Bilharzial arthropathy

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SUMMARY The joints of 124 young adult patients with bilharziasis were examined. Heels, sacroiliac joints, cervical spine, knee joint, dorsal spine, and tarsal joints were affected. Biopsy of the knee synovia showed synovitis, vasculitis, and the presence of bilharzial ova in three cases. Radiologically the heels and sacroiliac joints showed inflammatory changes. Schistosomiasis may cause an arthropathy.

Key words: infectious arthritis, schistosomiasis complications.

Bilharziasis is an endemic parasitic disease in many countries, which affects the urinary tract and the intestine. The main lesion is the granulomatous reaction that occurs around the living eggs.¹

There is a positive correlation between egg output and severity of disease.¹ Immunological studies showed that the various stages of the parasite within the vertebrate host are all potentially antigenic.¹ These antigenic substances are known to produce humoral and cellular responses. This work continues the study which was done by one of us to elucidate the joint status associated with this disease.³

Patients and methods

One hundred and twenty-four young adult male patients were seen all infected with bilharziasis and suffering from joint pains. Their age ranged from 10 to 30 years. The average duration of bilharzial infection was nine years (ranging from 12 months to 25 years). Routine analyses of stools, urine, egg count, blood picture, rheumatoid factor, ESR, and synovial fluid were done. Synovial biopsy by both the arthroscope and open surgery was done in these patients with high egg excretion in urine (> 50 eggs/ml urine, 11 patients). Radiological studies of the joints were carried out.

Results

CLINICAL FINDINGS

Low backache and painful knees were the main

complaints of the patients (Table 1). The large joints of the lower limb were involved. The joint pain or tenderness existed for periods ranging from a few days to several years. There was no morning hand stiffness, no effusion, heat, deformity, or loss of joint function. Mild synovial thickening was found in the knee joints of 22 patients. Joints were affected as shown in Table 2. The ESR was moderately raised: mean value 14.75 mm/h (Westergren).

Rheumatoid factor was negative in 120 patients and positive in four (3%). Microscopic examination of urine showed the presence of pus cells <8 high power field (HPF) in 33 patients (26.60%), > 8/HPF in 28 patients (22.6%); one patient had pus cells > 50/HPF.

Table 1 *Pattern of joint complaints*

<i>Joint complaint</i>	<i>Percentage</i>
Low back pain	44
Referred pain to thighs, legs	20
Pain in knees	32
Other joints	12
Pain in heels	10

Table 2 *Frequency of joint affection in bilharzial patients*

<i>Joints</i>	<i>Percentage</i>
Heels (52 patients)	41
Sacroiliac joints (52 patients)	41
Cervical spine	30
Knees	27
Dorsal spine	21
Tarsal joints	4

Accepted for publication 5 July 1984.

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The x-ray appearances of cervical and dorsal spine, knee, hands, and feet were within normal limits. However, sacroiliac and heel x-rays showed pathological changes of an inflammatory character.

The heels showed plantar fascitis and/or Achilles' tendinitis (26 patients), calcaneal spurs and periosteal reaction (26 patients). The sacroiliac joints showed loss of articular margins, erosion, and

Fig. 1 A section of synovial biopsy of the knee shows bilharzial ovum with fresh miracidium, its nucleus stained red (top right, arrow), pointed spine, and surrounded by inflammatory cells. The section also shows high vascularity with infiltration of inflammatory cells. (Haematoxylin and eosin, $\times 50$).

Fig. 2 High-power view of synovial section of bilharzial ovum, shows blood vessel and bilharzial ovum with refractile shell. There is loss of nuclear detail of muscular wall of blood vessels, which is an indication of early signs of vasculitis. (Masson trichrome stain, $\times 250$).

Fig. 3 No bilharzial ovum in this section, but blood vessel vasculitis, hypertrophy of the endothelial cells, and infiltration of inflammatory cells in blood vessel walls, lumen, and areolar connective tissue are seen. H and E, $\times 187$.

widening. Sclerosis of the sacral and iliac portions was increased on both sides in 52 patients, representing 41% of patients.

PATHOLOGICAL FINDINGS

Bilharzial ova were identified in the synovial biopsies of the knee joint in three patients. In one patient a fresh haematobium ovum with its nucleus of the miracidium stained red and surrounded by inflammatory cells was identified in serial sections stained with H and E (Fig. 1). This patient had had bilharziasis for 15 years; the egg excretion count was 100 eggs/ml in urine. In another patient, a male aged 27 years, the biopsy showed a bilharzial ovum with a refractile surface close to a blood vessel, which was hypertrophied, oedematous, and infiltrated with inflammatory cells. The patient had suffered from bilharziasis for 16 years (Fig. 2).

