Case Report

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

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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.

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) 8.0x10^9/L (4-11), haemoglobin 14g/dL (12.0-18.5), platelets 255x10^9/L (150-450x10^9/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. Serological tests for HIV, Hepatitis B/C were negative. Sputum for GeneXpert was positive. Serological tests for HIV, Hepatitis B/C were negative. Sputum for GeneXpert was positive. Serological tests for HIV, Hepatitis B/C were negative. Sputum for GeneXpert was positive. Serological tests for HIV, Hepatitis B/C were negative. Sputum for GeneXpert was positive. Serological tests for HIV, Hepatitis B/C were negative. Sputum for GeneXpert was positive.

<table>
<thead>
<tr>
<th>Test</th>
<th>Range</th>
<th>Value (Units)</th>
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<tbody>
<tr>
<td>ESR</td>
<td>0-10</td>
<td>106</td>
</tr>
<tr>
<td>CRP</td>
<td>0-7.4</td>
<td>14.27</td>
</tr>
<tr>
<td>WBC</td>
<td>4-11</td>
<td>8.0x10^9/L</td>
</tr>
<tr>
<td>Hb</td>
<td>12.0-18.5</td>
<td>14g/dL</td>
</tr>
<tr>
<td>Platelets</td>
<td>150-450</td>
<td>255x10^9/L</td>
</tr>
<tr>
<td>Serum Creatinine</td>
<td>57-113</td>
<td>68 umol/L</td>
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<tr>
<td>Liver Enzymes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspartate Transaminase (AST)</td>
<td>13-35</td>
<td>12 IU/L</td>
</tr>
<tr>
<td>Alanine Transaminase (ALT)</td>
<td>&lt;35</td>
<td>24 IU/L</td>
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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. No evidence of bacteriological joint involvement nor any other known cause of polyarthritis has been found in PD.

The duration of symptoms ranges from a few days to up to 6 years. 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. Rueda et al reported 63.3%, 58.8%, 29.1%, and 23.1% for ankles, knees, wrists, and elbows affection respectively. Also, oligoarthritis was observed in 40 percent of patients, polyarthritis 27.6 percent, and monoarthritis 24.6 percent.

PD pathogenesis remains unknown. An immunological reaction involving a hypersensitive response to tuberculoprotein and activation of CD4+ and CD8+ T cells has been considered. 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 DQBI*0301 alleles. 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.

**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. It should be differentiated from TB septic arthritis which is often monoarticular and in which Mycobacterium tuberculosis is isolated from the joint.

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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.4,15 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

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

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES


