

An Unusual Case of Gouty Achilles Tendinopathy

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Background

Gout is the most common inflammatory arthritis and is the result of monosodium urate precipitation into joints and soft tissue due to hyperuricemia. While the disease process is classified as a form of arthritis, monosodium urate crystal deposition into extra-articular soft tissues such as ligaments and tendons is a common manifestation. It is thought that this crystal deposition activates key mediators of the inflammatory response, including interleukin-1 beta, which may act to initiate tendinopathy by inducing gene transcription of inflammatory molecules. (1) Monosodium urate (MSU) crystals have also been linked to the downregulation of certain metalloproteases that can compromise tendon extracellular matrix and reduce expression of type 1 collagen. (2) 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 computerized tomography (DECT) are the most commonly described modalities in the literature for visualizing gout within tendons. Visualizing tophi and aggregates on these imaging modalities not only can provide an accurate diagnosis but can accelerate the treatment process for these patients and serve as a detection tool to assess the efficacy of urate-lowering therapy. Here we discuss a case report of a patient who came to the emergency department with an acute gout flare presenting as posterior heel pain and Achilles tendinitis with inflammation.

Case report

A 49 year-old male patient presented to the emergency department with acute tenderness and an erythematous, painful mass to the right heel. The patient had undergone a previous ipsilateral retrocalcaneal exostectomy with Achilles detachment and reattachment with suture anchors 5 years before presentation. Patient had an unremarkable post-operative course, however did endorse mild, intermittent inflammation to the Achilles tendon and posterior heel during physical activity. He denied recent or inciting trauma. Upon 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 which measured approximately 4cm x 4cm (Fig. 1A and 1B).



Figure 1a and 1B: clinical presentation of the patient in the emergency room.

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 (Fig 2A-2C). A computed tomography (CT) scan was obtained, which revealed a 2.4cm x2cm x 0.9cm septated fluid collection in the soft tissues superficial to the Achilles tendon (Figure 2C). Empiric piperacillin/tazobactam was initiated. An incision and debridement was performed under general anesthesia.

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 which had caseous material similar to that seen with gouty arthropathy. After mass removal, there was noted to be minimal residual fibers of the Achilles tendon and a significant portion was excised with the mass. The nodule was sent for pathologic analysis. Post-irrigation cultures were obtained, which were negative. Pathology confirmed a diagnosis of gouty arthropathy using hematoxylin and eosin stain (Fig 3A-3C). The patient was taken back to the OR 10 days post-operatively for a debridement of the Achilles tendon and Flexor Halluces Longus (FHL) tendon transfer performed in prone position. The Achilles tendon was significantly degenerated and was therefore detached off its insertion and debrided to healthy margins. A Flexor Hallucis Longus tendon transfer was performed through the same incision. (3) The FHL tendon was transferred under physiologic tension with a tenodesis screw



Figure 2A



Figure 2B



Figure 2C

Figure 2A and 2B: AP and mortise views of radiographs. Previous anchor can be visualized along with soft tissue edema. Figure 2C is a sagittal view of the CT of a 2.4cm x2cm x 0.9cm septated fluid collection in the soft tissues superficial to the Achilles tendon.

Pathology specimens

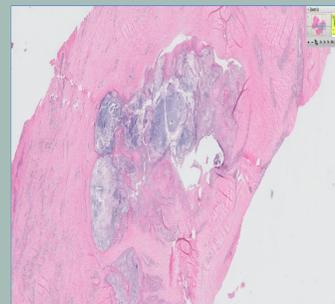


Figure 3A

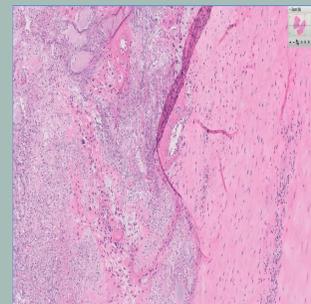


Figure 3B

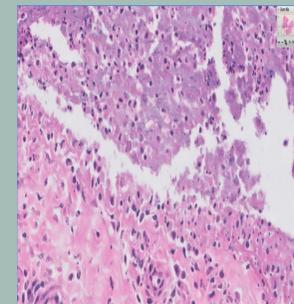


Figure 3C

Fig 3A: Hematoxylin and Eosin stain of tophus surrounded by chronically-inflamed tendon, mag 20X-1. Fig 3B: Magnification of figure 3A highlighting the chronic inflammation containing lymphocytes and the connective tissue reaction, mag of 400X-1. Fig 3C: Hematoxylin and Eosin stain highlighting the presence of neutrophils in the tophus and mixed into the surrounding tissue reaction, mag of 400X-1.

Post operative protocol

Post operative protocol: Patient was non-weightbearing 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. Allopurinol 100mg daily and colchicine 0.6mg twice daily are currently used for gout management. At 12 months follow up, the patient is able to ambulate without pain. Mild calf atrophy and expected weakness in plantar flexion was appreciated. Patient continues to function and perform activities of daily living without limitation.

Discussion

A study by Dalbeth et al concluded that MSU crystal deposition was observed in 10.8% of tendon/ligament sites. The Achilles tendon was the most commonly involved with an occurrence of 39.1%. (4) An ultrasound study by Ventura-Rios et al evaluating tendon involvement in patients with gout reported a nearly 50% prevalence of tophi and hyperechoic aggregates in the Achilles tendon. (5) However it was noted that some of these patients who had osteoarthritis also displayed these hyperechoic aggregates despite being asymptomatic and with normal uric acid levels, and this is due to the fact that these hyperechoic aggregates are not exclusive to gout.

Andia et. Al described a loss of tendon homeostasis secondary to inflammation and cell death as the link between hyperuricemia and tendinopathy. (1) The implications of a link between tendinopathy and hyperuricemia is reduction in tensile strength of the tendon which can lead to possible attenuation, tears, or even rupture of the involved tendon. Our patient in this report did not sustain a rupture of his Achilles however it has been documented that a common cause of pain in the Achilles tendon after trauma or surgery is gout. Our patient did have a remote history of Achilles tendinitis and underwent surgery for a painful retrocalcaneal exostosis with reattachment of the Achilles. The patient underwent surgical debridement of the Achilles and received oral treatment as an outpatient with improvement of symptoms as well as a referral to rheumatology. Patient has a follow-up of 12 months and has been fully ambulatory without any residual pain.

Conclusion

This case study on a 49 year old male patient with no acute Achilles injury who presented with a tender, erythematous right heel. Patient had debridement of the Achilles tendon and was placed on oral Allopurinol and Colchicine and referred to Rheumatology. Pathology specimen confirmed the diagnosis of gout. With over a 12 month follow-up, the patient is ambulating in regular tennis shoes with no complaints of pain. When a patient presents with acute tendon pain, gout should be considered in the differential diagnosis. It can be successfully treated with a staged protocol as noted in this case study.

References

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