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# Poncet's disease, an uncommon manifestation of a common disease: a case report

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## Abstract

Poncet's disease is a form of reactive arthritis caused by active extra-articular tuberculosis. Limited data available on this rare disease has hindered a complete understanding of it. Even though its prevalence was previously disputed, the incidence has recently been increasing, prompting the need for improved knowledge. Polyarthritis can be the only initial symptom preceding overt manifestation, and it can cause disabling pain, leading to restricted mobility and function. However, even in the context of active tuberculosis, polyarthritis of Poncet's disease can be misinterpreted as another rheumatological disease that presents similarly.

We report a 47-year-old woman, who presented with inflammatory polyarthritis in the background of recurrent sterile pyuria with obstructive uropathy which was due to active genitourinary tuberculosis. She showed a dramatic resolution of symptoms following the initiation of anti-tuberculous treatment.

We suggest that tuberculosis should be considered as a differential diagnosis for patients presenting with atypical joint pain, particularly in regions where tuberculosis is endemic.

**Key words:** Poncet's disease, tuberculous rheumatism, reactive arthritis

## Introduction

Tuberculosis caused by *Mycobacterium tuberculosis* is a significant contributor to global morbidity and mortality, with an annual death toll exceeding 1.6 million cases.[1] Poncet's disease (PD) is a form of reactive arthritis, resulting from a phenomenon of molecular mimicry between tuberculosis antigens and the host's cartilage in genetically susceptible individuals in the background of active pulmonary, extra-pulmonary, or miliary TB. This condition is characterized by symmetrical arthritis, predominantly affecting the knees and ankles, without any axial skeleton involvement. It is commonly associated with HLA-B27.[2]

Notably, the condition tends to improve after a few weeks of anti-tuberculous therapy, without any long-term sequelae. The diagnosis is typically not entertained unless the clinical manifestations lack a satisfactory explanation by any other concurrent disease processes.

We report a 47-year-old woman, who presented with multiple small and large joint polyarthritis in the background of recurrent sterile pyuria and obstructive uropathy which ultimately proved to be active genitourinary tuberculosis. She showed a dramatic resolution of symptoms following the initiation of anti-tuberculous treatment (ATT).

## Case report

A 47-year-old woman with a history of recurrent urinary tract infections over the past two years presented with bilateral small joint pain in both upper and lower limbs and pain in both wrist and ankle joints. These joint symptoms, which persisted for the last six months, were accompanied by another episode of dysuria, low-grade fever, and vomiting that lasted one week.

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She had symmetrical bilateral small joint pain, involving both proximal and distal phalanges, which worsens with immobility and in the early morning, lasting for hours. However, there were no associated morning joint stiffness, swelling, deformity, back pain, or heel pain.

Over the past two years, she has experienced recurrent urinary tract infections and has had multiple hospital admissions necessitating intravenous antibiotic treatment. However, despite urine cultures being negative in all instances, she was empirically administered intravenous antibiotics. There was no history of urolithiasis, childhood urinary tract anomalies, or gynecological abnormalities or interventions.

No discernible characteristics of extra-articular manifestations of rheumatoid arthritis such as skin nodules, pulmonary involvement, or vasculitic rashes were noted. She denies a history suggestive of skin thickening, Raynaud's phenomenon, dry mouth, proximal myopathy, or photosensitive rashes. There was no past or contact history of tuberculosis.

On examination, she appeared ill, febrile, pale, and BMI was 19.1 kg/m². Her pulse rate was 102 bpm, low volume, and blood pressure was 90/60 mmHg. Twenty-eight peripheral joints were tender encompassing bilateral metacarpophalangeal, proximal interphalangeal, distal interphalangeal, and metatarsophalangeal joints in both upper and lower limbs, and bilateral wrist, and ankle joints. Notably, there were no observable signs of joint erythema, swelling, or deformity. No extra-articular manifestations of rheumatoid arthritis. There is no evidence indicating the presence of connective tissue disorders such as SLE, scleroderma, or polymyositis. Abdominal examination revealed bilateral renal angle tenderness, but no organomegaly.

