
Poncet's arthritis: case report

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ABSTRACT

Poncet's disease is a rare clinical condition, characterized by polyarticular impairment in a patient diagnosed with tuberculosis, with no evidence of direct bacillary invasion of the joints, constituting reactive arthritis. We report a case of a 56-year old, white male from the city of Porto Alegre, with evidence of additive polyarthritis of the large joints, investigated for five years, and with no defined diagnosis. The patient had undergone unilateral nephrectomy five years before, and the anatomicopathological exam of the specimen revealed renal tuberculosis. The current tuberculin test was strongly reactive (PPD = 20 mm). Analysis of the synovial fluid showed no direct bacillary invasion. Tuberculostatic treatment was initiated and clinical remission occurred after two months. The diagnosis of Poncet's arthritis was established.

Keywords: reactive arthritis; tuberculosis; case report.

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INTRODUCTION

Tuberculous rheumatism, also known as Poncet's disease, is a rare syndrome described in 1897 by the Frenchman A. Poncet.¹ It is characterized by articular impairment in patients diagnosed with tuberculosis, not related to direct invasion by the micro-organism, but to an immune reaction to the tuberculo-protein, constituting a reactive arthritis. Few physicians know the disease, and the literature related to that syndrome is scarce and restricted to case reports, which contributes to its underdiagnosis. This study aimed at reporting a case of Poncet's arthritis diagnosed at our hospital, and at reviewing the diagnostic and therapeutic aspects involved.

CASE REPORT

The patient is a 56-year old, white male from the city of Porto Alegre, fisherman and musician, hypertensive, and diabetic. The patient reported undergoing unilateral nephrectomy five years before, whose reason he ignored. Of the exams then performed, abdominal echography showed right kidney hydronephrosis, and DMSA scintigraphy revealed lack of

scintigraphic imaging of the right kidney, and left kidney compensatory hypertrophy with slightly deficient function (right kidney, 0.8%, and left kidney, 25.8%). The anatomopathological report of the specimen revealed chronic suppurative pyelonephritis marked by parenchymal atrophy and presence of tuberculoid granulomas with caseous necrosis. However, no tuberculostatic treatment was then initiated. The patient was admitted to our hospital complaining of articular pain and edema for five months, vespertine fever, nocturnal sweating, and weight loss of 20 kg in five years. He had been previously hospitalized because of the same articular findings, with no response to colchicine therapy. The physical exam revealed arthritis of the right knee and ankle, and left wrist. The laboratory tests showed leukocytes of 11,400 (63% of segmented neutrophils) and C-reactive protein (CRP) of 154 mg/dL. The urine qualitative test was normal. Serum uric acid was 8.8. No alteration was observed in the radiographies of the thorax, hands, wrists, ankles, lumbosacral spine, hip, and sacroiliac joints. The antinuclear antibody and rheumatoid factor were negative. Sexually-transmitted diseases were ruled out, and the anti-HIV serology was negative. The analysis of the synovial fluid of the knee evidenced

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cloudy aspect, with 21,300 cells (70% of neutrophils; 8% of lymphocytes; 20% of monocytes; 30 leukocytes per field) and no birefringent crystals. Culture of the synovial fluid could not be performed due to scarcity of material. The intradermal skin test for tuberculosis reading was 20 mm, which is considered a strongly positive test. The presumptive diagnosis of active tuberculosis was made, with no clear focus. Because of the low pre-test probability of articular tuberculosis, the clinical diagnosis of Poncet's disease was made. Treatment with rifampicin-isoniazid-pyrazinamide was initiated, but ethambutol replaced pyrazinamide due to a significant increase in serum uric acid levels. For the initial treatment of pain, prednisone was used at the dose of 30 mg for three weeks, with a gradual dose reduction for three weeks more until drug suspension. After two months of treatment, the patient reported complete resolution of pain. In a 15-month follow-up, the patient gained 10 kg of weight and remained asymptomatic.

DISCUSSION

Tuberculosis is a very prevalent disease in developing countries. Approximately 10% to 11% of the extrapulmonary tuberculosis cases affect bones and joints, corresponding to 1% to 3% of all cases of tuberculosis.

The association of tuberculosis and rheumatologic diseases has been well recognized. Franco-Paredes *et al.* have established four different categories: (1) direct musculoskeletal involvement by the *Mycobacterium tuberculosis*, such as spondylitis, osteomyelitis, septic arthritis, and tenosynovitis; (2) *M. tuberculosis* as an infectious pathogen in rheumatologic diseases, particularly with the use of new therapeutic agents; (3) antituberculous-drug-induced rheumatologic syndromes, such as tendinopathies and drug-induced lupus; and (4) reactive immune phenomena, such as reactive arthritis, erythema nodosum.

