Spontaneous Rupture of the Tibialis Posterior Tendon

A Case Report and Review of the Literature

SAUL LIPSMAN, DPM*
JARED P. FRANKEL, DPM†
GARY W. COUNT, DPM‡

The authors investigate the literature on spontaneous rupture of the tibalis posterior tendon, noting the difficulty of satisfactory treatment. The case history they present agrees with previous experience. They discuss anatomic and biomechanic considerations, and plaster casting and subsequent neutral rigid orthoses, successfully used to provide relief for the patient.

Spontaneous rupture of the tibialis posterior tendon is a rare clinical finding. A review of the literature suggests that this condition is also difficult to treat satisfactorily. Kettlekamp and Alexander[1] reviewed four cases of spontaneous rupture of the tibialis posterior tendon. In these cases, three out of four patients were over the age of 40 and were initially diagnosed as having a tenosynovitis of the tibialis posterior tendon. None of these had a history of collagen disease or trauma. These patients were treated with conservative therapy and all had eventual exacerbations in the form of tendon rupture. The diagnosis of rupture was established by specific incidents relating to the patients' feeling the tendon rupture, loss of control and foot instability while weightbearing, unilateral discomfort in the region of the tibialis posterior tendon, fallen arches and painful, flexible flatfeet. The physical findings noted tenderness in the region of the tibialis posterior tendon, absent tibialis posterior function, swelling within the tendon sheath and ecchymosis.

The initial therapies employed for treatment of the tenosynovitis included plaster immobilization, arch supports, injection therapy and excision of tendon sheath. Treatment of the tendon rupture varied from the continued use of shoe corrections, surgical intervention in the form of tendon grafting of the deficit in the tibialis tendon and "Z" plasty lengthening of the posterior tibial followed by casting. Postoperatively, the patients did have diminished discomfort and increased activity when compared to their preoperative status. Whatever therapy was employed, the patients invariably had some residual discomfort and loss of power and efficiency, manifested as pes planovalgus on the affected side.

These cases illustrate that the combination of late repair and loss of tendon make a good functional result difficult to obtain.

Griffiths,[2] in a series of 20 tendon injuries around the ankle, reported four cases involving the tibialis posterior. Of these four, three were lacerations (two of which healed unsatisfactorily) and one was a spontaneous rupture with no repair in a patient with rheumatoid arthritis. The result here is listed as satisfactory, with the foot appearing and functioning normally, but with the tendon no longer functioning. In one case involving a tibialis posterior laceration, repair was not carried out until 3 years post-injury, which was when the patient first came to the physician. Surgical repair was recorded as unsuccessful.

* Diplomate of the American Board of Podiatric Surgery; Fellow, American College of Foot Surgeons. *Mailing address:* World Executive Building, 3500 North State Rd 7, Fort Lauderdale, FL 33313.

† Director of Residency Program at Las Olas General Hospital, Fort Lauderdale; Fellow, American College of Foot Surgeons.

‡ Resident, Las Olas General Hospital, Fort Lauderdale.

Goldner et al.[3] reviewed the case histories and treatment of nine patients with pathologic changes in the tibialis posterior tendon and medial plantar calcaneonavicular ligament.

Three patients, aged 11, 15 and 18, sustained traumatic lacerations of the tibialis posterior tendon. The remaining six patients were middle aged women and one 68-year-old male. These six were described as having chronic tenosynovitis and progressive stretching of the plantar calcaneonavicular ligament. The patients' complaints were similar to those reported by previous investigators.

The patients with lacerations were seen 7 years, 3 years and 5 months post-injury. In the first patient, age 11, seen 7 years post-injury, a transfer of the flexor hallucis longus tendon to the distal stump of the posterior tibial tendon, as well as a tightening of the plantar calcaneonavicular ligament, was performed.

This tightening was accomplished through the creation of a distally based flap which was advanced proximally; thus adducting the forefoot at the level of the talonavicular joint. The results are reported as a return to normal structure and function.

Another patient was treated 5 months post-laceration with transfer of the flexor digitorum longus tendon and medial reinforcement. The results are reported to be satisfactory in both structure and function.