During the investigations, she was found to have a white cell count was $9.9\times10^9$ /L and anemia with a hemoglobin of 8.6 g/dL. Blood picture showed features suggestive of sepsis and anemia of chronic disease. Her urine full report showed moderately field full pus cells, occasional red cells, and significant albuminuria. Renal functions were impaired with a serum creatinine level of 4.1mg/dL (0.5-1.1) and a blood urea nitrogen level of 120mg/dL (15-40). Serum sodium and phosphate levels were within the lower limit of the normal while serum potassium was 5.1mmol/L (3.5-5.0). Corrected serum calcium and serum uric acid were 8.4mg/dL (8.5-10.2) and 5.6mg/dL (3.5-7.2) respectively. She had normal liver biochemistry with alanine transaminase (ALT) 15 U/L (<40), aspartate transaminase (AST) 11U/L (<40), alkaline phosphatase 105 U/L (< 120) except low serum albumin level which was 2.6g/dL (3.4-5.4).

The inflammatory markers including a C-reactive protein of 70mg/L and an erythrocyte sedimentation rate of 132mm/hour in the first hour were noted. She was not a known patient with diabetes mellitus and her HbA1C was 5.7%.

X-rays of both hands were normal. The chest radiograph and high-resolution computed tomography of the lungs were reported as normal. An ultrasound scan of the abdomen revealed a swollen appearance, increased echogenicity, and prominent renal pyramids of bilateral kidneys suggestive of acute kidney injury. There were prominent bilateral hydronephrosis and multiple paraaortic lymphadenopathy.

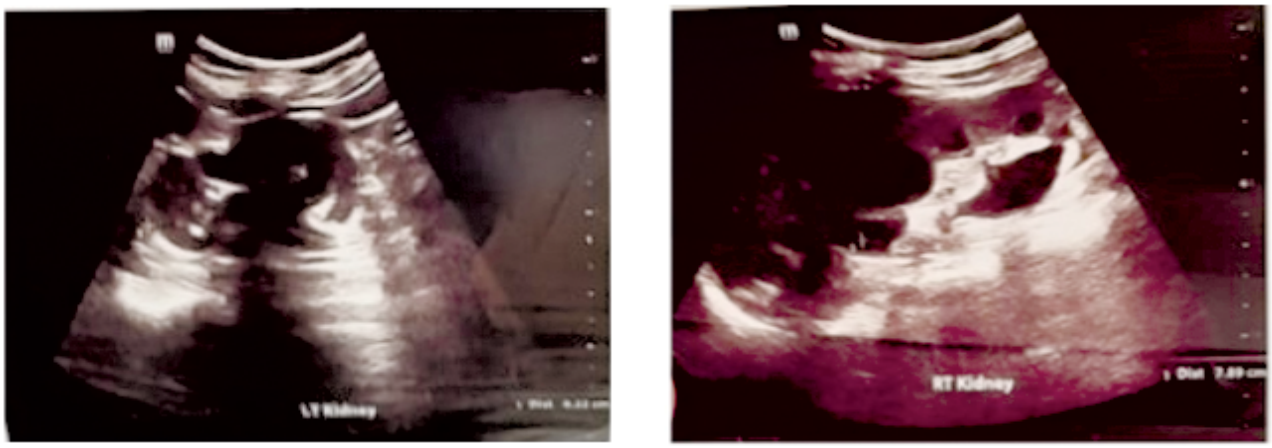


**Figure 1. Ultrasound scan of the abdomen a) Left kidney b) Right kidney. They are swollen in appearance with increased echogenicity and have prominent renal pyramids.**

Urine and blood cultures were negative. With the presence of integrated features such as recurring sterile pyuria with obstructive uropathy, persistent constitutional symptoms, and significant epidemiological impact, the diagnosis of genitourinary tuberculosis was strongly suspected. This was supported by a positive Mantoux test which showed an induration of >15mm. On the non-contrast CT scan of KUB, bilateral blunting of the calyceal system, multiple ureteric strictures with bilateral hydronephrosis, and uneven caliectasis were observed, all of which were suggestive of genitourinary tuberculosis. *Mycobacterium tuberculosis* (MTB) was identified after 06 weeks of incubation in two of her three early morning urine samples, subsequently validated by a positive result in a rapid urine culture for tuberculosis. The cystoscopy examination revealed an erythematous bladder base with contact bleeding and regions of nodularity exhibiting yellowish discoloration. Regrettably, the biopsy of the bladder wall did not yield satisfactory results.

Initially, the management of her urosepsis adhered to the guidelines set forth by the 2021 surviving sepsis campaign, involving the administration of intravenous fluids, empirical antibiotics, and inotropes.[3] Acute kidney injury necessitated the provision of hemodialysis support. Bilateral ureteric obstruction was alleviated by the insertion of a double J stent, intended to remain in place for a duration of 6 months, with periodic monitoring with ultrasonography. After the provisional diagnosis of genitourinary tuberculosis, she was commenced on renal-adjusted doses of ATT.

