CASE HISTORY
A 20 year old male university student presented with a one year history of a stiff painful swollen left ankle. He described morning stiffness of one hour and also night pain which occasionally kept him awake. He was unable to take part in sporting activities. There was no history of trauma. No other joints were painful. There was no history of back or heel pain, psoriasis, iritis, urethritis, or inflammatory bowel disease. There was no history of preceding gastrointestinal or genitourinary infection. He had had an episode of Stevens-Johnson syndrome at the age of 12 (but no recent skin or mouth lesions) and occasional migraine headaches. His health was otherwise good. There was no history of weight loss. Non-steroidal anti-inflammatory drugs provided more symptomatic relief than simple analgesics. His grandmother had rheumatoid arthritis. There was no other significant family history. Clinical examination demonstrated a large warm ankle effusion with generalised tenderness, stress pain, and significantly decreased ankle movement. No abnormality was found on examination of the remainder of the locomotor system. There was no evidence of psoriasis or other rash or nail lesion. The remainder of the clinical examination was unremarkable.

Investigations showed normal inflammatory markers: erythrocyte sedimentation rate was 2 mm in the first hour; C reactive protein <5 mg/l. A full blood picture was normal, with haemoglobin 144 g/l, white cell count 4.50×10⁹/l (differential white cell count normal), platelet count 250×10⁹/l. Urea and electrolytes, bone profile, and liver function tests were all normal. Serum ferritin was also normal at 97 μg/l (normal range 15–300). Serum urate was 0.34 mmol/l (normal range 0.16–0.43). Rheumatoid factor was negative. A plain radiograph of the left ankle demonstrated periarticular osteopenia and evidence of a joint effusion (fig 1). The joint spaces were preserved and no focal bone lesion was demonstrated. Overall, the appearance was suggestive of an inflammatory arthropathy.

The ankle joint was aspirated and 6 ml of amber coloured clear fluid was removed. Joint fluid analysis showed a total white cell count of 0.2×10⁹/l. Crystals were not identified, and no organisms were seen. There was no growth on joint fluid culture. The ankle joint was injected with 20 mg of triamcinolone acetonide. In view of the absence of a clinical history suggestive of inflammatory disease, a normal acute phase response, and non-inflammatory synovial aspirate, a magnetic resonance imaging (MRI) scan was requested.

The patient was reviewed three weeks later and he reported a temporary reduction in swelling of the ankle, although the pain and stiffness remained. The MRI scan disclosed a large effusion into the ankle joint and thickening of the synovium in the ankle (fig 2). There was marked oedema in the adjacent soft tissues around the ankle and in the sinus tarsi. Post-contrast studies confirmed the presence of synovial thickening. Bone oedema in the head and body of the talus was marked and there was also some oedema in the anterior
part of the calcaneum. No bone destruction was seen, but there was a small rounded lesion 1 cm in diameter in the subperiosteal region of the talar neck in keeping with an osteoid osteoma.

The patient was referred to an orthopaedic surgeon, and the osteoid osteoma was excised without complication. His expected prognosis is good.

**DISCUSSION**

This patient had persistent monarthritis of the ankle with no history of trauma. Joint aspiration was not in keeping with a septic joint or crystal arthritis. There were no features to suggest reactive arthritis or a monoarticular presentation of an inflammatory oligo- or polyarthritis. In particular, there was no acute phase response and the synovial aspirate was non-inflammatory. Although plain radiology was suggestive of an inflammatory arthropathy, atypical features included the description of significant night pain, and the duration of the symptoms. These atypical features prompted the request for MRI of the joint, which confirmed the underlying diagnosis of osteoid osteoma.

Osteoid osteoma is a benign osteoblastic lesion characterised by size (usually less than 1 cm), a clearly demarcated outline, and by the usual presence of a surrounding zone of reactive bone formation. Histologically it consists of cellular, highly vascular tissue, made up of immature bone and osteoid tissue.¹ Osteoid osteomas occur relatively frequently, usually in adolescence and early adulthood with a 2:1 male:female ratio. They most commonly occur in long bones such as the femur and tibia, but can occur in short bones such as the talus in this case. Location close to the joint as described here can lead to synovitis and effusion, and attribution of symptoms and signs to synovial pathology.² Radiologically the diagnosis may not be readily apparent, particularly when the nidus has an intra-articular location.³ Isotope bone scans are sensitive investigations but may be difficult to interpret in the presence of surrounding synovitis. Computed axial tomography is traditionally the preferred method for diagnosis and localisation of an osteoid osteoma. The lesion may also be well demonstrated by MRI, as in this patient, but recent evidence suggests that this is not always the case.⁴ Complete surgical removal of the nidus is curative, but incomplete resection may result in recurrence.

**THE LESSONS**

- In the absence of clinical or serological features suggestive of an inflammatory disease, a “monarthritis” in a young adult or adolescent demands further investigation.
- Synovitis and effusion may mask an underlying neoplasm.
- Plain radiology may not demonstrate small bony tumours, particularly if intra-articular in location; more sensitive imaging methods may be required.

**REFERENCES**

"Monarthritis" in a young man

A P Cairns, E M J Borthwick, H B Murtagh and G D Wright

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