The treatment of chronic tenosynovitis varied with the surgeon involved. Two patients who underwent the flexor hallucis tendon transfer as well as the medial ligamentous reinforcement had continued pain and therefore had the additional transfer of the flexor digitorum longus tendon for added support. The final results were satisfactory.

One patient with chronic degeneration was treated with tenosynovectomy, excision of an accessory navicular and re-attachment of the tibialis posterior tendon to the remaining navicular bone. The results were judged satisfactory, with the patient having some soreness at the region of the base of the fifth metatarsal as a result of the redistribution of body weight. In another patient treated with tenosynovectomy, resection and shortening of the tibialis posterior tendon without reinforcement of the medial ligaments, residual valgus of the heel and medial arch pain persisted.

The single male patient with chronic tenosynovitis was treated with tenosynovectomy and advancement of the tibialis posterior tendon to the tibialis anterior tendon. Upon 9-year follow-up, the patient had diminished pain but poor arch morphology which bothered him.

Case History

The single case of tibialis posterior tendon rupture which we have thus far encountered correlates with previous experience. The patient was a 66-year-old white male who complained of severe pain in the right foot for 2 days' duration (see Figs. 1 to 3). The patient reported he was playing golf, and following a forceful drive he experienced severe pain followed by swelling on the medial aspect of his right foot. The patient gave a past history of intermittent pain, of 2 weeks' duration, in the same location. The patient denied any past history relating to any collagen disorder.

Upon clinical examination, it was noted that the patient had pain, edema and erythema in the region of the insertion of the tibialis posterior tendon into the tuberosity of the navicular. Muscle testing revealed paresis of the posterior tibial muscle-tendon unit. The weight-bearing foot morphology showed

Figure 1. Comparison of non-weightbearing foot morphology, 2 days post-tendon rupture; note patient's left arch morphology.

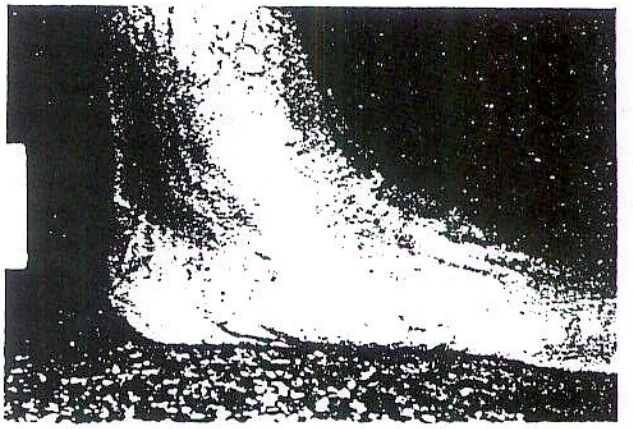

Figure 2. Weight-bearing view of affected left foot. Note fluid in posterior tibial tendon sheath posterior to medial malleolus, as well as edema in region of tuberosity of navicular. Compare morphology with Figure 3.

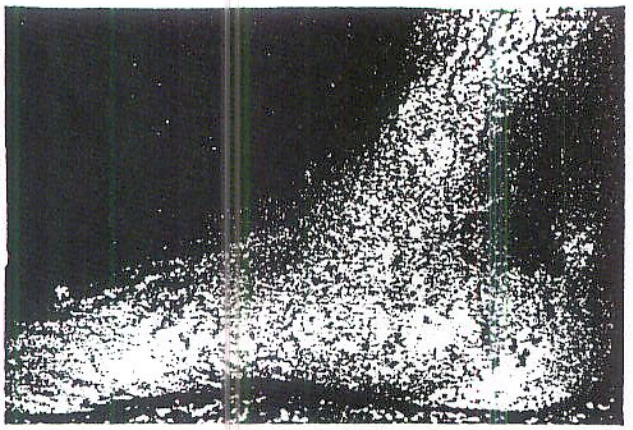

Figure 3. Weight-bearing view of non-affected right foot. Notice pes planus of lesser degree than left foot.

pes planovalgus deformity on the injured side. X-ray evaluation showed no bony pathology with the exception of increased talocalcaneal angle (dorsoplantar of the foot), right greater than left.

