Double Calcaneal Osteotomy in the Treatment of Posterior Tibial Tendon Dysfunction

This article presents an alternative treatment for tibialis posterior tendon rupture in a select group of patients with recent rupture and planovalgus foot structure. Double calcaneal osteotomy is used to realign weightbearing forces in situations where soft-tissue repair alone is not sufficient, and arthrodesis is premature. A single case is presented followed by a discussion, with encouraging results.

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Posterior tibial (PT) tendon rupture, or PT tendon dysfunction (PTD), has been reported extensively in the medical literature (1–6). The patient is typically a female >40 years of age who presents with a unilateral flatfoot (1, 2, 4, 6, 7). Occasionally, it is mentioned that the patient most often has bilateral flat feet and that the affected foot can be observed to be more severe (1, 7). The most common cause is believed to be related to tendon stress caused by an abnormal foot structure (1).

It is generally agreed that conservative treatment for this condition is often ineffective (1–3, 5). Most authors believe, then, that surgical treatment is preferred when dealing with PTD (1–5). Surgical procedures can generally be grouped into two categories. The first involves primary repair of the PT tendon and includes procedures such as reattachment of the tendon, bridging with the flexor digitorum longus tendon, and simple synovectomy of the tendon sheath (2–6). The patient receiving this treatment is usually in the acute or subacute stages of tendon rupture, with a short duration of symptoms (1, 2, 7). The second category involves joint fusion procedures, including subtalar fusion, subtalar/talonavicular fusion, or triple arthrodesis (1–3, 7). These patients present with long-standing deformity, often of several years. By this time, the tarsal joints have succumbed to abnormal loading forces and have developed significant degenerative joint disease (1–3). The only alternative is to fuse the arthritic joints, thus losing function (7).

It seems that this generalization of procedures has all but eliminated another category of patient: one that presents in the acute or chronic stage, but has not developed secondary arthritis, and yet has a foot structure that is contributory to PTD, where simple primary repair would produce less than desirable results. This article presents a case of PTD that combined primary tendon repair with double calcaneal osteotomy as the procedure of choice. Following the presentation is a discussion of the rationale for selecting the procedures.

Case Presentation

A 47-year-old moderately obese female presented to the authors. She complained of painful swelling below the medial ankle and along the medial side of the left foot. The swelling was noticed ~3 weeks previous to her visit. She was unable to recall any traumatic incident. The patient's general medical history was essentially unremarkable. She had a history of cholecystectomy and hysterectomy performed several years previously. She was not taking any medications, with the exception of "diet pills," and had no known allergies.

Physical examination revealed edema and tenderness along the course of the PT tendon, especially around the area of the navicular. No other areas of pain or edema were noted. Skin color and temperature were normal with no open lesions. Pedal pulses were palpable and equal bilaterally with no varicosities. Neurological findings were within normal limits. The patient had bilateral pes planovalgus deformity, greater on the left. The right foot was affected to a lesser degree and was asymptomatic. Biomechanically, the deformity was noted to be greatest in the transverse plane with significant forefoot

