

Gouty Achilles Tendinopathy

A Case Report

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Tendinopathy in the presence of gouty arthropathy is relatively common, yet the clinical suspicion for gout involvement in acute tendon pain remains low. A 49-year-old man presented with an acute, tender, erythematous mass to the right posterior heel. A computed tomographic scan was obtained, which revealed a septated fluid collection superficial to the Achilles tendon. The patient was taken to the operating room for an incision and drainage with debridement, and the abscess was found to be filled with caseous material. The diagnosis of gout was confirmed with pathology. The calcaneus was submitted to biopsy, and the results were negative for osteomyelitis. The patient was returned to the operating room for repair of the Achilles tendon with flexor hallucis longus tendon transfer. Postoperatively, the patient was nonweightbearing for 6 weeks. Oral colchicine was used perioperatively, and a steroid taper was administered. The patient was started on allopurinol and colchicine for chronic treatment. At 14 months, the patient was walking without pain or recurrence of the mass. Although the relationship between hyperuricemia and tendinopathy is not completely understood, it is apparent that tendon involvement may be a sequela in patients with gout. When a patient presents with acute tendon pain, gout should be considered in the differential diagnosis. (J Am Podiatr Med Assoc 109(4): 327-333, 2019)

Gout is the most common inflammatory arthritis and is the result of monosodium urate precipitation in joints and soft tissue caused by hyperuricemia. The most commonly affected areas include joints of the lower limb such as the knee, ankle, and first metatarsophalangeal joint.¹ Although the disease process is classified as a form of arthritis, monosodium urate (MSU) crystal deposition into extra-articular soft tissues such as ligaments and tendons is a common manifestation.² This is often more apparent in areas of mechanical stress to the tendons, including areas of pathology and enthesopathy.³ It is thought that this crystal deposition activates key mediators of the inflammatory response, including interleukin-1 β (IL-1 β), which may act to initiate tendinopathy by inducing gene transcription of inflammatory molecules.⁴ Monoso-

dium urate crystals have also been linked to the upregulation of certain metalloproteases that can compromise tendon extracellular matrix and reduce expression of type 1 collagen.⁴ This potential hyperuricemic tendinopathy may have significant implications. A review of the literature reveals such documented cases in the lower extremity involving tears of the peroneal tendons,⁵ partial rupture of the distal biceps,⁶ and enthesitis and tendinopathy of the Achilles tendon.^{7,8}

Uric acid is the end result of purine metabolism, which is a normal biochemical process when uric acid levels are maintained within a homeostatic range. Different values appear in the literature as a normal reference range and even vary by gender but generally are between 2 and 7 mg/dL.⁴ Hyperuricemia is defined as serum urate levels greater than 6.8 mg/dL.^{4,9} Increased levels are caused by one of two major etiologies: uric acid overproduction secondary to increased dietary intake of purine-rich foods, or uric acid underexcretion secondary to impaired renal function. These increased urate levels have

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been linked to certain immune responses. Monosodium urate crystals induce activation of caspase 1, which activates IL-1 β to promote other chemokines and cytokines that trigger the inflammatory pathway, causing cellular and metabolic distress and leading to tendinopathy and clinical pain, which can lead to partial tears or ruptures of the involved tendon.⁴ In acute reactive tendinitis, normal inflammatory mediators that promote healing such as leukocytes are not present. Instead, cytokines such as IL-1 β and prostaglandins, the same molecules that MSU crystals have been shown to activate, are observed. In addition, cell death occurs in tendinopathy, which is a process that releases a myriad of molecules into the extracellular space. One of these molecules is uric acid.

Several imaging modalities have been implicated in further assessing low-grade tendon inflammation in patients with hyperuricemia, suggesting a link between gout and tendinopathy. Ultrasonography and dual-energy computed tomography (DECT) are the most commonly described modalities in the literature for visualizing gout within tendons. Ultrasound is one of the best modalities for identifying tendon pathology, and it can accurately distinguish between two types of gout.^{2,10} The first type displays soft tophi with varying echogenicity. The second type has hard tophi that contain MSU crystals, which appear as a hyperechoic band with an acoustic shadow.¹¹ The emergence of DECT allows the detection of urate deposition in joints and soft tissues and uses a photoelectric effect to distinguish between urate and calcium to separate gout from chondrocalcinosis.^{3,12} The sensitivity and specificity of ultrasound to detect tophaceous gout within tendon is 69.6% and 92%, respectively,¹³ with the sensitivity and specificity of DECT both being between 78% and 100%.¹² Dual-energy computed tomography can facilitate the visualization and accurately quantify the MSU crystals with great accuracy. In a DECT study performed by Dalbeth et al², tendon involvement in the feet of patients with gout was examined. The study concluded that MSU crystal deposition was observed in 10.8% of tendon/ligament sites examined in the study. Of these sites, the Achilles tendon was the most commonly involved, with an occurrence rate of 39.1%. Visualizing tophi and aggregates on these imaging modalities not only can provide an accurate diagnosis but also can accelerate the treatment process for these patients and serve as a detection tool to assess the efficacy of urate-lowering therapy.

