First Presentation of Tophaceous Gout in the Achilles Tendon After Primary Repair: A Report of Two Cases

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INTRODUCTION

While it is unusual for the Achilles tendon to be the initial site of a gout flare in patients with no history of gout or hyperuricemia, up to 25% of patients with chronic gout have Achilles tendon involvement.[1] Gout flares may present initially as pain, swelling, and weakness around the Achilles tendon without trauma and can mimic infection at presentation.[2-6]

There are rare individual case reports of the development of gouty tophi as the initial presentation of gout years after the repair of a ruptured Achilles tendon.[7-9] We present two such cases, in a 42-year-old male and a 35-year-old male with no prior history of gout who each developed gouty deposition on and around retained suture material 6 and 8 years after initial Achilles repair. We hope to direct attention to the presentation of gout in this setting and start to delineate the progression of this presentation.

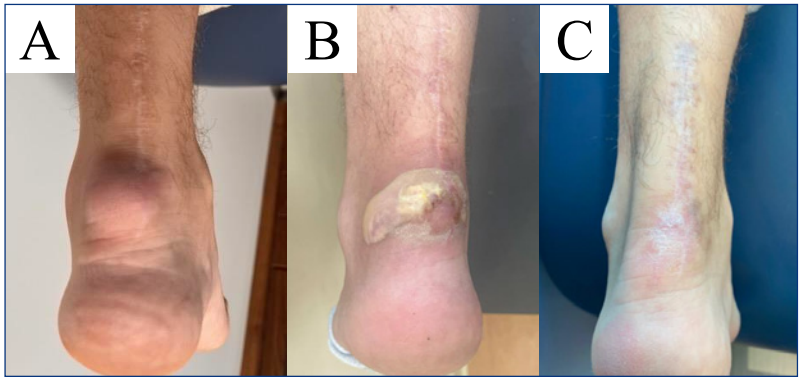
CASE REPORT

Case 1

A 42-year-old male, 6 years status post Achilles repair presented with 1 week of posterior Achilles pain and progressive ankle swelling. He was afebrile with normal vital signs and denied any trauma. His initial x-rays were normal. He was found to have a palpable golf ball-sized mass at the distal extent of the original incision [**Figure 1A**]. With concern for abscess and eruption of a previous occult infection, he was started on cephalexin and an MRI without contrast was obtained, demonstrating extensive tendinopathy with degenerative cystic change extruding through the dorsal distal Achilles into the subcutaneous tissue along with soft tissue swelling [**Figure 2A**]. The area of fluctuance was aspirated, yielding a white opaque fluid concerning for infection versus gout. Lab work revealed elevated C-reactive protein (CRP) (20.04 mg/L) and normal erythrocyte sedimentation rate (ESR) (11 mm/h). The patient subsequently developed drainage and additional skin changes at the aspiration site, and a surgical procedure for irrigation and debridement was scheduled [**Figure 1B**].

In the operating room, gray viscous fluid and white material exuded upon skin incision. The nonabsorbable suture and Achilles tendon were found to be embedded with white chalky material consistent with tophaceous gout. Irrigation and debridement was performed and all suture and friable tissue were removed. The patient was discharged the next day with empiric trimethoprim-sulfamethoxazole due to ongoing concern for superimposed infection. Preoperative aspiration cultures demonstrated no growth, and intraoperative cultures grew mixed cutaneous flora consistent with contamination. Pathology confirmed gouty tophi with fibrin as well as abundant acute inflammation. Upon further questioning at his 1-week follow-up, the patient confirmed a history of pain in his great toe and noted that his father had a history of gout. The patient's uric acid level was 7.9 mg/dL (normal 3.5–7.0 mg/dL) and he was started on allopurinol. Two weeks post-operatively, there were no signs of drainage or swelling. At 1 month, the patient could walk without limping. Six months post-operatively, the wound remained fully healed with baseline thickening of the Achilles tendon and no pain, swelling, or drainage [**Figure 1C**]. Physical exam demonstrated full range of motion with good plantar flexion strength, although single leg heel rise was weaker compared to the contralateral side, unchanged from prior to

