bpm, respiratory rate of 22 breaths/min, blood pressure of 120/80 mmHg, and oxygen saturation of 98% in room air. Respiratory examinations revealed features suggestive of consolidation in both lung fields. He also had tenderness at the knees and small joints of the hands. A preliminary consideration of tuberculous rheumatism to exclude rheumatoid arthritis was entertained.

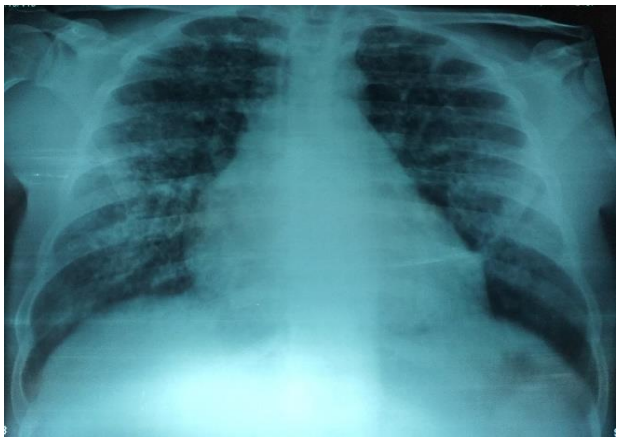

Figure 1: Chest radiograph showing bilateral inhomogeneous pin-sized nodular opacities with cystic changes involving all lung zones.

Laboratory findings included erythrocyte sedimentation rate (ESR) 106 mm/hr, c-reactive protein (CRP) 14.27mg/L (0-7.4), white blood cell count (WBC) 8.0×10^9/L (4-11), haemoglobin 14g/dL (12.0-18.5), platelets 255×10^9/L (150-450×10^9/L), serum creatinine 68 umol/L (57-113) and urinalysis was normal. Liver enzymes; aspartate transaminase; (AST) 12 IU/L (13-35) and alanine transaminase; (ALT) 24 IU/L (<35) were obtained. Serological tests for HIV, Hepatitis B/C were negative. Sputum for GeneXpert was positive for mycobacterium tuberculosis and Mantoux was 13 mm. Serum uric acid 0.28mmol/l (0.15-0.42), serum PSA 2.1 ng/ml (0-4) and total serum protein 6.5 mg/dl, albumin 3.2mg/dl, globulin 3.3mg/dl which were all normal. Joint fluid aspiration showed leucocyte count of 7000 cells/uL, no crystals, and the cultures were sterile. Rheumatoid factor (RF) 1.2 IU/mL (0-14), anti-cyclic citrullinated peptide (anti-CCP) 2.5U/ml (<5), antinuclear antibody (ANA) negative (<1:80), anti-double-stranded DNA (anti-dsDNA) 2.8IU/L (0-12). Radiograph of the chest showed bilateral inhomogeneous pin-sized nodular opacities with cystic changes involving all lung zones (Figure 1). Radiographs of the knee and hand joints were normal.

Based on the clinical findings and laboratory works, a definite diagnosis of PD was made. He was commenced on antituberculous drugs (isoniazid 300 mg daily, Rifampicin 600 mg daily, ethambutol 800 mg daily, and pyrazinamide 1.2 g daily). Three weeks into treatment, the patient showed good improvement in his articular pain and joint stiffness. At two months of treatment, the polyarthritis and stiffness had completely resolved. However, the patient is still on follow up.

DISCUSSION

PD is a rare condition that presents with polyarthritis of mainly large joints, fever, and malaise. It is slightly commoner in females and affects young individuals often. It is a reactive arthritis that is often missed and not well recognized by physicians and thus needs a high diagnostic suspicion index.[5,8] It should be differentiated from TB septic arthritis which is often monoarticular and in which Mycobacterium tuberculosis is isolated from the joint.[8]

In comparison to tuberculous septic arthritis that is monoarticular, infective and damaging, PD is a non-destructive parainfective polyarthritis that occurs in patients with active tuberculosis.[8] No evidence of bacteriological joint involvement nor any other known cause of polyarthritis has been found in PD.[9]