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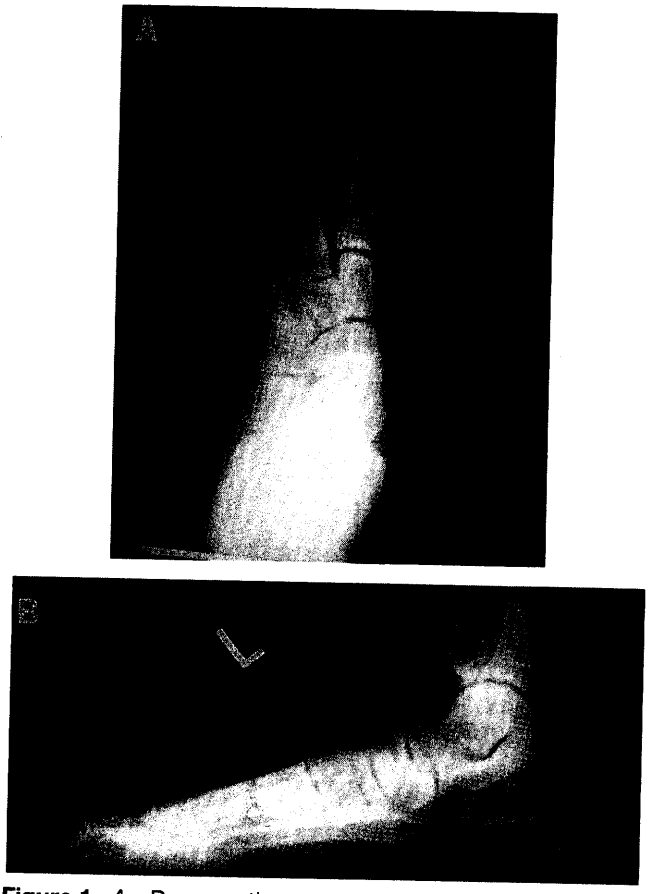

Figure 1. *A*, Preoperative anteroposterior view showing transverse plane deformity and talonavicular joint dislocation. *B*, Lateral view showing decreased arch height. Also note lack of any degenerative arthritis at subtalar joint or talonavicular joint.

abduction, with a moderate degree of calcaneal eversion. X-rays revealed no evidence of fracture or joint degeneration. There was, however, partial dislocation of the talonavicular joint (Fig. 1). The working diagnosis was partial PT tendon rupture or avulsion of the PT tendon from its insertion at the navicular tuberosity. The authors believed that her pre-existing pes planus foot structure induced the rupture. The patient was placed into a fiberglass nonweightbearing below-the-knee cast. She was to return to the office in 3 weeks, at which time she would be casted for orthotic devices.

The patient returned 3 weeks later with the cast intact, but she was still symptomatic. The patient was casted for neutral position accommodative orthotic devices, and the below-the-knee cast was reapplied. At her 1-week cast check, the patient stated that she was asymptomatic while in the cast. Two weeks later, the cast was removed, revealing a decrease in swelling and no pain. Orthotic devices were dispensed. The patient was advised to return if the symptoms recurred, and that a magnetic resonance imaging (MRI) study would be necessary to determine the extent of the injury. The symptoms began to return within 1 week. An MRI of the left foot was obtained that revealed irregularities of the PT tendon and edema within the tendon sheath (Fig. 2). Surgical intervention was discussed with the patient and agreed on. The elected procedure included repair of the PT tendon along with double calcaneal osteotomy to redistribute weightbearing forces. The authors believed that soft-tissue reconstruction by itself would, at best, provide temporary relief, because there would still be an existing pes planus deformity. Since the pes planus foot structure was thought to be the cause of the tendon rupture, it was determined that double calcaneal osteotomy would be necessary to reduce the deformity. Because of the patient's young age, active life-style and lack of degenerative findings on x-ray, joint fusion was not considered.

Operative Technique

Surgery was performed under general anesthesia, with a pneumatic thigh cuff set at 350 mm. Hg for hemostasis. The patient was given 1 gm. of cefazolin intravenously for antibiotic prophylaxis. A 12-cm. curved incision was placed along the lateral aspect of the foot beginning at the posterior aspect of the lateral malleolus and extending anteriorly to the calcaneocuboid joint. Tissues were separated anatomically to the level of the sural nerve and lesser saphenous vein that were retracted superiorly and the peroneal tendons that were retracted inferiorly. The body of the calcaneus was approached, and its superior and inferior margins were identified, along with the subtalar joint. A transverse through-and-through osteotomy was performed in the body of the calcaneus using a power sagittal saw. The osteotomy was made parallel to the peroneal tendons, with care being taken to avoid the subtalar joint. The posterior fragment was transposed medially and plantarly ~3 mm. and fixated into position using two Steinmann pins. This was done in an attempt to increase the calcaneal inclination angle and calcaneal varus.

