

Original Article

Tibialis anterior tendinosis: Clinical characterization and surgical treatment

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ABSTRACT

Background: Tibialis anterior (TA) tendinosis is rarely reported on in the literature. It is seen in patients older than 45 and causes weakness in dorsiflexion. This paper aims to describe surgical treatment and clinical outcomes.

Methods: Between 2015 and 2018, nine patients (six females, three males) with severe TA tendinosis with no tear (2), partial (1), or complete (6) underwent operative treatment. Patients underwent debridement and direct repair without augmentation, direct repair with fiber tape augmentation, tibialis posterior tendon (PTT) transfer, or tibialis anterior tendon (TAT) augmentation with a tendon autograft (n = 4). Autografts consisted of extensor digitorum longus (EDL) tendon, plantaris tendon, or both.

Results: Mean postoperative follow-up was 21.3 (range 8–31) months. All patients had a concomitant gastrocnemius recession, and three had hindfoot arthrodesis. Preoperative dorsiflexion strength was 0/5 for all and improved to 5/5 postoperatively in seven. The only current smoker developed wound dehiscence 2 weeks postoperatively and healed by 4. One developed marginal skin necrosis 3 weeks postoperatively and was treated successfully with casting.

Conclusion: Surgery reestablished function in individuals with TA tendinosis and allowed high level of satisfaction. Direct repair is possible. If the tendon gap is too large an autograft of EDL and plantaris tendon can be utilized.

Level of evidence: Level III Retrospective Comparative Study

1. Introduction

Tendinopathy is the generic term for the clinical syndrome of pain, swelling and impaired function of a tendon, regardless of etiology [1–3]. Abnormalities of the tibialis anterior tendon (TAT) are much less common than in other tendons because of its straight course and relatively minor exposure to mechanical stress [4]. Spontaneous subcutaneous TAT rupture is the most published condition, with 87 cases reported between 1975 and 2012. Some patients recall a specific moment of rupture, followed by pain, swelling, and functional deficits [5]. Others are asymptomatic and unaware of their injury due to compensation by other dorsiflexors of the toes, such as the extensor hallucis longus (EHL) and extensor digitorum longus (EDL). Demographically, patients with this condition are typically older males with medical comorbidities that have been associated with the rupture of other tendons, such as diabetes mellitus, rheumatoid arthritis, gout, and systemic or local steroid use [6–10]. Physical exam is often positive for

a palpable mass on the dorsal aspect of the ankle with decreased strength and range of motion (ROM). There is reduced ankle dorsiflexion, a drop foot with associated high-step gait, and recruitment of EHL and EDL with ambulation. If untreated, long-term sequelae can include clawing of the toes due to EHL and EDL overcompensation, gastrocnemius, and Achilles tendon contracture, and limited ambulation [7]. Imaging with ultrasound or MRI characteristically shows TAT discontinuity with retracted stump ends. Treatment ranges from no intervention to conservative management with an ankle-foot orthosis (AFO) or walking cast, plus activity modification and/or physical therapy, to surgical repair with or without a tendon graft [5,10].

Tendinosis often precedes tendon rupture, and many articles reporting “partial tears” or “chronic rupture” of a given tendon are in reality describing chronic tendinosis [2,3]. Severe tendinosis alone without tear can cause pain and functional deficits necessitating surgical intervention; however, with regards to the TAT, this has not been described, except for one report on distal TA tendinosis, which appears

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Table 1
General characteristics of diagnosis, surgery, and outcomes.

