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A rare cause of radial sided wrist pain: Atraumatic rupture of flexor carpi radialis

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Abstract
Background: atraumatic flexor carpi radialis (FCR) rupture is rare in the absence of direct trauma. In the majority of reported cases, the patient typically complains of pre-existing volar or radial sided wrist pain and swelling, followed by an acute 'giving way' sensation, with either loss of wrist flexion strength or an increase in regional wrist tenderness. Atraumatic FCR rupture occurs primarily in patients who have rheumatoid arthritis and secondary carpal collapse or severe degenerative joint disease, secondary to corticosteroid injections in the FCR tendon sheath and due to osteoarthritis (OA) of the scaphoid-trapezium-trapezoid (STT) joint. Atraumatic flexor carpi radialis rupture is a rare phenomenon and should be distinguished from other pathology. A review of the literature revealed only 15 case reports published since 1959 investigating this cause of radial sided wrist pain.
Case Presentation: MC, a 57 year old Caucasian female, presented to her GP after the sudden onset of pain in the right forearm while cooking. Examination revealed significant pain and swelling over the distal FCR tendon. Wrist flexion was greatly diminished in strength. Ultrasound revealed a full thickness tear of the FCR tendon at the myotendinous junction, with 31mm of retraction. The patient had an ultrasound some months earlier which revealed mild OA and synovitis in the 1st carpometacarpal joint (CMCJ), along with tenosynovitis of the FCR. The patient had been experiencing discomfort in the radial side of the right wrist, so a steroid injection of the FCR tendon was organised, with the aim of pain relief. At the time of injection, the radiologist proceeded with a base of thumb injection with a combination of Celestone and 0.75% Naropin, which was tolerated well. Three weeks after this injection, MC presented to the doctor with the symptoms described above, leading to a repeat ultrasound, which confirmed complete FCR rupture.
Conclusions: intra-sheath steroid injections are a known risk factor for all tendon ruptures, and this has been reported in the literature for the FCR previously. However, FCR tendon rupture incited by an adjacent joint injection appears to be a novel event.

Keywords: flexor carpi radialis (FCR); tendon rupture; wrist pain; joint anatomy; arthritis

Introduction

Background

Atraumatic flexor carpi radialis (FCR) rupture is rare in the absence of direct trauma or other antecedent events, such as a fall on an outstretched hand. In the majority of reported cases, the patient typically complains of pre-existing volar or radial sided wrist pain and swelling, followed by an acute 'giving way' sensation, with either loss of wrist flexion strength or an increase in regional wrist tenderness. Clinical examination usually demonstrates a visible and palpable tendon defect, and tenderness upon direct palpation along the length of the tendon. In cases of surgical exploration, fibrous neotendon or a gap filled with fibrous tissue is apparent and correlates well with advanced imaging, namely ultrasonography (USS) and magnetic resonance imaging (MRI). Atraumatic FCR rupture occurs primarily in patients who have rheumatoid arthritis and secondary carpal collapse or severe degeneration, secondary to corticosteroid injections in the FCR tendon sheath and due to osteoarthritis (OA) of the scaphoid-trapezium-trapezoid (STT) joint, which is thought to be secondary to a series of direct attritional microtears due to tendon abrasion against osteophytes and sharp bone surfaces [1]. Ultrasound is considered a primary radiological imaging modality for cases of suspected FCR rupture. Wrist flexor tendon ruptures may be managed non-operatively for the vast majority of patients. In this case report, a patient will be described with atraumatic FCR rupture in the presence of symptomatic OA and in the absence of any direct tendon sheath injection or inciting traumatic episode. Moreover, from an assessment of the limited studies on this cause of radial sided wrist pain, it appears that FCR tendon rupture incited by a local joint injection appears to be a novel event.

Case Presentation

MC, a 57 year old Caucasian female, presented to her GP after the sudden onset of pain in her right forearm while kneading cake icing. At this time, significant pain and swelling was noted over her distal FCR tendon. Wrist flexion was significantly diminished in strength, although functionally, the patient experienced no significant limitations in activities of daily living. Ultrasound at this time revealed a full thickness tear of the FCR tendon at the myotendinous junction, with 31mm of retraction. The muscle belly was retracted proximally. Distally, the tendon stump was degenerate and there was non-specific thickening of the empty tendon sub-sheath. All other tendons and nerves were intact. Examination revealed swelling and thickening around the STT

