

Case Report

Fluoroquinolone Induced Isolated Motor Neuropathy

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Abstract

Fluoroquinolone antibiotics are known to cause spontaneous tendinopathy and tendon rupture. Fluoroquinolones are not widely appreciated to cause neuropathy. Presented is a case of isolated motor neuropathy with prominent weakness and fasciculations caused by fluoroquinolone use. Recovery was incomplete at 18 months. Central nervous system (CNS) toxicity is well documented with fluoroquinolone use. Peripheral sensory neuropathy has been infrequently described; however, no well-documented case of isolated motor neuropathy appears in the literature. The mechanism is likely to be a direct axonal toxicity. With the increasing use of fluoroquinolones across all medical specialties, the recognition of potential neurotoxicity is important.

Keywords

- Fluoroquinolone
- Motor neuropathy
- Tendinopathy

INTRODUCTION

Fluoroquinolone antibiotics are widely used in clinical practice. They are known to cause spontaneous tendinopathy and tendon rupture [1]. Although CNS toxicity is well documented with a frequency of 1–7% of exposures, Fluoroquinolones are not widely appreciated to cause peripheral neuropathy, despite the FDA highlighting the need for increased awareness of neuropathy caused by fluoroquinolone use [2]. Case reports and small series have described typical sensorimotor neuropathy. There have been no case reports of isolated motor neuropathy. Presented is a case of serious isolated motor neuropathy caused by fluoroquinolone use.

CASE PRESENTATION

A 57-year-old male physician presented with septic olecranon bursitis. The only concomitant medication was omeprazole. Therapy was initiated with levofloxacin 500 mg daily and neurologic symptoms developed after 6 days of treatment. He was a recreational cyclist averaging 60 miles of cycling weekly. On day 6 of treatment, the patient experienced the sudden spontaneous onset of right posterior mid-calf pain not associated with strenuous activity, followed one hour later by identical symptoms on the left side. This was followed within 12 hours by the acute onset of bilateral calf weakness associated with prominent fasciculations involving the entire lower extremity musculature and extending proximally to the distal quadriceps. Calf weakness limited ambulation distance to 100 meters. There were no sensory symptoms. Extensive laboratory evaluation including chemistries, thyroid function, muscle enzymes and inflammatory markers were all unremarkable. MRI of the calves showed partial rupture of the musculo-tendinous junction of the gastrocnemius muscle on the left and partial rupture of the

musculo-tendinous junction of the soleus muscle on the right. Within 8 weeks, musculo-tendon pain resolved, and healing was confirmed by resolution of MRI findings. EMG/NCV was normal at week 12 after symptom onset. Weakness however, persisted. There was gradual but incomplete improvement over 12 months, with approximately 85% resolution at one year. Fasciculations had not resolved by 24 months.

DISCUSSION

Spontaneous tendinopathy and tendon rupture are well described complications of fluoroquinolone use. Estimation of the risk is hampered by underreporting in pharmacovigilance programs, but tendon rupture is estimated to occur with a frequency between 4–24 cases per 100,000 exposures, with the majority of cases affecting the Achilles tendon [3]. The limited data available suggests a direct toxic effect on the tendon as opposed to an inflammatory or vascular etiology [4]. CNS effects of fluoroquinolones occur in 1–7% of exposures, with seizures and psychosis as the most common serious events [5]. Peripheral neuropathy is described far less frequently, with data limited to case reports and small series, and the mechanism is unknown. From 1987–93, 37 cases of peripheral sensory disturbances attributed to fluoroquinolone use were reported to the Swedish Adverse Drug Advisory committee [6]. Symptoms were generally described as mild and of short duration. Most cases fit the description of a symmetric sensorimotor peripheral neuropathy with prominent sensory symptoms. Cohen reported a series of patients who were identified from a posting on the Quinalone Antibiotics Adverse Reaction Forum Website [7]. A request for cases involving the peripheral nervous system garnered responses from 45 patients, who provided detailed self-reported information. Of these patients, 47% reported both sensory and motor symptoms, 44% reported sensory symptoms alone, and

only 9% reported isolated motor symptoms. One patient had concomitant tendon rupture. Onset was within 7 days in 84% of patients. In 58% of these patients, recovery was incomplete at one year. More recently, a pharmacovigilance analysis was performed between the dates of 1997 and 2012, and noted 46,257 adverse event reports submitted for fluoroquinolones. Only 1% of these were reported to be peripheral neuropathy or Guillain-Barre Syndrome [8]. 28% of these cases reported physical disability and none had recovered at the time the events were collected. The median onset of neuropathy symptoms was 4 days post drug initiation. Isolated motor neuropathy was not described.

This case precisely fits the description of typical fluoroquinolone tendinopathy with sudden onset of bilateral tendon rupture at day 6 in the absence of exertion or trauma, and MRI confirmation of tendon rupture. Additionally, and more importantly from a severity and disability standpoint, there was clinical evidence of an isolated motor neuropathy with severe weakness and fasciculations and incomplete resolution at one year. The simultaneous onset of both tendinopathy and peripheral neuropathy suggests a common underlying mechanism. The severity and duration of fluoroquinolone neuropathy may be greater than previously recognized. Additionally, although the data is seriously limited by being self-reported, there is a suggestion that peripheral nerve involvement from fluoroquinolone use may not be a rare occurrence. Given our current understanding of quinolone tendinopathy and other forms of drug induced peripheral neuropathy, the underlying mechanism may be a direct axonal toxicity.

CONCLUSION

Isolated motor neuropathy must be added to the list of

toxicities of fluoroquinolone antibiotics. In addition to the well documented tendinopathy and CNS toxicity of fluoroquinolones, peripheral nerve toxicity is likely to be much higher than previously reported, and is likely underrecognized. Given the widespread use of fluoroquinolones, increased awareness of the potential neurotoxicity is important.

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