Patient	Symptom duration (months)	Diagnosis	Tendinosis segment length (cm)	Repair technique ^{GR}	Additional diagnosis	Dorsiflexion strength		Final F/U (months)	Complications
						Pre-op	Post-op		
1	3 month	Chronic TAT rupture/tendinosis	4	Direct repair + fibertape augmentation	Equinus contracture	0/5	5/5	29	
2	6 month	Chronic TA tendinosis without rupture	1.5	PTT transfer to midfoot	Foot drop	0/5	4/5	25	
3	6 month	Acute on chronic TA tendinosis with partial tear of TAT	8	EDL tendon autograft	Equinus contracture	0/5	5/5	8	
4	3 month	Complete TAT rupture	4	Plantaris tendon autograft*	Severe talonavicular cuneiform arthritis, achilles tendon rupture, equinus contracture	0/5	5/5	14	Wound dehiscence (2 weeks)
5	3 year	TA tendinosis without rupture	3	Direct repair	None	0/5	5/5	18	
6	3 month	Complete TAT rupture	6	Direct repair	Foot drop, tibial varus deformity	0/5	N/A	23	
7	3 week	Chronic TA tendinosis with rupture	8	EDL tendon autograft ^{SF}	Equinus contracture, PTT dysfunction	0/5	5/5	22	
8	3 week	Complete TAT rupture	N/A	Direct repair + fibertape augmentation ^{SF}	PTT dysfunction, planovalgus deformity, mid foot arthritis, equinus contracture	0/5	5/5	31	Subtalar nonunion (21 months)
9	3 week	Chronic TA tendinosis with rupture	3.5	EDL and plantaris tendon autograft	Equinus contracture	0/5	5/5	N/A	Marginal skin necrosis (3 weeks)

* Concomitant talonavicular fusion, subtalar fusion, and naviculocuneiform fusion performed.

to be a distinct condition [11–16]. In the present study, nine patients with TA tendinopathy requiring surgical management are reported: two with tendinosis alone, one with a partial tear, and six with complete tears. The senior author's preferred operative techniques and the respective outcomes for treating TA tendinopathy are presented.

2. Materials and methods

2.1. Data collection

A retrospective chart review was performed for nine patients with tibialis anterior tendinosis managed by the senior author between 2013 and 2018. Collected data included: dexterity (left or right foot), demographics [age, sex, race, and body mass index (BMI)], medical comorbidities [history of diabetes mellitus (DM), hypertension (HTN), rheumatoid arthritis, other inflammatory arthropathy, immunosuppressive drug use, peripheral vascular disease (PVD), chronic obstructive pulmonary disease (COPD), peripheral neuropathy, congestive heart failure (CHF), other pulmonary disorder, chronic kidney disease (CKD), Human Immunodeficiency virus (HIV status), smoking, or substance use], history of present illness (symptoms, duration of symptoms prior to decision for surgical treatment, prior foot or ankle injury, and prior foot or ankle surgery), physical exam findings of the foot and ankle, diagnostic imaging, preoperative diagnosis, postoperative diagnosis, surgical details (gross TAT appearance, length of tendinosed segment, surgical repair technique, and concomitant surgeries), early postoperative complications (superficial surgical site infection (SSI), deep SSI, wound dehiscence, or sepsis), other complications, preoperative and postoperative strength and ROM with dorsiflexion, and time to final follow-up.

2.2. Surgical technique

After administration of general anesthesia, a tourniquet was applied around the thigh and inflated to a pressure of 280 mmHg. First, a gastrocnemius recession consistent with the standard Strayer technique was performed in all patients [17]. In all cases, intraoperative exam confirmed that approximately 15–20° of dorsiflexion could be achieved after gastrocnemius recession.

Next, a second incision was made over the anteromedial aspect of the tibialis anterior tendon, extending from the level of the superior extensor retinaculum to the level of the medial cuneiform. Severe tendinosis of the TAT was identified in all cases; one also had a distinct partial tear, and six had a complete tear. Visual inspection and evaluation were performed to determine the length and severity of the degenerated tendon segment under neutral ankle dorsiflexion. Next, the tendinosed segment was excised such that only healthy tendon remained. If the remaining healthy tendon end or ends were no less than half of their initial thickness and could be approximated or brought to its insertion, then direct tendon repair without augmentation was performed. If not, then the tendon was augmented with an autograft consisting of EDL tendon, plantaris tendon, or both. In one case, tibialis posterior tendon (PTT) transfer to the midfoot was performed.

