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ACHILLES TENDINITIS AS A RARE COMPLICATION OF CIPROFLOXACIN TREATMENT – CASE REPORT AND CURRENT RECOMMENDATIONS

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ABSTRACT

Fluoroquinolones are broad-spectrum antibiotics widely prescribed for respiratory, urinary, gastrointestinal, and systemic infections. Despite their efficacy, these agents have been increasingly linked to tendinopathy and tendon rupture, most frequently affecting the Achilles tendon. The underlying mechanisms are thought to involve impaired collagen synthesis, extracellular matrix degradation, and tenocyte apoptosis. We present the case of a 51-year-old female lung transplant recipient who developed acute bilateral Achilles tendinitis after three days of intravenous ciprofloxacin therapy. Prompt discontinuation of the antibiotic, coupled with conservative management using rest and topical aluminum acetate gel, led to complete symptom resolution within one week. This case illustrates the potential for rapid onset of fluoroquinolone-induced tendinopathy, particularly in high-risk populations, and underscores the importance of early recognition and withdrawal of the offending drug. Clinicians should remain vigilant when prescribing fluoroquinolones, reserving their use for situations where safer alternatives are unavailable.

KEYWORDS

Achilles Tendinopathy, Fluoroquinolones, Tendinitis, Ciprofloxacin

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Introduction

Fluoroquinolones, including ciprofloxacin, constitute a widely prescribed class of antibiotics characterized by high tissue penetration and broad-spectrum activity against numerous pathogens. Since the 1990s, multiple reports have documented their association with tendon injuries—most notably involving the Achilles tendon—manifesting as tendinitis or rupture.

The magnitude of this adverse effect has prompted major regulatory authorities, including the U.S. Food and Drug Administration (FDA) and the European Medicines Agency (EMA), to implement substantial restrictions and warnings in official product labeling. This article presents a case of ciprofloxacin-associated Achilles tendinitis, accompanied by a review of current evidence regarding its epidemiology, risk factors, pathophysiological mechanisms, and clinical management.

Fluoroquinolones are a class of synthetic antibiotics with a broad spectrum of activity. Currently used in clinical practice are agents such as: ciprofloxacin, levofloxacin, moxifloxacin, gemifloxacin, delafloxacin, norfloxacin, and ofloxacin. In Poland and worldwide, the most commonly prescribed are: ciprofloxacin, levofloxacin, and moxifloxacin. Fluoroquinolones exert a bactericidal effect by inhibiting type II enzymes - DNA gyrase and topoisomerase IV. This leads to the induction of irreversible, toxic double-strand breaks in bacterial chromatin, resulting in cell death. This mechanism is selective, with minimal interaction with human enzymes. Fluoroquinolones are used to treat a wide range of infections, including lower and upper respiratory tract infections (pneumonia, bronchitis), urinary tract infections, gastrointestinal infections, skin and soft tissue infections, bone and joint infections, peritonitis, sepsis, typhoid fever, sexually transmitted infections, as well as tuberculosis and anthrax.

Some agents (e.g., moxifloxacin, levofloxacin) are also used off-label in difficult cases of drug-resistant tuberculosis [9].

The most common adverse effects include dyspepsia, nausea, diarrhea, headache, dizziness, insomnia, and skin rashes. Other less common but serious complications include tendinopathy and risk of tendon rupture (particularly the Achilles tendon), neurological disorders such as peripheral neuropathy (which may be permanent), dizziness, psychosis, seizures, and exacerbation of myasthenia gravis; cardiac rhythm disturbances including QT interval prolongation, torsades de pointes, and, rarely, life-threatening arrhythmias; phototoxic reactions; hematologic effects (leukopenia, anemia); and psychiatric effects (depression, suicidal ideation) [10].

Pathophysiology and literature review:

The basic pathophysiology of tendinopathy is not fully understood. Animal and in vitro studies have proposed several possible mechanisms. Ciprofloxacin may affect fibroblast metabolism within tendon structures by reducing collagen synthesis and increasing degradation of the extracellular matrix [4]. The chelating properties of fluoroquinolones may also disrupt physiological interactions between cells and the extracellular matrix [5]. There is also evidence that fluoroquinolones increase apoptosis in human tenocytes. Monolayers of human tenocytes were incubated with ciprofloxacin or levofloxacin at various concentrations (0, 3, 10, 30, and 100 mg/L of medium) for up to 4 days. At concentrations achievable during fluoroquinolone therapy, 3 mg/L of ciprofloxacin significantly reduced type I collagen content; similar changes were observed at concentrations of 3 mg/L ciprofloxacin or 10 mg/L levofloxacin for β(1)-integrin receptors. The effects intensified with higher concentrations and longer incubation times. In addition, a time- and concentration-dependent increase in matrix metalloproteinases, as well as in the apoptosis marker activated caspase-3, was observed. Fluoroquinolones induced typical changes such as chromatin condensation in the nucleus, swelling of cellular organelles, apoptotic bodies, and vesicles on the cell membrane [6].

The association between fluoroquinolones and tendon injury has been confirmed in numerous observational studies and meta-analyses. One meta-analysis included fifteen studies. Fluoroquinolone treatment was associated with an increased risk of ATR (Achilles tendon rupture) (OR 2.52, 95% CI 1.81–3.52, $p < 0.001$, $I^2 = 76.7\%$), an increased risk of AT (Achilles tendinitis) (OR 3.95, 95% CI 3.11–5.01, $p < 0.001$, $I^2 = 0\%$), and an increased risk of ATD (Achilles tendon disorders) (OR 1.98, 95% CI 1.62–2.43, $p < 0.001$, $I^2 = 84.5\%$). Initial risk estimates remained statistically significant among patients aged ≥60 years. Risk estimates were not significantly altered after accounting for concomitant corticosteroid use or methodological study quality assessment. Analysis by type of fluoroquinolone was possible only for ATR, showing that ofloxacin and norfloxacin increased the risk of this complication, whereas ciprofloxacin and levofloxacin did not. The results of this meta-analysis confirm the risk of tendon injury associated with fluoroquinolone use. Advanced age and concurrent corticosteroid therapy appear to be additional risk factors for tendinopathy [1].