In this article, we discuss a case of a patient with an acute gout flare presenting as posterior heel pain

and Achilles tendinitis with inflammation. This case report shows that when a patient presents with red hot, tender heel pain, gout should be considered in the differential diagnosis. To our knowledge, this is the first reported case of gout destruction of the Achilles tendon so extensive that it required reconstruction with a flexor hallucis longus (FHL) tendon transfer.

Case Report

A 49-year-old man presented to the emergency department with acute tenderness and an erythematous painful mass in the right heel. The patient had a surgical history of an ipsilateral retrocalcaneal exostectomy with Achilles detachment and reattachment with suture anchors 5 years prior to the current presentation. The patient had an unremarkable postoperative course, but did relate mild, intermittent inflammation to the Achilles tendon and posterior heel during physical activity. He denied recent, inciting trauma. On presentation to the emergency department, the patient was febrile and tachycardic. Neurovascular status was intact. There was a firm, nodular mass located posterior to the Achilles tendon at the level of the retrocalcaneal bursa. This measured approximately 4 $\times$ 4 cm (Fig. 1). There was associated erythema that surrounded the mass 3 cm circumferentially. Mild warmth was noted. No fluctuance, crepitus, or open wounds were appreciated. Muscle strength was graded +5/5 in eversion, inversion, and dorsiflexion. Plantarflexion strength examination was limited because of guarding and poor effort.

Plain radiographs did not reveal signs of osteomyelitis to the calcaneus. Soft-tissue edema was appreciated in the soft tissues adjacent to the Achilles tendon (Figs. 2 and 3). Evidence of a radiopaque suture anchor from previous surgery was retained in the calcaneus. Magnetic resonance imaging was unable to be performed because of retained hardware in the cranium. Therefore, a computed tomographic scan was obtained, which revealed a 2.4 $\times$ 2 $\times$ 0.9-cm septated fluid collection in the soft tissues superficial to the Achilles tendon (Fig. 4). Inflammatory markers are listed in Table 1.

Given his fever, tachycardia, physical examination, and computed tomographic findings, infection was suspected. Empiric parenteral piperacillin/tazobactam was administered. An incision and drainage with debridement was performed under general anesthesia. The patient was positioned in prone position. An incision was made to the posterior aspect of the Achilles in line with the



Figure 1. Posterior to anterior view (A) and lateral view (B) at clinical presentation in the emergency room.

previous surgery. Dissection was carried through subcutaneous tissue, where a nodular mass was noted within the posterior soft tissues. This was contiguous with the Achilles tendon. After complete excision, an intratendinous abscess was identified that had caseous material. After excision of the mass, minimal healthy fibers of the Achilles tendon were noted, and a significant portion was excised with the mass. The nodule was sent for pathologic analysis. Postirrigation cultures were obtained,

which were negative. The wound was closed with 3-0 nonabsorbable suture. Oral amoxicillin/clavulanic acid was continued on an outpatient basis until culture results had finalized. Bone cultures were obtained to evaluate for osteomyelitis and were negative. Pathologic evaluation verified a diagnosis of gout (Figs. 5–7).

After verifying negative cultures, at 10 days postoperatively, the patient was taken back to the operating room. Clinical erythema was significantly improved. The patient consented to a debridement of



Figure 2. Lateral radiograph. Previous anchor can be visualized along with soft-tissue edema.



Figure 3. Anteroposterior ankle radiograph.



Figure 4. Sagittal view computed tomographic scan showing a $2.4 \times 2 \times 0.9$ -cm septated fluid collection in the soft tissues superficial to the Achilles tendon.

the Achilles tendon and FHL tendon transfer. This was performed under general anesthesia and in prone position. The previous incision was opened and the Achilles tendon and calcaneus were visualized. The Achilles tendon was significantly degenerated and was therefore detached off its insertion and debrided to healthy margins. An FHL tendon transfer was performed through the same incision.¹⁴ The FHL tendon was transferred under physiologic tension