The senior author's preferred technique was to augment the TAT with a tendon autograft comprised of EDL and/or plantaris tendons. The plantaris tendon was identified during the gastrocnemius recession and harvested using a tendon stripper. Next, incisions were made over the second and third metatarsal heads, the EDL tendon slips were released at the level of the metatarsal heads, and the tendon was harvested proximally through the same incision used for the TAT exposure. Then, the plantaris tendon and EDL tendons were incorporated together to form a single tendon graft, connecting medial cuneiform and distal end of the proximal TAT. In all cases, the proximal end of the distal tendon was incorporated with the free graft. Approximation of proximal and distal ends was attempted in all cases and the tendon ends were brought together whenever possible.

Postoperatively, all patients followed a three-phase rehabilitation protocol. Beginning immediately after surgery, patients remained non weightbearing in a cast for 4–6 weeks, until complete healing of the skin and soft tissue occurred (Phase 1). Outpatient physical therapy was started at the beginning of Phase 2. In this phase, patients remained in a boot for 6 weeks, progressing to partial- and full-weightbearing status as tolerated. In Phase 3, the boot was exchanged for an AFO at 10–12 weeks, which was maintained for a minimum of 3 months to protect the repair.

3. Results

General characteristics of diagnosis, surgery, and outcomes are shown in Table 1. All patients were evaluated and treated surgically by the senior author between 2013 and 2018. Of the nine patients (nine feet), six were women (six feet) and three were men (three feet), with a mean age of 54 (range, 32–62) years at time of surgery. Six were Caucasian and three were African-American. Four cases were left-sided and five were right-sided.

The duration of symptoms prior to decision for surgical treatment ranged from 3 weeks to 3 years (mean = 33 weeks). Three patients reported a prior ankle sprain injury related to symptom onset, one recalled the possibility of an ankle sprain, and one had a history of iatrogenic TAT laceration. The other four patients recalled no acute injury, although one noted acute onset of pain and swelling after stretching her foot and hearing a “pop”. In terms of medical comorbidities, four patients had diabetes mellitus, four had hypertension, two had gout, two had rheumatoid arthritis, one had ankylosing spondylitis, and one was an active smoker. All patients had a documented history of at least two medical comorbidities. Two patients reported systemic use of immunosuppressive medications, one patient with methotrexate, and one with methotrexate, prednisone and rituximab. The latter patient also reported receiving a cortisone shot in the midfoot region three months prior to presentation. All nine patients reported a history of peripheral neuropathy. According to BMI classification, five patients were obese (greater than 30), three patients were overweight (25–29.9), and one patient was normal weight (18.5–24.9).

All nine patients had tenderness to palpation at the dorsomedial midfoot of the affected extremity. Six patients had documented drop foot and high-step gait, and six had equinus contracture. Five patients had a palpable lump along the anterior aspect of the affected ankle. Preoperative ankle active dorsiflexion was zero degrees (normal = 20°) for four patients, and undocumented for five patients. Preoperative ankle dorsiflexion strength was 0/5 for all nine patients.

All nine patients had imaging with X-ray (n = 9). Additional imaging was obtained in seven patients using ultrasound (n = 3), or MRI (n = 4) conducted at our facility. One patient had ultrasound imaging at another facility. The MRI findings included: TAT thickening without discrete tear (n = 1), severe TA tendinosis with partial tear (n = 1), chronic TAT rupture with tendinosis (n = 1), and complete TAT tear with thickening and retraction to the tibiotalar joint. MRI's and images of tendinosis are shown in Figs. 1 and 2. The ultrasound findings suggested: no tear (n = 1), full thickness TAT tear (n = 2), and full-thickness TAT tear with retraction to tibiotalar joint (n = 1).