The calcaneocuboid joint was then approached. Dissection proceeded anatomically to the level of the extensor digitorum brevis muscle. The muscle was detached from its lateralmost origin site and reflected dorsally and anteriorly. Peroneal tendons were reflected inferiorly. The calcaneocuboid joint was identified and a site 1.5 cm. posterior to it was marked. Using a power sagittal saw, a transverse osteotomy was performed at this level, with care being taken to preserve the medial cortex of the calcaneus. The osteotomy was distracted using an osteotome, and a 1-cm. cortical allograft (tibia) was impacted into the site. Intraoperative x-rays were taken at this time, which showed good position of both osteot-

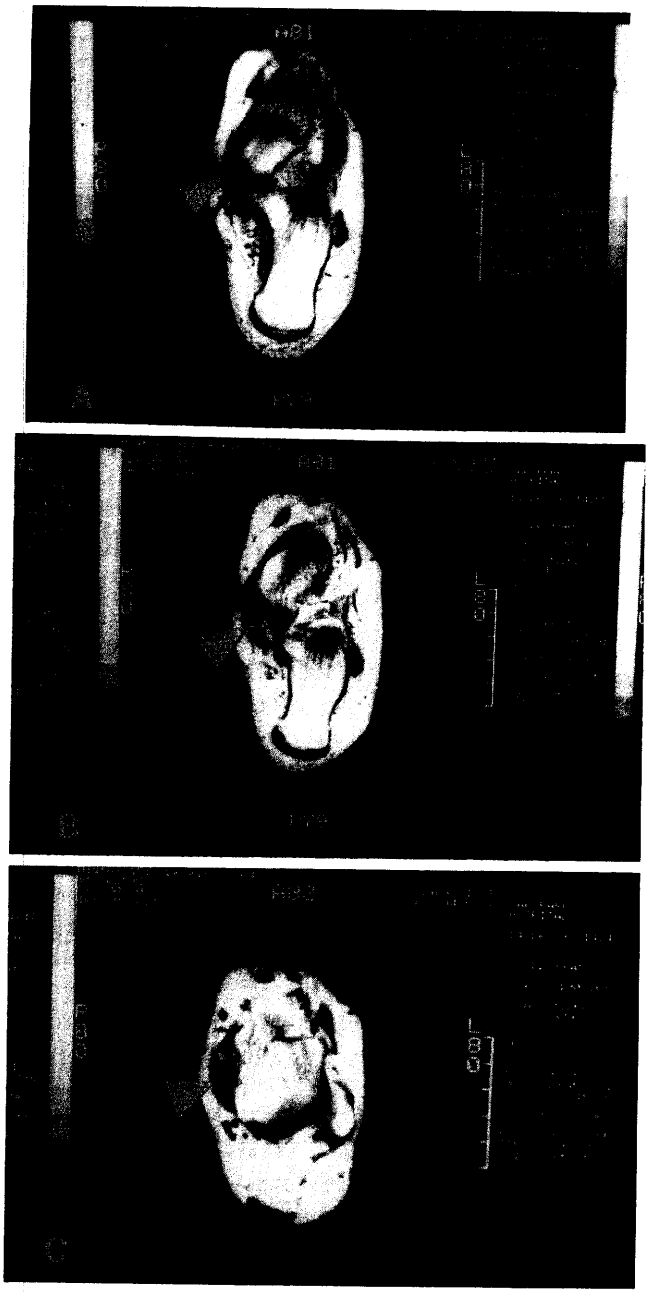

Figure 2. *A*, MRI showing thickening of PT tendon on coronal view (*arrow*). *B*, MRI demonstrating irregularities in PT tendon (*arrow*). *C*, MRI axial view showing edema within PT tendon sheath.

omies, graft, and fixation devices (Fig. 3A). A dramatic reduction in transverse plane foot deformity was also observed (Fig. 3B). Both sites were copiously irrigated, and deep tissues and skin were reapproximated. This procedure was performed to reduce transverse plane deformity and realign the talonavicular joint, as well as assist in increasing calcaneal inclination by stabilization of the lateral column.