joint with pain and crepitus on loading, but no palpable tenderness. The patient had an ultrasound some months earlier which revealed mild OA and synovitis in the 1st carpometacarpal joint (CMCJ), along with tenosynovitis of the FCR. Moreover, investigations at that stage did not reveal flexor carpi radialis brevis or any other anomalous muscles in the distal forearm which may coexists in 2.6-8.6% of presentations; when such anatomical variants are present, which are largely asymptomatic, a good clinical response can be expected, in terms of return to normal function [16]. The patient had been experiencing pain in the radial side of the right wrist, thought to be driven FCR tendinitis, so a steroid injection of the FCR tendon sheath was organised by the patient's doctor. At the time of injection, the interventional radiologist instead recommended a CMCJ injection, which MC agreed to. The procedure, which involved administration of a combination of Celestone and 0.75% Naropin, was tolerated well and there were no immediate complications. The accuracy of the procedure was not reported in the case notes. Three weeks after this injection, the patient presented to her local doctor with worsening radial sided wrist pain, leading to a repeat ultrasound examination, which confirmed complete FCR rupture, with a significant degree of tendon retraction.

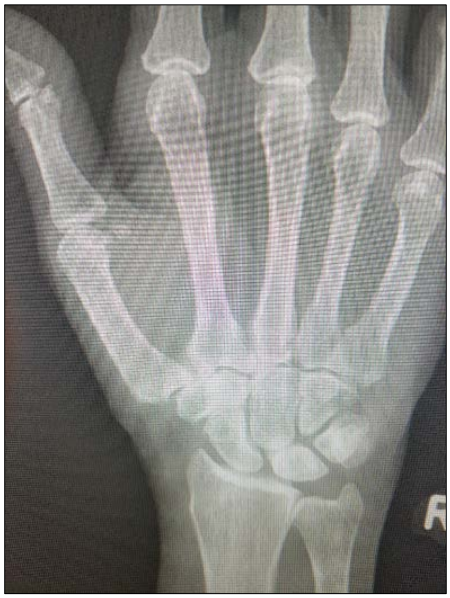

Fig 1: AP XR of the right hand demonstrating 1st CMCJ and STT arthritis, in keeping with early to moderate disease at this level.

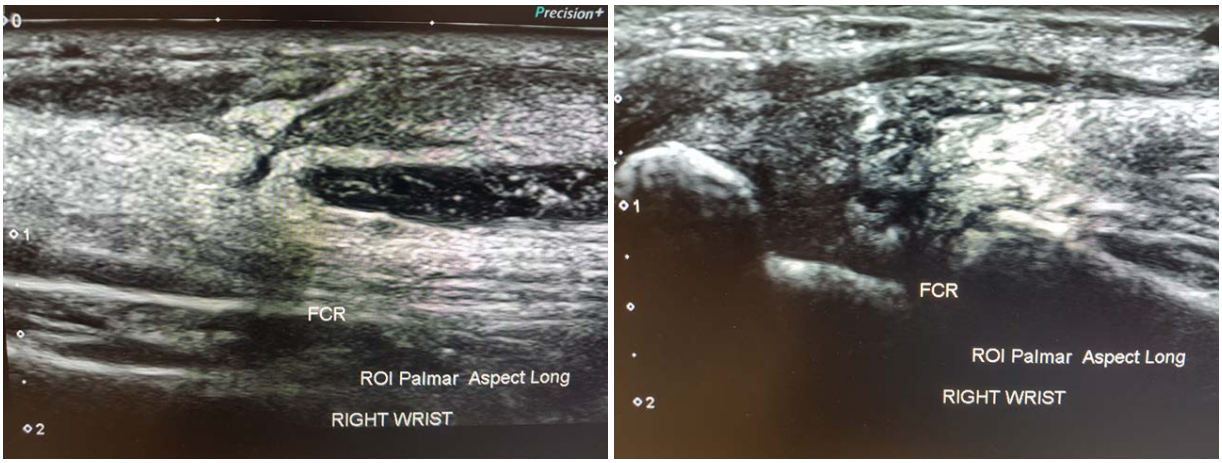

Fig 2a and 2b: dynamic ultrasound examination of the right polar wrist demonstrating a complete full thickness tear of the FCR tendon at the myotendinous junction with retraction. The other flexor and extensor tendons appear within normal limits.

This patient's medical history was largely unremarkable regarding her FCR tendon, with no previous trauma or inflammation. However, it must be noted that she was diagnosed with CLL in 2011, for which she underwent chemotherapy with fludarabine, cyclophosphamide, and rituximab. She completed four courses of this before entering remission in 2014. Her only inciting risk factors appear to have been CMCJ OA and her single steroid injection outside of the FCR tendon sheath, but around the area of clinical interest.

