

Case Report

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Not Every Hot, Tender, Inflamed Joint is Infected (or Gout)!

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ABSTRACT

Calcific tendinitis is a rare condition that classically involves the rotator cuff. Only isolated case reports exist of the disease affecting other anatomical locations. Our case is noteworthy because it describes calcific tendinitis in an uncommon location; the flexor pollicis longus of the thumb.

Calcific tendinitis has a prevalence of 3-10% in the general population, and is most commonly seen among women in their 4th to 6th decade of life. Calcific tendinitis is best diagnosed by imaging where plain radiography and ultrasound are both helpful for detecting calcifications. Blood tests are generally not used for making the diagnosis. However, elevated white blood cells and inflammatory markers (e.g. sedimentation rate and C-reactive protein) may be seen in the acute phase of calcific tendinitis, but are usually normal. Although the exact pathophysiology of calcific tendinitis is unknown. Calcific tendinitis condition is often self-limited and interventions (i.e. anti-inflammatory medications, analgesics, glucocorticoid injections, physical therapy) are used with a “try it and see” mindset. Other treatment options (extracorporeal shock wave therapy, therapeutic ultrasound, iontophoresis, platelet rich plasma) have been tried.

Acute calcific tendinitis should be on the differential diagnosis for acute musculoskeletal pain—even pain that is not at the rotator cuff. This case demonstrates that pain that is associated with a tender, inflamed joint can be more than either infection or gout. It is important to recognize calcific tendinitis as a potential diagnosis, as this could prevent unnecessary interventions and therapy such as intravenous antibiotics.

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Introduction

Calcific tendinitis is a rare condition characterized by an inappropriate deposition of calcium hydroxyapatite crystals in tendons [1]. The exact pathophysiology of this disease remains to be understood, but data shows that trauma or overuse of the affected tendon is not the culprit. Most cases of calcific tendinitis are seen in women between the ages of 40 years and 60 years [2]. Calcific tendinitis classically affects the tendons of the rotator cuff. There have only been isolated case reports of the disease affecting other anatomical locations, such as the rectus femoris and gluteus maximus [3]. Our case describes a young woman with no significant past medical history who developed acute calcific tendinitis of the flexor pollicis longus in the right thumb.

Case

A 36-year-old female without any significant medical history presented to the rheumatology team with right thumb pain for 2 days. She had woken up that morning with mild pain (4/10) of the thumb. Thumb pain was constant and exacerbated by movement. Pain progressed throughout the day reaching 6/10 in intensity, was throbbing in nature, and localized to the interphalangeal (IP) joint of the thumb. Pain was later associated with swelling, warmth, and redness of the finger (Figure 1 & 2). In addition, the pain's intensity increased the next day and started affecting the

ability of the patient to sleep at night. She tried Tylenol, Ibuprofen, and immobilization of the thumb without any relief. The patient denied fevers, chills, night sweats, skin rashes, and other joint pain. She also denied trauma to the thumb or any strenuous activities.



Figure 1: Right thumb with swelling



Figure 2: Right thumb with swelling and erythemaPhysical

Physical Exam

On exam, patient was in mild distress due to the pain. Her right thumb was swollen, in extension, and slightly erythematous on the volar aspect of the IP joint. No erythema or edema was present over the MCP joint. The skin around the thumb and the nail were intact. There was warmth and severe tenderness on palpation of the IP joint. There was no tenderness on the MCP joint but there was minimal tenderness over the thenar eminence. Active and passive flexion of the right thumb IP joint was limited due to pain, but extension was intact. Flexion and extension of the MCP joint was also intact. The rest of the joint exam was unremarkable.

Additional Testing

No blood tests were recommended given the lack of systemic symptoms and the isolated joint involvement. An x-ray of the right hand was done and was unremarkable (Figure 3 & 4).



Figure 3: Normal X-ray of R hand



Figure 4: Normal X-ray of R hand

Treatment Course

Given the physical exam findings, the rheumatologist thought the patient had an infectious process of the first right IP joint. She began a course of Augmentin 875 mg twice a day and Ibuprofen 800 mg three times a day, as needed, for pain.

Despite antibiotic and Ibuprofen use, the patient's symptoms progressed over the next 2 days. She then saw a hand surgeon, who recommended urgent exploratory surgery for possible flexor tenosynovitis of the thumb. The patient went to an orthopedic surgeon for a second opinion and another x-ray was performed. The results of this x-ray showed flexor pollicis longus tendon calcification (Figure 5).



Figure 5: X-ray showing flexor pollicis longus tendon calcification

The patient was started on a Medrol dose pack, and Ibuprofen use was recommended for pain as needed.

Her symptoms improved significantly within the first 24 hours. Full ROM of the thumb was achieved by the third week post-treatment.

The patient's pain is gradually resolving with steroid therapy (2/10). Conservative treatment of calcific tendinitis most commonly includes rest of affected tendon, anti-inflammatory medications, or a steroid injection. Only 10% of patients do not respond to conservative treatment and thus seek surgery to remove calcium deposits [4].

Discussion

Calcific tendinitis has a prevalence of 3-10% in the general population, and is most commonly seen among women in their 4th to 6th decade of life. Our case is noteworthy because it describes calcific tendinitis in an uncommon location; the flexor pollicis longus of the thumb. As previously mentioned, calcific tendinitis classically involves the rotator cuff. In fact, calcific tendinitis is considered a painful "shoulder disorder." Nevertheless, there have

been documented cases of calcific tendinitis in other anatomical locations [3].

