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CASE REPORT

Rupture of adductor longus tendon due to ciprofloxacin

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We present a rare case of spontaneous rupture of the adductor longus tendon induced by ciprofloxacin. A 35-year-old man was diagnosed with pneumonia and was recommended ciprofloxacin 500 mg iv twice a day for 7 days. Three days after receiving the initial dose, he developed discomfort in his left medial thigh, and pain and swelling in the same area followed ten days later. He consulted us when he noted a palpable mass on the medial side of his left thigh, and MRI study revealed adductor longus tendon rupture. There was no obvious underlying disease or other factor causing fragility of his adductor longus tendon. We review the pathophysiological mechanisms leading to fluoroquinolone-related tendon rupture as well as the risk factors and discuss proper management.

Key words : ciprofloxacin ; adductor longus ; tendon ; rupture.

INTRODUCTION

Shortly after their introduction, fluoroquinolones were associated with reports of tendonitis and tendon rupture. During the past years, the number of reports has risen, possibly because of an increased use of fluoroquinolones. More than 200 reports of quinolone related tendinopathy have been published in the English literature.

Although the pathogenesis of this rare effect is unknown, the clinicians must recognise this condition early and interrupt the treatment with quinolones.

CASE REPORT

A 35-year-old man was admitted to the emergency department with acute dyspnoea and fever. Clinical and laboratory examinations revealed pneumonia, so he was treated for seven days with ciprofloxacin 500mg iv, twice a day. He suffered from diabetes mellitus, treated with insuline. He had not previously received quinolones.

Discomfort in the medial side of the left thigh was noted three days after he received the initial dose of ciprofloxacin. No injury was mentioned. He had non-pitting oedema and tenderness on palpation of the adductor muscles and experienced pain on thigh adduction.

There was no radiological evidence of a fracture and a Doppler study was negative for thrombosis.

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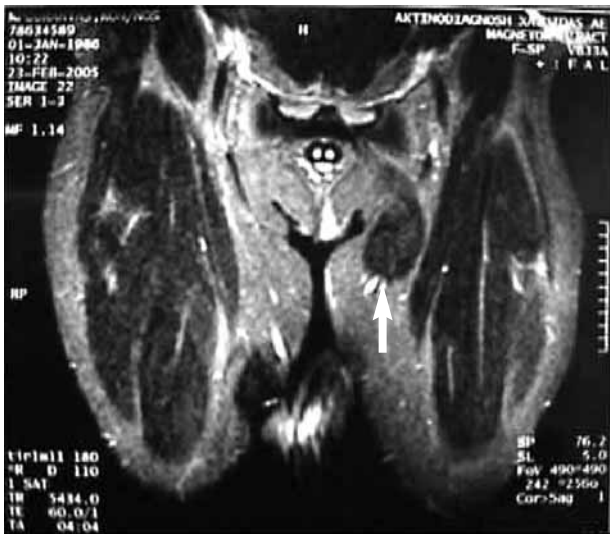

Fig. 1. — Rupture of the distal tendon of the adductor longus muscle (arrow).

The diagnosis of muscle strain was made and he was recommended to rest.

Ten days later (6 days after discontinuing ciprofloxacin) he was again evaluated in the emergency department because of severe pain in the medial side of his left thigh. He heard a snap while he was sitting down in an arm-chair. Pain was elicited while he was walking. The medial thigh felt warm and tender, and local oedema extended to the knee. He was again diagnosed as presenting a strain and was discharged.

Two weeks later, he was admitted to our department because of a palpable mass in the left thigh. On examination, the mass was painless, soft, mobile and not tender on palpation. He was able to walk and bear weight without pain. We suspected a tendon rupture induced by the quinolone. The suspicion was confirmed by MRI, which revealed a distal rupture of the adductor longus tendon (fig. 1).

The patient ultimately denied operative repair, because he was free of pain.

DISCUSSION

Fluoroquinolone antimicrobials are currently in wide use because of their strong activity against Gram-negative, Gram-positive and anaerobic bacteria. They are indicated in the treatment of community-acquired pneumonia, acute exacerbations of chronic bronchitis, and urinary infections.

Iatrogenic tendinopathy and tendon rupture are uncommon but well documented effects of fluoroquinolone therapy. No other drugs are known to induce tendon rupture, apart from corticosteroids. Mc Ewan reported the first case of tendon rupture associated with ciprofloxacin *(7)*. The Achilles tendon is the most frequent site of involvement, and both sides are affected in half of the cases. In addition ruptures of the biceps, rotator cuff, triceps, flexor tendon sheath of fingers and thumb, patellar tendon, supraspinatus tendon, quadriceps, and subscapularis have been also reported *(4)*.

To the best of our knowledge, this is the first description of an adductor longus tendon rupture due to ciprofloxacin.

The incidence of fluoroquinolone-induced tendon injury in healthy population ranges from 0.14% to 0.4% *(8)*, but it is increased in patients with concomitant pathologic conditions. Predisposing factors include renal failure, haemodia lysis, renal transplant, corticosteroids, diabetes mellitus, hyperparathyroidism, rheumatoid arthritis, gout, sports activity, advanced age, magnesium deficiency, peripheral vascular disease and diuretics *(6)*.

Although the onset of symptoms typically occurs within 12 weeks, the first symptoms may appear as early as 2 hours after the first dose and as late as 6 months after treatment discontinuation *(4)*. The majority of cases occurred with a perfloxacin dosage of 800 mg daily and ciprofloxacin dosage of 500 mg daily.

Usually quinolone induced tendinitis starts with sharp pain occurring spontaneously with walking or palpation, accompanied by oedema and erythema. When tendon rupture occurs, the patient feels a snap, following which the pain diminishes. The clinical diagnosis may be confirmed by MRI which reveals fiber discontinuity, oedema and haemorrhage.

When the rupture affects the adductor longus tendon, conservative therapy with physiotherapy is recommended. In athletes acute repair with suture anchors has been suggested *(9)*.

The pathogenesis of fluoroquinolone-induced tendinopathy remains unknown, but seems to be multifactorial. Studies have implicated ischaemic, toxic, and matrix degrading processes. Jorgensen *et al* suggested degenerative lesions, fissures, interstitial oedema without cellular infiltration, necrosis, and neovascularisation due to an ischaemic process *(3)*. Beuchard *et al* described thickening of the vasculature, hyaline deposits, and inflammatory infiltration without degenerative lesions, induced by ischaemia *(1)*. Necrosis has also been noted in patients who have received norfloxacin and ciprofloxacin *(2)*. Le Huec *et al* suggested direct toxicity on collagen fibers because of the rapid onset of tendon injury *(5)*. In an *in vitro* study, Williams *et al*. showed decrease in fibroblast proliferation and collagen and proteoglycan synthesis, and enhanced cytokine and metalloprotease activity in the tendons after exposure of tendon tissue to ciprofloxacin *(10)*.

When tendonitis develops during fluoroquinolone therapy, the physician should consider the possible association with fluoroquinolones. Special care must be taken when prescribing fluoroquinolones to patients at risk. Discontinuation of the medication and immobilisation of the affected muscle should be instituted to avoid tendon rupture.

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