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Surgical Techniques for Repair of Atraumatic Tibialis Anterior Tendon Ruptures: A Report of Two Cases

Romy Patel, DPM [1], Lawrence Fallat, DPM, FACFAS [2]

[1] Second-Year Resident, Podiatric Surgical Residency, Beaumont Hospital Wayne, Podiatric Surgery, Wayne, MI
[2] Director, Podiatric Surgical Residency, Beaumont Hospital Wayne, Podiatric Surgery, Wayne, MI

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ABSTRACT

Tendon ruptures of the foot and ankle are overwhelmingly due to direct or blunt force trauma; however, spontaneous tendon ruptures have been less commonly documented in the published data. Surgical techniques for the repair of atraumatic ruptures differ from those for acute traumatic ruptures owing to the delayed patient presentation. Spontaneous tendon ruptures usually result from predisposing factors that have compromised the structural integrity of the tendon before the rupture occurs. We present 2 cases of atraumatic rupture of the tibialis anterior tendon, each repaired using a different surgical technique. A unique surgical procedure was selected after preoperative planning and individual patient considerations. Each patient had a minimum follow-up period of 12 months after surgery. Both patients returned to their previous functional status with no long-term sequela.

Ruptures of the tibialis anterior tendon (TAT) are quite rare, accounting for only 10 of 1014 foot and ankle tendon ruptures, as reported by Anzel et al (1). Most commonly, they are noted in males aged 50 to 70 years (2). Injuries to the TAT can occur from both traumatic and atraumatic mechanisms. Traumatic mechanisms usually involve direct injury such as an acute laceration or blunt trauma. These types of ruptures have frequently been reported. Spontaneous nontraumatic ruptures of the TAT have been sparingly reported, with the published data limited mostly to isolated cases.

Often, spontaneous ruptures occur with concomitant factors that predispose the tendon to failure. These factors include diabetes mellitus, inflammatory arthropathy, a history of steroid injections, or the use of fluoroquinolones (3). A history of diabetes with poor glycemic control has also been associated with increased tendon glycation. The end products of tendon glycosylation contribute to changes in collagen crosslinking within the tendon, which increases the risk of spontaneous rupture from tendon contracture and rigidity. Patients with diabetic-induced neuropathy are also known to have an increased risk of rupture of the flexor tendons (4).

Gout and other inflammatory arthropathies have been documented as a potential cause of a spontaneous TAT rupture. Patten and Pun (2) and Jerome et al (5) reported histologic findings of chalky white deposits within the substance of a ruptured TAT. When examined microscopically, gouty tophi were noted to have displaced the normal fibers of the tendon. The deposition of the uric acid crystals had completely compromised the structural integrity of the TAT tendon. Polarized light microscopy revealed pathognomonic findings of negatively birefringence needle-shaped crystals.

Localized steroid injections in the area of tendon insertion or along its course increase the risk of tendon rupture. The steroid causes tenocyte death, which reduces the tensile strength of the tendon. The most commonly documented cause of local steroid-associated TAT rupture is injections into the first tarsometatarsal joint (6).

The mechanism of failure for spontaneous rupture is an eccentric load of a degenerated, weakened TAT against a plantarflexed ankle (2). Although injury from blunt trauma and laceration is easily diagnosed, spontaneous rupture of the TAT is often associated with vague symptoms, such as mild pain, that subside shortly after the initial injury (7). After rupture of the TAT, the long extensors are recruited to assist in dorsiflexion (8), and this, along with no clear traumatic event, makes it difficult for patients to notice any acute pathologic features after injury.

Often, the vague symptoms can lead to the patient presenting past the acute stage of the tendon rupture. It is important to carefully evaluate the extremity and make an accurate diagnosis. On visual examination, a pseudocyst will commonly be noted along the anterior aspect of the ankle (i.e., the distal aspect of the ruptured tendon after it has retracted along its sheath). Extensor substitution can be seen when the patient attempts to dorsiflex the ankle. A marked weakness in dorsiflexion at the ankle joint on manual muscle testing is usually

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Address correspondence to: Lawrence Fallat, DPM, FACFAS, Podiatric Surgery Residency, Beaumont Hospital Wayne, 20555 Ecourse Road, Taylor, MI 48180.
E-mail address: lfallatdpm@aol.com (L. Fallat).

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noted compared with the contralateral limb. Pain is not a reliable marker in the subacute stage of the injury owing to the slow resolution of the symptoms as time passes. On gait examination, the patient will be seen to have a mild "foot slap," and firing of the long extensors will be seen during the swing phase to help compensate for the loss of dorsiflexion.

The common differential diagnoses for a TAT rupture include a common peroneal nerve lesion causing foot drop or L5 radiculopathy. A combination of imagining modalities and a careful physical examination will easily lead to the exclusion of these causes in a spontaneous TAT rupture. The medical imaging findings can provide a quick and accurate confirmation of the diagnosis once a thorough physical examination has been performed. Although ultrasonography can be used, magnetic resonance imaging (MRI) is considered the reference standard because of its diagnostic ability to evaluate the TAT and the anatomic structures surrounding it (9).

The TAT is the primary dorsiflexor of the foot (10). During the swing phase of gait, contraction of the TAT prevents stumbling or tripping by clearing the foot over the ground. In the contact phase at heel strike, it aids in balancing plantarflexion of the foot by eccentric contraction and preventing foot slap (2). A prompt diagnosis and appropriate treatment of a rupture can prevent long-term sequela such as lost dorsiflexion, which can result in drop foot and Achilles tendon contracture (11). Drop foot alters the normal gait and can lead to abnormal ambulation and increase the risk of future injury.

