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Case report: A delayed extensor pollicis longus tendon repair, following a rupture secondary to multifactorial causes

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Abstract

Introduction: Spontaneous tendon ruptures have been widely reported in the literature with well documented risk factors. This includes but is not limited to, long term fluoroquinolone antibiotic therapy. The fluoroquinolone family of antibiotics includes levofloxacin, ciprofloxacin and pefloxacin amongst others. The incidence of tendinopathy associated with fluoroquinolone antibiotic usage is reported as being between 0.14-0.4%, with the Achilles tendon being most commonly affected. Although this complication is reported as rare, concomitant corticosteroids treatment and end stage renal failure are both noted as independent factors that further increase the risk for tendon injury. End stage renal failure predisposes to secondary hyperparathyroidism which has also been shown to play a major role in the pathogenesis of tendon rupture

Case report: We present a rare case of Achilles tendon and Extensor Pollicis Longus (EPL) tendon ruptures following long-term fluoroquinolone antibiotic therapy in a 65-year-old patient, with the co-existing risk factors mentioned above.

Discussion: The cause of the tendon rupture in the case we present was multifactorial. The patient had a history of long term fluoroquinolone therapy for recurrent urinary tract infections. This was superimposed on end stage renal failure treated by haemodialysis for over 15 years and a subsequent development of secondary hyperparathyroidism. It is important to realise that although literature may suggest individual causes for tendon rupture, the cumulative effect of these factors increases this risk even further as evidenced by the patient's history of multiple tendon rupture.

Conclusion: Although tendon rupture is commonly due to isolated traumatic injury, it is important to be aware of other causes – especially in the presence of multiple risk factors. It is reasonable to consider alternative antibiotic therapies in this cohort, as the risk of tendon rupture from fluoroquinolone will be compounded upon existing risk factors.

Keywords

quinolone; tendon rupture; chronic renal failure; hyperparathyroidism; extensor pollicis longus

Introduction

Spontaneous tendon ruptures have been widely reported in the literature. There have been many causes attributed to this phenomenon which includes uraemic tendinopathy secondary to regular haemodialysis as a result of End Stage Renal Failure (ESRF) [1,2]. This cohort exhibits various other risk factors which contribute to this presentation; secondary hyperparathyroidism, in particular, has been shown to play a major role in the pathogenesis of tendon rupture [3]. Furthermore, medications such as the fluoroquinolone group of antibiotics and corticosteroids are well known to increase the rate of tendinopathy [4].

The fluoroquinolone family of antibiotics includes levofloxacin, ciprofloxacin and pefloxacin amongst others. They are commonly used to treat a wide range of infections, mainly respiratory tract and urinary tract infections. The incidence of tendinopathy associated with fluoroquinolone antibiotic usage is reported as being between 0.14-0.4%, with the Achilles tendon being most commonly affected [5]. Although this complication is reported as rare, concomitant corticosteroids treatment and increasing age are both noted as independent factors that further increase the risk for tendon injury [6]. There is a spectrum of presenting symptoms from mild pain around the tendon, to complete rupture requiring surgical intervention.

We present a rare case of Achilles tendon and Extensor Pollicis Longus (EPL) tendon ruptures, due to a multifactorial aetiology comprising of chronic fluoroquinolone antibiotic therapy in a patient with multiple pre-existing risk factors.

Case Report

We present a case of a left-handed, 65-year-old, Caucasian gentleman who presented in August 2015 to the plastics outpatients trauma clinic as a routine referral. During the initial assessment it was clinically deemed that he had ruptured a tendon in his left thumb. His extensive past medical history included: polycystic kidney disease, chronic renal failure of over 15 years requiring haemodialysis via an arterio-venous fistula, ulcerative colitis, primary sclerosing cholangitis, ischaemic heart disease, inflammatory arthropathy and prostatic adenocarcinoma. The only medication of note was long-term (5 years) ciprofloxacin on an intermittent basis for recurrent urinary tract infections. The patient had no known allergies. He was also noted to have a history of untreated Achilles tendon rupture. Subsequent ultrasound imaging confirmed a distal Extensor Pollicis Longus (EPL) rupture in his left hand. (See Figures 1 [longitudinal view] and *2* [transverse view], the red arrows indicating the location of the rupture.) Following this, he was advised to wear a volar thumb splint for a period of 4-6 weeks and to return to clinic to assess any improvements or the need for further treatment.