The diagnosis at this time was tenosynovitis followed by spontaneous rupture of the tibialis posterior tendon. Initial therapy consisted of posterior splint application followed 2 days later by below the knee casting with the foot inverted and adducted. At 3 weeks, the cast was changed and physical examination at that time revealed a palpable defect in the posterior tibial tendon of approximately ½ to 1 cm. in length.

A below the knee cast was reapplied for 3 weeks, at which time, upon cast removal, the defect was gone and there was a palpable thickening of the posterior tibial tendon or tendon sheath. At this time, the patient was treated with ultrasound, electrogalvanic stimulation, and strapping. The patient was casted for neutral orthoses which were dispensed at the subsequent visit. Once weightbearing, the patient was asymptomatic, but his foot morphology remained pes planovalgus. Further follow-up evaluation is needed to judge the long-term results of therapy.

Initial surgical intervention was not carried out for a number of reasons. First, the diagnosis was made primarily by history; the edema initially present prevented palpation of a deficit in the tendon. Once this defect was found, i.e., 3 weeks post-injury, it was deemed proper to attempt conservative management.

This decision was based upon a number of factors. First, the patient's life style was basically sedentary. A complete return to nonpathologic structure and function was not mandatory, as long as the patient's symptoms of pain and weakness were relieved. Second, unless the procedure described by Goldner et al.[3] was performed, the prognosis for significant improvement by surgical means was poor. If conservative management was ineffective, then the time devoted to this therapy would not significantly alter the prognosis if a tendon transfer and medial ligamentous reconstruction was performed.

Anatomic Considerations

A thorough knowledge of the anatomic relationships of the tibialis posterior tendon and muscle is required in order to discuss the pathologic changes which occur during its loss. The tibialis posterior originates from the posterior surface of the interosseous membrane between the tibia and fibula, from the posterolateral surface of the tibia, and from a section on the posterior, proximal fibula. As it proceeds down the leg (as one of the four deep posterior muscles of the lower leg), it passes deep (anterior) to the flexor digitorum longus and enters a groove on the posterior surface of the medial malleolus. (The tendon is within its own tendon sheath from 4 cm. proximal to the malleolus, distally to the region just proximal to its insertion on the tuberosity of the navicular.) From the posterior surface of the medial malleolus, it passes deep to the laciniate ligament and superficial to the deltoid ligament and then inferior to the plantar calcaneonavicular ligament.

Here, the tendon bifurcates into two slips; the larger, superficial section is attached to the tuberosity of the navicular, from which fibers continue to the plantar surface of the medial cuneiform. The deeper more lateral division of the tendon, which arises inferior to the plantar calcaneonavicular ligament, forms part of the origin of the medial head of the flexor hallucis brevis and then extends to its insertion on the second cuneiform and the bases of the second, third and fourth metatarsals.[4]

It should be noted that if the tendon rupture occurs proximal to the level of the plantar calcaneonavicular ligament, the action of the tendon on all its insertions will be eliminated. This is important when considering the future development of a myriad of forefoot deformities.

Biomechanical Considerations

The role of the tibialis posterior muscle as an invertor and plantar flexor of the foot is well documented.[5,6,7] The muscle is an antagonist of the peroneous brevis and longus.[6] This fact is readily seen in the child suffering from poliomyelitis who has paralysis or paresis of the tibialis posterior and develops severe pes planovalgus. This condition is

corrected with transfer of the antagonistic peroneus longus to the insertion of the tibialis posterior.[8]

The tibialis posterior also acts to stabilize the midtarsal and subtalar joint in a supinatory direction (primarily the midtarsal joint). The force which it produces is primarily adductory[9] with a somewhat lesser posterior axial force and slight inversion force of the forefoot on the rearfoot.[5]

Its fulcrum of activity is the posterior groove in the tibial malleolus. The fulcrum is important when considering the action of the muscle as well as the pathologic clinical findings, such as the bowstringing of the tendon. This is readily seen in any pronated hypermobile foot. It certainly seems plausible that this bowstringing and the concomitant irritation can be contributory to the development of tibialis posterior tenosynovitis.

