CHAPTER 1

TIBIALIS POSTERIOR DYSFUNCTION
An Overview

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Over the past decade, there has been increasing attention paid to the entity of tibialis posterior dysfunction (TPD). This entity can create significant and severe disability for patients by the creation of a collapsed foot. Unlike other tendon ruptures, there is usually a long prodrome of tendinitis before the tendon actually ruptures. During this time, there are many mechanical and anti-inflammatory strategies that may serve to preserve the function of tibialis posterior. However, once the dysfunction is identified, it is important to proceed aggressively with treatment of the condition in order to prevent total collapse of the foot.

LITERATURE REVIEW

E. Dalton McGlamry, DPM and Terrance Mueller, DPM[1,2] both deserve credit for increasing the awareness of podiatric physicians to the condition of tibialis posterior dysfunction. When the author was in residency in Tucker, Georgia (between 1980 and 1983) Dr. McGlamry was lecturing extensively on this topic at the national level. He emphasized the need to have a high index of suspicion for the condition, particularly in the older patient, or in one with a unilateral flatfoot. Dr. McGlamry also discussed the use of triple arthrodesis as a stabilizing procedure in the severely collapsed tibialis posterior dysfunction foot.

In 1982, Mueller submitted an article to the *Journal of the American Podiatric Medical Association* (published in 1984) that reviewed 25 cases from the literature and his personal experience regarding this entity.[1] He recommended soft tissue procedures for initial surgery ranging from primary repair to tendon grafts and/or tendon transfers. In 1991, Mueller proposed both a classification system and a staging system. The classification system included direct injury, pathologic rupture, idiopathic rupture and functional rupture. The concept of functional rupture is important, because the majority of TPD patients have an intact tendon that is hypertrophic from scarring. The tendon, although intact, has no functional strength, presumably from healing in a lengthened position. Although the tendon is palpable, the consequence is the same as if there was a rupture with a gap in the tendon.

In a 1987 article, Banks and McGlamry discussed the etiology of TPD, and noted that in TPD, bilateral collapsing pes plano valgus is the rule.[3] This observation correlates well with the concept of TPD as the end-stage of collapsing pes plano valgus. The other significant contribution of this article was to advocate surgical repair for the majority of patients with this condition. Prior to this, many patients had undergone prolonged trials of unsuccessful conservative therapy. The authors recommended triple arthrodesis as a treatment to improve stability and function. They noted that soft tissue procedures would not effect the underlying initial collapsing pes plano valgus, and that triple arthrodesis can correct in multiple planes. They also contributed a recommendation that when indicated, tendo Achillis lengthening should accompany surgical stabilization of the TPD foot.

PATHOLOGY

Kannus and Jozsa conducted a controlled study on the tendons from 891 patients.[4] They reported that healthy tendon structure was not demonstrated in any spontaneously-ruptured tendon. They concluded that degenerative changes were common in the tendons of people over the age of 35, and that these changes were associated with spontaneous rupture. Therefore, it was likely that a ruptured tendon had some pre-existing deterioration. In the case of TPD, this is probably the chronic tenosynovitis from mechanical strain caused by chronic collapsing pronation of the foot.

If the statement of Kannus and Jozsa is correct, then the question must be raised of how are degenerative changes produced in tibialis posterior? Other authors, including Banks and McGlamry[3] have previously described the mechanical demands on the

tendon and the underlying collapsing pes plano valgus pathology that contributes to tendon deterioration. The author is conducting a retrospective study at the Pennsylvania College of Podiatric Medicine. In this study, a previously unreported high incidence of os tibiale externum in TPD was observed. The incidence of os tibiale externum is normally reported at 10% to 15%, in the author's study the incidence of os tibiale externum associated with TPD was reported to be approximately 60%. The mechanical disadvantage caused by insertion of tendon fibers into the ossicle may be a major contributing factor to TPD in many patients.

Another contributing factor may be hypovascularity of a segment of the tendon. In a 1990 article, Frey et al. described the presence of abundant vascularity at the osseous insertion and musculotendinous junctions of tibialis posterior.[5] They also described a zone of hypovascularity, located posterior and distal to the medial malleolus. This avascularity, combined with mechanical tendon degeneration, may be the predisposing factor in TPD and rupture.

MAKING THE DIAGNOSIS

The greatest difficulty with tibialis posterior dysfunction is the delay that frequently occurs prior to making an accurate diagnosis. This delay in diagnosis is probably based upon two factors: the condition results from a chronic mechanical overload on the foot and ankle, and the course of the process is quite gradual; and unless the clinician has a high index of suspicion, it is very easy to miss the diagnosis.

