

Bilateral simultaneous spontaneous rupture of the Achilles tendon

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INTRODUCTION

Ciprofloxacin is an antibiotic with a broad spectrum of activity. It belongs to the fluoroquinolone group, which are relatively safe and effective antibacterial agents. The adverse reactions most commonly occur in the gastrointestinal tract, skin and the CNS (Ball et al, 1999). Its use is restricted in children because of the risk of damage to the cartilage. Inflammation and rupture of the Achilles tendon is a rare but potential side effect of fluoroquinolones (Jorgensen et al, 1991; LeHuec et al, 1995; Ball et al, 1999; van der Linden et al, 1999).

Long-term use of steroids, especially in the elderly, is also a recognized cause of Achilles tendon rupture (Haines,

1983; Baruah, 1984). This article reports a case of bilateral simultaneous spontaneous rupture of the Achilles tendon following a short course of ciprofloxacin and prednisolone.

DISCUSSION

The Achilles tendon is the thickest and strongest tendon of the body, formed by the union of the gastrocnemius and soleus tendons. Spontaneous rupture of this tendon is unusual. Excessive loading of the tendon during physical training is the main cause of rupture (James et al, 1978). Inflammation and rupture of the Achilles tendon has been described as a rare but potential adverse effect of fluoroquinolones and long-term steroid therapy (Haines,

1983; Baruah, 1984; Jorgensen et al, 1991; LeHuec et al, 1995; Ball et al, 1999; van der Linden et al, 1999).

Spontaneous tendon rupture is known to be associated with systemic disorders including Cushing's disease, rheumatoid arthritis, systemic lupus erythematosus, chronic renal failure requiring haemodialysis, hyperparathyroidism, type II hyperlipoproteinaemia and gout.

The pathophysiology of tendon rupture in relation to fluoroquinolones is still unclear. It may be multifactorial, including ischaemic factors (van der Linden et al, 1999; Williams et al, 2000), interleukin-1 β -induced reduction in prostaglandin E₂ output in tendon-derived cells affecting various cellular

CASE REPORT

A 76-year-old man presented to the accident and emergency department with a 2-week history of gradually increasing difficulty with walking and mild pain in both ankles. He gave no history of trauma.

He had a history of asthma, chronic obstructive airway disease and paraproteinemia (consistent with a plasma cell disorder, but not diagnostic of multiple myeloma and hence was not on any treatment for it). There was no history of diabetes or gout. He was an ex-smoker, having given up smoking 13 years previously. He gave no history of other tendon or joint problems.

He had been discharged from the hospital 3 weeks previously after being treated for pneumonia. During his previous admission he was treated with oral ciprofloxacin 500 mg twice daily for 4 days, intravenous benzylpenicillin 1.2 g four times a day for 7 days and oral prednisolone 30 mg once a day. He was discharged on prednisolone, which he gradually reduced at a rate of 5 mg every 4 days and was on 1 mg/day when his symptoms started.

On admission he had bruising, swelling and mild tenderness proximal to the insertion of Achilles tendons bilaterally. The plantaris was palpable on the right side but not on the left. A palpable gap and a negative Simmond's test/squeeze test was also present on both sides. He had no active plantar flexion on the left side but with difficulty was able to demonstrate some degree of active plantar flexion on the right. His full blood count, urea and electrolytes, blood sugar and liver function tests were within the normal range.

Ultrasound scan of both the Achilles tendons demonstrated a defect of several centimetres proximal to the distal insertions, consistent with complete ruptures (Figures 1–4). The defects were filled with material of mixed echogenicity. Intact tendon material was noted medial to the defect on the right, consistent with the plantaris tendon.

He underwent surgical exploration, which confirmed the clinical and radiological findings. The plantaris was found to be intact on the right side. The operation did not pose any problems in spite of the injury being 2 weeks old. The tendons were repaired with No.1 polydioxanone (PDS) suture using a Kessler stitch. Postoperatively the ankles were immobilized in an equinus below-knee plaster followed by serial plasters to bring the ankles to neutral at 6 weeks. The plasters were removed at 8 weeks and a mobilization programme initiated.

