

Modified Kelikian Procedure for Delayed Presentation of Large Gap Deficit Tibialis
Anterior Tendon Rupture

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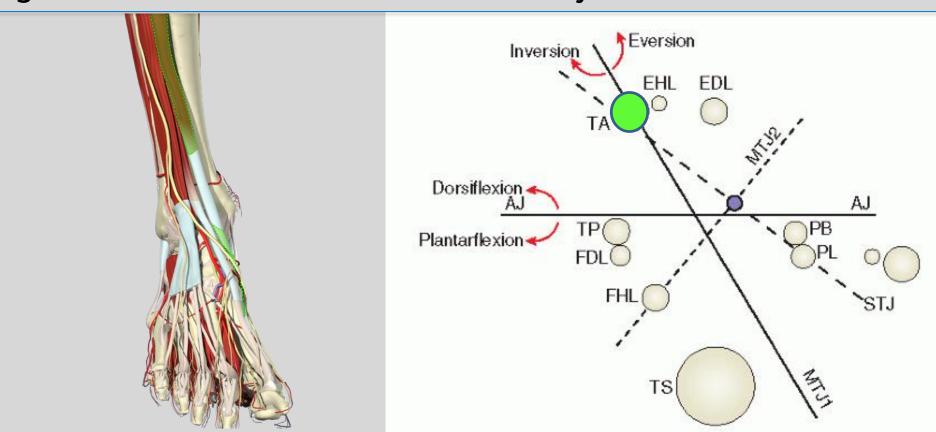
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# Purpose

Little literature exists regarding treatment options for tibialis anterior tendon (TA) rupture. We present a previously undescribed modification to the Kelikian procedure for repair of a chronic tibialis anterior tendon rupture with a large gap deficit.

Figure 1: Mechanical Axis of Lower Extremity



### Literature Review

Approximately 80% of dorsiflexion strength is produced by the TA<sup>1</sup> (Figure 1). If this tendon is unable to function the extensor hallucis longus and the extensor digitorum longus are recruited to assist with dorsiflexion.

The gait cycle consists of two phases; stance phase (60%) and swing phase (40%). The TA is active twice during this cycle. First, at heel strike, the TA eccentrically controls the progression of the foot to flat position in stance phase. Secondly, the TA concentrically contracts during the swing phase of gait to allow for dorsiflexion of the ankle and clearance of the foot from the ground.

The proximal blood supply of the TA comes from the anterior tibial artery and the myotendinous junction while the distal supply comes from branches of the medial tarsal artery<sup>2</sup>. Petersen et al.<sup>3</sup> described the distribution of blood vessels to the TA as non-homogenous. The posterior half of the TA tendon has a complete vascular network from the myotendinous junction to the insertion. The middle and anterior part of this tendon has a relatively avascular zone between 5-16mm from the insertion to 45-68mm proximally. This coincides with the area of tendon that is not covered by the extensor retinaculum. The vast majority of ruptures occurs at this avascular zone.

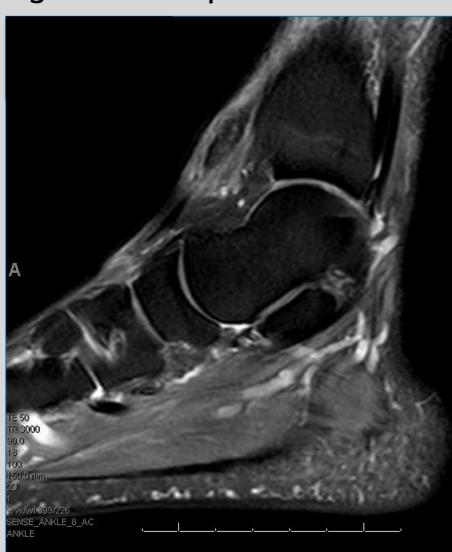
Tibialis anterior tendon ruptures are extremely uncommon injuries.

