Spontaneous Tibialis Anterior Tendon Rupture: Delayed Repair With Free-Sliding Tibialis Anterior Tendon Graft

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A spontaneous rupture is even rarer.³ The rarity of the condition and the subtle physical signs⁴ make the diagnosis difficult. A high level of suspicion and meticulous clinical evaluation are required.

We report on a case of spontaneous TA rupture in a 51year-old man with diabetes. The tendon defect was 8 cm long, and reconstruction was performed with a free-sliding TA tendon ipsilateral autograft.

CASE REPORT

A man in his early 50s with diabetes presented to the outpatient clinic with painful bulging over the anteromedial aspect of the right ankle. He could not recall any associated injury. Physical examination revealed a normal range of active dorsiflexion and minimal footdrop. This movement was achieved through recruitment of the extensor hallucis longus (EHL) and the extensor digitorum longus (EDL), resulting in a maximal extension of the great toe. If recruitment of the EHL was abolished by active flexion of the hallux or by blocking the metatarsophalangeal joint dorsiflexion, then the range of active dorsiflexion was reduced. Foot pronation was maximal in the swing phase of gait and reducing toward the end of this period, as the hallux extended maximally, elevating the arch and increasing the dorsiflexion of the ankle. Internal rotation of the tibia and foot was noticed just before heel strike. The TA muscle was wasted, and its tendon continuity was lost. A painful TA tendon stump was palpable above the ankle level. Ultrasound and magnetic resonance imaging (MRI) confirmed the diagnosis of total rupture of the TA tendon (Figure 1).

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SURGICAL TECHNIQUE

A longitudinal incision was made medial to the EHL and was carried from the level of the tendon stump to the EHL insertion to the first metatarsal. A proximally retracted, degenerated, and scurry stump of the TA tendon was recognized as indicating an avulsion-type injury (Figure 2). The tendon sheath was opened carefully, and the degenerated



Figure 1. Magnetic resonance imaging of right foot shows tibialis anterior tendon stump.



Figure 2. Tibialis anterior tendon was proximally retracted, leaving a very small distal stump.



Figure 3. Proximal stump of tendon after longitudinal incision of its thick inflammatory sheath.



Figure 4. Free-sliding tibialis anterior tendon graft harvested after longitudinal incision of its thick inflammatory sheath.

tissue of the tendon substance was excised, leaving an 8-cm tendon gap (Figure 3). Then, the incision was proximally extended, and the healthy part of the remaining tendon was split proximally up to the musculotendinous junction. The half of the split tendon was then harvested and used to bridge the defect (Figure 4). Proximally, an end-to-end anastomosis was performed. The superior extensor retinaculum was initially released by a stepped incision (Figure 5). A stepped repair was then performed to ensure smooth gliding of the repaired tendon. Distally, the graft was fixed into the base of the first cuneiform with use of a bone anchor (Figure 6). During the distal fixation, the graft was kept under tension with the foot in the neutral position. Finally, no equinus contracture was revealed, and we did not perform any gastrocnemius lengthening to balance the muscles.

An ankle-foot plaster was then applied, and the patient was instructed not to load the right foot for 2 weeks. At the end of that period, the plaster was removed, and a functional ankle-foot orthosis (blocking plantar flexion) was applied for 4 weeks. After participating in a physiotherapy program, the patient regained active use of TA, and ankle dorsiflexion no longer required compensatory action of EHL and EDL (Figure 7). One year after surgery, the patient had normal gait and was symptom-free.



Figure 5. Step-cut release of superior retinaculum.



Figure 6. The graft was inserted into the base of the first cuneiform and fixed with a bone anchor.

We have obtained the patient's informed, written consent to publish his case report.

DISCUSSION

The TA tendon, the main dorsiflexor and invertor of the ankle, acts primarily during the swing phase, heel strike, and early-stance phase of the gait cycle, providing controlled plantar flexion and preventing slapping of the foot.³ The TA tendon originates from the lateral tibial condyle, the anterolateral aspect of the proximal half of the tibial diaphysis, and the proximal half of the interosseous membrane. Its musculotendinous junction is at the margin of the middle and distal thirds of the tibia. The tendon runs underneath the superior (transverse) and inferior extensor retinaculum and inserts into the medial aspect of the first (medial) cuneiform and inferomedial aspect of the base of the first metatarsal. Loss of TA function causes dorsiflexion inability and footdrop. Gait abnormality is characterized by toe-catching during the swing phase and foot slap after heel strike. High-stepping gait is advocated to avoid toe-catching.