Repair of the soft tissues was then initiated. Attention was directed to the medial aspect of the foot where an ~10-cm. incision was performed, extending from the inferior aspect of the medial malleolus distally to the first metatarsal-cuneiform articulation. Dissection proceeded anatomically to the level of the laciniate ligament that was incised exposing the tibialis posterior (TP) tendon and its sheath. Dissection was continued anteriorly to the level of the navicular tuberosity. It was noted that the TP tendon had no attachments to the navicular. Attachments were, however, observed into the first cuneiform and deep attachments to the lesser tarsus. The navicular and tendon were both examined, with no evidence of avulsion of the tendon or any previous attachments to the navicular. This was suspected to be an abnormal tendinous insertion. It was not evident on MRI.

At a level immediately proximal to the navicular tuberosity, the tendon was degenerated over a span of ~2 cm. This section of tendon was noted to be very narrowed, white, and fibrotic. The remainder of the tendon was of normal appearance. The dystrophic section of tendon was excised, leaving proximal and distal tendon stumps. A portion of the navicular tuberosity was resected generating a surface of bleeding cancellous bone. A Mitek G-II[4] anchoring device was inserted into the navicular, and the proximal tendon stump was secured into its normal position on the navicular tuberosity. Additional simple interrupted sutures of 0 polyglycolic acid were used to attach the proximal and distal tendon stumps as the foot was held in an inverted position. The tendon was found to be securely anchored, the site was irrigated, and tissues closed anatomically.

Medial and lateral incision sites were infiltrated with 0.5% bupivacaine plain and 0.4% dexamethasone phosphate. SILASTIC[5] drains were inserted into the wounds, and a dry sterile compressive dressing was applied. The tourniquet was released, with normal color returning immediately to all digits. A fiberglass posterior splint was applied, and the patient was admitted for 23-hr. observation.

The patient's first postoperative visit was 3 days later when drains were removed and a new fiberglass posterior splint applied with instructions for nonweightbearing with crutches. The wound appeared healthy at this time. Ten days later, sutures were removed and a below-the-knee fiberglass nonweightbearing cast was applied with follow-up in 1 month. At 7 weeks, external pins were removed and x-rays showed continued healing of osteotomies, with early consolidation of the bone graft (Fig. 4). At 9 weeks, the patient was given a

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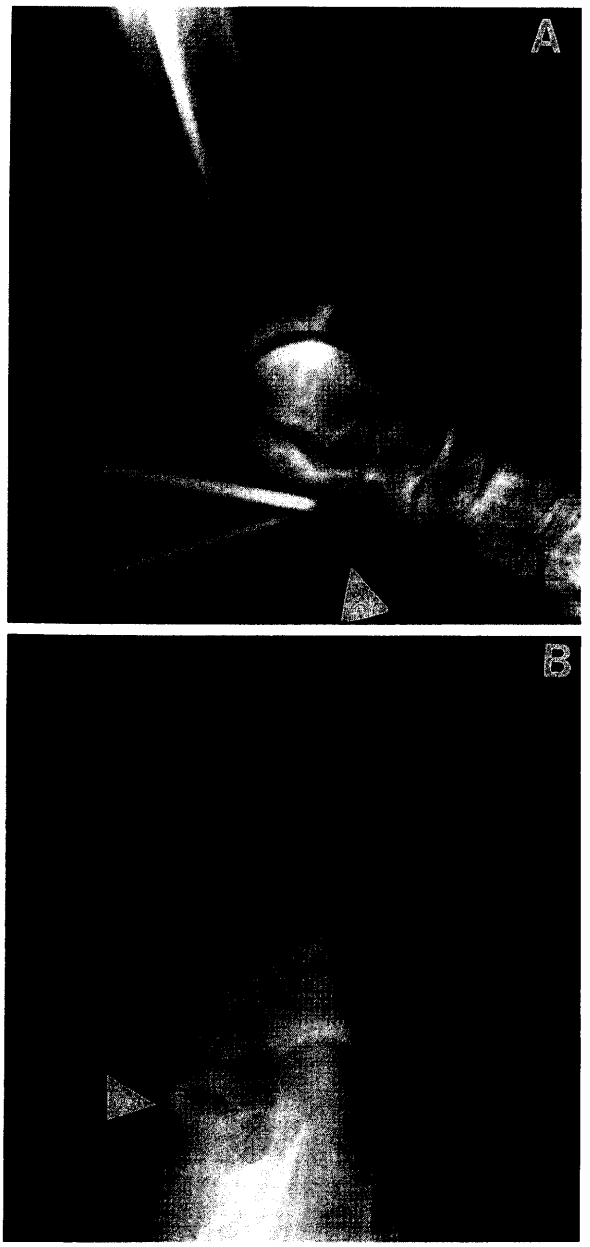