Figure 1. [**A**] Palpable golf ball-sized mass at the distal extent of the original incision of Case 1. **[B]** Site of tophaceous gout and drainage 1 day before surgery. **[C]** Fully healed wound 6 months after surgery.



the gout flare. At 1 year follow-up, the patient had continued taking allopurinol with no additional symptoms reported. The Foot and Ankle Outcome Score (FAOS) at one year was 85%, broken down into the following subtotals: Symptoms + Stiffness: 71%; Pain: 92%; Function, daily living: 96%; Function, sports and recreational activities: 90%; Quality of life: 44%.

Case 2

A 35-year-old male, 8 years post-Achilles repair presented with 4 days of new left ankle pain and progressive swelling after light exercise. Exam revealed swelling and tenderness along the Achilles tendon posteriorly, as well as decreased dorsiflexion range of motion and decreased sensation in the sural nerve distribution. X-rays demonstrated posterior calcaneal bone spurring and mild lateral and posterior soft tissue swelling. Deep vein thrombosis was excluded through ultrasound. The patient's ankle was made weightbearing in a tall boot and he was given a 6-day methylprednisolone taper with transient relief. Lab work revealed normal CRP (<3 mg/L), elevated ESR (15 mm/h), and normal uric acid (6.7 mg/dL). Given continued pain with attempted weightbearing, an MRI with and without contrast was ordered, revealing edema in the flexor hallucis longus muscle and fluid along the medial Achilles tendon [**Figure 2B**]. Due to concern for deep infection, he was started on trimethoprim-sulfamethoxazole. However, swelling and tenderness increased over the following week, along with the development of erythema about the posterior ankle. A second methylprednisolone taper was initiated, and the patient had transient improvement; however, symptoms returned after discontinuation. He was taken to the operating room 3 weeks after initial presentation, where an incision along the Achilles tendon exuded a chalky white fluid. Both suture and Achilles tendon were found to be embedded with white chalky material consistent with tophaceous gout. The suture and all friable tissue were removed, and the tendon was thoroughly irrigated. Intraoperative cultures were negative. Pathology confirmed gouty tophi with fibrin. Two weeks postoperatively, there were no signs of drainage or swelling. At 1 month, the patient could walk without pain in a tall boot. Three months postoperatively, he had significant improvement in symptoms and was able to walk without a boot. At 6 months, he had returned to his baseline function and pain level, and physical exam demonstrated good plantar flexion strength and full range of motion. The Foot and Ankle Outcome Score (FAOS) at 6 months was 84%, broken down into the following subtotals: Symptoms + Stiffness: 93%; Pain: 83%, Function, daily living: 87%; Function, sports and recreational activities: 80%; Quality of life: 63%.

Figure 2. T2-weighted MR images: [**A**] In Case 1, sagittal and corresponding axial views. [**B**] In Case 2, sagittal and corresponding axial views.

DISCUSSION

Four other reports of surgical site gout surrounding retained suture material after Achilles tendon repair that share similarities with our cases were found in the literature: in a 58-year-old female 12.5 years after repair,[9] in a 49-year-old male many years after repair,[6] in a 32-year-old male 7 years after repair,[8] and in a 53-year-old male during revision surgery 2.5 years after initial repair.[7] All six patients had no prior diagnosis of gout or hyperuricemia and at presentation were afebrile with progressive pain and swelling around the original incision. Three patients presented with an abscess that drained a significant amount of chalky white material, although similar material was found embedded in the nonabsorbable suture in all patients, and suture removal appeared to resolve all redness, swelling, and pain.