The delay in diagnosis may be substantial, because of the variety of symptoms that can be reported with this condition. Some patients will present with plantar fascitis and heel pain as a result of the collapse of the foot. Other patients may report symptoms consistent with tarsal tunnel syndrome. In addition, the development of flexor substitution hammertoes can cause the focus to be directed toward the distal forefoot rather than the main area of pathology. The result of this delay in diagnosis is that there is usually a progression in the collapse of the foot. The medial column will collapse in the sagittal plane, and there will be a transverse plane abduction occurring at the midtarsal joint. The development of degenerative joint disease in these areas can further complicate the deformity.

If the clinician has a high index of suspicion, diagnosis by means of manual muscle testing is possible. The difficulty with this procedure, however, is that the patient may effectively substitute with tibialis anterior, and thus possess a strong inversion strength, even when placed in some mild degree of plantarflexion. The author's technique consists of placing the patient in a fully plantarflexed and adducted position, and then having the patient resist an eversion force.

Other clinical signs include an asymmetric flatfoot, particularly with a greatly-increased abduction of the forefoot on the rearfoot. This abduction creates the "too many toes sign," whereby the examiner will see more of the lateral digits when observing the patient walking from behind because of the amount of forefoot abduction. There may also be difficulty with raising up on the affected limb without support. This test can also be used to help determine the flexibility of the deformity. If the heel inverts when the patient stands on his toes, this indicates that there is residual flexibility of the deformity.

The history on these patients is classic. They generally are aware that they have had a decrease in the height of the arch, and an increase in pain in the foot, generally over a period of months or years. They may also be aware of a change in shoe wear on the affected side.

Signs of acute inflammation or injury may often be absent. There will be swelling and increased skin temperature only if there is a continuing tendinitis. Frequently, these symptoms are absent because the tendon has already completely ruptured. The radiographic findings are variable. If the disorder has progressed long enough, there may be sagittal plane collapse of the medial column, and there may be transverse plane abduction at the metatarsal joint with an increase in the cuboid abduction angle and an increase in the talocalcaneal angle.

Additional imaging can include CT scans or MRI. However, the majority of the literature states that MRI is more sensitive and specific. The MRI is helpful in confirming whether or not a rupture is present, and also in identifying the location and extent of the rupture. This assists in pre-operative planning.

The most common MRI findings are an increase in diameter of the tendon, particularly near the insertion at the navicular. An effusion

within the tendon sheath may also be present and will be visible as a clear space surrounding the tendon.[6] The majority of tibialis posterior tendon injuries are classified as Type I, meaning that tendon response is hypertrophic. The tendon itself is intact, and no gap will be evident either clinically to palpation, or on the MRI. The imaging of the hypertrophic area of the tendon is likely to demonstrate significant heterogeneity in the color of the tendon. The tendon will be imaged with areas of gray or white rather than demonstrating a uniform intense dark coloration. These markers demonstrate areas of intramural tendon degeneration. Because these lesions may not be visible to the naked eye, the MRI is much more helpful than surgical inspection in order to demonstrate the true quality of the tendon. Conti et al. proposed an MRI classification scheme for TPD.[7] (Table 1)

Table 1

MRI CLASSIFICATION FOR TIBIALIS POSTERIOR DYSFUNCTION

Type I A	One or two fine longitudinal splits without intrasubstance degeneration. This probably correlates with a short period of symptomatology.
Type I B	This type shows more longitudinal splits and a mild increase in tendon diameter.
Type II	This morphologic type demonstrates narrowing of the tendon, with significant longitudinal tears and intramural degeneration.
Type II A	Diffuse swelling of the tendon and uniform degeneration. This correlates with a longer duration of symptoms and greater physical findings.
Type II B	Complete rupture of the tendon and replacement with scar tissue.

In their study reviewing surgical versus MRI grading of tibialis posterior dysfunction, Conti et al. showed that MRI grading was predictive of outcome for a particular soft tissue repair technique. Surgical grading did not correlate with outcome.[7] This is a key point. If the patient is to have a surgical repair that relies on the mechanical integrity of the TP tendon, then a pre-operative MRI may be very important.