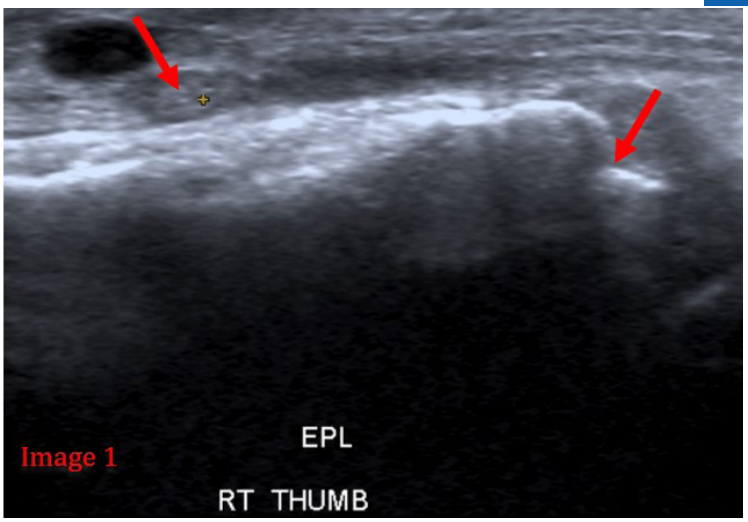

Figure 1: longitudinal view of the EPL tendon, with red arrows indicating the location of the rupture.

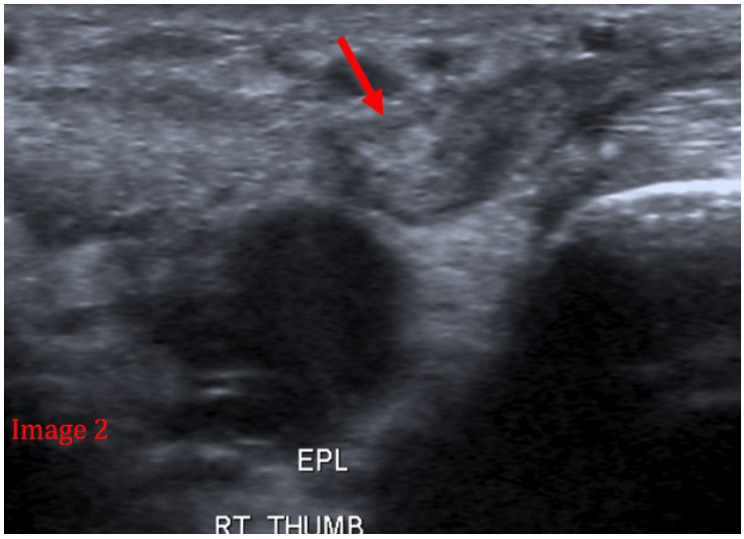

Figure 2: view of the EPL tendon, with red arrows indicating the location of the rupture.

A follow-up clinic appointment at 2 months revealed that the patient had no functional improvement and was still unable to extend his thumb. Understandably, this was causing him increasing frustration, as he had difficulty with daily activities. He denied any other sensorimotor symptoms. During this appointment the patient was advised of the possible options for his left EPL tendon repair and the risks of delayed treatment. He then underwent a primary suture repair in October 2015. Surgical intervention was delayed until this point, owing to the initial underlying inflammation.

The patient underwent a repair of his EPL tendon with a transfer of the Extensor Indicis Propius (EIP) tendon. The distal end of the EPL tendon was revealed by a sigmoid incision at its distal attachment. The operation was complicated by various factors, primarily the presence of an Arterio-Venous (AV) fistula in proximity to the operative field. This prevented the use of a tourniquet and the limited the extent of dissection in view of tracing the proximal end of the EPL tendon. The EIP tendon was found via an incision near its distal end and divided at a level proximal to the metacarpophalangeal joint and tunneled to lie in line with the distal end of the EPL. The free ends were sutured in place and the tendon function was tested intra-operatively.

