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Levofloxacin-induced gastrocnemius tendon rupture: a case report

Sara Ileri[1*]

Abstract

Background Fluoroquinolone group antibiotics are frequently used in various infections such as urinary system, respiratory system, skin, and soft tissue infections owing to their broad spectrum and good antibacterial activity. Various tendon pathologies, especially tendinopathy, have been reported in literature most commonly in the Achilles tendon, but gastrocnemius tendon rupture has never been documented.

Case presentation A 58-year-old Caucasian white female patient had knee and calf pain that started 10 days after levofloxacin treatment. Various differential diagnoses were excluded, and finally a knee magnetic resonance image scan was performed owing to the patient's history of diabetes and levofloxacin use 10 days prior. A significant effusion was detected in the lateral gastrocnemius muscle, and millimetric hypointense free tendon fragments were seen within the effusion. Therefore, gastrocnemius tendon rupture was diagnosed, and bed rest, extremity elevation, 900 mg acetaminophen, and cold application therapy were provided. During follow-up, the patient's extremity pain subsided, and walking function improved.

Conclusion By presenting this case, we aim to remind clinicians to keep in mind the rare tendon rupture possibilities other than Achilles tendon during fluoroquinolone group antibiotic therapy.

Keywords Fluoroquinolones, Tendon rupture, Risk factors, Tendinopathy prevention

Introduction

Fluoroquinolones (FQ), first used in the 1960s, are favorable antibiotics in clinic treatment of airway and urinary tract infections because of their broad spectrum; however, they have severe side effects—tendinopathy and tendon rupture. This adverse effects can occur sometimes within hours after initial treatment, sometimes 6 months after withdrawal. FQ-induced tendinopathy was first reported in 1983; since then, more than 100 cases have been published [1]. However, specific gastrocnemius tendon rupture has never been documented.

The underlying pathophysiology of FQ-induced tendinopathy is not entirely known [2].

Most cases have been associated with corticoid use; diabetes mellitus; vascular and rheumatic conditions; smoking; steroids; repetitive stress; renal diseases; and anything related to peripheral vascular disease [3, 4].

Patient's age and sex are also considered predisposing risk factors [6].

The incidence rate for tendinopathy is low, ranging from 0.1 to 0.01%, and the incidence rate for tendon rupture is even lower, below 0.01% [5].

However, predisposing factors facilitate encounters with tendon pathologies more frequently.

The aim of this case report is to present the challenging diagnosis of a rupture of gastrocnemius tendon in a patient with diabetes owing to the uncommon nature of the presentation and location of a FQ-induced tendinopathy.

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Case description

A 58-year-old Caucasian white female patient was admitted to the emergency department with sudden onset of knee and calf pain after walking for 1 hour during her routine morning exercise. The patient had no previous history of trauma. In her anamnesis, tobacco use, chronic steroid use, repetitive stress activities, or signs of peripheral vascular disease were not stated, but it was learned that she used levofloxacin 500 mg for 7 days due to upper respiratory tract infection 10 days prior.

Aside from primary hypertension and diabetes, her past medical history was noncontributory. Medications included insulin and an angiotensin-converting enzyme inhibitor (10 units of subcutaneous glargine insulin per day and 10 mg of oral ramipril per day).

The patient had type 1 hypertension and diabetes, and she was also using insulin and an angiotensin-converting enzyme inhibitor.

Since Homan's sign was positive on physical examination, venous Doppler ultrasound was performed to rule out deep vein thrombosis. No pathologic findings were detected on ultrasound. Biochemical tests of the patient, including inflammatory tests such as C-reactive protein (CRP), sedimentation, and hemogram were within normal limits apart from slightly high glucose and HbA1c levels (fasting glucose, 145 mg/dl; HbA1c, 8%).

Grade 1 gonarthrosis was detected on knee radiograph, so magnetic resonance imaging was planned for the patient. In T2-weighted axial, coronal, and sagittal sections, partial loss of tendon integrity compatible with partial rupture at the right lateral gastrocnemius head and popliteus tendon and effusion and millimetric free tendon fragments within the effusion were observed. The effusion was located primarily in the lateral head of gastrocnemius and extended to the neighborhood of the peroneal nerve and biceps femoris (Figs. 1, 2, and 3). Partial tendon rupture of gastrocnemius was diagnosed. Surgical consultation was initially obtained, 900 mg/day acetaminophen for 7 days was prescribed, and cold compression therapy was applied as the conservative treatment.

In addition, bed rest and extremity elevation were recommended for 15 days. During 2 weeks of the follow-up period, a rehabilitation protocol was applied, and the patient's extremity pain subsided. Her walking function improved with a splint and a controlled ankle motion (CAM) boot.

Discussion and conclusion

Levofloxacin-induced tendinopathy and/or rupture, other than Achilles tendon rupture, is a rare complication, but it should be kept in mind in the differential diagnosis. Concomitant use of FQ with steroids may augment

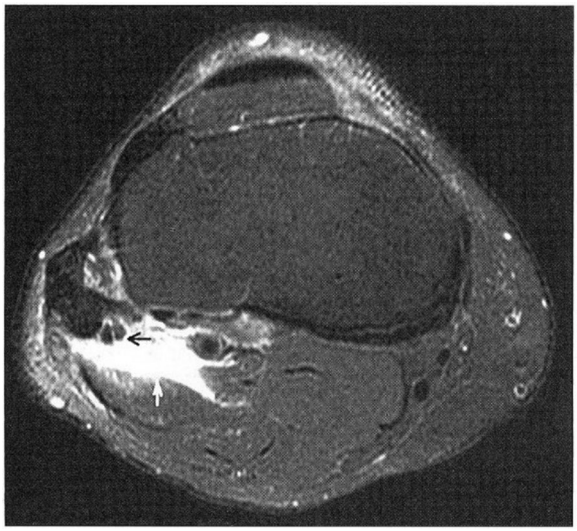

Fig. 1 Axial section of T2-weighted magnetic resonance imaging of gastrocnemius. Partial tendon loss and effusion (white arrow). Minimal tendon fragmentation within the effusion (black arrow)

