Levofloxacin-Induced Tendon Rupture: A Case Report and Review of the Literature

Liana Gold, ARNP, MSN, and Helena Igra, MD

Treatment with the fluoroquinolone class of antibiotics has become increasingly popular. Clinician preference for quinolones stems from their excellent gastrointestinal absorption, superior tissue penetration and broad-spectrum activity. However, this has led to widespread and indiscriminate use, affecting microbial resistance patterns and increasing drug-related morbidity. Although quinolone-induced tendonopathy and tendon rupture have been previously described in the literature, reports of tendon rupture in association with newer quinolones such as levofloxacin are now emerging. We describe a patient with levofloxacin-induced partial rupture of the Achilles tendon and review the literature, pathophysiology, predisposing factors, and treatment recommendations.

Case Report
An 88-year-old man was admitted with a recurrent draining abdominal abscess. A dye study confirmed an enterocutaneous fistula for which a right colon resection was performed. Past medical history included hypothyroidism, parkinsonism, dementia, osteoporosis, and prostate cancer previously treated with leuprolide. Medications at admission included levothyroxine, carbidopa/levodopa, donepezil, alendronate sodium, calcium, fludrocortisone, baby aspirin, and vitamin E.

Oral levofloxacin, 500 mg/day, was initiated 5 days before admission. After surgery, the patient received 500 mg of intravenous levofloxacin daily with cefazolin for 5 days and then was continued on daily oral levofloxacin at the same dosage for an additional 5 days.

Discomfort in the left ankle was noted 2 days after the initial dose of levofloxacin and progressed throughout the peri- and post-operative period. One week after discharge, he presented to the emergency department with mild, nonpitting edema of the left calf and ankle, and pain on plantar flexion. No Homan sign could be elicited. A Doppler study failed to detect a thrombus, and there was no radiologic evidence of a fracture. The patient was diagnosed with a muscle strain and discharged from the hospital with a prescription for 100 mg of celecoxib twice per day for pain and 20 mg of furosemide per day for edema.

Two weeks later, he was re-evaluated in the emergency department because of severe left leg pain, increased edema, erythema, and warmth extending to the knee, with tenderness on palpation of the Achilles tendon. A second Doppler study was negative, and the patient was admitted for a presumed cellulitis that resolved rapidly on intravenous cefazolin. However, he continued to complain of ankle pain on weight bearing.

We suspected a quinolone-induced tendonopathy, so magnetic resonance imaging (MRI) was performed, which confirmed a 2-cm segment of partial tearing of the Achilles tendon, approximately 4 cm above the calcaneal insertion with surrounding soft tissue edema (Figure 1). Because the patient was not a good surgical candidate, a below-the-knee ankle-foot orthosis was applied for 3 months. He received regular physical therapy while remaining non–weight-bearing at home. Full recovery ensued without any sequelae of tendinopathy.

Discussion
Although quinolone-induced tendon rupture has been described in the literature, case reports of complete or partial tendon rupture specifically attributed to levofloxacin are rare thus far. Quinolone-induced tendonopathy was noted as early as 1983; Bailey et al reported a norfloxacin-related tendinitis in a patient with kidney failure. The first case of a tendon rupture associated with ciprofloxacin was reported in 1987. A pefloxacin-related

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From the Departments of Internal Medicine (LG) and Dermatology (HI), Mount Sinai Medical Center, Miami Beach, Florida, and Private Practice of Dermatology, Miami Beach, Florida. Address correspondence to Helena Igra, MD, 4045 Sheridan Ave, #253, Miami Beach, FL 33140 (e-mail: drhelena@the-beach.net).
Figure 1. MRI of a 2-cm segment of an Achilles tendon partial tear, 4 cm above the calcaneal insertion with surrounding soft tissue edema.

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Patient to a quinolone-related tendon rupture include advanced age, prior tendonopathy, magnesium deficiency, hyperparathyroidism, diuretic use, peripheral vascular disease, rheumatoid arthritis, diabetes mellitus, and strenuous sports activities.1,11,18 The patient in this case report noted symptoms of tendonitis within days after oral levofloxacin was initiated. MRI confirmed the diagnosis of tendon rupture 3 weeks later. His risk factors included advanced age, steroid therapy, a brief course of diuretics, and prolonged exposure to levofloxacin.

Conclusion
The deleterious effects of fluoroquinolones on tendons have been documented since the 1980s. A concomitant rise in tendon rupture incidence has been observed during this same time span.19,20 With the increasing use of levofloxacin and other quinolone antibiotics, we should expect to encounter a growing number of patients experiencing tendonopathy. Patients presenting with joint tenderness and swelling, especially those in a high-risk category, should be questioned as to quinolone use dating as far back as 6 months or more. Discontinuation of the medication and immobilization of the affected joint should be foremost. Preventative measures include avoiding indiscriminate use of quinolones, recognition of risk factors, and adherence to renal dosing. Finally, an emphasis on patient awareness can further reduce the morbidity associated with quinolone-induced tendonitis and/or rupture by prompting earlier presentation, evaluation, and intervention.

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References