The patient is progressing well and is presently mobilizing with the aid of two walking sticks. He continues to receive physiotherapy to further improve the strength and movement in his ankles.

Figure 1. Ultrasound scan showing the proximal end of the ruptured right Achilles tendon (arrow).



Figure 2. Ultrasound scan of the right Achilles tendon showing the mixed echogenic material within the defect.



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activities of interleukin-1 β (Corps et al, 2003), selective enhancement of matrix metalloproteinase 3 expression by interleukin-1 β in tendon-derived cells (Corps et al, 2002) and/or as a result of their magnesium antagonist effects (Shakibaei et al, 2001). Incubation of canine Achilles tendon fibroblasts with ciprofloxacin showed a significant reduction in cell proliferation compared with the control cells. An increase in matrix-degrading protease activity from the fibroblasts and an inhibitory effect on fibroblast metabolism (Williams et al, 2000) was also demonstrated.

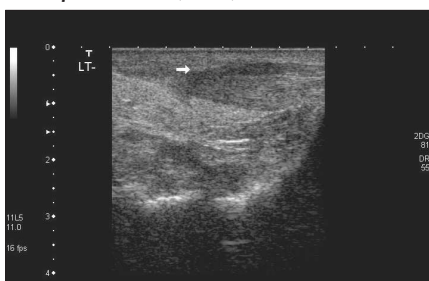
The pathophysiology with regards to corticosteroids is also controversial and may include inhibition of fibroblast proliferation and maturation (Baruah, 1984), inhibition of collagen synthesis and/or ischaemic factors (Rose and Frassica, 2001).

The Achilles tendon has a hypovascular area 2–6 cm proximal to its insertion into the calcaneum as shown by angiographic studies (Lagergren and Lindholm, 1958). One or more of the above factors may be responsible for further compromising the blood supply to the Achilles tendon, causing it to rupture during the use of fluoroquinolones and corticosteroids.

Figure 3. Ultrasound scan showing the proximal end of the ruptured left Achilles tendon.



Figure 4. Ultrasound scan showing the distal portion of the ruptured left Achilles tendon with a defect proximal to it (arrow).



The ruptured Achilles tendon can either be treated conservatively in plaster or surgically. The choice of treatment depends on the surgeon's preference and the patient's need. After surgery the repair is often protected by an equinus plaster for 6–8 weeks to allow the approximated tendon to heal. The patient is then given a heel raise or a range of motion brace and mobilization is commenced under the supervision of the physiotherapist.

Pain, swelling and tenderness in the region of the Achilles tendon may be caused by insertional tendinitis, peritendonitis, tendinosis, partial or complete rupture and retrocalcaneal bursitis. These conditions, even though distinct, often occur in combination (Maffulli and Kader, 2002; Schepsis et al, 2002). Fluoroquinolone-associated tendon disorders have been shown to be common in patients over 60 years of age (van der Linden et al, 2001). Ultrasonography is effective in differentiating between these conditions (Blankstein et al, 2001).

Histopathological analysis of the ruptured tendon was not performed in the above case but previous work has shown that the microscopic changes in the ruptured tendon secondary to ciprofloxacin are similar to those seen in overuse (Movin et al, 1997).

In the above case, the patient received a short oral course of both ciprofloxacin and prednisolone. It is not possible to say whether either drug alone caused the rupture or whether it was a combined effect.