The prognosis in terms of the development of further pathologic foot changes is undocumented in the literature. With the loss of the tibialis posterior activity, the subsequent excessive pronation of the subtalar joint in gait will have far reaching manifestations.[10]

With subtalar joint pronation, the axes of motion of the talonavicular joint and the calcaneocuboid joint become more parallel and thus allow unlocking of the midtarsal joint. This unlocking results in a positional forefoot valgus.[5] In gait, the first ray prematurely receives the stress of body weight while in an abnormal position and prior to the stabilizing contraction of the peroneus longus. (A stable calcaneocuboid articulation and lateral column must be present for the cuboid to be an effective fulcrum of activity for the peroneus longus and its approach to the first ray.[5]) The result of function in this position is inversion and dorsiflexion of the first metatarsal with subsequent eversion and abduction of the hallux accompanied by hypermobility of the first ray. The development of hallux abducto valgus, plantar keratosis and hammer toe deformities is also inevitable.

The result of the loss of pull of the tibialis posterior tendon on its insertion which forms part of the origin of the medial head of the flexor hallucis brevis may have unknown implications in the development of hallux abducto valgus.

The continuation of these subluxatory changes will eventually result in talonavicular joint arthritis,[10] with the typical x-ray findings which are consistent with degenerative joint disease. In addition, the subtalar joint pronation will produce a functional limb length discrepancy and predispose the patient to the development of knee derangements secondary to internal rotation of the leg and subsequent hip and low back dysfunction.

Any biomechanical condition which produces subtalar joint pronation and subsequent unlocking of the midtarsal joint will result in increased stress on, and increased activity of, the posterior tibial and long flexors of the toes. When they are under this tension, they develop tenderness along their course.[6] Such biomechanical conditions include compensated gastrocnemius equinus and compensated forefoot varus.

Pathology

Along with this tenderness, "one finds thickening and occasionally some palpable fluid in the sheath of the tibialis posterior tendon."[11] This swelling, pain and tenderness (manifestations of tenosynovitis) can be present distal or well proximal to the medial malleolus. This tenosynovitis can be a precursor to tendon rupture. Such symptoms of the nonspecific tenosynovitis seem to worsen with a day's activities and be least symptomatic upon awakening in the morning. The pain is often neuritic in nature and has been mistaken for a neuralgia.

The histologic picture produced is thickening and fibrosis of the tendon sheath. The tendon itself may show thickening or thinning, and may be covered with granulation tissue. In some cases, there is proliferation of the synovial lining, while in others it is worn away.

The vascularity of the tendon also varies at this point. The blood supply to a normal tendon is minimal to begin with, in comparison to other tissues. The vascular supply comes from sparse arterioles within the interfascicular intervals and from external, efferent vessels. With tenosynovitis, the vascularity varies. According to Steindler,[6] we know that early degenerative changes occur within tendons. The central artery disappears as early as the third decade, so the tendon becomes entirely dependent upon vascular supply from the surrounding (edematous) area. Therefore, with such a limited vascular supply, spontaneous tendon rupture is nothing unusual. Unless the trauma is obviously the primary causative factor, the tendon is likely to rupture due to the degenerative changes.

Clinical Signs and Symptoms of Rupture

The rupture of the tendon itself is usually accompanied by pain and swelling. The patient and examiner will note unilateral or greater severity of pes planovalgus on the affected side as well as some instability and apropulsive changes in gait. Any

history of nonspecific pain followed by a dramatic increase in severity of pain in this region is a clue to the diagnosis of rupture of this tendon.

Treatment of Tendon Rupture

Indicated treatment of tibialis posterior tendon rupture varies somewhat with the duration of time passed since the actual rupture. Late repair performed following rupture has been shown to be unsuccessful. (Late repair meaning repair performed after the tendon has healed in a pathologic position.) If one considers tendon healing complete in 4 to 6 weeks, plus additional time after the third decade of life, then one can extrapolate that repair attempted after 4 to 6 weeks post-injury would have a lesser chance of producing a good functional result. After such a period of time, the muscular and tendinous atrophy, plus scarring and fibrosis of the tendon, would result in diminished functional return, even with the most ideal surgical repair of the tendon itself.