The most common anatomic area of rupture correlates directly with the zone of hypovascularity within the TAT. The vascularity of the tendon is nonhomogeneous, with the anterior half avascular between the superior and inferior extensor retinaculum. This area measures 45 to 67 mm long (12).

Appropriate management of these injuries will differs according to each patient's case. Markarian et al (8) found no significant differences in the outcomes between operative and nonoperative treatment of patients with a TAT rupture. However, in their study, the average age of the operative group was 19 years younger than that of the nonoperative group. Also, a predominance of acute ruptures was present in the operative group. Operative treatment of a TAT rupture for young and active patients for both acute traumatic and subacute spontaneous ruptures remains favored for long-term functionality (13). The mechanism and acuity of the injury are important factors to consider in preoperative planning. A direct surgical approach with end-to-end repair of the tendon with nonabsorbable suture is common for acute traumatic injuries. Alternatively, if the distal portion of the ruptured tendon is nonviable, the tendon can be directly attached to the proximal aspect of the medial cuneiform using a screw and washer, pull-out wire, interference screws, or suture anchors (14). Chronic injuries that present >1 month after injury require more complex surgical management. Often, end-to-end repair of the tendon will no longer be possible because the tendon has retracted proximally along its sheath. Numerous techniques can be used to overcome the deficit left by tendon retraction and to aid in the overall strength of the tendon, including slide lengthening, tendon turndown, interposition or tendon grafting, and augmentation (13,15,16).

Case Reports

We present 2 cases, each outlining an atraumatic, spontaneous rupture of the TAT and the appropriate treatment method, with a focus on technique. A different surgical technique was used to repair the tendon in each case. To the best of our knowledge, the combination of differing acuity, method of injury, and surgical technique is unique.

Both patients in our reported cases were placed in the supine position on the operating table. A thigh tourniquet set at 325 mm Hg was used for hemostasis. The patients underwent the procedure under general anesthesia. Cefazolin 2 g intravenously was administered for surgical prophylaxis.

Postoperatively, both patients were placed in a well-padded below-the-knee fiberglass cast, with the foot positioned at 90°. They were instructed to be non-weightbearing on the surgical extremity. Crutches were used for ambulation. Oral deep vein thrombosis prophylaxis and pain medication was prescribed for both patients.

Patient 1

The first patient was a 50-year-old female with a medical history of type 2 diabetes and social history of smoking. Her diabetes was rather poorly controlled. Her medications included glipizide (Glucotrol) 10 mg, metformin 1000 mg, and sitagliptin (Januvia) 100 mg. Her hemoglobin A1c was 8.3, with a mean plasma glucose of 216 mg/dL on the day of surgery. She presented to the clinic with a chief complaint of mild pain and weakness along the anteromedial aspect of the right ankle. The patient stated that she remembered the pain beginning about 8 weeks before her presentation to our clinic. She stated that she had "felt a pop," followed by instant severe pain to the anterior aspect of the right ankle while getting out of bed 1 night. Soon after, the pain had subsided, but the weakness in her ankle remained.

On examination in our clinic, we noted a distinct weakness in dorsiflexion of the right ankle. Absence of contracture of the TAT when dorsiflexing was also noted (Fig. 1). During the gait examination, her toes dragged on the ground, and a mild foot slap was noted. An MRI scan was obtained to confirm the working diagnosis of a TAT rupture. The MRI study showed complete rupture of the TAT, with a deficit of 7.8 cm between the proximal and distal stumps (Fig. 2). The large deficit had resulted from the subacute nature of the presentation of the patient, which had allowed the ruptured tendon to retract

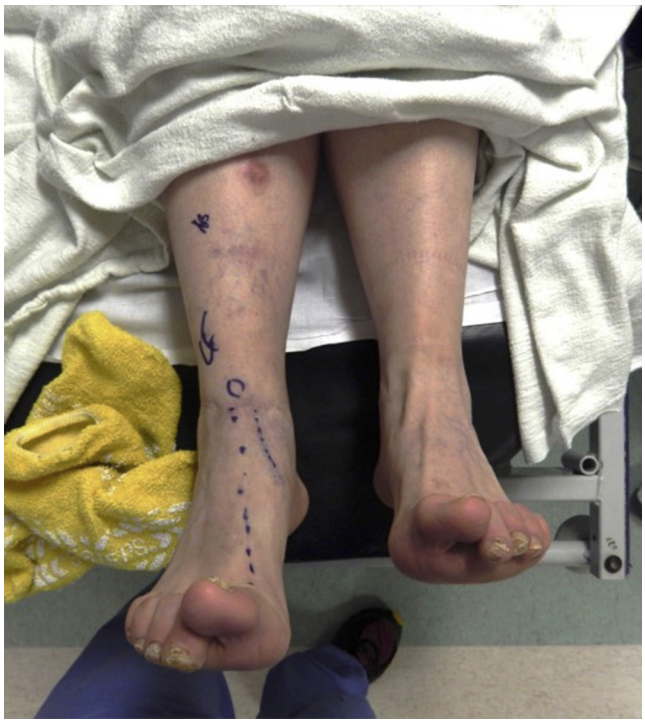

Fig. 1. View of right foot of patient 1 showing significant loss of dorsiflexion at the ankle joint with hyperextension of the hallux. Note the absence of contracture along the course of the tibialis anterior tendon on the right foot.