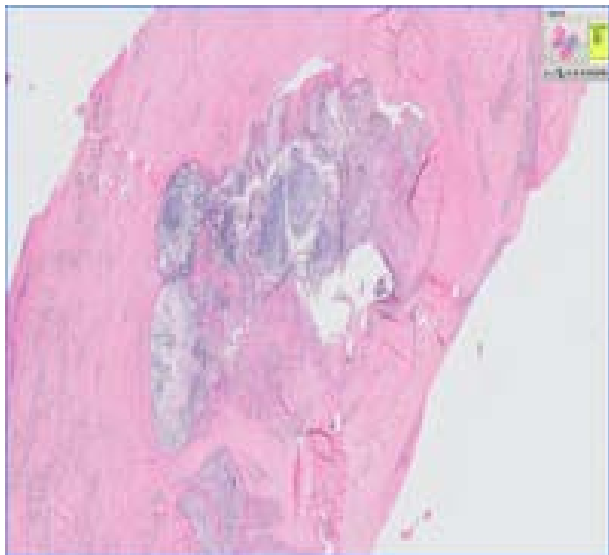


Figure 5. Tophi surrounded by chronically inflamed tendon (H&E, $\times 20$).

Table 1. Laboratory Markers at Admission

Marker	Value
White blood cell count ($\times 10^9$ /liter)	11.6
Creatinine (mg/dL)	1.52
Erythrocyte sedimentation rate (mm/h)	14
C-reactive protein (mg/dL)	89
Uric acid (mg/dL)	10

into a bone tunnel in the calcaneus with a tenodesis screw. A well-padded posterior splint was applied. The patient was nonweightbearing for 6 weeks and transitioned into a walking boot. Oral colchicine was prescribed for management of acute gout in the perioperative setting in addition to an oral steroid taper. An outpatient referral was made to a rheumatologist for long-term management of gout. The patient continues a regimen of oral allopurinol 100 mg daily and colchicine 0.6 mg twice daily. At the 8-month follow-up, the patient was able to walk without pain. Mild calf atrophy and expected weakness in plantarflexion was appreciated. At the final 14-month follow-up, the patient continues to walk in regular tennis shoes without a brace (Fig. 8). The patient continues to function and perform activities of daily living without limitations.

Discussion

Tendon involvement in gout, especially tendons of the lower extremity, is a relatively common

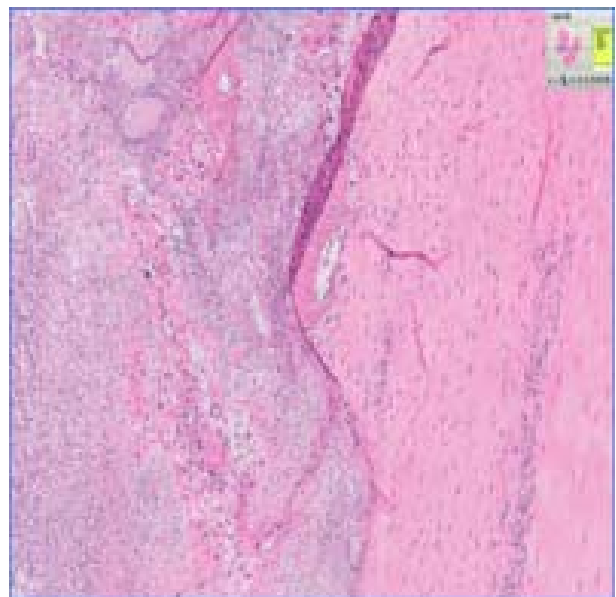


Figure 6. Chronic inflammation, lymphocytes, and the connective tissue reaction (H&E, $\times 400$).

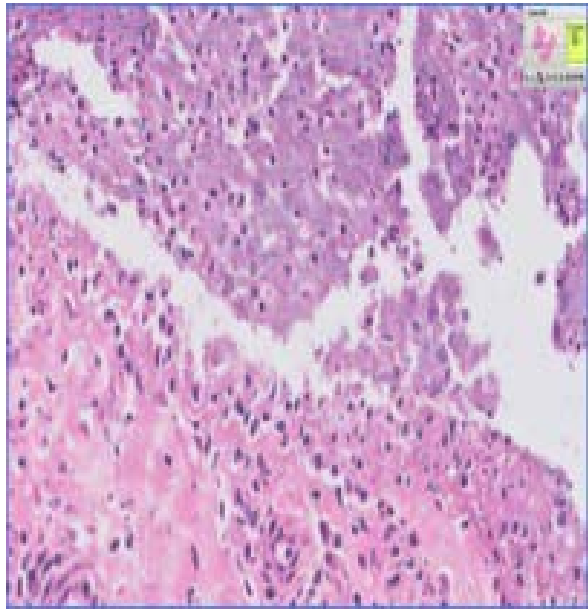


Figure 7. The presence of neutrophils in the tophi and mixed into the surrounding tissue reaction is highlighted (H&E, $\times 400$).

