Spontaneous Tibialis Anterior Tendon Rupture with Gout Predisposition: A Case Report

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Statement of Purpose

Tendon ruptures of the foot and ankle are most commonly due to direct trauma. Spontaneous tendon ruptures are less commonly documented in the current published literature, but typically result from predisposing factors which have compromised the structural integrity of the tendon. In current literature, there is minimal research on the correlation between gout and spontaneous tendon rupture. For those with recurrent gout, uric acid deposition and inflammation contributes to tendon weakening. Atraumatic rupture of tendons often results in retraction making end-to-end repair difficult. This case report documents the spontaneous anterior tibialis tendon (TAT) rupture in a patient with significant gout history.

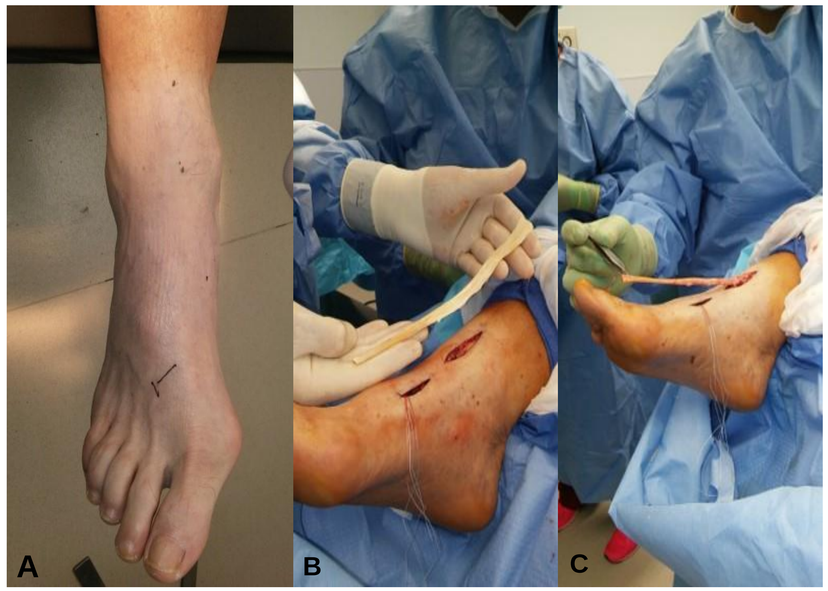

Figure 1: A) Clinical presentation of Right Foot B) Fresh Frozen Tendon Graft C) Insertion of Fresh Frozen Tendon Graft

Literature Review

Gout is an inflammatory arthritic disorder characteristic of monosodium urate (MSU) crystal accumulation within the articular and subcutaneous structures (1). Joints become inflamed and degenerative with chronic prolonged high levels of uric acid collection within tophi (2). In examination of the lower extremity, the first metatarsal phalangeal joint is most commonly affected, leading to pain out of proportion and disability. However, there is minimal medical literature available supporting gouty tophi infiltration within tendons and if this can be a predisposing factor leading to spontaneous tendon rupture within the lower extremity (3,4,5). Utilizing dual energy CT, Dalbeth et al. observed the presence of tophaceous gout within the feet of 92 patients, which included 199 affected tendons and ligaments. The Achilles tendon was the most commonly involved ligament/tendon site (48% of all patients) followed by peroneal tendons (27%), and tibialis anterior involvement at 14% of all patients (6). Furthermore, Stewart et al. found all affected muscle groups crossing the ankle joint had significant reduction in muscle strength (7,8). To conclude, tophaceous deposition in these tendinous structures leads to mechanical strain allowing for spontaneous ruptures.