The duration of symptoms ranges from a few days to up to 6 years.[3] It is non-destructive aseptic polyarthritis/ oligoarthritis of mainly large joints, ankles being most common followed by knees and wrist joints. The sacroiliac joints are normally spared.[3,4] Rueda et al reported 63.3%, 58.8%, 29.1%, and 23.1% for ankles, knees, wrists, and elbows affectation respectively. Also, oligoarthritis was observed in 40 percent of patients, polyarthritis 27.6 percent, and monoarthritis 24.6 percent.[4]

PD pathogenesis remains unknown. An immunological reaction involving a hypersensitive response to tuberculoprotein and activation of CD4+ and CD8+ T cells has been considered.[5] The hypothesis that a genetic predisposition may be involved is also present. There is a strong correlation of reactive arthritis with human leukocyte antigen HLA-B27. A study by Lugo-Zamudio et al observed a significantly higher incidence in PD patients of HLA-B27 and DQB1*0301 alleles.[10] In certain patients with PD, HLA DR4+ has also been seen and DR4+ patients are hyperresponsive to mycobacterial antigens. PD may therefore result from a genetically defined hyperresponsiveness to disseminating mycobacterial antigens into joint spaces.[11,12] Immunological cross-reactivity from molecular mimicry between host tissues and mycobacterial antigen can also

play a role in the pathogenesis of PD. It has been shown that a fraction of mycobacterium tuberculosis and human joint cartilage have antigenic resemblance.[4,11]

PD primarily occurs in patients with extrapulmonary tuberculosis, and one of the hallmarks of the disease is the presence of erythema nodosum.[9,13] Other related clinical features identified include urticaria, conjunctivitis, Bazin's erythema induratum and oral ulcers.[14,15] There were however, a few studies on known sterile polyarthritis with acute lung or urogenital system tuberculosis.[9,16,17] Our case is among the few with PD presenting with acute lung TB. Our patient presented with cough, fever, and polyarthritis. The presence of Mycobacterium tuberculosis was revealed by sputum GeneXpert, and the synovial fluid examination was sterile. After effective treatment with antikochs, there is usually an improvement in the joint condition of patients with PD.[5,9,16] Our patient's symptoms completely resolved with antituberculous treatment in a short time. It is important to note that diagnosis is confirmed by complete resolution of symptoms with antituberculous therapy within weeks to months.

The PD diagnosis is mainly clinical and there are no standard criteria for diagnosis. However, in 2015 Sharma and Pinto proposed some simplified diagnostic criteria for PD based on 23 patient's characteristics in their report. (Table 1).[3]

Table 1: Sharma and Pinto's diagnostic criteria for Poncet's disease.[3]

Diagnostic criteria	
Essential criteria	Inflammatory, non-erosive, non-deforming arthritis**,** Exclusion of other causes of inflammatory arthritis
Major criteria	Concurrent diagnosis of extra-articular tuberculosis Complete response to antitubercular therapy
Minor criteria	1. Mantoux positivity 2. Associated hypersensitivity phenomenon, such as erythema nodosum, tuberculids, or phlyctenular keratoconjunctivitis 3. Absence of sacroiliac and axial involvement
For diagnosis	
Definite	Essential + two major
Probable	Essential + one major + three minor
Possible	Essential + one major + two minor, or essential + three minor

A diagnostic criteria had previously been proposed by Novaes et al, which, however, are not commonly used in routine clinical practice.[18] Our patient fulfilled the criteria for a definitive diagnosis of PD by applying the Sharma and Pinto's criteria. He had inflammatory non-erosive arthritis; had other causes in inflammatory arthritis excluded and he had a concurrent active extraarticular TB and had a full response to antituberculous drugs in keeping with the essential and major criteria for a definitive diagnosis of Sharma and Pinto's criteria.

In PD, the prognosis is usually good if the cause is treated promptly. We believe that due to the high prevalence of TB in Nigeria, PD may be more prevalent than is reported in literature. We therefore, recommend clinicians have a high diagnostic suspicion index.

CONCLUSION

In any case of active tuberculosis presenting with arthritis, PD should be considered as a differential. The diagnosis is clinical and established in a patient with TB after excluding other possible causes of arthritis. The accurate and timely diagnosis of this condition by clinicians is crucial with the aim of instituting the appropriate therapy.

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