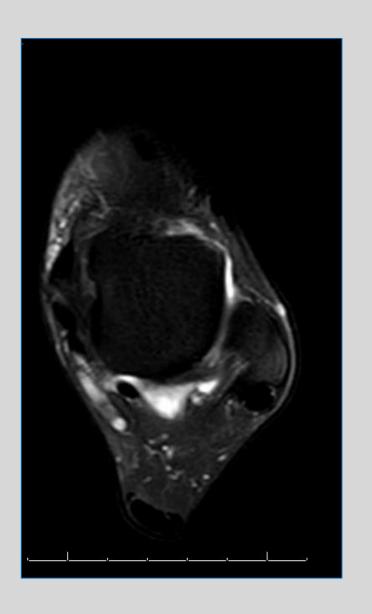
Patients often present with vague symptoms of anterior ankle pain or the complaint of "foot slap" during gait. Ambiguous symptoms often lead to a delayed diagnosis. Markarian et al. reported the average delay in presentation of 10 weeks due to seemingly retained muscle strength<sup>4</sup>.

# Literature Review

The goal of TA rupture treatment is to relieve pain and restore function. No consensus exists regarding operative vs non-operative management. Conservative treatment offers an attractive option for elderly or lowdemand patients, although there exists a 60% non-compliance rate with AFO bracing<sup>5</sup>. Surgical treatment options include: primary repair, non anatomic repair, reconstruction with adjacent tendon, free autograft, or allograft<sup>2</sup>. The first comparative study of operative and non-operative treatment was published in 1998<sup>4</sup>. They report on 16 patients, 8 operative an 8 non-operative. Out of the 8 operative patients, there were 5 separate procedures performed to achieve repair. They were unable to show statistical difference for outcome score of operative vs nonoperative. Recently, a large systematic review of TA rupture treatments was performed<sup>6</sup>. A total of 87 patients were included in this review. Seventy two were surgically treated and 15 were conservatively treated. The authors noted that there was a large variation in operative techniques, primary repair being the most common (N=23). Upon closer analysis, ruptures repaired within 3 months, primary repair was most common. Ruptures repaired greater than 3 months, autograft was used<sup>6</sup>. Mean post-operative treatment scores were able to be directly compared using the FAOS score. Surgical repair, regardless of technique, scored 84.4 while conservative treatment averaged a score of 69.4.