Table 1 shows the operative techniques and outcomes for each patient. Intraoperatively, all nine patients were found to have TA tendinosis, and all underwent debridement and excision of the tendinosed segment. Two patients underwent direct repair of the TAT without augmentation. Two patients underwent direct repair with fiber tape augmentation: one with TAT transfer to middle cuneiform, and one with re-approximation of the tendon ends. One patient underwent PTT transfer. This patient had two previous TAT surgeries at an outside hospital: first, repair for an iatrogenic TAT laceration; and second, debridement for chronic TA tendinosis. Four patients underwent TAT augmentation with tendon autograft: two using EDL tendon, one using plantaris tendon, and one using both EDL and plantaris tendon, as

described in the surgical methods. All nine patients had a concomitant gastrocnemius recession. Three patients had concomitant hindfoot arthrodesis. Fig. 3 depicts intra-operative clinical picture of tibias anterior tendinosis and Fig. 4 shows the harvested tendon graft for augmentation.

The mean duration of postoperative follow-up was 21.3 (range, 8–31) months. Postoperative ankle dorsiflexion strength was 5/5 for seven patients, 4/5 for one patient, and unavailable for one patient, due to insufficient follow-up at the time of this study. Postoperative ankle dorsiflexion ROM was 20° for four patients, two improving from a preoperative ROM of zero degrees.

One patient had wound dehiscence, 2 weeks postoperatively, with subsequent healing by 4 weeks postoperative time. Another patient developed marginal skin necrosis 3 weeks postoperatively, which healed with casting and local wound dressing. This patient was also the only current smoker. One patient had chronic subtalar nonunion, status-post concomitant subtalar fusion.

4. Discussion

TA tendinopathy is a rare clinical entity that has primarily been studied through case reports and low-volume case studies limited to patients with complete TAT rupture. Mechanisms of TAT rupture include direct trauma, closed indirect trauma, an applied dorsiflexion force, and spontaneous subcutaneous rupture, the last being the least reported [5,18]. Most cases of spontaneous rupture appear to be preceded by chronic degeneration, often related to underlying medical problems, such as diabetes mellitus, diabetic-induced neuropathy, inflammatory arthropathy, gout, or previous steroid injections [10,18]. Despite its apparent pathophysiologic role in TAT rupture, TA tendinosis has not been studied, except for one paper on distal TA tendinosis, and one on its operative management. Considering the sample size of previously published literature and the novelty of this condition, a sample size of nine of this retrospective study should be considered adequate to add important data in literature.

There has been one report characterizing distal TA tendinosis without rupture in 29 patients [19]. The patients were mostly elderly, overweight women without other medical comorbidities. They reported burning medial midfoot pain and swelling, but no physical exam deficits. The senior author later reported on 11 patients who underwent surgery for this condition [16]. Six received debridement reinforced with a suture anchor, and six received augmentation with an EHL transfer into the medial cuneiform. Aside from these two reports on distal TA tendinosis, symptomatic TA tendinosis without rupture has not been described.

In a 2015 systematic review of 87 cases of spontaneous subcutaneous TAT rupture, the average age was 65.8 years [5]. Our sample of patients with TA tendinosis was an average of 10 years younger (mean age 54, range 32–62 years), supporting the concept that degeneration precedes complete tear. Many authors consider age an important factor for selecting conservative versus surgical treatment of TAT rupture [5,20,21]. Christman-Skieller et al. (2015) reported that although patients treated conservatively tend to be older (mean age 72.4 years) than those treated surgically (mean age 63.9 years), age itself is not a contraindication for operative repair; many authors have described positive outcomes for active, elderly (greater than 70 years) patients who wanted to maintain their activity level and therefore opted for operative repair [5]. Surgical repair provides very good outcomes, and patients who are operative candidates should be counseled that surgical treatment leads to fewer sequelae, such as persistent drop foot and flatfooted gait, with a strong likelihood of return to baseline activity status. Surgical options include primary repair, non-anatomic repair, reconstruction with adjacent tendon transfer, free autograft reconstruction, and allograft reconstruction. Patients who are not surgical candidates or who are strongly against surgery can be offered conservative treatment with AFO, physical therapy, and activity



Fig. 1. Sagittal MRI of tendinosis.

modification, however, persistent gait abnormalities and weakness in dorsiflexion are likely outcomes [5,6].

