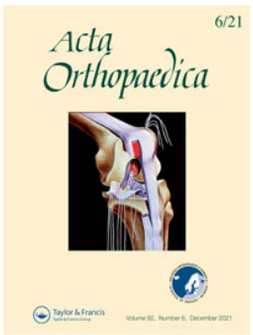

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Gouty tenosynovitis simulating an infection

A case report

Gouty tenosynovitis in the hand may be misdiagnosed as infectious or rheumatoid synovitis. Our case initially was treated as infectious tenosynovitis, but at reoperation gouty deposits were found penetrating the flexor tendon sheath. Tenosynovectomy and excision of intratendinous tophi were performed with a good result. Gouty synovitis is diagnosed in specimens fixed with ethanol because formalin destroys the typical crystals of urate.

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Introduction

Gouty involvement of flexor tendons in the hand is a rare condition. The tendon sheath provides a barrier against tophaceous infiltration (Straub et al. 1961, Primm & Allen 1983). However, Hankin et al. (1985) and Moor & Weiland (1985) recently described 2 cases of gouty involvement of the tendon sheath. We report a case with gouty tenosynovitis that initially was misdiagnosed as septic tenosynovitis.

Case report

A 74-year-old man with podagra 3 years earlier presented a history of increasing pain in the middle finger of the right hand during the last 2 days without preceding trauma. He was taking furosemide, and because of his gout, he took indomethacin intermittently. At examination, the middle finger was swollen and tender along the volar aspect with reduced motion, and there was a stitch mark on the pulp. The body temperature was 38.8° C and the ESR was 38 mm/h. Infectious tenosynovitis was suspected and drainage was performed. A sample was taken for bacterial culture, and antibiotic treatment was initiated. The initial postoperative course was encouraging, but on the fifth day, the symptoms recurred and a reoperation was performed. The tendon sheath was completely explored and a profuse tenosynovitis with infiltrating tophi was found (Figure 1). Tenosynovectomy, with excision of all tophi, was per-

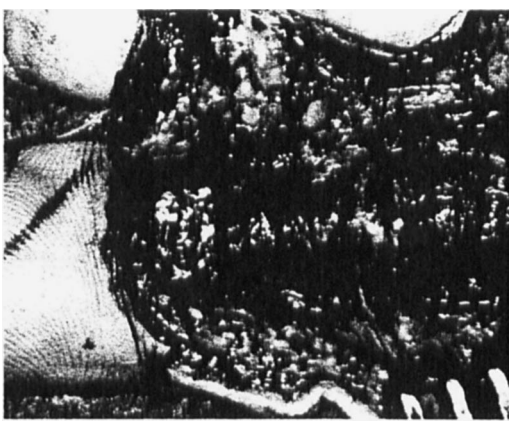

Figure 1. Macroscopic appearance of flexor tenosynovitis in gout. Tophaceous infiltration of the deep flexor tendon and the distal tendon sheath.

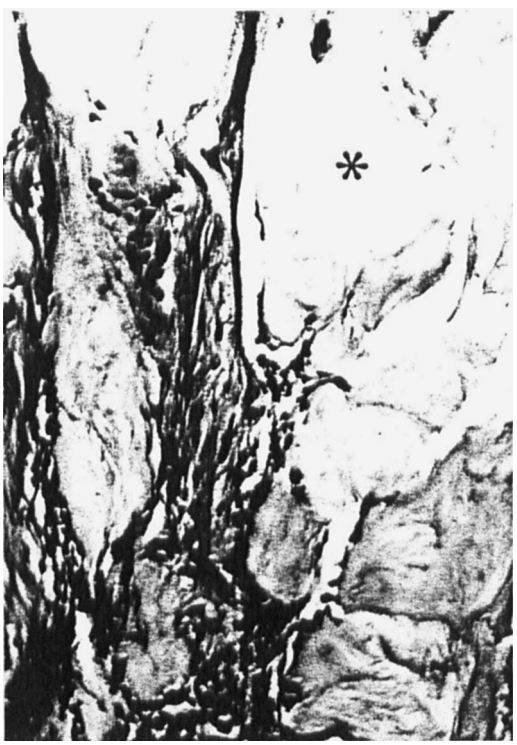

Figure 2. Photomicrograph of the lesion. Irregular material of urate crystals (•) surrounded by fibrous connective tissue with macrophages. Formalin fixation. HE, ×120.

formed and the specimens were fixed in formalin. Sections were stained with hematoxylin erythrosin and examined with light microscopy (Figure 2). No bacterial growth was found; the serum urate was 585 μmol/l and the ESR 95 mm/h. Indomethacin and allopurinol replaced the antibacterial therapy; active motions were encouraged, and the inflammatory symptoms subsided. Six months later the active range of movement was almost normal.

Discussion

The differential diagnosis of gouty tenosynovitis includes infectious and rheumatoid synovitis. Because 10–20 per cent of patients with rheumatoid arthritis have raised levels of serum urate (Straub et al. 1961), rheumatoid arthritis may be difficult to distinguish from gout. Hyperuricemia may also be present in myelofibrosis, leukemia, and polycythemia vera (Straub et al. 1961). Acute attacks of gout may be initiated by trauma (Hankin et al. 1985), drugs, and surgical operations (Straub et al. 1961). Detailed history, clinical examination, laboratory tests (ESR, leucocytes, serum urate, rheumatoid factors), crystal analysis, and bacterial cultures help in making the diagnosis. Specimens fixed in ethanol and examined with light microscopy show the typical crystals of urate. When fixed in formalin, irregular material surrounded by connective tissue, macrophages, and single foreign-body giant cells is characteristic of gout, since formalin destroys the typical urate crystals.

Treatment with probenecid and allopurinol reduces serum urate and prevents further depositions and should immediately be initiated. Acute exacerbations are best treated with indomethacin. In 5 per cent of the cases, medication has, however, no effect, and later chronic manifestations, such as skin perforation, joint destruction, tendon ruptures, infiltration of flexor tendons, and median nerve entrapment, may develop (Straub et al. 1961, Pledger et al. 1976, Green et al. 1977, Gelberman et al. 1980, Moore & Weiland 1985). In such cases, surgery is advocated, and indications have been discussed by Linton & Talbott (1943), Straub et al. (1961), and Larmont (1970). The surgical treatment of choice is complete exploration of the tendon sheath, thorough tenosynovectomy between the pulleys, excision of tophi, and complete closure of the wound. An immediate active motion program postoperatively is important.

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