Acute tropical polyarthritis in Zimbabwe: a prospective search for a gonococcal aetiology

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SUMMARY Fifteen consecutive patients with acute arthritis were studied. Careful culture and microscopy of swabs from mucosal sites were performed to examine the hypothesis that the acute tropical polyarthritis commonly seen in Zimbabwe is due to undiagnosed gonococcal infection. Rheumatic fever and Reiter’s disease accounted for two cases each. Gram negative intracellular diplococci were found in three patients who would otherwise have been diagnosed as having tropical polyarthritis. Except for two of the remaining eight patients, who had raised antistreptolysin O titres suggesting recent streptococcal infection, no explanation for the arthritis was found. The clinical entity of tropical polyarthritis may not be due to a single aetiological agent.

In Zimbabwe the most common acute arthritis necessitating admission to a medical ward is a disorder known as acute non-specific arthritis, acute non-specific polyarthritis, or acute tropical polyarthritis. The aetiology of this condition is unknown, but an infective cause is suspected. In the United States, and to a lesser extent Britain, Neisseria gonorrhoeae is one of the more common causes of infectious arthritis in young adults. Urogenital gonococcal infection is common in Zimbabwe. We therefore investigated patients with acute arthritis to examine the hypothesis that tropical polyarthritis is a manifestation of unrecognised infection with N gonorrhoeae.

Patients and methods

All patients admitted to the medical wards of Parirenyatwa or Harare hospitals in Harare, Zimbabwe during the period July 1983 to February 1984 with arthritis of less than two weeks’ duration and who had not received antibiotics were studied. One of the authors saw each patient, took a careful history, and performed a detailed physical examination. A full blood count, erythrocyte sedimentation rate, antistreptolysin O (ASO) titre, Venereal Disease Research Laboratory (VDRL) test, brucella agglutination titre, rheumatoid arthritis (RA) latex test, and blood cultures were performed.

Swabs were taken from the throat, urethra (men), endocervix (women), and rectum. Gram stains of these were performed and each specimen was immediately plated onto blood agar and Thayer Martin media which were incubated at 37°C in a CO₂ enriched environment using candle extinction jars. Synovial fluid was aspirated whenever possible and Gram stains and cultures performed in the same way. Microscopy was performed by both authors and by technicians of the microbiology laboratory service. Diagnosis of a particular rheumatic condition was made in patients who fulfilled accepted criteria.

Results

Fifteen consecutive patients with acute arthritis were studied. The typical triad constituting Reiter’s syndrome was present in two patients, and two patients fulfilled criteria for the diagnosis of rheumatic fever. These four patients are not discussed further. The remaining 11 patients were thought to have tropical polyarthritis. Gram negative intracellular diplococci were identified in specimens from three patients whose case summaries follow. Synovial fluid and blood cultures yielded no growth, and none of these three patients had a tenosynovitis.

CASE 1
A 22 year old woman had arthritis involving both knees, the right elbow, right ankle, and clinical evidence of pelvic inflammatory disease. A maculopapular evanescent purplish rash was visible on the
inner aspect of the thighs. Gram negative intracellular diplococci were cultured from the endocervical swab. A rapid response to parenteral penicillin and oral anti-inflammatory drugs was noted.

**CASE 2**

A 52 year old man had a week’s history of a urethral discharge followed by four days of acute arthritis involving the right shoulder and knee and the left ankle. Gram negative intracellular diplococci were seen in both the knee aspirate and the urethral smear, but the organisms failed to culture. Response to parenteral penicillin and oral anti-inflammatory drugs was slow with satisfactory resolution of symptoms only occurring after two weeks.

**CASE 3**

A 28 year old man with no genitourinary symptoms had arthritis affecting both wrists, knees, and several metacarpophalangeal joints of both hands. Gram negative intracellular diplococci were seen and cultured from the urethral swab. A rapid response to parenteral penicillin and oral anti-inflammatory drugs was noted.