One such case described acute calcific tendinitis of the index finger [5]. In this case, an otherwise healthy 9-year-old male presented with 2 days of pain, erythema, and swelling in his left index finger. Plain radiography showed a calcific mass in his proximal interphalangeal joint. The hospital was still concerned for infection, and surgical exploration was suggested. The patient sought a second opinion and during this time, the swelling and erythema improved. Ultrasound of the area showed a radiodense mass moving in synchrony with the tendon. Once acute calcific tendinitis of the flexor tendon was suspected, the patient received a finger splint and therapy with NSAIDs. His symptoms completely resolved a few weeks later. If the patient had not gotten a second opinion, he most likely would have endured an unnecessary surgical procedure [5].

Another unique presentation of calcific tendinitis occurred in the gluteus maximus tendon [3]. This case involved a 48-year-old woman with lupus nephritis and pulmonary hypertension. She presented with acute onset left buttock pain for 3 days and a palpable, tender mass over her left gluteal region. Fine needle aspiration revealed rhomboid-shaped, positively birefringent crystals under polarized light microscopy. A diagnosis of calcific tendinitis of the gluteus maximus secondary to calcium pyrophosphate dihydrate crystal deposition disease (CPPD) was made and she was treated with a steroid and lidocaine injection [3].

Our patient's presentation was similar to the typical presentation seen in acute calcific tendinitis of the shoulder: sudden onset of pain unassociated with trauma. In fact, those involved in manual labor or athletic activities are actually not at an increased risk of developing the condition [2]. Likewise, our patient was not involved in such activities—she is a physician. Of note, the patient does have a history of fracture in that digit, but this took place 12 years ago.

The diagnosis upon clinical presentation was unclear. Patient had acute pain with swelling and erythema at the site. In the absence of trauma, gout and infection were also considered. Calcific tendinitis is best diagnosed by imaging where plain radiography and ultrasound are both helpful for detecting calcifications. In our case, diagnosis was confirmed with plain radiography, after visualizing calcifications. Repeat plain radiographs can be sought during follow-up visits to assess calcium degradation. Blood tests are generally not used for making the diagnosis. However, elevated white blood cells and inflammatory markers (e.g. ESR and CRP) may be seen in the acute phase of calcific tendinitis, but are usually normal [5]. The diagnosis of calcific tendinitis in the hand has a higher rate of missed or delayed diagnosis, as its presentation shares features with other conditions. Furthermore, there is a lack of familiarity with its clinical presentation [5].

Although the exact pathophysiology of calcific tendinitis is unknown, it is thought to have 3 stages of “calcification.” They are pre-calcific, calcifying, and post-calcific. In the pre-calcific stage, patients generally do not have symptoms [5]. During this stage, certain sites in the body undergo cellular changes (i.e. tenocytes become chondrocytes) [6]. These changes predispose tissues to calcium deposition. In the calcific stage, cells secrete calcium that coalesce into calcium deposits. Once calcification is complete, the “resting phase” begins. This phase is not painful, but is followed by the “resorptive phase,” which is the most painful phase of this condition. It is unknown what triggers the switch

from the resting phase to the resorptive phase [7]. However, during the resorptive phase, macrophages, polymorphonuclear cells, and fibroblasts phagocytose the calcium deposit. The last stage of calcific tendinitis, the post-calcific stage, is painless. The calcium deposit eventually disappears and is replaced by granulation tissue [7].

Treatment of calcific tendinitis starts with conservative management. Our patient was started on oral steroid therapy and her symptoms started resolving. In calcific tendinitis of the shoulder, the condition is often self-limited and interventions (i.e. anti-inflammatory medications, analgesics, glucocorticoid injections, physical therapy) are used with a “try it and see” mindset [5]. Given that the affected site has a smaller joint space than that of the shoulder, a glucocorticoid injection and physical therapy may not be as beneficial. If our patient had not responded to steroids or anti-inflammatory oral therapy, what other options would have been available?

In particular, extracorporeal shock wave therapy (ESWT) has been shown to reduce pain and improve joint mobility in calcific tendinitis of the shoulder [8]. High-energy extracorporeal shock waves are used in urology for kidney stone breakdown. Whereas in urology the waves are used to disintegrate stones, in musculoskeletal disorders, these waves are used to stimulate tissue repair [9]. In ESWT, shock waves travel through tissue and promote revascularization and repair at sites of tissue interface (i.e. where waves detect calcium) [7]. Another therapy used is called iontophoresis, or transdermal drug delivery using the voltage gradient on the skin [7]. The drug that is often used is acetic acid, because hydroxyapatite calcium crystals are soluble in acidic solutions [7].

Therapeutic ultrasound has also been tried [7]. It is thought that ultrasound can promote calcium breakdown in several ways. Ultrasound activates endothelial cells, and activated endothelial cells release chemoattractant substances. These chemoattractants promote the migration of macrophages that induce phagocytosis of calcium. Ultrasound can also trigger disruption of apatite-like microcrystals, which are then removed by macrophages. The increase in tissue temperature combined with ultrasound increases blood flow and metabolism, which in turn, also aids calcium dissolution [7].

Platelet-rich plasma (PRP) has also been tried as a treatment option. There was a case of a 44-year-old woman with calcific tendonitis of the shoulder that was treated with a PRP injection in her subacromial area (3 injections over 6 weeks). She had improvement of symptoms in 2 weeks and eventually became asymptomatic [10].

Our case highlights the fact that acute calcific tendinitis should be on the differential diagnosis for acute musculoskeletal pain—even pain that is not at the rotator cuff. This case demonstrates that pain that is associated with a tender, inflamed joint can be more than either infection or gout. It is important to recognize calcific tendinitis as a potential diagnosis, as this could prevent unnecessary interventions and therapy such as intravenous antibiotics.

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