Although three of nine patients in our patient sample did not have complete tear of the TAT, they exhibited findings similar to those with TAT rupture, including drop foot and high step gait, decreased ROM and strength with dorsiflexion, and recruitment of EDL and EHL with ambulation. In this regard, chronic TA tendinosis may be considered anatomically distinct from, but functionally similar to TAT rupture. Indeed, our approach to treatment of TA tendinosis paralleled that of TAT rupture. Based upon our patients' considerable functional deficits, relatively young age, and desire to return to their baseline activity status, all nine patients were offered, and opted for, surgical treatment. The surgical approach for these patients was similar to that of patients with TAT rupture: debridement followed by direct repair, reconstruction with adjacent tendon transfer, and/or augmentation with a tendon graft.

The surgical technique for repair of TAT rupture can often be predicted based upon injury chronicity but is ultimately chosen based upon the length between the two ruptured ends. Harkin et al. report that 2 cm–4 cm defects can be repaired directly or with TAT sliding graft, but defects greater than 4 cm require a free tendon graft [6]. Chronically ruptured (greater than 3 months) TAT's tend to have more severe stump retraction and tissue necrosis, requiring a free tendon graft for repair; whereas acutely ruptured (less than 3 months) TAT's have less retraction and are often repairable through direct end–end anastomosis. In our study of TA tendinosis, there is no relationship between the symptom chronicity, and the length of the tendinosed segment or the

surgical approach. For example, Patient 2 reported 3 years of pain, and was found to have 1.5 cm tendinosed segment treatable through direct repair; whereas Patient 3 reported only 3 weeks of pain but was found to have an 8 cm tendinosed segment requiring a free tendon autograft. This finding reinforces the concept that tendinosis can be largely asymptomatic, and symptom chronicity does not reflect the degree of the underlying structural degeneration. Additionally, the length of the tendinosed segment did not predict the use of direct repair versus tendon free graft. Patient 9 had a 3.5 cm tendinosed segment excised and required a free tendon autograft; however, Patient 6 had a 6.0 cm segment removed and was able to undergo direct repair. Thus, the length of tendinosed segment does not predict the distance between the two healthy tendon ends under tension, or the ability for direct end–end anastomosis.

Several etiologies of TAT rupture have been proposed, one being inherent hypovascularity of the distal end of the TAT. Avascular tissue is bradytrophic and has poor healing potential, which makes it vulnerable to repetitive microtrauma and subsequent degeneration [4]. In a study of the normal blood supply to the TAT, Petersen et al. discovered a 45–67 (mean 56.6) mm avascular zone within the TAT, ranging from 5 to 17 mm (mean 10.1 mm) from its insertion site [22]. The inferior half of the avascular zone corresponds to the most frequent site of spontaneous TAT rupture, which is within 5–30 mm from its insertion [10,23]. While hypovascularity may play a role in the degeneration that precedes TAT rupture, inherently insufficient blood supply is probably not responsible for the TA tendinosis in our study. In our sample, the length of tendinosis ranged from 1.5 cm to 8 cm, with



Fig. 2. Transverse MRI of tendinosis.

the average tendinosed segment (4.75 cm) being 8.4 times the size of the average avascular zone in Petersen et al.'s study of normal TAT blood supply [22].

The degeneration seen in our sample is more likely related to systemic medical disease and use of immunosuppressive medication. The aforementioned factors have been shown to cause tendinosis and appear to be highly prevalent in cases of TAT rupture [3,23–25]. As predicted, these medical problems were remarkable in our study of patients with TA tendinosis. Two patients reported use of the immunosuppressive medication methotrexate, one also receiving prednisone, rituximab, and a recent cortisone injection to the midfoot of the affected extremity. All nine patients in our series had systemic disease: five had HTN, four had DM, two had gout, two had RA, one had ankylosing spondylitis, and all nine had peripheral neuropathy from diabetes. This last point is highly noteworthy because diabetic neuropathy indicates chronically poor glycemic control, and uncontrolled DM induces notable structural, inflammatory, and vascular changes in tendons, predisposing diabetic patients to a greater risk of chronic tendinopathy and/or rupture [26]. Pursuing a surgical option in diabetic patients with tendinopathy is important to decrease the long-term morbidity, and restore normal gait function and plantar pressures to prevent ulceration [18].