Financial Disclosures: None

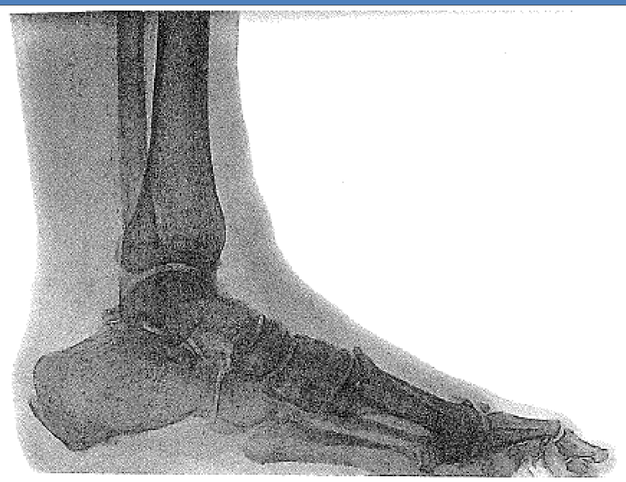

Figure 2: Lateral X-Ray No acute osseous findings

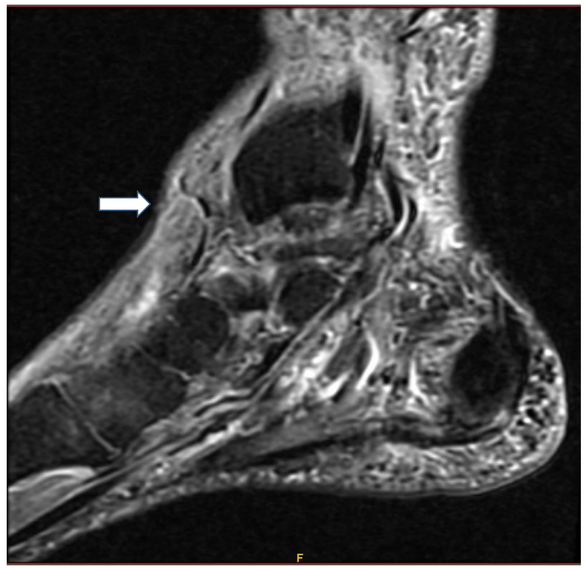

Figure 3: T2 MRI Tibialis anterior tendinosis with 2.7cm rupture and retraction of the thickened, edematous and frayed tendon edge to the level of the talonavicular joint.

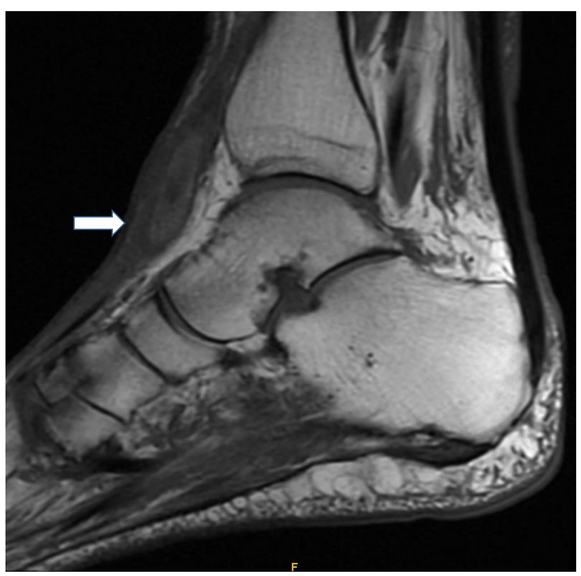

Figure 4: T1 MRI Tibialis anterior tendinosis with 2.7cm rupture and retraction of the thickened, edematous and frayed tendon edge to the level of the talonavicular joint.

Case Study

A 74 year old male with history of gout, presented with right foot pain of four weeks duration, denying any trauma. Patient had pain and 3/5 muscle strength when dorsiflexing his ankle. A palpable knot was noted at the medial dorsal foot along the course of the tibialis anterior tendon (Figure 1 A). No fractures or acute osseous changes were present on X-Ray (Figure 2). As seen in figures 3 and 4, the MRI revealed tibialis anterior tendinosis with rupture and 2.7cm retraction of the thickened, edematous and frayed tendon edge to the level of the dorsal margin of the talonavicular joint. The ruptured tendon was repaired two months after the initial visit. It was repaired utilizing fresh frozen tendon graft with an 3.5mm corkscrew bone anchor, which was inserted into the first cuneiform, along with resection of hypertrophic first cuneiform (Figure 1 B,C). Postoperatively, the patient was non weight bearing for 6 weeks in a below knee cast. After 6 weeks, patient was placed in a CAM boot and was to remain non weight bearing from weeks 6-8. Patient experienced symptoms of gout at postoperative week 2 for which he received Allopurinol, Indomethacin, and Colchicine. At the 3 month postoperative visit, patient had completed 7 weeks of physical therapy. Through the postoperative course, patient did not experience any unexpected pain or swelling. In addition, patient was back to normal ROM compared to the contralateral extremity, 5/5 muscle strength of all muscle groups crossing the ankle joint without pain or crepitus and there were no deformities or muscle wasting noted. At 13 month follow up, patient has full return of ankle dorsiflexion with 5/5 muscle strength.

Analysis & Discussion

Spontaneous rupture of the tibialis anterior tendon is a rare condition, with few cases reported. However, it may be noted more commonly in men in their 5th -7th decades of life. While traumatic rupture of the tibialis anterior tendon may be due to sudden plantar flexion of the ankle, spontaneous rupture can be the result of added strain on the tendon where there is history of systemic degenerative disease, or use of local steroid injections. It has been documented that gout can infiltrate tendons resulting in the deposition of urate crystals which can cause reduction of tendon tensile strength. This reduced strength can lead to degeneration or inflammation of the tendon and even rupture. Our case report describes a patient who had decreased dorsiflexion and non-traumatic tibialis anterior rupture with history of gout attacks, including an attack during the postoperative course. After repair with a fresh frozen graft the patient was able to regain full range of motion and strength. Currently, there is a lack of research for gout predisposing spontaneous tibialis anterior rupture and thus, we believe that our case can help contribute to the literature.

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