Discussion

The primary function of the FCR is flexion of the wrist; it also contributes to radial deviation. Loss of FCR function leads to decreased wrist flexion fatigue resistance and tripod pinch strength [14]. Anatomically, this bipennate muscle originates from the medial epicondyle of the humerus and runs laterally to the flexor digitorum superficialis (FDS) in the anterior forearm, inserting onto the base of the second metacarpal and with small attachments to the third metacarpal and tuberosity of the trapezium. The average length of the muscle is 15 cm, with a range of 12-17cm in most cadaveric series. Its tendinous part commences along with its sheath at approximately 8 cm (with a range of 6-9cm) proximal to the distal radioulnar joint (DRUJ) [13]. Flat and broad, the musculotendinous junction of the FCR tendon gradually becomes elliptical in the hand and passes in distance less than 1mm over the scaphoid tubercle, trapezium and the STT joint line; the latter of which is important due to its association with FCR tendon rupture when significantly arthritic [1]. Arthritis of the 1st CMCJ is a less likely, but still plausible cause of chronic irritation of the FCR tendon due to its more proximal location, with an average distance from tendon to joint of 3mm. The distal tendon is contained within the transverse carpal ligament and essentially constitutes its radial side wall. Moreover, the FCR tendon angulates slightly before entering the tunnel, where it is flanked by superficial and deep laminae, ramifying its supportive components [13].

As had been established, FCR rupture is a relatively rare phenomenon and should be distinguished from other pathology

causing radial sided wrist pain. A review of the literature has revealed only 15 case reports published since 1959 investigating this unique 'pain driver'. The most common factors cited were STT OA or RA, appearing as the primary contributor in five of the studies, with chronic tendon degradation proposed as the mechanism of rupture. Trauma was identified in the remaining five cases, two of which involved comminuted distal radius fractures lacerating the tendon directly, as has been previously identified for the extensor pollicis longus (EPL) tendon at the dorsal aspect of the distal radius, where direct tendon damage is incurred in the ulnar sided groove formed by Lister's tubercle [2, 3, 20]. Indeed, Vaughan-Jackson first described a mechanism of tendon rupture around the wrist in 1948, although the majority of this preliminary work focused on pathology of extensor tendons, including the EPL. Subsequently, Mannerfelt and Norman explored causes of rupture in flexor tendons of the wrist [15]. However, more than half of century later, there is no universally accepted classification of atraumatic rupture of wrist flexor tendons. Most attempts to describe this abnormality are based on anatomical location and/or pathophysiological processes often connected to other local or systemic pathology. Whether or not an episode of atraumatic tendon rupture represents an intrinsic process or extrinsic trauma or overload remains unclear. Netscher described a number of different types of atraumatic flexor tendon rupture: attritional, infiltrative and iatrogenic or drug related, which can occur secondary to local bone or joint pathology, infiltrative synovitis and local or systemically administered pharmacological agents, respectively [18]. It has previously been hypothesised that the final common pathway in all of these pernicious effects on the quality of the tendons themselves and on their functional characteristics is an absolute and quantifiable reduction of water content and diameter of collagen fibres [19].

The case report patient presented here had imaging confirmed OA, and thus had at least one chronic risk factor for atraumatic FCR rupture. The mechanism behind this is likely long-term irritation of the tendon sheath due to osteophytes which may be present in the mild stage of OA and not obvious on USS [3]. Regular wear and tear leads to thinning of the protective sheath, and eventually the tendon is liable to fail under minor stress. In this case, kneading icing was potentially enough to cause the injury. However, Stoop *et al*. 2018 stated that STT OA does not correlates with radial volar sided wrist pain which MC had prior to FCR rupture; the age of patient, however, does correlate (>55 years) [17]. Therefore, the patient's STT OA may be a purely incidental, non-contributor to her radial sided wrist pain. It should also be noted that up to 55% of patients with STT OA have symptoms consistent with tendinopathy of the ipsilateral FCR tendon [17]. Age and location of pain are independent risk factors for changes in the FCR tendon. Exploring the age, it was found that 97% of subjects over 55 years of age with atraumatic quadriceps tendon rupture had degenerative changes in their tendon on histological investigation, whereas 34% of the population in the same age range had degenerative tendons without rupture. To extrapolate, it can be concluded that the age of our case report patient may be as much a contributing factor in her FCR tendinopathy as the presence of STT arthritis [17, 19].