CONCLUSIONS

This report is presented to remind prescribers that tendon rupture is a well-known adverse reaction of fluoroquinolones, particularly ciprofloxacin, and also of steroid therapy. The risk of tendon rupture may increase when the two are used in combination. As the use of these drugs is increasing, the prescription should be carefully considered and the dosage accurately managed, especially in patients with associated risk factors. In order to reduce the risk of tendon rupture, the authors recommend that in patients on steroids requiring antibiotics, one

should use fluoroquinolones cautiously and monitor these patients closely for pain, swelling and tenderness in the region of the Achilles tendon. An ultrasound examination of the Achilles tendon should be considered if the above symptoms appear. **HM**

- Ball P, Mandell L, Tillotson G (1999) Comparative tolerability of the newer fluoroquinolone antibacterial. *Drug Saf* **21**: 407–21
- Baruah DR (1984) Bilateral spontaneous rupture of the Achilles tendons in patient on long-term systemic steroid therapy. *Br J Sports Med* **18**: 128–94
- Blankstein A, Cohen I, Diamant L et al (2001) Achilles tendon pain and related pathologies: diagnosis by ultrasonography. *Isr Med Assoc J* **3**: 575–8
- Corps AN, Harrall RL, Curry VA, Fenwick SA, Hazleman BL, Riley GP (2002) Ciprofloxacin enhances the stimulation of matrix metalloproteinase 3 expression by interleukin-1 beta in human tendon-derived cells. A potential mechanism of fluoroquinolone-induced tendinopathy. *Arthritis Rheum* **46**(11): 3034–40
- Corps AN, Curry VA, Harrall RL, Dutt D, Hazleman BL, Riley GP (2003) Ciprofloxacin reduces the stimulation of prostaglandin E2 output by interleukin-1 beta in human tendon-derived cells. *Rheumatology (Oxford)* **42**(11): 1306–10
- Haines JF (1983) Bilateral rupture of Achilles tendon in patients on steroid therapy. *Ann Rheum Dis* **42**: 652–4
- James SL, Bates BT, Osternig LR (1978) Injuries to runners. *Am J Sports Med* **6**: 40–50
- Jorgensen C, Anaya JM, Didri C (1991) Arthropathy with Achilles tendon involvement induced by pefloxacin. *Rev Rheum Mal Osteoartic* **58**: 623–5
- Lagergren C, Lindholm A (1958) Vascular distribution in the Achilles tendon: an angiographic and microangiographic study. *Acta Chir Scand* **116**: 491
- LeHuec JC, Schaevebeke T, Chauveaux D (1995) Epicondylitis after treatment with fluoroquinolones antibiotics. *J Bone Joint Surg (Br)* **58**: 293–5
- Maffulli N, Kader D (2002) Tendinopathy of Tendo Achilles. *J Bone Joint Surg Br* **84-B**: 1–11
- Movin T, Gad A, Guntner P, Foldhazy Z, Rolf C (1997) Pathology of the Achilles tendon in association with ciprofloxacin treatment. *Foot Ankle Int* **18**(5): 297–9
- Rose PS, Frassica FJ (2001) Atraumatic bilateral patellar tendon rupture, A case report and review of the literature. *J Bone Joint Surg Am* **83-A**(9): 1382–6
- Schepsis AA, Jones H, Haas AL (2002) Achilles tendon disorders in athletes. *Am J Sports Med* **30**: 287–305
- Shakibaei M, de Souza P, van Sickle D, Stahlmann R (2001) Biochemical changes in Achilles tendon from juvenile dogs after treatment with ciprofloxacin or feeding a magnesium-deficient diet. *Arch Toxicol* **75**: 369–74
- van der Linden PD, van de Lei J, Nab HW, Knol A, Stricker BH (1999) Achilles tendinitis associated with fluoroquinolones. *Br J Clin Pharmacol* **48**: 433–7
- van der Linden PD, van Puijenbroek EP, Feenstra J et al (2001) Tendon disorders attributed to fluoroquinolones: a study on 42 spontaneous reports in the period 1988 to 1998. *Arthritis Rheum* **45**: 235–9
- Williams RJ III, Attia E, Wickiewicz TL, Hannafin JA (2000) The effect of ciprofloxacin on tendon, paratenon and capsular fibroblast metabolism. *Am J Sports Med* **28**: 364–9