Poncet's Disease: Reactive Arthritis Due to Tuberculosis

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Abstract

Poncet's disease (PD) is a rare aseptic form of polyarthritis occurring with active tuberculosis (TB) infection. Signs and symptoms of arthritis resolve with antituberculous treatment. Clinicians, especially in the countries of high disease burden, should be aware of PD as one of the differential diagnoses, even in patients without typical symptoms of TB. Here we report a case of migratory polyarthritis diagnosed as active pulmonary TB and Poncet's disease. *(J Pediatr Inf 2016; 10: 72-5)*

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Introduction

Poncet's disease (PD), also called tuberculous rheumatism, is still not a well-known entity. There is uncertain information about its prevalence. Diagnosis can be established only after exclusion of other etiologies of polyarthritis and tuberculous arthritis itself (1).

It has been suggested that a vigorous immune response to mycobacteria, an arthritogenic microorganism, contributes to the arthritic process in PD (2). T-cell mediated cross reactivity between mycobacterial antigens and host cartilage is accepted as the main pathogenesis. Genetic predisposition seems to be the explanation of why some patients with develop PD and others do not (3).

Herein, we report a case of migratory polyarthritis diagnosed as pulmonary TB infection and PD, with a dramatic response of arthritis to antituberculosis treatment.

Case Report

A 9-year-old boy presented with arthralgia and edema for 2 months on the bilateral ankles and right elbow in a migratory pattern. He had fever for 2 days and decreased appetite without weight loss. He denied any respiratory symptoms. His medical history was unremarkable.

He was febrile on admission up to 38.2°C. His height and weight were appropriate for his age. Physical examination revealed pain and swelling of the right elbow. He had a BCG scar < 3 mm. The ophthalmological examination was normal.

Initial laboratory tests showed anemia (hemoglobin: 10 gr/dL, hematocrit: 30.5%, and

mean corpuscular volume: 74 fL), thrombocytosis (715,000/mm^3), mild leukocytosis (12,000/mm^3), elevated erythrocyte sedimentation rate (93 mm/h), C-reactive protein (156 mg/L), and antistreptolysin O (619 IU/mL) levels. Acute rheumatic fever was excluded because of non-completion of modified Jones criteria. For the differential diagnosis of rheumatologic diseases, tests such as rheumatoid factor, complement 4, antinuclear antibody, anti-double-stranded DNA, cytoplasmic antineutrophil cytoplasmic antibodies, and perinuclear antineutrophil cytoplasmic antibodies were negative. Tissue typing for human leukocyte antigen (HLA) B27 was negative. Additional laboratory tests ordered for infectious etiologies were negative for mononucleosis, toxoplasmosis, brucellosis, cytomegalovirus, parvovirus, salmonellosis, and borreliosis. Examination of bone marrow aspiration performed to exclude malignity was normal. Routinely ordered chest X-ray showed bilateral hilar lymphadenomegaly (Figure 1). Evaluation of the patient on chest computed tomography (CT) revealed multiple mediastinal and hilar lymph nodes with a maximal size of 4×2.5 cm, a primary focus on the upper lobe of right lung, and granulomas with a diameter of 1 cm on bilateral lungs (Figure 2). On abdominal CT, ordered for pathological lesions of the spleen noticed on lower sections of the chest CT, three hypodense splenic nodules and multiple mesenteric lymphadenopathies were observed. These findings on CT were interpreted as a TB infection. The tuberculin skin test was measured as 18 mm. An interferon gamma (IFN-γ) release assay, namely QuantiFERON®-TB Gold test (QFT-G, Cellestis Limited; Carnegie, Victoria, Australia), was found to be positive. Synovial fluid was inflammatory in character with a leucocyte count of 5×10^9/L and no microorganisms were seen on Gram stain. Standard culture, acid-fast staining, and tuberculosis culture of the synovial fluid yielded no pathogen. Tuberculosis culture of gastric lavage fluid was positive for *Mycobacterium tuberculosis*. Antituberculous treatment (isoniazid, rifampicin, ethambutol, and pyrazinamide) was initiated. On family screening for TB, the father had also been diagnosed as having TB. After 3 weeks of antituberculosis treatment, signs and symptoms of arthritis disappeared.

Informed consent was obtained from the patient described in this report.

Discussion

A sterile reactive arthritis that may complicate TB is a less known entity than septic arthritis and is therefore