Other synovial biopsies showed reactive synovitis or normal synovium. The main pathological changes were (Fig. 3): increased vascularity with infiltration of inflammatory cells, mainly lymphocytes and plasma cells, with thickening of the walls of arterioles and hypertrophy of endothelial cells; infiltration of inflammatory cells of the loose areolar connective tissue of the synovium with deposition of fibrinous material.

Discussion

This appears to be the first finding of bilharzial ova in the synovium of the knee joint of a patient suffering from bilharziasis—convincing evidence that bilharziasis can be a cause of arthropathy. The

pathological changes of synovitis and vasculitis with or without the presence of bilharzial ova were found in synovial biopsies which were taken from the knee. The x-ray changes of the heels and sacroiliac joints were of diagnostic value.

Some other investigators⁴⁻⁸ have reported reactive arthritis after parasitic infestation. The pathogenesis was not known. Persistence of parasites (the mean duration of infection in our patients was 9 years) and capacity to release large quantities of antigens may be responsible for a variety of immunological reactions.

An individual predisposition also might explain the occurrence of complications in some people despite a high prevalence rate among certain populations.⁶ The secondary invasion of bacteria and bacterial toxins that are usually associated with bilharziasis may also play a role.

Bassiouni³ first suggested that bilharzial arthropathy may mimic the clinical picture of reactive arthropathy.

The present study has shown that the joints of the lower limb were particularly affected. A history of multiple attacks of arthritis without residual deformity but with calcaneal spurs was sometimes found. These spurs were of an inflammatory character, like those of Reiter's syndrome.⁹

Sacroiliac changes occurred in 41% of patients and were similar to the sacroiliac changes described by Forrester *et al.*¹⁰ The changes were asymmetrical, with irregular widening of joint space, irregular sclerosis present bilaterally but more extensive on one or other side, and rarely bony ankylosis. The duration of arthritis was variable, lasting from a few days to three years.

Finally, we propose clinical criteria for the diagnosis of bilharzial arthropathy: the identification of bilharzial ova or granulomatous changes in any organ of the human body after exposure to bilharzial infection; involvement of one or more large joints of an inflammatory character without effusion, deformity, or loss of joint function; histopathological evidence of reactive synovitis, vasculitis with or without the presence of bilharzial ova in the synovial biopsy of a joint; negative RF, slightly or moderately raised ESR, and pus cells in urine; reactive inflammatory x-ray changes in the heels or sacroiliac joints.

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Book review

Clinics in Rheumatic Diseases Vol. 8. No. 1. Systemic Lupus Erythematosus. Edited by G. R. V. Hughes. Pp. 323. £10.75. Saunders. London. 1982.

It is seven years since an issue on systemic lupus erythematosus appeared in the *Clinics in Rheumatic Diseases*, and the present volume is a welcome survey on the recent advances in the understanding of this disease. The volume may be arbitrarily divided into three sections - basic immunology, major system involvement, and the treatment of SLE. The greatest advances are evident in the first section, which includes reviews on the immunogenetics, cellular and humoral immunity, complement deficiency states in SLE, and the modulation of immune responses by sex hormones. There follow chapters on renal, central nervous system, skin, cardiac and intrathoracic manifestations of SLE, together with clinically orientated reviews of antinuclear antibody (ANA)-negative, drug induced, and juvenile SLE. The last section discusses the use of plasmapheresis, pulse methylprednisolone, and antimalarial therapy, concluding with a chapter by the editor reviewing the management of SLE. He contrasts the benign prognosis for most of these patients with the

adverse effects of treatment, particularly corticosteroids, and pleads for a conservative therapeutic approach.

Each of the authors is both a clinician and a scientist, and this balance is evident throughout most of the book. The better chapters in the section on basic immunology open with an explanation of current understanding of the normal immune mechanisms before discussing the immunopathology, and the best chapters end with a summary. It is a pity not all these chapters were arranged this way. The material is comprehensible to those unfamiliar with current knowledge and may be rapidly reviewed by those not wishing to read details. The clinical section is equally well balanced, with a notable contribution by Dr Maddison on ANA-negative SLE.

Apart from the concluding chapter those dealing with treatment did not warrant the space they took. Admittedly the authors discussing the use of antimalarials, pulse methylprednisolone, and plasmapheresis were hampered by the absence of conclusive data, but there is little value in a detailed review of uncontrolled or anecdotal material. Nevertheless, all in all this is an excellent volume, the best brief review available on current knowledge of SLE.

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