Risk factors:

The best-documented risk factors include advanced age, concomitant kidney disease or organ transplantation, as well as concurrent glucocorticoid therapy, which further increases the risk of tendon injury. According to EMA recommendations, fluoroquinolones should be avoided in combination with steroids in at-risk patients. Due to serious, potentially long-lasting adverse effects, the EMA restricted the indications for fluoroquinolone use (2018/2019) and recommended immediate discontinuation of therapy at the first signs or symptoms involving muscles, tendons, or joints [2]. The U.S. Food and Drug Administration updated its"Boxed Warning" (the highest level of safety-related warning that can be assigned to a medication). These warnings are intended to draw the consumer's attention to the major risks associated with the drug and recommend reserving fluoroquinolones for situations where no alternative treatment options are available [3].

Clinical symptoms:

Swelling may occur in the tendon area, along with tenderness on palpation and redness of the overlying skin. Discontinuity of the tendon is often palpable. The Thomson test and tendon percussion test are performed, as well as assessment of Hoffa's sign, gait abnormalities, and the ability to rise onto the toes. Ultrasound imaging is useful in the evaluation of such injuries [8].

Management and treatment:

In cases where antibiotic therapy is truly necessary, fluoroquinolones should be avoided in patients with identified risk factors. Individuals with a history of fluoroquinolone-associated tendinopathy should not be prescribed drugs from this class [7]. In clinical practice, for fluoroquinolone-induced Achilles tendinitis, the primary intervention is discontinuation of the antibiotic; the decision to initiate alternative antibiotic therapy is based on microbiological indications. The inflammation itself should be managed with cold compresses (ice therapy) [7] or aluminum acetate gels, which help reduce local inflammation and pain. Limiting movement and unloading the affected limb is also essential. After the acute phase of inflammation has subsided, gradual rehabilitation - such as eccentric exercises, can be introduced. Surgical tendon repair is often required in cases of significant damage or when there is no improvement after conservative treatment. The choice of management approach always depends on the patient's profile and the preferences of the treating center.

Case report

A 48-year-old female patient was admitted to the Department of Internal Medicine at the Regional Clinical Hospital No. 2 in Rzeszów with leukopenia, anemia, and symptoms of lower respiratory tract infection. She had undergone bilateral lung transplantation in December 2024 for hypocomplementemic urticarial vasculitis syndrome (HUV) and chronic obstructive pulmonary disease with emphysema.

Her past medical history included: arterial hypertension, proteinuria, irritable bowel syndrome, gastroesophageal reflux disease, and thoracic spine spondylosis. Maintenance therapy consisted of: tacrolimus 5 mg twice daily, prednisone 10 mg once daily, mycophenolate mofetil 720 mg twice daily, valganciclovir 450 mg once daily, and cotrimoxazole 480 mg every other day.

Due to suspected infection of unclear etiology, empirical intravenous ciprofloxacin was initiated at 400 mg every 12 hours. After approximately three days, the patient reported bilateral ankle pain, initially attributed to her regular physical exercise. The following day, pain intensified, and erythema developed along both Achilles tendons. Ciprofloxacin-induced tendinopathy was suspected, and the antibiotic was promptly discontinued.

Ceftriaxone (1 g every 12 hours) was initiated, and an orthopedic consultation was requested. Ultrasound revealed bilateral swelling of Kager's fat pad with hyperemia extending into the proximal third of the Achilles tendon, as well as a small effusion in the deep bursa on the right side. The diagnosis of fluoroquinolone-associated Achilles tendinitis was confirmed. Conservative management with strict rest, cessation of physical activity, and local cooling gel containing aluminum acetate was recommended.

After two days of therapy, erythema subsided, and the patient reported a reduction in pain. Concurrently, active cytomegalovirus infection was diagnosed and treated with intravenous ganciclovir, with no relation to the tendinopathy. Symptoms resolved completely within seven days of ciprofloxacin withdrawal, without sequelae.

Discussion

Fluoroquinolone-induced tendinopathy is a well-documented but relatively rare adverse effect, most commonly affecting the Achilles tendon. The pathophysiology is not fully understood but may involve oxidative stress, collagen degradation, and impaired tendon repair mechanisms. Risk factors include: renal impairment, solid organ transplantation, and concomitant corticosteroid therapy - all relevant in this case.

Our patient developed tendinitis after only three days of ciprofloxacin exposure, highlighting the potential for early onset. Prompt recognition and discontinuation of the offending drug are crucial to prevent tendon rupture. Conservative management is usually effective, although recovery may take weeks to months in some cases. In this patient, symptoms resolved within one week, without long-term consequences.

This case underscores the need for caution when prescribing fluoroquinolones in transplant recipients, particularly those on long-term corticosteroids. Alternative agents should be considered whenever possible, and patients should be informed of the potential risk of tendon-related adverse events.

Conclusions

Ciprofloxacin-induced Achilles tendinitis should be considered in transplant recipients presenting with acute ankle pain and erythema, particularly in the context of corticosteroid therapy. Early recognition, discontinuation of the fluoroquinolone, and conservative management are essential to avoid tendon rupture and long-term morbidity.

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