The intensive phase commenced by administering isoniazid, rifampicin, pyrazinamide, and ethambutol for a duration of 2 months. This was followed by the continuation phase consisting of isoniazid and rifampicin for an additional 4 months. After the initiation of ATT, the patient exhibited significant improvement in her polyarthritis symptoms, and her inflammatory marker levels decreased. In conjunction with ATT, a daily oral dose of 40 mg of prednisolone was prescribed to address bilateral ureteric obstruction resulting from tuberculous strictures. As the urosepsis and obstructive uropathy resolved, her renal function showed improvement; however, it did not fully recover and resulted in stage 2 chronic kidney disease. Upon the completion of 6 months of ATT, TB urine cultures were repeated, confirming their negativity.

During the follow-up period, she has been monitored for renal function, adverse effects of ATT, and relapse of urogenital TB. Annual urine TB rapid cultures and ultrasonography were scheduled to detect a relapse.

## Discussion

Poncet's disease (PD), also known as tuberculous rheumatism, is a relatively infrequent expression of tuberculosis infection. Acute symmetrical polyarthritis affecting both large and small joints is a characteristic feature of PD associated with active pulmonary, extra-pulmonary, or miliary TB.[2,4,5] Arthritis associated with PD typically resolves within a few weeks of ATT and does not demonstrate chronicity or articular damage. In addition, extra-articular manifestations are rare, and erythema nodosum occurs in only a small percentage of patients.[2] The exact pathogenesis of tuberculous rheumatism is currently unknown. However, it is hypothesized that the condition may be related to immune mechanisms triggered by infection with *M. tuberculosis*, potentially in conjunction with the patient's genetic susceptibility.[2,4]

The prevalence of joint involvement in patients with PD is highest in the ankles (63.3%) and knees (58.8%), followed by the carpal joints (29.1%) and elbows (23.1%), whereas joints such as the proximal interphalangeal and metacarpophalangeal joints are less frequently affected.[2] Among the PD cases reported in the medical literature, tuberculosis infection was found to be predominantly extrapulmonary in 56.8% of cases, with lymph node involvement present in 42% of patients.[2]

Our patient fulfilled most of the diagnostic criteria outlined by Rueda et al. for diagnosing PD.[2] These criteria include the presence of active extra-articular tuberculosis, rheumatic manifestations in multiple joints, absence of axial involvement, complete remission following ATT, absence of chronicity or articular sequelae, and exclusion of rheumatological diseases.[2]

She showed symptoms of inflammatory-type arthritis that affected both small and large joints, with some atypical characteristics for rheumatoid arthritis, including the absence of joint swelling, stiffness, or deformity, along with negative anti-CCP antibodies. Nevertheless, she has met the 2010 ACR/EULAR criteria by scoring 7, which includes joint involvement, symptom duration, and acute-phase protein. There was no other clinical or serological evidence for connective tissue disorders or vasculitis to account for polyarthritis. The investigation profile including double-stranded DNA antibody, P-ANCA, C-ANCA, and complement components 3 and 4 levels were found to be negative.

The diagnosis of PD can be challenging and delayed in some cases. Similar cases have been reported in the literature where the diagnosis is made by exclusion and confirmed by the response to anti-

tuberculous therapy.[4] According to studies by Rueda et al. the diagnosis of Poncet's disease can sometimes be delayed from 19 months to 12 years.[2]

## Conclusion

The diagnosis of Poncet's disease was made in our patient with atypical polyarticular rheumatism in the presence of active genitourinary tuberculosis and the marked resolution of symptoms following the initiation of anti-tubercular treatment (ATT). Furthermore, the diagnosis was reinforced by the exclusion of other connective tissue disorders and vasculitis through serological testing.

Accurate diagnosis of PD poses a challenge for clinicians; however, it should be included in the differential diagnosis for patients presenting with atypical joint symptoms, particularly in regions with high TB prevalence.

## Author declaration

### Consent for publication

The corresponding author obtained informed written consent of the patient to publish the patient's details.

### Competing interests

The authors declare that they have no conflicts of interest.

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### Criteria for authorship

All authors contributed to the conceptualization and writing of the manuscript. Management of the patient, follow-up and writing of the manuscript was done by CTWM. AHNF and NPS supervised the management of this case and contributed to writing the manuscript. All authors read and approved the final manuscript.

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