

indices, and we hope that this can be achieved rapidly and with agreement between centres.

DAVID L SCOTT  
Department of Rheumatology  
St Bartholomew's Hospital  
London EC1A 7BE

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### Trauma and seronegative spondyloarthropathy: rapid joint destruction triggered by physical injury in HLA-B7

Sir: Several cases of HLA-B27 positive patients who developed peripheral arthritis immediately after injury have been reported in the last few years.<sup>1-4</sup>

The role of trauma in the initiation of seronegative spondyloarthropathy has been discussed.<sup>1-5</sup> Trauma may alter or release antigens, or both, from connective tissue,<sup>2-6</sup> and may lead in predisposed subjects to the development of an autoimmune reaction. This predisposition was associated with HLA-B27 in all reported cases.<sup>1-4</sup>

A new case of peripheral arthritis triggered by physical injury is reported here. Unlike previously reported cases of peripheral arthritis precipitated by trauma this patient was B27 negative, but had the cross-reactive antigen B7. No other precipitating factors were identified. After two different injuries the same patient developed arthritis, which caused rapid deterioration in two different joints.

In May 1985, after a minor injury to his left elbow, a 48 year old man developed arthritis of the same joint, without fever, diarrhoea, or urethritis. Arthrocentesis yielded a yellow, turbid fluid, whose culture proved negative. He was treated with anti-inflammatory drugs and intra-articular corticosteroids, but joint effusion persisted and flexion deformation developed in two months. In July 1989 he knocked his left knee, and a week later he developed arthritis with large effusion. He was admitted to another hospital and again the articular fluid was inflammatory and the culture was negative. Blood examination showed: erythrocyte sedimentation rate (Westergren) 140 mm/1st h, white cell count  $14.5 \times 10^9/l$  with 70% polymorphonuclear leucocytes, and haemoglobin 140 g/l; the tuberculin test (purified protein derivative 5 U) was positive.

He had no previous history of fever, urethritis, diarrhoea, or mucocutaneous lesions. Arthroscopy and synovial biopsy of the left knee were carried out, and the results were consistent with chronic synovitis without granulomas. After four weeks of anti-



Left elbow. Severe joint space narrowing and marginal bone erosions were evident.

inflammatory treatment the swelling persisted, and synovectomy by means of arthrotomy was carried out. He was then transferred to our rheumatology department. Physical examination showed seborrhoeic dermatitis and a swollen painful left elbow with limitation of movement (extension 60°—flexion 90°). The left sacroiliac joint showed tenderness, and movement caused pain. We also found decreased lumbar flexion (Schober test <3 cm). The left knee progressed well after operation, without swelling. Laboratory tests showed: erythrocyte sedimentation rate 29 mm/1st h, white cell count  $6.6 \times 10^9/l$ , haemoglobin 130 g/l. Antinuclear antibody, anti-DNA, latex, and Waaler-Rose tests and tests for complement were negative or normal. Serology tests for salmonella, shigella, yersinia, and toxoplasma proved negative. HLA was B7 positive. A radiograph of the left elbow showed joint destruction (figure). Rectification and syndesmophytes were found after radiographic examination of the lumbar spine. A technetium-99m bone scan showed uptake in the left sacroiliac joint. Another arthrocentesis of the elbow was carried out, but bacterial and mycobacterial cultures proved negative. After an infiltration of intra-articular corticosteroids the patient greatly improved for one month, and three months later a rhenium chemical synovectomy was carried out. Five months later he continued to be free from pain in the elbow (extension 50°, flexion 120°).

In this case the temporal relation between both physical injuries and the development of peripheral arthritis is striking. There is little doubt about the diagnosis of seronegative

spondyloarthropathy in this patient. He had clinical and radiological evidence of lesions in the lumbar spine and the left sacroiliac joint. He developed rapid joint destruction in the left elbow and left knee after both physical injuries.

Our patient, unlike previous cases<sup>1-4</sup> of peripheral arthritis precipitated by trauma, did not have the HLA-B27 antigen, but was B7 positive. We think this is the first case of spondyloarthropathy related to trauma in a B27 cross-reactive antigen (CREG group). At least 12 well described cases of spondyloarthropathy triggered by trauma have been reported in recent years. Some presented as Reiter's syndrome,<sup>3-7</sup> others as peripheral arthropathy,<sup>1-4</sup> and all of them were B27 positive. Seronegative spondyloarthritides include a heterogeneous group of disorders occurring in genetically predisposed subjects after exposure to environmental triggers.<sup>9-10</sup> We agree with those who suggest that physical injury may precipitate this arthropathy.<sup>1-4</sup> Genetic predisposition is clearly marked by HLA-B27, and perhaps by other antigens of the cross-reactive group (CREG), but to establish this fact more studies are necessary.

LOURDES MATEO  
JORDI DEL BLANCO  
XAVIER JUANOLA  
JOSÉ VALVERDE  
DANIEL ROIG  
Servicio de Reumatología  
Hospital de Bellvitge 'Princesps d'Espanya'  
Feixa Llarga s/n  
L'Hospitalet de Llobregat  
Barcelona, Spain

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## Trauma and seronegative spondyloarthritis: rapid joint destruction triggered by physical injury in HLA-B7.

L Mateo, J Del Blanco, X Juanola, et al.

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