Discussion

In this specific case, there are multiple relevant predisposing factors. The patient is diagnosed with ESRF requiring dialysis, with subsequent development of secondary hyperparathyroidism (current PTH levels are within normal range: 31pg/ml due to treatment with a calcimimetic). In addition, the patient had only stopped his long-term steroid therapy and ciprofloxacin before his initial presentation. It is important to realise that although literature may suggest individual causes for tendon rupture, the cumulative effect of these factors increases this risk even further as evidenced by the patient's history of multiple tendon rupture.

Gao et al. demonstrated that tendinopathy was more common in haemodialysis patients although rupture was a rare end-point [7]. Case series, reports and literature reviews suggest that end stage renal failure and haemodialysis are major predisposing factors for tendinopathy and tendon rupture [1,2]. Other factors also significantly contribute; the duration of dialysis (15 years in the presented case), secondary hyperparathyroidism, accumulation of beta-2-microglobulin, corticosteroids, quinolone antibiotics, malnutrition and chronic acidosis have all been identified [8].

It is postulated that secondary hyperparathyroidism is important in the process of tendon rupture [3]. The elevated levels of parathyroid hormone seen in ESRF patients leads to sub-tendinous bone resorption at the insertion sites. This leads to a weakened insertion site with eventual tendon rupture [9]. Many case reports and series suggest secondary hyperparathyroidism as the most important predisposing factor in development of tendon ruptures, however without evidence from trials or case-control studies, the sole effect of untreated secondary hyperparathyroidism is difficult to ascertain, nonetheless patients with secondary hyperparathyroidism should be optimally managed with physicians exercising caution with fluoroquinolone and steroid use in patients with background risk factors.

Moreover, the use of high dose fluoroquinolone is known to precipitate tendinopathy [4], with one study suggesting a dose dependent relationship [10]. The Achilles tendon is the most common tendon affected and was the first tendon to rupture in the presented patient [11]. Fluoroquinolone induced tendinopathy was initially published by Bailey et al. and has since gained increased recognition as an independent risk factor [12]. Pefloxacin and ciprofloxacin are most frequently implicated, but tendon injury has also been reported from others within the fluoroquinolone group [6,13]. The underlying pathophysiology of the above process is not fully understood but has been linked to local ischemic and mechanical stressors [14].

Long-term corticosteroid use has been associated with tendon rupture, with one study concluding that the risk of Achilles tendinopathy was over 6-fold in patients above the age of 60 and on fluoroquinolone/corticosteroid therapy when compared with non-fluoroquinolone users of the same age group [10]. In fact, the risk of Achilles tendinopathy increases 3-fold when comparing concomitant corticosteroid and fluoroquinolone use to fluoroquinolone alone [14]. Therefore, it would be valid to consider alternative antibiotic therapy in these patients.

Diagnosis for tendinopathy is mainly clinical and supported by radiological findings through sonography or Magnetic Resonance Imaging (MRI), of which sonography has a higher sensitivity but lower specificity [15]. This may demonstrate tendon thickening and hypoechoic areas within the tendon.

An important consideration of fluoroquinolone and corticosteroid tendinopathy is that it is frequently abrupt in onset and typically presents within a median of 6 days from start of antibiotic therapy. However, this is reported to be in the range of days, to even years [16]. Fluoroquinolone tendinopathies in particular, are shown to be bilateral in over 50% of cases [17]. Consequently, this highlights the importance of bilateral clinical examination and imaging upon suspicion of tendon rupture in this subset of patients. Features which may be present on the contralateral side to the rupture, include pain and tendon swelling. A series of case reports, investigating the effectiveness of prophylactic tendon decompression on the contralateral side to a tendon rupture, exhibits positive outcomes in the 3 cases outlined [18]. Their results support prophylactic release if clinical and radiological evidence suggests the tendon may be at risk.

Conclusion

Although tendon rupture is commonly due to isolated traumatic injury, it is important to be aware of other causes – especially in the presence of multiple risk factors. It is reasonable to consider alternative antibiotic therapies in this cohort, as the risk of tendon rupture from fluoroquinolone will be compounded upon existing risk factors. Furthermore, in the cohort of patients on fluoroquinolone therapy presenting with tendon rupture, investigation of the contralateral tendon may be indicated. This may reveal scope for prophylactic management, ultimately preventing the complications of a second tendon rupture.

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