In addition, MRI can serve to greatly assist in the identification of other pathology in the rearfoot that is accompanying the rupture. Schweitzer et al. demonstrated that talo-navicular pathology is a sensitive and specific sign of TPD.[8]

TREATMENT

The treatment for tibialis posterior dysfunction depends significantly on the patient's age and weight, the period of time from injury to diagnosis, other systemic factors, and most importantly, the extent of the collapse of the foot. In addition to the foot pathology, it is critical to evaluate the status of the tendo Achillis. Many of these patients have a significant equinus deformity on the affected side. It may be that the equinus begins as a primary deformity adding to the mechanical stress on the foot. Once the foot collapses, however, there is an additional secondary shortening to the tendo Achillis which makes it extremely difficult to effectively realign the foot without adding greater tension to the tendo Achillis itself. For this reason, a heel cord lengthening of some type is a major component of tibialis posterior repair in the majority of cases.

The typical tibialis posterior disfunction patient is somewhat older (55+), tends to be overweight, and does not have a very high functional demand.[9] There is also a subset of younger patients who have seronegative inflammatory disease.[10] These patients should be counselled regarding effective weight control as part of their therapy. In general, patients with advanced collapse of the foot from posterior tibial dysfunction benefit most from an osteotomy or fusion type of procedure. The author uses a combination of procedures, including a tendo Achillis lengthening as needed, Evans calcaneal osteotomy and/or talonavicular fusion with autogenous calcaneal bone graft, and a Cotton osteotomy of the first cuneiform, when necessary. When properly performed, this combination of procedures reduces tension on the midfoot by reducing the powerful heel cord influence, stabilizing the medial column and increasing the height of the arch. It also serves to reduce the amount of forefoot abduction.

There are many treatment approaches for TPD. Stabilization of the subtalar joint by means of

fusion or a STA-peg device, serves to stabilize the midtarsal joint and prevents any further medial column collapse at the midtarsal joint. Soft tissue treatment approaches can vary. Primary repair of the tendon can be performed when the rupture is treated in the acute phase. More commonly, treatment is delayed. Soft tissue procedures are useful for those patients in whom the deformity has remained flexible without secondary degenerative joint changes. Some soft tissue approaches include:

1. Flexor transfer. The transfer of the flexor digitorum longus, or flexor hallucis longus to augment tibialis posterior. This can be done by re-routing directly to the navicular or suture alongside tibialis posterior tendon, when it is viable.
2. Young suspension. The transfer of intact tibialis anterior through the navicular. This reinforces the plantar navicular-cuneiform joint with a new ligament. It also allows peroneus longus to function unopposed on the first ray.
3. Cobb procedure. This procedure uses a portion of tibialis anterior as a graft to the healthy portion of tibialis posterior.
4. Other procedures such as plication of the spring ligament can also be performed.

In addition to the soft tissue procedures, a number of osseous procedures can be performed prior to considering a triple arthrodesis. These include:

1. Navicular-cuneiform fusion. This is useful as a procedure to stabilize a navicular-cuneiform fault or treat degenerative joint disease.
2. Cotton osteotomy. This uses an opening wedge graft of the medial cuneiform to plantarflex the medial column.
3. Evans osteotomy. This procedure uses an opening wedge graft of the calcaneus anteriorly to relocate the midtarsal joint, stabilize the lateral column, decrease heel valgus, decrease forefoot abduction, and increase calcaneal pitch. In older patients, fusion of the CCJ using a bone graft has met with success.
4. Talo-navicular fusion. This procedure is used alone or in combination with a CCJ fusion, Evans osteotomy, or other procedures.
5. Other combinations. The Evans procedure can be performed with medial column soft tissue procedures etc.

Triple arthrodesis is reserved for those patients in whom there is very severe rearfoot collapse, usually with degenerative joint disease or chronic joint pain. The above combinations of procedures have reduced the need for triple arthrodesis.

It is important to rule out any radiculopathy which might be causing weakness to tibialis posterior. If the diagnosis of tibialis posterior rupture is made based upon clinical examination alone (and not MRI), then it is even more important to rule out any radiculopathy causing weakness to the tibialis posterior muscle.

SUMMARY

Many patients suffer from undiagnosed tibialis posterior dysfunction. These patients know that they tire more easily when walking or standing, and have significant foot pain. They may often present to the practitioner with complaints of other symptoms including those in the forefoot and from compression of the posterior tibial nerve. The physician must have a high index of suspicion so that proper testing and evaluation can be performed. For advanced cases, predictable treatment seems to best be afforded by a fusion type procedure.

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