The tuberculous septic monoarthritis form, in which the mycobacterium can be isolated from the culture of the affected joint, is widely known.³ However, according to Franco-Paredes *et al.*, active tuberculosis can also be complicated with a sterile reactive arthritis, the so-called Poncet's rheumatism. It is always worth remembering and reporting that, especially now with the resurgence of tuberculosis among us and worldwide. The potential of tuberculosis, even when subclinical, to trigger reactive conditions should be remembered. That possibility becomes increasingly important as the careless use of corticosteroids, immunosuppressants or biologics can trigger the reactivation or dissemination of the disease.

The etiopathogenesis of that disease is still very controversial. The more accepted hypotheses include molecular mimicry and thermal shock proteins.⁴

In molecular mimicry, there is interaction between antigens of the infectious agent and the components of the articular tissue. The arguments confirming that theory are as follows:⁵ 1) Pearson's model of adjuvant arthritis (1963), in which a simple intradermal inoculation of Freund's adjuvant (a macerate of heat-killed *M. tuberculosis* or *Mycobacterium bovis*, emulsified in mineral oil) induces the appearance of murine arthritis 11 to 13 days after inoculation; 2) development of bone granulomas and articular lesions after intravesical instillation of BCG as an alternative therapy for urinary bladder cancer. In such cases, oligoarticular or polyarticular arthritides can occur in 3% of the individuals treated one to three months after beginning therapy.

Thermal shock proteins represent a group of proteins produced by all species in face of varied stimuli, such as heat, radiation, viral infection, and cytokines. Several pathogens, including mycobacteria, have antigens homologous to proteins, suggesting autoimmunity. Bacillary antigens share amino acid sequences with cartilage proteoglycans, producing cross reactivity, mediated by T lymphocytes, and lesion in host cells. There must be genetic predisposition, because the HLA-DR3 and HLA-DR4 genotypes show an exacerbated T-lymphocyte-mediated response to mycobacterial antigens, and can represent the expression of an immune response that also occurs with tuberculin.^{5,6}

In Poncet's disease, the oligo- or polyarticular impairment is more frequent than the monoarticular impairment, similarly to other reactive arthritides, involving mainly the large joints, such as knees, ankles, and hips, often accompanied by articular effusion. There is no microbiological evidence of the mycobacterium invasion in the affected joint,⁷ the serological tests for autoimmunity are negative, and the tuberculin test, as well as acute phase proteins, are altered.⁸

Gilberto Santos Novaes⁹ has proposed the following diagnostic criteria after assessing a series of 25 patients with Poncet's disease: a) evidence of active extra-articular tuberculosis; b) rheumatic manifestations in more than one joint; c) absence of personal and family antecedents; d) lack of axial, vertebral column and sacroiliac impairment; e) unspecific laboratory findings; f) complete remission of the rheumatic manifestations with antituberculous chemotherapy, and no permanent articular sequelae; g) exclusion of other rheumatic diseases.

It is worth noting that, with modern diagnostic methods for tuberculosis infection, Poncet's reactive arthritis might prove to be more prevalent than supposed. Currently, two diagnostic

methods are used: CRP in sterile samples, such as synovial fluid; and interferon gamma release assays. Valleala *et al.*¹⁰ have reported a case of tuberculosis diagnosed by use of that method, with confirmation through therapeutic response.

Our patient underwent radiographies of the chest and affected joints, which showed no alterations characteristic of tuberculosis. In articular tuberculosis, the radiography usually shows enlargement of the articular space, articular effusion and *pannus*. Shortly, cartilage destruction and subchondral bone erosion occur.

Our patient had a history of gout and mildly elevated uric acid, which initially confounded and delayed the diagnosis. However, gouty arthritis usually has monoarticular impairment, the first toe being the most frequently affected site.

Gouty arthritis tends to occur in crises and go into remission with specific treatment with colchicine and anti-inflammatory drugs, which did not happen with our patient. In addition, the patient had had renal tuberculosis five years before, and had not been treated. The current tuberculin test reading of 20 mm is considered strongly positive. Unfortunately, our service does not count on modern diagnostic methods to search for the mycobacterium, and culture of the synovial fluid could not be performed due to scarcity of material. However, with the data available, the major hypothesis considered was Poncet's arthritis. The early identification of that condition is important so that the specific treatment can be instituted, because arthritis is resolved completely with tuberculostatic therapy in a few weeks.

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