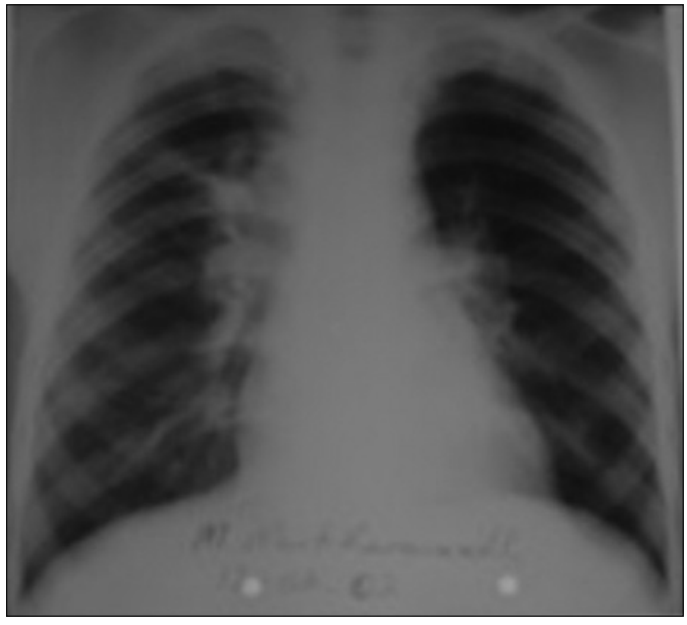

Figure 1. Chest X-ray of the patient showing bilateral hilar lymphadenomegaly

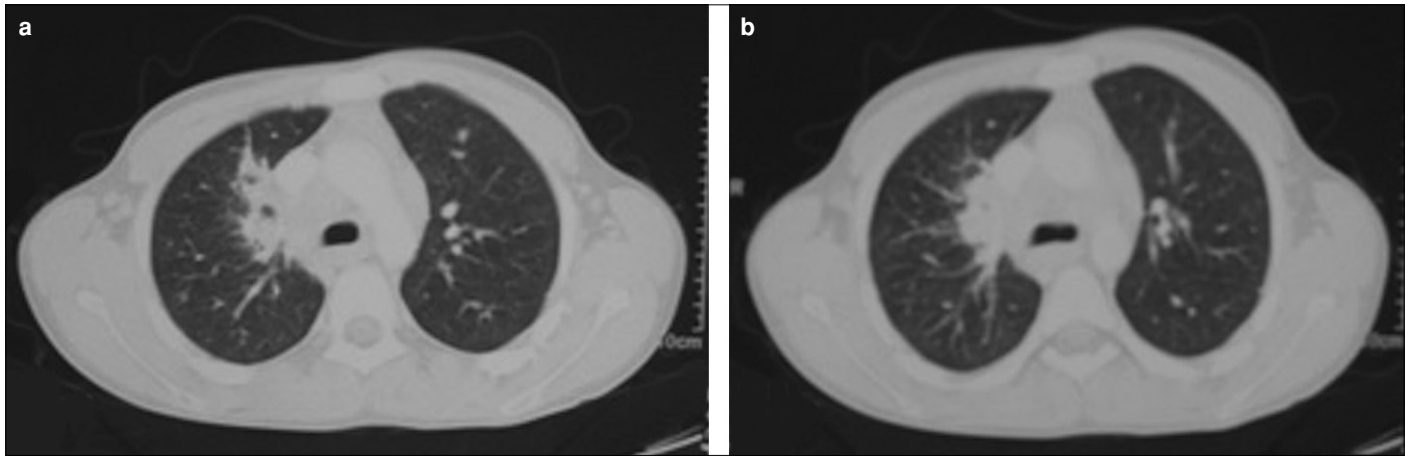

Figure 2. a, b. Primary focus extending from the upper lobe to the apical segment of the right lung, and multiple granulomas on bilateral lungs

Table 1. The main differences between tuberculous arthritis and Poncet's disease

Tuberculous septic arthritis	**Poncet's disease**
Generally chronic presentation	Generally acute or subacute presentation
Generally monoarticular	Generally poly-oligoarticular
Generally large and medium weight-bearing joints are affected.	Generally large and small joints are affected.
Sacroiliitis may occur.	Generally not associated with sacroiliitis
Synovial fluid cultures frequently yield the pathogen.	Standard cultures and cultures for tuberculosis are negative.
No association with HLA-B27 positivity.	Sometimes may be associated with HLA-B27 positivity.
Responds rather slowly to antituberculous treatment.	Generally responds to antituberculous treatment within a few weeks.
May cause joint destruction.	No residual joint destruction
HLA: Human Leukocyte Antigen	

often missed and underdiagnosed. The characteristic different features of septic arthritis and PD are shown in Table 1. Acute presentation of arthritis together with acute onset of TB is the most commonly observed presentation (4). However, in a review of cases, it is stated that six out of seven patients had developed arthritis after initiation of antituberculous treatment (5). On the other hand, arthritis appeared before presentation of TB infection in our case. Accordingly, it is proposed that PD develops in a variable pattern during the course of active TB infection.

Because PD is a diagnosis of exclusion, synovial fluid analysis, culture, or even biopsy should be obtained to prove the sterility of the joint. This is important because the prognosis of tuberculous arthritis and PD is different. However, this condition may not always be available. In a review of PD, synovial fluid culture and histology were performed for only 30% of cases. In our case, this requisite for diagnosis is met by sampling the synovial fluid of the affected joint (6).

The presented case was asymptomatic for the primary site of TB. The diagnosis of such cases is more difficult than of cases of polyarthritis with a clear primary site for TB. This has resulted in a wide range of differential diagnoses, including other rheumatologic diseases, other infectious agents causing polyarthritis, and malignancies.

Rapid resolution of arthritis is generally expected once antituberculous treatment is initiated (4, 6), which may be accepted as a therapeutic confirmation of the clinical diagnosis. Signs and symptoms of our patient were relieved in 3 weeks from the beginning of the antituberculous treatment.

It is believed that genetic predisposition plays a key role in the development of PD in patients with TB. Several studies in the literature have found the presence of some Human leukocyte antigen (HLA) alleles in patients with PD (4, 7, 8). On the other hand, in a majority of cases in the literature, the presence of HLA-B27 was not tested (5, 6). Accordingly, the association of HLA-B27 remains uncertain and controversial. We tested for HLA-B27 in a diagnostic work-up of rheumatological arthritis and it was negative in our patient.

In conclusion, we think that especially, in countries with a high prevalence of TB, diagnostic work up of arthritis should also include TB, because signs and symptoms of arthritis may develop before signs and symptoms of the primary TB infection itself, as in our case. A thorough search for the primary site of infection with adequate history, physical examination, and simple chest X-rays should be performed. This approach may prevent the initiation of immunosuppressive drugs in patients with an improper diagnosis of rheumatological disease, which would worsen the underlying TB infection.

Informed Consent: Written informed consent was obtained from the patient.

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