Figure 2: Pre-Operative MRI





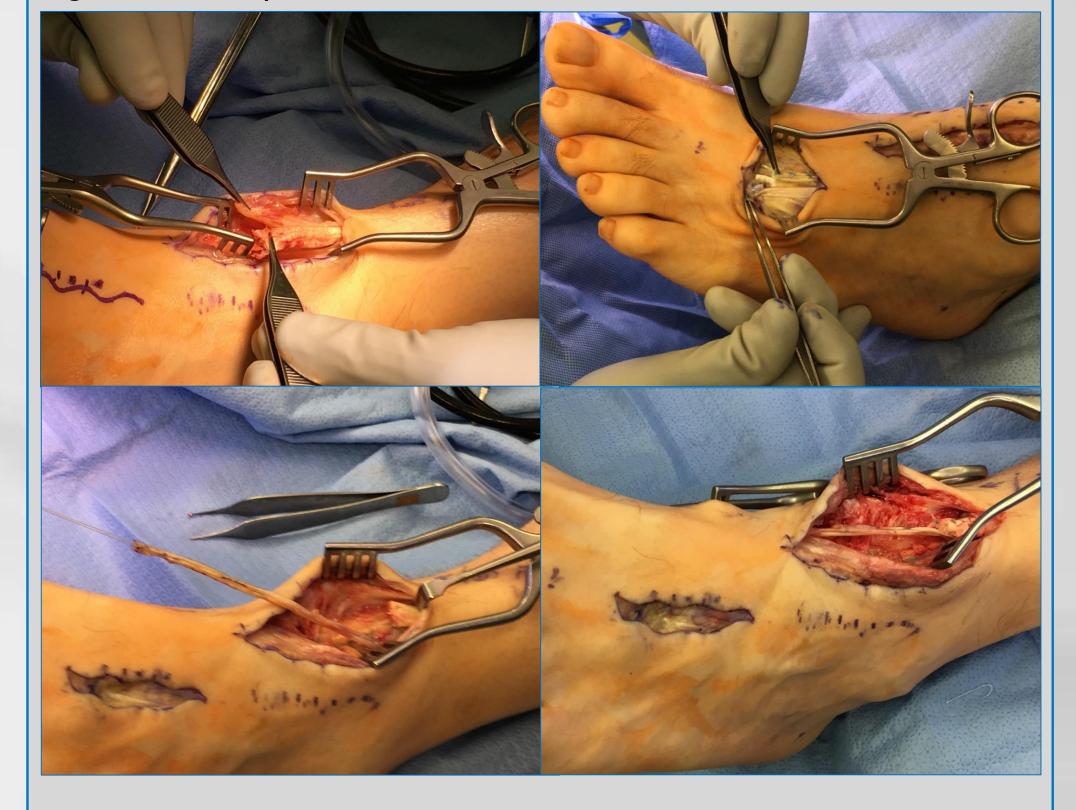
# Procedure

Patient is a 55 year old male with a PMHx of Type II DM (HbA1C 7.4), HTN, and HLD who sustained a complete TA rupture following a ground level fall while on carpet. Initiation of treatment was delayed by 3 months as PCP believed tendon was intact due to persevered dorsiflexion. During Podiatric evaluation patient was noted to have a palpable mass in area of TA just proximal to ankle and a dorsiflexion muscle strength of 4-/5 with recruitment of lesser toes and hallux. Additionally, a high steppage gait was noted. MRI confirmed a complete TA rupture with a 7cm deficit at the level of the ankle joint (Figure 2).

#### Procedure

Patient was taken to the operating room for TA tendon repair. Incision was placed over the TA tendon proximal to the ankle joint. Proximal stump was identified and debrided back to healthy appearing tendon. This tendon stump was noted to glide freely within the sheath. The same was performed at the insertion. This left an 8cm deficit between the healthy tendon ends. A primary repair was impossible. Decision was made to perform an autograft repair using the EDL tendon slips. An incision was made in the midfoot over the EDL tendon of the 3<sup>rd</sup> digit. The EDL tendon slips of 2, 3, and 4 were harvested and combined using fiberwire in a side to side fashion. The distal stumps of these tendons were tenodesed to the intact 5<sup>th</sup> slip of the EDL. The harvested bundle of EDL tendons were passed beneath the retinaculum and tenodesed to the proximal TA stump. This, again, was then passed under the retinaculum and anchored to the medial cuneiform with a tendodesis screw and tenodesed to the intact TA insertion stump. (Figure 3).

Figure 3: Intra-Operative Photos



# **Analysis & Discussion**

We present a surgical technique utilized to restore the function of a chronic TA rupture. Our patient had a large gap deficit which made primary repair impossible. In contrast to the originally described Kelikian procedure, this modification used EDL tendon slips while maintaining their native function. The patient was able to regain normal TA function.

# **Analysis & Discussion**

This case presented a delayed diagnosis TA rupture of 3 months. A study Markarian et al., found that the average delay in diagnosis was 71 days<sup>4</sup>. They postulated that delay in diagnosis was due to a low index of suspicion. In our case, the rupture was missed by the patient and the PCP. A systematic review of TA rupture treatments was performed in 2015<sup>6</sup>, they found that ruptures greater than 3 months, autograft was used most commonly. Our case holds true to this. Due to the patient's large gap deficit an autograft was used. The Kelikian procedure was originally described in 1985 and consisted of 2,3 EDL tendon Pulvertraft to the TA tendon and a tenodesis of 2,3 EDL to the EDB<sup>7</sup>. We decided to perform a side to side tenodesis of the 2, 3, 4, EDL slips to the TA to avoid anterior ankle bulk. Additionally, the distal stumps of the harvested tendons were tenodesed to the intact 5<sup>th</sup> EDL slip to maintain anatomic function as we believe the EDB was unable to perform the bulk of the dorsiflexion of the lesser digits.

The goal of this surgery was to avoid as much insult to the retinaculum as possible and give the patient a normal gait pattern. This was achieved. At 14 month follow up patient was noted to have 5/5 muscle strength with dorsiflexion without evidence of extensor substitution. Gait was non-antalgic, symmetric, and propulsive. No clawing of digits was noted. A gait analysis study was performed of TA rupture repair using a Z-plasty<sup>8</sup>. This study noted that TA rupture repair does not restore physiologic gait pattern fully with significantly greater plantarflexion in terminal swing phase. They believed this was due to scar tissue formation. Our study avoided significant scar tissue formation by decreasing the retinaculum insult by using tendon passers.

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## **Financial Disclosures**

Jacob Russell Hagenbucher - none

Michael Gentile - Flower Orthopedics - intellectual property, surgeon advisory board, consultant; Tyber Orthopedics - surgeon advisory board, consultant; Depuy/Synthes - consultant