PONCET’S DISEASE:
A REPORT OF FOUR CASES AND REVIEW OF
THE LITERATURE

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ABSTRACT

Four cases of polyarthritis concomitant with active tuberculosis is reported. In three patients pulmonary tuberculosis was confirmed by identification of Mycobacterium tuberculosis in the sputum and bronchoalveolar lavage specimens, and in another one tuberculous lymphadenitis was confirmed by excisional biopsy. In all patients arthritis resolved by tuberculosis treatment and did not recur during a follow up period of 18 months to 12 years. The findings of the presented cases are compatible with Poncet’s disease (tuberculous reactive arthritis).

INTRODUCTION

Poncet’s disease (PD) is described as a polyarticular arthritis which occurs during active visceral tuberculosis (TB) infection. Despite the many case reports the concept of PD is not accepted as a specific disease. The following four cases of reactive arthritis (ReA) associated with Mycobacterium tuberculosis (MtB) infection present additional information in supporting the existence of PD. These patients attended or referred to Shaheed Beheshti Hospital in Babol.

Case 1

An 18-year-old girl presented in September 1980 with arthritis in her ankles, right knee, left elbow and right wrist of three month’s duration. In physical examination the knee joint was swollen and tender, the ankle joints were also tender and slightly swollen, and the wrist and elbow joints were painful in palpation and movement. The remainder of the examination was normal. A complete blood count (CBC) and routine laboratory tests including the latex agglutination test for rheumatoid factor (RF) and antinuclear antibody by immunofluorescence (ANA) were negative. The erythrocyte sedimentation rate (ESR) was 39 mm/hour, and C-reactive protein (CRP) was positive. The radiographs of the wrist and knee joints were unremarkable. Ibuprofen was administered for symptomatic treatment but she did not respond to full doses of ibuprofen during the subsequent visits. Two months later physical examination revealed enlarged cervical lymph nodes which were excised and showed tuberculous lymphadenitis. One month after anti-TB drug therapy the arthritis resolved and did not relapse up to 12 years after discontinuation of the drugs.

Case 2

A 14-year-old girl presented with acute polyarthritis involving the right hip and knee joints, left ankles and bilateral elbow joints, and the left wrist for three days in 1988. Over two weeks of observation, the arthritis showed a migratory pattern similar to rheumatic fever. Physical examination showed tenderness, swelling and effusion in the knee joints, movement of the hip joint was very painful and severely limited, and the other joints were also tender and
swollen. CBC showed a mild leukocytosis, the ESR was 59 mm/h, the CRP was positive, the antistreptolysin O (ASO) titer was 333 Todd units, ANA and RF were negative, and other laboratory tests were unremarkable. Administration of 100 mg/kg aspirin for two weeks was not effective and other non-steroidal anti-inflammatory drugs (NSAIDs) also had limited benefits. A diagnosis of juvenile chronic arthritis was established and during the next six years, arthritis was partially controlled by chloroquine phosphate or sulphasalazine and NSAIDs. During the follow up period, episodes of partial remission and exacerbation were seen. In July 1994, joint symptoms progressed and severe polyarthriti...
was excluded and PD was confirmed.

A rheumatoid arthritis-like presentation with symmetric involvement of small finger joints accompanying bacteriologically confirmed TB has been reported by Dlugovitzky et al. Three out of the five patients remained arthritic by the time of bacteriologic conversion and fulfilled the criteria for RA. In the two remaining patients sputum negativity was accompanied by disappearance of rheumatic manifestations in favour of PD.

PD is an entity characterized by ReA developing in the presence of active TB elsewhere. Any joint can be affected, however the knees, ankles and elbows are involved more often than small joints of the hands and feet. ReA is mediated by an immune response against whole bacteria or their fragments, which is carried to the joint. The responsible antigen may be HLA-B27 complexed with peptides derived from proteins of arthritis causing bacteria such as heat shock protein (hsp) family. The hsp, a ubiquitous protein in a wide range of species from bacteria to mammals, is strongly immunogenic and can induce autoimmune disease. Mycobacterium tuberculosis (Mtib) antigen in adjuvant arthritis is an hsp which can cross react with cartilage proteoglycan and induce autoimmunity and result in cytokine secretion by CD4 lymphocytes which has been implicated in the pathogenesis of autoimmune arthritis. Intra-articular administration of recombinant Mtib hsp can induce joint inflammation in Mtib sensitized recipients.

In conclusion, the immune response against Mtib hsp in the genetically predisposed host may explain the mechanism of ReA in PD. The pathogenic similarity of PD and erythema nodosum which is applicable clinically to the third case of the present study has been demonstrated in a case report. Further investigation is required to elucidate the relationship between active TB and ReA.

REFERENCES
