**Case report**

Coexistence of Reiter’s syndrome and rheumatoid arthritis in a genetically susceptible individual

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**SUMMARY** A patient is presented who had features of Reiter’s syndrome for 10 years before developing features of rheumatoid arthritis. Diagnostic criteria for both diseases are fulfilled, and HLA typing revealed the presence of both B27 and DR4 antigens. The coexistence of Reiter’s syndrome and rheumatoid arthritis appears to have occurred in an individual genetically susceptible to both diseases.

The coexistence of Reiter’s syndrome (RS) and rheumatoid arthritis (RA) has been reported infrequently.1–4 Only one report5 gives information on complete histocompatibility antigen typing. We present an additional such case with complete tissue typing information showing 2 distinct susceptibility genes, one known to be associated with Reiter’s syndrome and the other with rheumatoid arthritis.

**Case report**

A 32-year-old white male developed nonspecific urethritis in 1969 and was treated with tetracycline for 2 weeks. Two weeks after cessation of antibiotics he developed a recurrence of urethritis associated with conjunctivitis, ankle synovitis, and dactylitis of the third right toe. Tetracycline was reinstituted along with nonsteroidal anti-inflammatory drugs, with slight improvement. Eighteen months later he developed low back pain, with flattening of the normal curvature of the lumbar spine, and continued to have dysuria, bilateral conjunctivitis, and pauciart-ritis. Laboratory tests including serum uric acid, latex fixation test for rheumatoid factor (RF), and urine analysis were within normal limits. Synovial fluid obtained by arthrocentesis did not show crystals. He responded poorly to salicylate therapy but improved significantly with phenylbutazone.

Four months later ankle synovitis, heel pain, dysuria, conjunctivitis, and low back pain recurred. Physical examination was unchanged except for the presence of a tender prostate. Pertinent laboratory findings included a negative RF titre and sterile pyuria. Roentgenography of the feet revealed joint space narrowing of the right third metatarsophalangeal joint without erosion or periostitis. The sacroiliac (SI) joints were normal on anteroposterior radiography of the pelvis. His course over the next 10 years was characterised by recurrent low back pain, metatarsalgia, talalgia, asymmetrical pauciartitis involving the ankles and toes, nonspecific urethritis, conjunctivitis, and superficial painless mucosal ulcerations of the mouth and glans penis. Repeated determinations of RF and antinuclear antibody (ANA) gave negative results. Radiographs subsequently revealed evidence of erosion of the calcaneous superiorly at the insertion of the Achilles tendon and periostitis inferiorly at the insertion of the plantar fascia (Fig. 1). Unilateral sacroiliitis of the left SI joint was found.

In the autumn of 1980 the patient complained of bilateral shoulder pain for the first time. Serology now disclosed a positive RF titre by latex fixation of 1:1280 and a negative ANA. Six months later he presented with a 3-month history of an additive symmetrical polysynovitis, prolonged morning stiffness, and midday fatigue. On examination active symmetrical synovitis of shoulders, elbows, wrists, knees, ankles, and small joints of the hands and feet was found as well as decreased motion of the neck.
and flexion contractures of both elbows. Rheumatoid nodules were not identified, but RF titre remained positive at 1:5120. Tissue typing by the conventional microlymphocytotoxicity method for antigens A, B, C, and DR B cell alloantigen gave positive results for these HLA antigens: A24, A31, B27, B60, CW2, DR4. The other C and DR antigens could not be typed. The joint fluid had very poor mucin clot and was negative for crystals. At the time of this report the patient has experienced significant improvement after receiving 1 g of chrysotherapy. The improvement is shown by an increased sense of well-being, a decrease in severity and length of morning stiffness, and a decrease in degree of synovitis. Talalgia and metatarsalgia have failed to respond and remain a problem.

Discussion

Defining Reiter's syndrome as an episode of peripheral arthritis of more than 1 month's duration occurring in association with urethritis and/or cervicitis has been recently shown to yield a high degree of sensitivity (84.3%) and specificity (98.2%). Our patient had over a period of 10 years recurrent episodes which fulfilled these criteria. In addition he had other symptoms and signs such as conjunctivitis, heel pain, back pain, dactylitis, and mucosal ulcerations, which together constitute a typical pattern for RS. Erosion at the posterior margin of the calcaneous in association with a plantar calcaneal spur is not specific but is suggestive of RS.

Eleven years after the initial symptoms a new pattern of joint involvement, symmetrical upper extremity polyarthritis, emerged in this patient. Prolonged morning stiffness and fatigue were new complaints. He became seropositive for RF and had synovial fluid findings compatible with RA. Response of the RA features but not the RS components to chrysotherapy is another indication of the coexistence of both RS and RA in this patient.

The associations of HLA B27 antigen with RS and HLA DR4 antigen with RA have been established. There are only a few reports describing the coexistence of RS and RA, and among them only one describes the complete HLA antigen typing. In that prospective study of 184 patients with ankylosing spondylitis and/or Reiter's disease Alexander et al. reported 5 men with concomitant RA; 2 had RS and both were positive for B27 and DR4 antigen. In our patient both B27 and DR4 antigens are present, and both related diseases are expressed. Although these
susceptibility genes are not considered to be the cause of these diseases but only predisposing factors, cases such as this emphasise the need for the continued search for possible initiating factors in the environment.

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References


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