Figure 3. *A*, Intraoperative lateral view showing pin placement and location of bone graft (*arrow*). *B*, Intraoperative anteroposterior view showing graft placement (*arrow*) and reduction of transverse plane deformity.

fiberglass below-the-knee walking cast and was relatively asymptomatic. At 12 weeks, the patient was placed into a removable lower leg walker, with instructions for active and passive range of motion exercises. The patient continued to improve and had no complaints. At 16 weeks, the patient returned to normal shoe gear and orthotic devices, continuing physical therapy. At 6 months, the patient had progressed to stationary bicycling and step aerobics. At 11 months of follow-up, she remains pain-free and completely asymptomatic with a

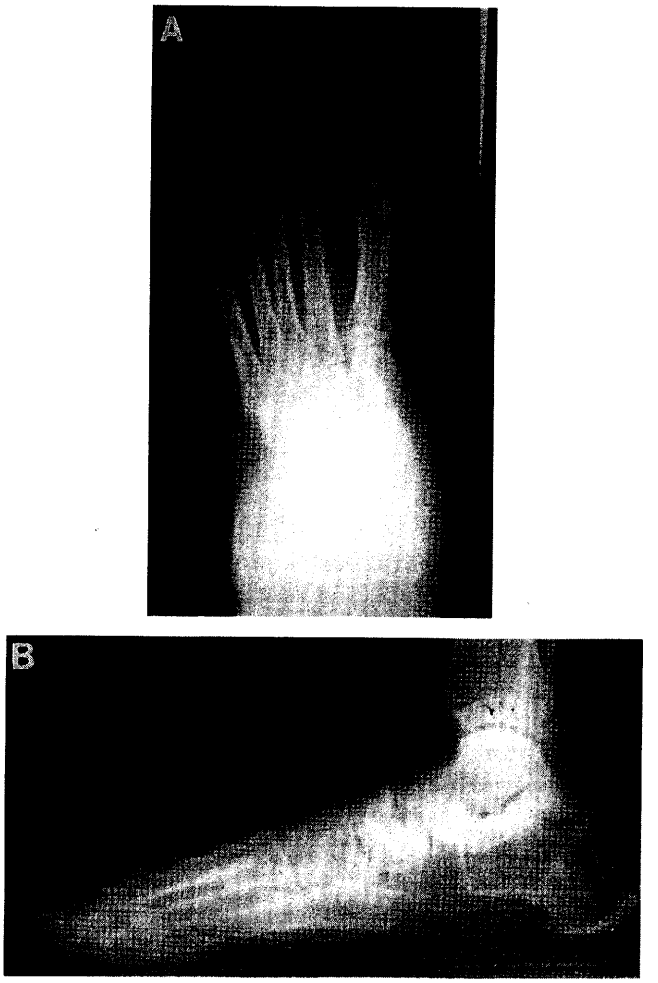

Figure 4. *A*, Anteroposterior view showing transverse plane stability of forefoot 5 months postoperative. *B*, Lateral view showing healing of osteotomies, incorporation of bone graft, and sagittal plane stability 5 months postoperative.

normal gait pattern, and has returned to preinjury levels of activity.