occurrence, especially in diseased tendons and areas of other mechanical stress, such as insertional attachments and enthesopathies.³ This is well documented in the literature as mentioned by Dalbeth et al² in a DECT study where they reported that the Achilles tendon was the most common tendon involved in MSU crystal deposition. Further corroborating the prevalence of the Achilles tendon in gouty arthropathy is another DECT study by Fritz et al¹² that reported a 36% occurrence rate of urate deposits within the Achilles tendon. An ultrasound study by Ventura-Rios et al¹³ evaluating tendon involvement in patients with gout reported a nearly 50% prevalence of tophi and hyperechoic aggregates in the Achilles tendon. However, it was noted that some of the patients who had osteoarthritis also displayed these hyperechoic aggregates despite being asymptomatic and with normal uric acid levels, and this could be because these hyperechoic aggregates are not exclusive to gout. Dual-energy computed tomography is now included in the 2015 criteria of the American College of Rheumatology/European League Against Rheumatism. It also serves as a good tool for monitoring the effectiveness of urate-lowering drug therapy in gout patients.¹² Dual-energy computed tomography does have a higher sensitivity and specificity to detect MSU deposits; however, in the absence of DECT, ultrasound remains an effective imaging



Figure 8. Lateral foot radiograph taken at 14-month follow-up.

modality to detect MSU deposits in patients with gout.¹

The findings of these studies and others attest to the prevalence of gout in lower-extremity tendons; however, much more remains to be elucidated regarding the pathogenesis of urate deposition within tendinous fibers. Andia and Abate⁴ outline tendon involvement in gout from a biochemical perspective and attribute the link between tendinopathy and hyperuricemia to an inflammatory response initiated by MSU crystal deposition. They further describe a loss of tendon homeostasis secondary to inflammation and cell death as the link between hyperuricemia and tendinopathy. Robust cell death seen in tendinopathy can release a significant amount of uric acid into the intratendinous space that will eventually precipitate into MSU and present as acute gout within the tendon.

The implications of a link between tendinopathy and hyperuricemia is a reduction in tensile strength of the tendon, which can lead to possible attenuation, tears, or even rupture of the involved tendon. Several reports in the literature document such cases. In a case report by Mahoney et al,¹⁵ a spontaneous rupture of the Achilles tendon is described in a patient with a history of gout in whom gouty tophi had been observed in the severed ends of the tendon at the time of surgical repair. Our patient did not sustain a rupture of his Achilles tendon; however, it has been documented that a common cause of pain in the Achilles tendon after trauma or surgery is gout.¹⁶ Our patient did have a remote history of Achilles tendinitis and

underwent surgery for a painful retrocalcaneal exostosis with reattachment of the Achilles tendon. Other reports document rupture of the quadriceps tendon in gout,¹⁷ longitudinal tears of the peroneal tendons,⁵ and partial rupture of the distal biceps tendon, although this is in the upper extremity.⁶

The present report describes a case of symptomatic hyperuricemia involving the Achilles tendon in a patient who presented with pain, swelling, and erythema to the right posterior heel and ankle in addition to an elevated serum uric acid level. We believe that the elevation in C-reactive protein (CRP) is due to the sensitive nature of this marker to an acute inflammatory process, whereas the erythrocyte sedimentation rate (ESR) will often have high false-negative and false-positive results in an acute situation, as it is a nonspecific substitute marker for the acute phase reaction. As much as 100-fold or more increase is noted to be found in CRP in comparison to ESR. In addition, ESR is slower to respond to an inflammatory process than CRP, which is another reason for its low value when measured early in the inflammatory process. The patient then underwent surgical debridement of the Achilles tendon, requiring complete removal of the tendon and resulting in an FHL tendon transfer to reconstruct the Achilles tendon. Using the FHL to augment the Achilles has the advantage of supplemental blood circulation to the avascular distal Achilles tendon, which is crucial for eliminating infection and improving wound healing.¹⁸ The patient was referred to a rheumatologist and was treated with oral allopurinol 100 mg daily and colchicine 0.6 mg twice daily, with improvement of symptoms. At 12-month follow-up, the patient is fully ambulatory without any residual pain. This patient's presentation is consistent with many other differential diagnoses including but not limited to rheumatoid arthritis, tumor, pseudogout, and infection. In such cases, a high degree of suspicion is needed, especially in patients who have a history of gout.

Conclusions

The exact relationship between hyperuricemia and tendinopathy is still not fully understood. The differential diagnosis for a patient presenting with acute tendon pain of the lower extremity, such as the patient presented in this case study, should include the possibility of gout, and this possibility should be explored measuring serum uric acid levels in addition to proper imaging and

surgical intervention if warranted. With further research, the link between tendinopathy and gout can be better understood and more effectively treated.

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Conflict of Interest: None reported.

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