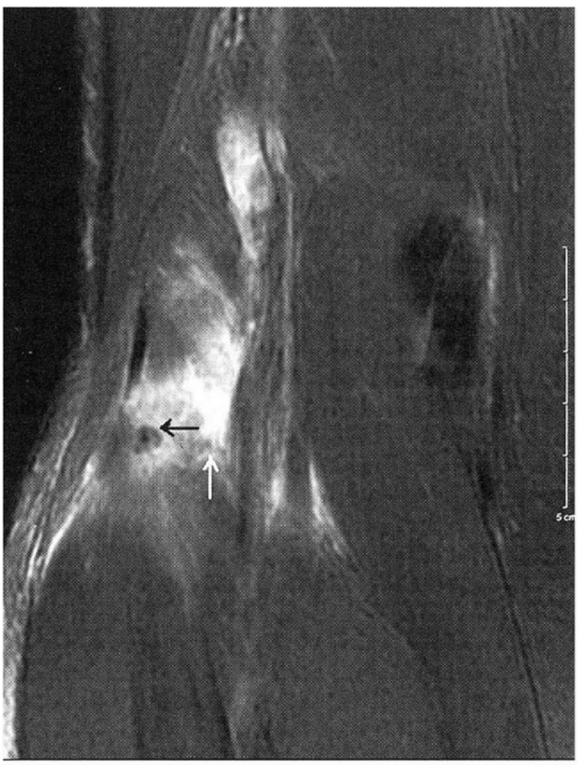

Fig. 2 Sagittal section of T2-weighted magnetic resonance imaging of gastrocnemius. Prominent effusion (white arrow) and millimetric hypointense free tendon fragments (black arrow)

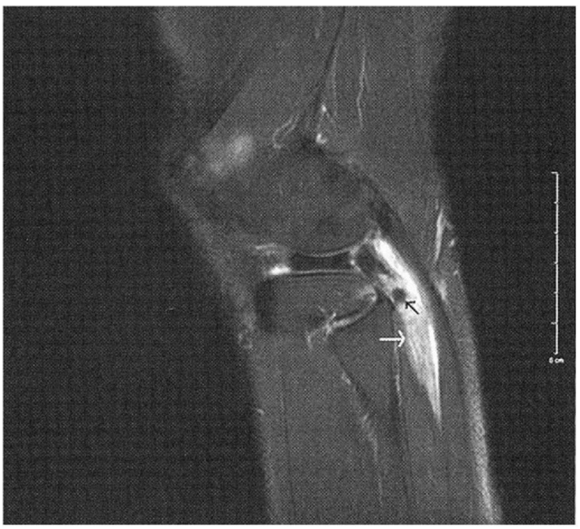

Fig. 3 Coronal section of T2-weighted magnetic resonance imaging of gastrocnemius. Evident effusion along the lateral gastrocnemius (white arrow) and millimetric tendon fragments within the effusion (black arrow)

this harmful side effect. Considering these complications, fluoroquinolones should be avoided in patients with risk factors, especially in patients with diabetes.

Tendinopathies generally occur within the range of therapeutic doses of fluoroquinolones, but, according to literature, the risk appears to be present up to 60 days following treatment with an adjusted incidence rate ratio [aIRR] of 1.61, 95% confidence interval (CI) 1.25–2 for any tendon rupture and aIRR 3.14, 95% CI 2.11–4.65 for Achilles tendon and unfortunately increases by ~6% with each day of current exposure. People with prior tendon rupture, comorbidity, increased body mass index (BMI), diabetes mellitus, and individuals prescribed oral corticosteroids were at an increased risk of tendon rupture [6].

In 1995, the US Food and Drug Administration (FDA) updated fluoroquinolone product labeling with a warning about the possibility of tendon rupture [7].

In addition, it conducted a review of disabling and potentially irreversible serious side effects of fluoroquinolones, resulting in a restriction of their use in less severe infections [8].

The underlying pathophysiology of FQ-induced tendinopathy is not fully understood. Fluoroquinolones have toxic effects on collagen chelation of magnesium and free-radical formation and may impair the integrity of tendons. The most reported fluoroquinolones are ciprofloxacin pefloxacin, but levofloxacin, ofloxacin, and norfloxacin have also been reported.

In our case, diabetes mellitus was the major predisposing factor. Therapy duration also contributed to the process of tendinopathy. Atypical localization complicates the diagnosis, but a proper anamnesis and relevant imaging methods can shed light on the underlying etiology.

Therefore, clinicians should be aware of this harmful side effect even in a rare tendon such as gastrocnemius and advise patients to stop the medication when they experience tendinopathy-related symptoms.

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Author contributions
SYI diagnosed the case and wrote and revised the manuscript.

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Availability of data and materials
The supporting data can be supplied by the author via email upon reasonable request.

Declarations

Ethics approval and consent to participate
Not applicable. Informed consent of the patient was obtained before the manuscript preparation. This study did not require ethical board approval because it was a case presentation.

Consent for publication
Written informed consent was obtained from the patient's next-of-kin for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The author has no conflict of interest to declare.

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