**OTHER CASES**

In the remaining eight cases no firm diagnosis other than tropical polyarthritis was made. There were five men and three women with ages ranging from 19 to 52 years (mean 30 years). Two of these patients volunteered symptoms suggestive of preceding viral upper respiratory tract infection, otherwise arthritis had been the first symptom noted. A history of preceding urethritis, conjunctivitis, dysentery, or rash was specifically sought. This was invariably negative. Apart from one patient with clinical evidence of pelvic inflammatory disease and an urticarial rash and another with plantar fascitis, there was no clinical evidence of urogenital infection, conjunctivitis, enthesitis, or rash.

The number of joints affected ranged from two to more than 10. The joints most commonly involved were knee (100%), ankle (50%), and wrist (50%). The small joints of the hand (25%) and hip (25%) were less commonly affected. Six of the eight patients were pyrexial, often with temperatures of 38–39°C, and seven patients were treated with antibiotics, most commonly penicillin, as well as aspirin. The acute symptoms and fever usually settled within 72 hours and follow up records were available for six patients. The arthritis had completely resolved in two weeks (one patient), one month (one patient), two months (three patients), and six months (one patient).

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A full blood count and erythrocyte sedimentation rate (ESR) determination were performed in all eight patients. The white cell count ranged from 6·5 to 11·7 × 10⁹/l and the ESR (Westergren) from 3 to 91 mm/1st h, with a mean of 61 mm/1st h. The VDRL test was negative in all eight patients. The ASO titre was estimated in seven patients, and in two patients titres of 800 and 1600 units suggested recent streptococcal infection. One of these patients had a history of recent upper respiratory tract symptoms, but neither had any accompanying features of rheumatic fever. The RA latex test was performed in 6/8 patients, the brucella agglutination titre in 6/8, and antinuclear factor in 4/8; the results were all negative. Blood cultures (8/8) and synovial fluid cultures (6/8) did not yield any growth.

**Discussion**

An illness similar to the tropical polyarthritis reported in Zimbabwe has been reported from Malawi, Uganda, Kenya, Nigeria, and Papua New Guinea. The clinical definition and aetiology of this illness are still ill defined. Prospective culture of joint fluid for *N gonorrhoeae* was performed by Jeremy and colleagues without success. It has since been noted, however, that cultures of synovial fluid are often negative in patients with gonococcal arthritis, and a diligent search for evidence of mucosal infection with *N gonorrhoeae* is a more important aid to diagnosis.

We found bacteriological evidence of mucosal infection by Gram negative intracellular diplococci in only three of the patients initially thought to have tropical polyarthritis. Two of the three had clinical evidence of genitourinary infection and an urticarial rash. The distribution of arthritis may be of some help in differentiating the two conditions. The knee joint is the joint most commonly involved in both conditions, but involvement of the small joints of the hand is common in gonococcal arthritis and relatively uncommon in tropical polyarthritis. In our study involvement of the small joints of the hand was not a useful discriminating feature. The ESR is often markedly raised in gonococcal arthritis, but this is often also a feature of tropical polyarthritis.

Prompt response to antibiotics has been used as a criterion for the diagnosis of gonococcal arthritis. As most patients in our study received both antibiotics and anti-inflammatory drugs, and as both conditions may be self limiting it is difficult to evaluate response to antibiotics as a useful differentiating feature.

An interesting finding was a high ASO titre in two patients with tropical polyarthritis. Riley also noted this in four of his patients. McDanald and Weisman
have suggested that adults may develop arthritis without the carditis of rheumatic fever after streptococcal infection.17

Although tropical polyarthritis may be a reactive arthritis, the condition may not be as homogeneous as previously supposed. Unrecognised gonococcal and perhaps streptococcal infection may account for some cases, but in most the aetiology is still undetermined. Although tropical polyarthritis still exists as an ill defined clinical entity and further detailed investigation is likely to show several causes of a clinically similar picture, the unusual prevalence of this condition in certain geographic areas remains puzzling.

**References**

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