Actual repair of the patient's tendon would seem to be indicated after early recognition of rupture (within a few days), and in a situation where a good functional result is imperative. Late repair is generally unsuccessful as evidenced by the literature. However, late surgical repair utilizing the technique described by Goldner et al.[3] seems to be a viable alternative treatment in the patient seen early or late after initial injury. With the knowledge of their technique, one can attempt conservative management, especially in the geriatric patient, with full realization that a surgical technique is available which has shown great promise in one series.

An approach should be formulated for the treatment of the patient seen late after initial insult. Conservative therapy available consists of cast immobilization, physical therapy modalities to relieve the patient of any acute pain, followed by rigid or semi-rigid foot orthoses. With this type of therapy, symptomatic relief is possible, but functional activity of the tibialis posterior is not to be expected.

If the patient is seen soon enough after the injury, or has continued or intractable pain, then surgical intervention may be warranted, as some functional activity may be returned as well as a further relief of pain and other subjective symptoms. A complete return to "normal" structure and function cannot be expected with repair of the injured tendon alone.

The surgical repair which Kettlekamp and Alexander[1] report as giving the best functional result is end to end suture of the tendon fragments following "Z" plasty lengthening of the proximal tendon tag. In addition, tendon transfers may also prove valuable. The peroneus longus, flexor digitorum longus or flexor hallucis longus appear to be suitable, although much consideration must be given to the results of a further alteration of structure in a previously nonpathologic foot.

Synovectomy is another procedure used to obtain relief of symptoms secondary to hypertrophic synovium and granulations.

An additional procedure which may be of some assistance in this situation is a Young tenosuspension. By rerouting the tendon of the tibialis anterior through a drill hole in the navicular, the dorsiflexory force is removed from the medial column of the foot. Thus the tibialis anterior supplements other procedures used to treat the weakened medial arch of the foot.[12]

Goldner et al.[3] have described a series of surgical procedures which they feel are indicated in a variety of situations which differ in the duration of time since the initial insult and the etiology of the injury. In the case of traumatic laceration of the posterior tibial tendon, reinforcement of the injured tendon with the flexor hallucis longus or the flexor digitorum longus is reported to return to satisfactory function.[3]

If the laceration is long standing (present for several years) then the recommended therapy is tendon reinforcement with the flexor hallucis or digitorum longus and medial ligamentous reconstruction. The middle aged patient with chronic tenosynovitis of the posterior tibial tendon and subsequent progressive equinovalgus is treated in the same basic manner as those with an old laceration. The six patients in the last category of chronic tenosynovitis were treated with a variety of procedures, all short of the recommended plication of medial ligaments; the results were uniformly less than perfect. This is the expected result after one has reviewed the literature.[1, 2]

Discussion

In our case which is similar in etiology to those described by Goldner et al. (the difference being that there were no spontaneous ruptures in their series) it would now seem in retrospect that an open repair with plication of the medial ligamentous structures would result in satisfactory function. However, we feel that when dealing with geriatric patients, where complete functional return isn't necessarily mandatory, conservative therapy should be given a trial. This is especially true when a successful procedure has been described which can be used late after initial insult, without any

direct evidence to indicate detrimental effects from the delay in surgical repair.

Summary

Diagnosis is aided by clues in the history as well as characteristic physical findings. While chronic tibialis posterior tenosynovitis and subsequent symptomatic pes planovalgus is not an uncommon finding, the progression to frank tendon rupture is rare.

A review of the literature shows generally poor results obtained with surgical intervention in the form of late repair. The exception to this is the procedure described by Goldner et al.[3] If initial conservative therapy is unsuccessful in the relief of symptoms, then a return of function as well as a relief of symptoms may be achieved with the method of ligamentous plication and tendon reinforcement.

A case is documented where plaster casting and subsequent neutral rigid orthoses were used successfully to provide subjective relief in the patient's condition. Further follow-up of this case and others is needed to provide a more definitive statement regarding this treatment modality.