TA tendon ruptures are traumatic or spontaneous. Few traumatic ruptures have been reported,⁵⁻⁸ and even fewer spontaneous ruptures. The spontaneous ruptures were related to local corticoid injection,⁹ uric acid sediments,¹⁰ psoriatic



arthritis,¹¹ and rupture in a 65-year-old man with diabetes.¹² Petersen and colleagues¹³ studied the vascularity of the TA tendon and hypothesized that an avascular area of 45 to 67 mm over the anterior half of the tendon may be one of the predisposing factors of the spontaneous tendon rupture. Diabetic angiopathy leading to compromised tendon blood supply seemed to be the main reason for the spontaneous TA rupture in our patient's case.

Diagnosis of TA rupture is based on careful clinical examination. All individual muscle functions have to be meticulously tested.¹⁴ The examiner has to keep in mind the compensatory mechanism of ankle dorsiflexion by the action of the EHL and EDL muscles.⁴ The TA deficiency can be easily demonstrated by isolating the compensatory action of these muscles.

The diagnosis is easier in traumatic TA rupture than in spontaneous rupture. Concomitant injury to adjacent extensor tendons and neurovascular structures must be excluded. However, lacerations over the tibia may give the false impression of security, as they are often thought to be skin lacerations. As the TA has a 6- to 7-cm excursion, a tendon injury that occurs when the ankle is dorsiflexed may have the distal tendon stump lying several centimeters distal to the skin wound and may not be apparent on initial exploration.¹⁵ Spontaneous ruptures require a high index of suspicion; when doubt exists, ultrasonography or MRI can be used to confirm the diagnosis.¹⁶ In addition, both imaging modalities are useful in differentiating partial from complete ruptures and in estimating defect size.



Figure 7. At the last follow-up, 1 year after surgery, the patient was able to make his right foot actively perform (A) flexion, (B) inversion, and (C) eversion.

The literature shows a tendency toward early repair in acute traumatic TA ruptures.^{17,18} Early exploration may enable primary tendon repair without the need for tendon grafts and transfers. For spontaneous cases, most authors have advocated early tendon repair and have reported satisfactory results.^{6,19} However, spontaneous ruptures are usually neglected, and muscle atrophy and retraction, as well as adhesions, make the surgical repair very difficult and the clinical outcome poor.¹²

In the case of our patient, an active person who had a symptomatic functional deficit, we decided on surgical management. The massive, 8-cm defect excluded the possibility of primary tendon reattachment to navicular bone. In addition, insufficiency of the distal tendon stump made Kelikian transfer²⁰ unfeasible. Reconstruction of the TA tendon with free-sliding TA tendon autograft seemed a

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promising alternative. This technique had been reported as bridging 2- to 7-cm defects with satisfactory results.²¹ The TA tendon has a large diameter and maintains adequate tensile strength for ankle dorsiflexion even when split into halves. The harvested free tendon graft can be transposed and can bridge the defect, resulting in anatomical repair. Another advantage of this technique is that no other donor tendons are involved, and there is no donor-site morbidity. Compared with side-to-side anastomosis, end-to-end proximal repair maximizes the bridging length capacity and reduces the diameter of the anastomosis.

Anatomical closure of the superior extensor retinaculum is highly associated with adhesion formation and restricted tendon gliding. Leaving the superior retinaculum unrepaired (to facilitate TA gliding) may result in tendon bowstringing, biomechanical alterations, and strength loss. The stepped repair of the superior retinaculum (as described by Wong²¹), leading to retinaculum lengthening, minimized constriction and restriction of the tendon excursion over its repaired ends. Early mobilization with blocked plantarflexion provided satisfactory protection, enhanced remodeling of the repaired tendon, and minimized adhesion formation.

CONCLUSIONS

Spontaneous TA rupture is very rare. In this report, we have described the repair of a very large tendon defect. Tendon repair with a free-sliding TA tendon autograft and superior extensor retinaculum stepped release seems to be an effective technique in cases of large and neglected defects.

AUTHORS' DISCLOSURE STATEMENT

The authors report no actual or potential conflict of interest in relation to this article.

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