5. Conclusion

In summary, the TA tendinosis described in this study is clinically and pathologically different from the syndrome of distal TA tendinosis described by Beischer et al., whose cohort had pain at the TAT insertion site, but none of the functional deficits associated with TAT rupture [19]. This study is the largest to characterize TA tendinosis (3 of 9 without complete rupture) severe enough to cause the same functional deficits as a complete tear. The results indicate that surgical treatment was effective for reestablishing function in all nine individuals with TA tendinosis. By this rationale, surgery offers great results, with a high level of satisfaction to patients with TA tendinosis, despite possible complications in this medically morbid patient group. Direct repair may be possible in many cases, but if the tendon gap is too large, then an

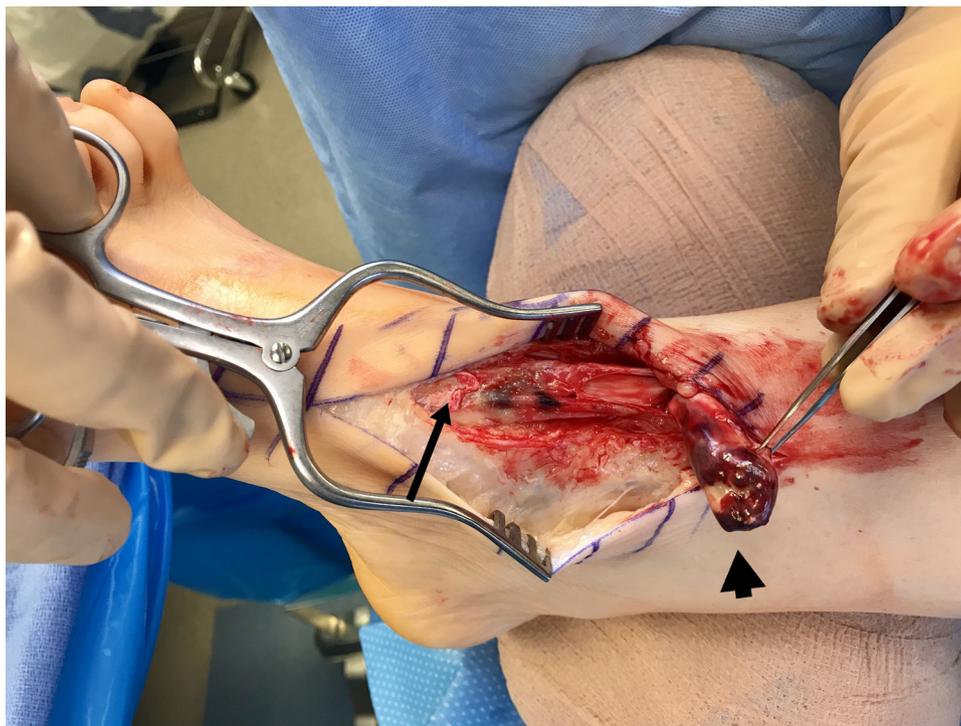


Fig. 3. Intra-operative tibialis anterior tendinosis: arrow head-proximal stump of the tibialis anterior tendon, arrow-distal stump of the tibialis anterior.



Fig. 4. Tendon graft—2nd and 3rd slip of extensor digitorum longus.

autograft of EDL tendon and plantaris tendon can be efficiently utilized.

Author declaration

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The authors confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed.

The authors confirm that due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing the authors confirm that the regulations of their institutions concerning intellectual property have been adhered to.

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