Case report

Spontaneous rupture of the Achilles tendon in a patient with gout

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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'¹ and a textbook attributes one of the causes of tendon rupture to gout,² recent reports of tendon rupture in gout are rare.³ 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 μ mol/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 discolouration 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 mm³ (5.7×10^9 /l), serum uric acid 8.8 mg/100 ml (528 μ mol/l), blood urea 31 mg/100 ml (5.0 mmol/l).

X-rays showed osteoarthritic changes in both knees and the right shoulder. He was given allopurinol 100 mg b.d. increasing to t.d.s., and within 3 months the serum uric acid fell to 5.6 mg/100 ml (336 μ mol/l).

His subsequent course was uneventful until April 1978, when while walking across a road he developed sudden severe pain in the right lower leg and felt something give in his calf. The diagnosis of ruptured Achilles tendon was confirmed later that day at an emergency operation. The rupture had occurred 5 cm from the insertion of the tendon, and numerous nodules were found in the area; the paratenon was hypertrophied and there was an underlying haematoma. The ends were trimmed and repaired with nylon sutures. The specimens removed were 6 pieces of fibrofatty tissue, the largest being $4.5 \times 1 \times 0.8$ cm. Within the fibrous tissue there were round deposits up to 1 cm in diameter which were chalky white on section. The tissue was fixed in 10% formal saline. Histological sections stained with haematoxylin and eosin showed numerous collections of amorphous eosinophilic material in which needle-like spaces were seen. The amorphous deposits were surrounded by chronic inflammatory cells and numerous giant cells. The lesions were of variable size. Some were quite large and were found to be within dense collagen tissue consistent with part of a tendon (Fig. 1).

Elastic van Gieson stains confirmed the presence of collagenous fibrous tissue and no elastotic degeneration was seen. Examination of sections under polarised light showed no evidence of crystalline material, urate crystals being water soluble; the appearances were typical of gouty tophi. Four days after surgery he had an acute attack of gout involving

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

the left great toe which was treated with colchicine and continuation of the allopurinol. Since then there has been normal function of the right Achilles tendon, no further episodes of gout have occurred, and a recent serum uric acid was $270 \mu\text{mol/l}$ (4.5 mg/100 ml).

Discussion

Normal tendon has great tensile strength,⁴ and spontaneous tendon rupture is uncommon. Although rupture of normal tendons has been achieved experimentally,⁵ most authors have identified a pre-existing condition in man. Several systemic disorders have been associated with spontaneous tendon rupture: primary hyperparathyroidism,⁶ secondary hyperparathyroidism,⁷ rheumatoid arthritis,⁸ systemic lupus erythematosus,⁹ steroid medication in SLE,¹⁰ and steroid medication of other conditions.¹¹ 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.¹² Angiographic and microangiographic studies have shown that an area 2–6 cm from the place of insertion into the calcaneum is relatively avascular¹³ and a particularly vulnerable area for such degeneration.

After trauma gout has been described as the commonest cause of pain in the Achilles tendon.¹⁴ 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.¹⁵ Alternatively local factors such as tissue vascularity and collagen matrix composition¹⁶ 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 gout³ later re-examination of the histological specimens of the severed ends of the tendon showed urate crystals but also the process of elastosis.¹⁷ Connective tissue elastosis was first described in the skin¹⁸ following observations on the transformation of degenerating collagen fibrils into elastin.¹⁹ Elastin fibres have been found interspersed with collagen fibrils in tendons of patients who have had tendon rupture with renal tubular acidosis²⁰ and renal disease on chronic haemodialysis.²¹

The previously reported cause of tendon rupture attributed to gout³ 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.

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