Intra-sheath steroid injections are a known risk factor for all tendon ruptures, and this has been reported in the literature for the FCR as well [2, 3]. However, FCR tendon rupture incited by a nearby joint injection appears to be a novel event, especially if rupture was in the musculotendinous junction [13]. Indeed, the majority of papers that have been reviewed as part of this unique case report focused on pathological processes leading to tendon rupture and failed to uniformly describe the precise anatomical location of the tendon rupture. In a study of hand tendon ruptures, four patients were reported with various finger extensor tendon ruptures after receiving injections into their MCP joints for RA [4]. Another study found that injections outside of tendon sheaths carried a similar risk profile compared with intra-sheath injections, noting spontaneous ruptures in young athletes who had received multiple steroid treatments in adjacent joints; indeed, during the athletes' tendon repair procedures, four out of five subjects were found to have local deposits of steroid at the site of their rupture [5]. This study also observed a 2-8 week latency between injection and rupture, which is in line with the case report patient's presentation. Several other studies have also reported ruptures following local joint injections, including the tibialis anterior tendon [10], supraspinatus tendon [11], and patellar tendon [12].

Regarding the case report patient's identified chronic medical condition, chronic lymphocytic leukemia, it is unlikely that the condition itself increased her risk of FCR rupture without an inflammatory component. Only one case report was found of quadriceps tendon rupture linked to chronic lymphocytic leukemia. Indeed, chronic lymphocytic leukemia can manifest with local and systemic changes. Both phenomena aren't well described in the literature. From a single study, a histological investigation of a subject's tendon revealed extensive B cell chronic lymphocytic leukemic infiltration, along with mucoid degeneration. Perhaps altered synovial lining has a high affinity for neoplastic lymphocytes, which potentiates the local inflammatory response, making rupture more likely [19]?

There is no level 1-3 evidence linking the case report patient's chemotherapy regimen with tendinitis, either, as she did not receive any corticosteroids, which have been implicated more broadly in leading to a reduction in the quality of tendon tissue, even when administered systemically [8]. However, there is a case report on bilateral rupture of Achilles tendons in a previously asymptomatic patient who received combination therapy with gemcitabine, cisplatin and vincristine. However, it was revealed that this patient, in addition to the above triple chemotherapy for non-small cell lung carcinoma, was also taking 16 mg of dexamethasone, a proven, independent risk factor for tendon degeneration, particularly at sustained, high parenteral doses [21]. Moreover, recent studies have implicated fluoroquinolone antibiotics in various cases of tendon rupture, including wrist tendons [6, 7, 18]; this antibiotic is regularly prescribed for patients with haematological malignancies. It is unclear from our patient's history whether or not she was prescribed this agent, but if this were the case, it may have predisposed her to an atraumatic FCR tendon rupture. The patient has been pain free after six months of follow-up, with full range of motion, achieved from a sustained period of physiotherapy. This outcome demonstrates that timely detection and non-operative treatment of flexor carpi radialis tendon rupture can achieve good functional results.

In summary, FCR tendon rupture is a rare phenomenon, although the literature supports trauma [2, 3], steroid injections [9], local arthritis [1], and certain systemic drugs [6, 7] as risk factors. These identified factors are applicable to many tendons throughout the upper and lower limbs, and this needs to be taken into account

when considering whether or not to offer steroid injections for 'musculoskeletal pain drivers' without a clear diagnosis; a clear understanding of the anatomy is key, and knowing what pain source to target is an important facet of clinical sports medicine practice. Sheath injections with cortisone have been identified as a risk factor as well, with several cases of tendon rupture reported in the sports medicine literature. It is likely that steroid deposition in and around tendons is similarly damaging to their strength, though further research will need to be done to confirm this. In the interim, one should err on the side of caution when offering high-risk patients steroid injections, regardless of the intended location and the anticipated therapeutic benefits.

Conclusions

Rupture of FCR is associated with significant reduction in quality of life due to pain and altered wrist and hand function, at least in the short to medium term; long term restoration of function and quality of life is to be anticipated, provided the patient received the appropriate sustained period of physiotherapy. From the available studies, which largely constitute case reports, FCR tendon rupture is a relatively rare phenomenon in terms of being a driver of radial or solar sided wrist pain, but it is associated with a host of identified risk factors. Atraumatic rupture of FCR needs to be considered in the differential diagnosis list when reviewing patients in the clinical setting. The clinical picture in cases of atraumatic wrist pain is often obscure. Detailed history taking followed by thorough examination are the key points in the treatment of the underlying pathology. Experienced sports medicine clinicians should not rely entirely on radiological investigations, as they can be misleading and findings are often incidental. In the case of patient MC, it was difficult to define if her previously severe and clinically symptomatic STT osteoarthritis (although only modest on plain film imaging) was the independent risk factor for her subsequent FCR rupture or whether a more complex multifactorial pathological process should be implicated.

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