Case report

Spontaneous rupture of the Achilles tendon in a patient with gout

PATRICK G. MAHONEY, PETER D. JAMES, CHRISTOPHER J. HOWELL, AND ANTHONY J. SWANNELL

From Nottingham City Hospital and the Queen's Medical Centre, University of Nottingham

SUMMARY A 49-year-old man with long-standing gout suffered a spontaneous rupture of the Achilles tendon. Surgical repair was performed, and gouty tophi were found in the severed end of the tendon. The possible causes of this spontaneous rupture are discussed.

Although an early treatise on gout describes 'violent stretching and tearing of ligaments' and a textbook attributes one of the causes of tendon rupture to gout, recent reports of tendon rupture in gout are rare. The purpose of this paper is to report a case of tendon rupture through gouty deposits.

Case report

A 49-year-old policeman with a family history of long-standing gout in both grandfathers first developed mild attacks of gout himself at the age of 31 years. These attacks were initially infrequent and treated with colchicine and phenylbutazone. A random serum uric acid after 3 years was 10·3 mg/100 ml (618 μmol/l). Over the next few years attacks occurred about once a year but after 13 years the site and frequency increased involving the great toes, ankles, knees, and shoulders. He was started on allopurinol 100 mg t.d.s. by his general practitioner. Unfortunately this caused flushes and dizziness and was stopped.

He was referred for a rheumatological opinion in 1974. Clinical examination revealed a large 18 stone (114 kg) man with a handlebar moustache, some discoloration of the right great toe, and limitation of movements of the right shoulder and both knees. There were no subcutaneous tophi. He was normotensive with no evidence of renal or cardiovascular disease. Haemoglobin 14·5 g/100 ml (14·5 g/dl), ESR 12 mm/hour, white blood count 5700 mm$^3$ (5·7 × 10$^9$/l), serum uric acid 8·8 mg/100 ml (528 μmol/l), blood urea 31 mg/100 ml (5·0 mmol/l).

Accepted for publication 20 August 1980

Correspondence to Dr P. G. Mahoney, City Hospital, Hucknall Road, Nottingham NG5 1PB.
vascularity augmented by urate and association of fortuitous Deposition of commonest cause generation.

a and insertion into and shown requisite of in the absence steroid medication hyperparathyroidism, rheumatoid arthritis, erythematosis, rupture: primary condition existing rupture of normal tendons has spontaneous tendon has been normal mg/100 episodes of steroid medication secondary hyperparathyroidism, spousal medication in SLE, and steroid medication of other conditions. Even in the absence of systemic conditions local degeneration of the tendon has been stressed as a prerequisite of rupture of the free part of the tendon. Angiographic and microangiographic studies have shown that an area 2–6 cm from the place of insertion into the calcaneum is relatively avascular and a particularly vulnerable area for such degeneration.

After trauma gout has been described as the commonest cause of pain in the Achilles tendon. Deposition of urate crystals may be on the basis of fortuitous association of altered connective tissue metabolism and the supersaturation of tissue by urate augmented by proteoglycans as an active factor. Alternatively local factors such as tissue vascularity and collagen matrix composition may account for urate deposition in other tissues and could explain the urate deposit in the tendon of our patient. The urate crystals themselves may have caused a reduction of tensile strength in the tendon on account of their number and size. In a reported cause of spontaneous tendon rupture attributed to gout later re-examination of the histological specimens of the severed ends of the tendon showed urate crystals but also the process of elastosis. Connective tissue elastosis was first described in the skin following observations on the transformation of degenerating collagen fibrils into elastin. Elastin fibres have been found interspersed with collagen fibrils in tendons of patients who have had tendon rupture with renal tubular acidosis and renal disease on chronic haemodialysis.

The previously reported cause of tendon rupture attributed to gout was in renal failure and had elastosis affecting the tendon. Our patient had normal renal function and did not have elastosis. Whether the tendon weakness in our case was due to the urate crystals themselves or the process which facilitated their deposition remains unclear.

Fig. 1 Transverse section of a severed end of the Achilles tendon showing gouty tophi. (H & E, × 54).

References


