- and mechanisms of action. *Am J Med* 1970; 48: 443-8.
- 4 Markand O N, D'Agostino A N. Ultrastructural changes in skeletal muscle induced by colchicine. *Arch Neurol* 1971; 24: 72-82.
 - 5 Riggs J E, Schochet S S, Gutmann L, Crosby T W, DiBartolomeo A. Chronic human colchicine neuropathy and myopathy. *Arch Neurol* 1986; 43: 521-3.
 - 6 Kuncel R W, Duncan G, Watson D, Alderson K, Rogawski M A, Peper M. Colchicine myopathy and neuropathy. *N Engl J Med* 1987; 316: 1562-8.
 - 7 Kuncel R W, Duncan G. Chronic human colchicine myopathy and neuropathy. *Arch Neurol* 1988; 45: 245-6.
 - 8 Kuncel R W, Cornblath D R, Avila O, Duncan G. Elektrodagnosis of human colchicine myoneuropathy. *Muscle Nerve* 1989; 12: 360-4.
 - 9 Lazaro R P, Kirshner H S. Proximal muscle weakness in uremia. *Arch Neurol* 1980; 37: 555-8.
 - 10 Lin H, Rocher L L, McQuillan M A, Schmaltz S, Palella T D, Fox I H. Cyclosporine-induced hyperuricemia and gout. *N Engl J Med* 1989; 321: 287-92.

Reactive arthritis and group G streptococcal pharyngitis

Sir: A previously healthy 29 year old man presented to his general practitioner with a week long history of fever, sore throat, and polyarthralgia. A throat swab was taken and treatment was started with ampicillin 250 mg and ibuprofen 600 mg, both four times daily. He was seen two days later with more profound malaise, and a synovitic left wrist, and was referred to us. There was no history of rash, dysuria, bowel disturbance, or eye symptoms. He had a stable heterosexual relationship. On examination his temperature was 39.0°C, with generalised tender cervical lymphadenopathy and an inflamed throat. There was synovitis in the left wrist and restricted movement. Neck movement was globally restricted, with evidence of capsulitis of the right shoulder and supraspinatus tendonitis in the left. The results of the rest of the examination were entirely normal.

The throat swab (taken by the general practitioner before starting treatment with antibiotics) showed a heavy growth of Lance-

field group G streptococci. A left wrist aspirate showed no organisms on Gram staining, and no subsequent growth even after enrichment culture. Five sets of blood cultures were sterile. Antistreptolysin O titre was >1000 international units (IU) per millilitre (normal <200). White cell count was $17.5 \times 10^9/l$ (neutrophils 14.9), haemoglobin 143 g/l, platelets $327 \times 10^9/l$, erythrocyte sedimentation rate 75 mm/h, C reactive protein 375 mg/l (normal <100), normal biochemistry except for alkaline phosphatase 412 IU/l (normal 80-280), γ -glutamyltransferase 82 IU/l (normal 10-48), ferritin 839 $\mu g/l$ (normal 10-385). Rheumatoid factor, antinuclear antibodies, and anti-neutrophil cytoplasmic antibodies were negative. The following were normal or negative: left wrist and chest radiographs; urine analysis and mid-stream urine culture; electrocardiogram; echocardiogram; viral antibody screen; hepatitis B serology; yersinia, brucella, and borrelia antibodies. HLA class I typing was A1 B8 B15.

He was admitted to hospital. Treatment was continued with ampicillin and indomethacin 50 mg three times daily. Over the next 10 days he experienced prolonged morning stiffness and a swinging fever. The neck pain and capsulitis of the right shoulder rapidly settled. There was sequential painful involvement of the left wrist, both supraspinatus tendons, left then right hip, right wrist, and bilateral adductor enthesitis, with each episode lasting 24-72 hours. The erythrocyte sedimentation rate peaked at 128 mm/h four days after admission, and the platelets at $859 \times 10^9/l$ nine days later, returning gradually to normal thereafter. The C reactive protein slowly fell to normal. Indomethacin was discontinued when a mild hepatic picture developed on the liver function tests, and prednisolone 40 mg daily was introduced. Thereafter, no new joint or enthesopathic symptoms developed, and results of liver function tests were normal. He was discharged 16 days after admission. Within two months all inflammation had subsided with a full return to normal function of all joints. The prednisolone has been tapered rapidly, and the patient has returned to full time employment.

Group G streptococci have been associated with previous reports of septic arthritis,¹ but the low virulence confines most serious disease to patients with a predisposition to infection.² There are case reports of sterile reactive arthritis in septicæmic patients.^{1,3} In this patient there was no evidence of septicaemia, joint aspiration of the wrist showed no growth, and he had been previously well. His subsequent arthritis and enthesitis was flitting and short lasting, which is much more characteristic of reactive and rheumatic fever arthritis⁴ than of multifocal sepsis, though the distribution of joint and enthesitis involvement was unusual. Furthermore, bacteraemia rarely complicates pharyngitis.⁵ Host antibody responses are poor in streptococcus group G pharyngitis, so that until now no cases of post-streptococcal sequelae have been described in group G pharyngitis.¹ As far as we are aware this represents the first reported case of reactive arthropathy complicating such an infection in the absence of septicaemia. It appears therefore that group G streptococcal infection may be yet another cause of reactive arthritis and enthesitis.

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- 1 Gaunt P N, Seal D V. Group G streptococcal infection of joints and joint prosthesis. *J Infect* 1986; 13: 115-23.
- 2 Gaunt P N, Seal D V. Group G streptococcal infections. *J Infect* 1987; 15: 5-20.
- 3 Rogerson S J, Beeching N J. Reactive arthritis complicating group G streptococcal septicaemia. *J Infect* 1990; 20: 155-8.
- 4 Homer C, Shulman S T. Clinical aspects of rheumatic fever. *J Rheumatol* 1991; 18 (suppl 24): 2-13.
- 5 Barnham M. Bacteraemia in streptococcal infections of the throat. *J Infect* 1983; 7: 203-9.