References

1. Kettlekamp, D. B. and Alexander, H. H.: Spontaneous rupture of the posterior tibial tendon. J. Bone Joint Surg., 51A: 759, 1969.
2. Griffiths, J. C.: Tendon injuries around the ankle. JBJS 47B: 686, 1965.
3. Goldner, I. L., Keats, P. K., Bassett, F. H., et al.: Progressive talipes equinovalgus due to trauma or degeneration of the posterior tibial tendon and medial plantar ligaments. Orthop. Clin. North Am., 5: 39, 1974.
4. Lewis, O. J.: The tibialis posterior tendon in the primate foot. J. Anat., 98: 209, 1964.
5. Sgarlato, T. E.: Compendium of podiatric biomechanics. California College of Podiatric Medicine, San Francisco, March 1971.
6. Steindler, A.: *Kinesiology*, p. 407, Charles C Thomas, Springfield, IL, 1935.
7. Warwick, R. and Williams, P. C. (Editors): Gray's Anatomy, 35th Br. Ed., p. 577, W. B. Saunders Co., Philadelphia,
8. Fried, A. and Hendel, C.: Paralytic valgus deformity of the ankle. Replacement of the paralyzed tibialis posterior by the peroneus longus. J. Bone Joint Surg., 39-A: 921, 1957.
9. Duchenne, G. B.: *Physiology of Motion*, (Translated by G. B. Kaplan), J. B. Lippincott, Philadelphia, 1949.
10. Schneider, M. and Balon, K.: Deformity of the foot following anterior transfer of the posterior tibial tendon and lengthening of the Achilles tendon for spastic equinovarus. Clin. Orthop., 125: 113, 1977.
11. Lipscomb, P. R.: Nonsuppurative tenosynovitis and paratendinitis. In *Instructional Course Lectures, Americal Academy of Orthopedic Surgeons*, Vol, 7, p. 254-261, J. W. Edwards, Ann Arbor, 1950.
12. Beck, E. and McGlamry, E. D.: Modified young tenosuspension technique for flexible flatfoot. In *Reconstructive Surgery of the Foot and Leg*, Edited by E. Dalton McGlamry, p. 293-326, Intercontinental Medical Book Corp., 1974.

Additional References

Anzel, S. H., Covey, K. W., Weiner, A. D., et al.: Disruption of Muscles and Tendons: An analysis of 1,014 cases. Surgery, 45: 406, 1959.

Basmajian, J. V. and Stecho, G.: The role of muscles in arch support of the foot. An electromyographic study. J. Bone Joint Surg., 45-A: 1184, 1963.

Bunnell, S.: *Surgery of the Hand*, p. 437-439, J. B. Lippincott Co., Philadelphia, 1956.

Conwell, H. E. and Alldredge, R. H.: Ruptures and tears of muscles and tendons. Am. J. Surg., 35: 22, 1937.

DuVries, H. L.: *Surgery of the Foot*, C. V. Mosby Co., St. Louis, 1965.

Chormley, R. K. and Spear, I. M.: Anomalies of the posterior tibial tendon. Arch. Surg., 66: 512, 1953.

Key, J. A.: Partial rupture of the tendon of the posterior tibial muscle. J. Bone Joint Surg., 35-A: 1006, 1953.

Milgram, J. E.: Muscle ruptures and avulsions with particular reference to the lower extremity. In *Instructional Course Lectures, American Academy of Orthopedic Surgeons*, Vol. 10, p. 233-243, J. W. Edwards, Ann Arbor, 1953.

Peacock, E. E., Jr. and Van Winkle, W., Jr.: *Surgery and Biology of Wound Repair*, W. B. Saunders Co., Philadelphia, 1970.

Schneider, M. and Balon, K.: Deformity of the foot following anterior transfer of the posterior tibial tendon and lengthening of the Achilles tendon for spastic equinovarus. Clin. Orthop., 125: 113, 1977.

Tachdjian, M. O.: *Pediatric Orthopedics*, Vol. 2, W. B. Saunders Co., Philadelphia, 1972.

Williams, R.: Chronic non-specific tenosynovitis of tibialis posterior. J. Bone Joint Surg., 45-B: 542, 1963.

Young, C. S.: Operative treatment of pes planus. Surg. Gynecol. Obstet., 68: 1099, 1939.