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Gouty tenosynovitis – more common than we think?

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We report a case of acute, gouty flexor tenosynovitis in a Maori farmer, initially diagnosed as infective tenosynovitis. There was no previous history of gout and the serum urate was normal. Suppurative tenosynovitis caused by bacterial infection is a common condition. Tenosynovitis mimicking bacterial infection may be caused by gout, calcific tendonitis, rheumatoid arthritis and amyloidosis.1 Gouty tenosynovitis has been reported to mimic tuberculous tenosynovitis,2 and has been implicated in tendon rupture.3 Only one case of gouty tenosynovitis mimicking bacterial infection has been previously reported.4 It is important to recognise gout as a differential diagnosis of infective tenosynovitis, particularly where no organism is found. This is especially important in New Zealand where a high prevalence of gout has been reported in Maori men and men from the South Pacific Islands.5,6

Case report

A 52-year-old, Maori male farmer presented with a two-day history of fusiform swelling of the right ring finger. This had become increasingly painful, with difficulty in grip. On examination he had a temperature of 37.2 °C, and a pulse rate of 82/min. His finger was hot, red and swollen, with a tense effusion of the flexor sheath. The finger was held in a slightly flexed position and any attempts at passive extension were extremely painful. He also complained of some pain around the metacarpo-phalangeal (MCP) joint of the middle finger, which on examination showed moderate soft-tissue swelling over the dorsal aspect. There was no history of trauma and the patient was in good health with no history of rheumatoid arthritis, diabetes or alcoholism. He was not on any regular medication and had a normal diet. C-reactive protein was raised at 30 mg/l (normal <9 mg/l) but other laboratory tests were normal: white cell count 9.8, neutrophils 62%, erythrocyte sedimentation rate 10 mm/hour. The blood urate level was also normal at 0.35 mmol/l (normal 0.20 to 0.40 mmol/l). Radiological examination of the hand was unremarkable.

Clinical signs were suggestive of an infective tenosynovitis and so the flexor sheath was explored under general anaesthetic. A classical double-incision technique was used. There was inflammation of the synovium and a small amount of straw-coloured fluid but no other abnormality was seen. The MCP joint of the middle finger was aspirated and was found to contain similar straw-coloured fluid. The joint was opened through a dorsal incision and exploration revealed some chalky material in the joint. This raised a suspicion of gout and aspirates from both the tendon sheath and the joint were sent for polarising microscopy as well as Gram's stain and culture. Aspirates were sent in both plain and EDTA tubes.

Gram's stain did not reveal any organisms and results of blood cultures and culture from the tendon sheath were negative. The aspirates of both the tendon sheath and MCP joint were, however, positive for uric acid crystals. Antibiotics were therefore stopped and the patient was commenced on indomethacin and then allopurinol. The patient received hand physiotherapy and four months later had 80% range of movement and returned to work as a farmer. Serum urate was 0.41 mmol/l (normal 0.20 to 0.40 mmol/l) at eight weeks.

Discussion

It is not uncommon to see a patient with a diagnosis of 'infective' tenosynovitis where no organism is found in the aspirate. Three such cases of 'sterile flexor tenosynovitis' have been reported,7 but the incidence in wider practice is uncertain. Strong suspicion is required if

cases of gouty flexor tenosynovitis are not to be missed. It is important to differentiate gout from infection although they may coexist.8 Diagnosis is made by synovial fluid examination for urate crystals and it is recommended that specimens are fixed in alcohol. In 5–15% of cases of gouty arthritis, crystals may be absent in the synovial fluid aspiration and a synovial biopsy may be needed to confirm the diagnosis.9 Gouty tenosynovitis in the hand can be present without visible tophi or previous involvement of the upper limb,1 and is difficult for the clinician to differentiate from infective tenosynovitis. Serum urate is often normal at the time of an acute attack of gout. In this patient there was no history of gout, serum urate was normal at presentation and the diagnosis of gouty tenosynovitis would have been missed without the finding of chalky material on exploration of the MCP joint.

The exact incidence of gouty tenosynovitis is unknown. We believe that gout mimicking infective tenosynovitis may be more common than is currently recognised and should be considered in the differential diagnosis. This is of particular note in our New Zealand population where the prevalence of gout is high.

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