Case Reports

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).


**Keywords:** Reactive arthritis, Tuberculosis, Poncet’s disease.

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. 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.
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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 arthri-
tis 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 poly-
arthritis involving bilateral proximal interphalangeal (PIP),
metacarpophalangeal (MCP) and metatarsophalangeal
(MTP) joints, elbow and knee joints developed. The right
hip and both sacroiliac joints were also affected. In physical
examination hand joints were severely distended with effu-
sion in most PIP joints. The knees and elbows were tender
and swollen, movement of the right hip was painful and lim-
lit and sacroiliac joints were tender, aspiration of the knee
joints revealed inflammatory fluid which was negative in
culture. The ESR was 76 mm/h, CRP was ++++, ANA and
RF were negative, radiographs of the knees and hands showed soft tissue swelling and radiographs of the pelvis showed normal sacroiliac joint appearance but degenera-
tive changes in the right hip with severe joint space narrowing
and subchondral bone sclerosis in the femoral head.

Oral methotrexate 7.5 mg weekly was added. During the
next six weeks, joint signs subsided but cough and dyspnea
appeared. Chest auscultation revealed wheezes and rhon-
chi, and the chest radiograph revealed pulmonary parenchym-
al infiltration. Ziehl Neelson staining of the sputum re-
vealed Mtb.

Antirheumatic drugs were discontinued and anti-TB
therapy was substituted. Joint symptoms and signs subsided
completely after six weeks and did not recur up to five years
after treatment of TB.

Case 3

In March 1995, a 33-year-old woman who had been
well previously presented with arthritis of the left ankle, left
shoulder and right wrist, pain in the right forearm, and the
appearance of erythema nodosum on the anterior surfaces
of the tibia bilaterally over a two weeks period. In physical
examination the ankle joint was severely tender and slightly
swollen. There were several tender, red and erythematous
nodules on the tibial surfaces and the results of clinical ex-
amination in the remainder of the musculoskeletal system
were unremarkable. Routine blood tests including CBC, RF,
ASO, ANA and CRP were negative. The ESR was 19 mm/
h, and the chest radiograph showed left hilar adenopathy.

Pulmonary and mediastinal CT scanning revealed multiple
bilateral adenopathies in both hilar and para-aortic areas.
The specimen obtained by fiberoptic bronchoscopic alveo-
lar lavage revealed Mtb on smears and culture. Arthritis sub-
sided after six weeks of anti-TB treatment and did not re-
lapse up to three years later.

Case 4

A 49-year-old man was admitted in January 1997 with
fever, weakness, arthralgia and arthritis. The temperature
ranged between 37.5-39°C over two weeks of hospitaliza-
tion. There was tenderness and slight swelling in both ankles
and mild localized edema on the dorsal regions of the tarsal
joints bilaterally and moderate difficulty in walking. The
knee joints were slightly tender. Blood count, urine culture,
urinalysis, blood cultures and liver function test were nor-
mal. RF, ANA, ASO, Coombs wright agglutination and PPD
test were negative. Other febrile illnesses were ruled out by
appropriate tests. Chest radiograph and CT scanning revealed
bilateral hilar adenopathies. Fiberoptic bronchoscopic study
showed no endobronchial lesion. The specimen obtained by
bronchoalveolar lavage and the sputum staining and cul-
tures revealed Mtb. Fever and joint pain subsided two weeks
later without any treatment, but swelling and edema disap-
peared after two months of anti-TB therapy. The joint signs
did not recur over an 18 month follow up period.

DISCUSSION

Four cases of simultaneous ReA and active TB infec-
tion are reported here. In three patients pulmonary TB was
confirmed by bacteriologic studies and in the fourth case,
TB lymphadenitis was established by histologic examina-
tion of the lymph node. The diagnosis of the presented cases,
regarding the pattern of joint involvement affecting predomi-
nantly the large joints of the lower limbs and the rapid re-
sponse of arthritis to antituberculous therapy is consistent
with tuberculous ReA (PD). The diagnosis of PD remains
clinical and is established on excluding other potential causes
of arthritis in a patient with active TB.3 The complete reso-
lution of arthritis symptoms on anti-TB therapy also furn-
ishes further proof of the diagnosis.

Involvement of the musculoskeletal system, such as
spondylitis, osteomyelitis or arthritis occur in only 2% of
patients with TB19 but should be considered in any patient
with rheumatic symptoms. Unlike some reported cases2,4,5
with direct involvement of bone by Mtb, the presented cases
show no evidence of direct skeletal involvement. The time
from the onset of arthritis to diagnosis of TB in this study
ranged from two weeks to six years. In other reported cases
disease duration ranged from two weeks up to one
year,3,5,7,11,12,15 but cases with a prolonged disease course
similar to our second patient have not been reported up to
now. The clinical picture of this patient in particular when
she presented with symmetric polyarthritis was indistinguish-
able from rheumatoid arthritis (RA); however, with regard
to the rapid response of arthritis to anti-TB treatment, RA

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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 negativization 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 (Mt) 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 Mt hsp can induce joint inflammation in Mt sensitized recipients.

In conclusion, the immune response against Mt 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