Case Report

A 65-year-old man complained of swelling and pain of his left foot since one day (figure 1). He had a history of rheumatoid arthritis, for which he was taking azathioprine and oral corticosteroids. He had not experienced symptoms in this foot before, neither had he received local corticosteroid therapy. At the time of presentation he did not complain of pain in the other joints. Physical examination of all joints was normal. However, on the dorsum of the left foot there was localised redness of the skin with a palpable fluctuating mass. Ultrasound examination showed signs of tenosynovitis or peritendinitis, without signs of arthritis. Needle aspiration of the palpable mass revealed a red-yellow substance. Microscopic examination of the aspirated fluid is shown in figure 2.

What is your diagnosis?

See page 293 for the answer to this photo quiz.

Figure 1. Left foot of the patient showing localised swelling and redness caused by the tenosynovitis or peritendinitis

Figure 2. Microscopy of fluid obtained by fine needle aspiration (250x)
Microscopic examination showed leucocytes and square-shaped ('plate-like') crystals. This shape is typical for cholesterol crystals. Cholesterol crystals are large, flat and rectangular with notched corners. Polarised light microscopy demonstrated that the crystals were moderately birefringent. Culture of the aspirated fluid was negative. Cholesterol concentration in the aspirated fluid was not measured. Serum total cholesterol, LDL cholesterol and triglycerides were normal. HDL cholesterol was slightly reduced (0.88 mmol/l, normal value >0.90 mmol/l). Apolipoprotein values were not determined. The patient had no cardiovascular symptoms, but received acenocoumarol because of recent venous thrombosis and pulmonary embolisms. The patient improved spontaneously.

Cholesterol crystals have been described to localise to arthritic fluid, bursal fluid and rheumatoid nodules of patients with rheumatoid arthritis. The pathophysiology of cholesterol crystal formation in these sites is not well known. Systemic causes that have been proposed are dyslipoproteinemia or formation of lipoprotein-directed antibodies, followed by deposition of antibody-antigen complexes in for example synovial fluids. However, deposits of cholesterol are usually considered to arise due to local factors, such as cell membrane degeneration, disturbed local cholesterol/lipoprotein metabolism and clearance or increased permeability to cholesterol because of chronic inflammation.

REFERENCES