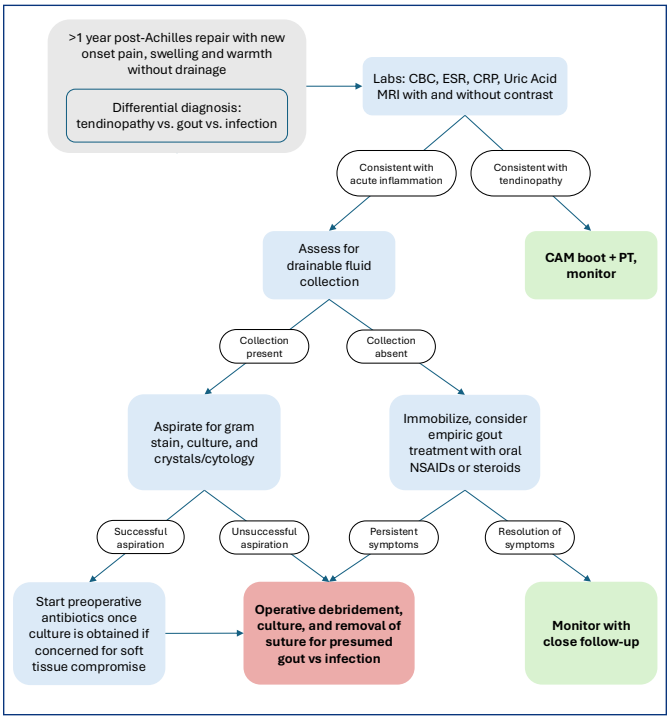
There may be a relationship between gout and Achilles rupture even without suture repair. Studies have shown that asymptomatic hyperuricemia may be a risk factor for Achilles tendon rupture as elevated serum uric acid levels may disrupt the functional integrity of tendon tissue.[10–12] In tendon stem/progenitor cells (TSPCs), high uric acid levels have been shown to suppress the AKT-mTOR signaling pathway, which plays a critical role in sensing cellular stress and metabolic abnormalities, thus impeding the function of TSPCs in regulating tendon homeostasis and increasing risk of rupture.[12] Again, neither patient in this study had prior uric acid levels available to reference as a potential risk factor for their initial ruptures.

Even without prior rupture or repair, tophaceous gout may have a specific predilection for the Achilles tendon. Lower temperatures and limited blood supply make the tendon more prone to monosodium urate (MSU) crystal buildup due to decreased clearance, and repeated mechanical stress with microtrauma can create local inflammation or microtears that serve as a nidus for MSU crystal accumulation.[13]

Another factor involved may be the suture material. Nonabsorbable sutures are frequently used in tendon repairs because they have greater tensile strength and may reduce the risk of re-rupture compared to absorbable sutures.[14,15] While no association between retained suture material and gout flares has been established, it is possible that the sutures can act as foreign bodies that increase inflammation, contributing to the development of gout. In particular, both of our patients' initial repairs were done with FiberWire (Arthrex, Naples, FL), a braided nonabsorbable silicone-coated suture, which has been associated with inflammatory and foreign body reactions.[16] Mechanical friction from shoes and chronic irritation by the suture against the overlying skin may lead to further inflammation, precipitating the formation of MSU crystals.[9] The combination of nonabsorbable sutures and MSU crystals has been hypothesized to result in a more robust inflammatory response than induced by either process alone.[7] Two similar cases have been reported in the upper-extremity literature, with tophi formation surrounding nonabsorbable suture 7 and 33 years after initial tendon repair in the forearm and hand in patients with no history of gout or hyperuricemia.[17,18]

These cases underscore the need for heightened clinical awareness and consideration of gout in the differential diagnosis for late post-surgical Achilles repair complications. Earlier diagnosis can lead to expedited operative intervention prior to potential skin breakdown or superimposed infection. The presentation for infection and tophaceous gout is similar and delineating the two pathologies is critical for appropriate treatment. If a fluid collection is present, aspiration for gram stain, culture, and cytology/crystals prior to initiating oral antibiotics would be ideal. Empiric antibiotics following aspiration should be considered if there is concern for infection causing destruction of the tenuous soft tissue over the Achilles tendon. We recommend early surgical intervention if there is a large fluid collection or failure to respond to oral NSAIDs or steroids as surgical debridement is indicated if either infection or gout is present. The treatment algorithm outlined in **Figure 3** provides a useful framework, but individual patient care should ultimately be guided by clinical judgement.

Figure 3. Algorithm for workup and management of new inflammatory symptoms at repair site >1 year after initial Achilles repair.



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