Discussion

Anatomy and Physiology

In the normal foot, the TP functions as an adductor of the midtarsal joint, directly opposing the actions of peroneus brevis (7, 8). It is also considered a major invertor of the rearfoot (8). The muscle becomes active early in the stance phase of gait, controlling rearfoot eversion by its relation to the subtalar joint axis. The muscle also aids in subtalar joint inversion and heel lift in the propulsive phase, then becomes inactive after heel lift (7).

TP is also believed to act as a stabilizer of the foot during propulsion. The multiple insertions of the TP tendon have been well-described previously. They include attachments at the navicular, first cuneiform joint,

sustentaculum tali, 2nd and 3rd cuneiforms, cuboid, and bases of 2nd, 3rd, and 4th metatarsals. There are also soft tissue attachments into the naviculocuneiform capsule, inferior band of the extensor retinaculum, long and short plantar ligaments, spring ligament, and dorsal ligamentous complexes (7). Mueller (8) notes that the tendon does not seem to glide at these secondary insertions and thus probably acts as a bony stabilizer. He also feels that the interweaving of the TP and peroneus longus insertions helps to balance frontal plane action of each muscle, where weakness of either muscle results in overpowering by the other.

Pathology

A number of theories have been published regarding the etiology of PTD. Mueller (8) lists four categories of dysfunction, including direct injury, pathological rupture, idiopathic rupture, and functional rupture. Banks and McGlamry (1) use two categories, traumatic and degenerative, whereas Johnson (2) adds spontaneous rupture to this list. However, one should probably be suspicious of degenerative changes being present in any case of idiopathic or spontaneous rupture (1, 7). A study by Frey *et al.* (9) examined the possibility of a zone of hypovascularity that normally occurs in the PT tendon. The hypovascular region was noted to be where the tendon courses around the medial malleolus, a common site of rupture. They suggest that PT rupture arises from a variety of causes (9).

Banks and McGlamry (1) state that the most common cause of PT rupture arises from inflammation that results in an already-stressed tendon that is trying to stabilize a hypermobile foot. The PTD patient most often presents with a bilateral pes planus, with the involved foot being more progressively so (7). Hall (7) feels that obesity, pes planus, and hypovascularity of the tendon itself are predisposing factors toward PTD. Hall cites a study by Garrett that demonstrates increased stress on the PT tendon as it tries to control pronation in a pes planus foot with a valgus heel. This problem is compounded by obesity, which can then result in failure of the tendon and ligamentous stretching along the medial column of the foot (7). This results in the characteristic progressive unilateral flatfoot, with greater deformity in the transverse plane, so well described in the literature.

Diagnosis

Symptoms and signs will vary with the onset, extent, and duration of the PT tendon lesion (1–3, 7). Several authors have divided PTD into stages based on the duration of the lesion, presenting symptoms, and condition of the tendon when examined surgically or with MRI, currently the standard for assessing tendon integrity (3, 8). Table 1 represents a summary of the stages of PTD as described by Johnson and Strom (3) and Mueller (8).

TABLE 1. **Stages of PT tendon rupture [as described by Johnson and Strom (3) and Mueller (8)]**

	Clinical Signs	Radiographic Signs
Stage I	Pain and edema along course of tendon; possible weak heel-rise test; relief with rest	No changes
Stage II	Increasing pain; minimal relief with rest; medial arch begins to flatten; increasing heel valgus; "Too-many-toes" sign; most common stage of presentation	Forefoot abducted to rearfoot; subluxed talonavicular joint; decreased arch height; MRI shows tendon irregularities
Stage III	Long-standing deformity; increasing forefoot abduction; increasing heel valgus; no rearfoot inversion; minimal heel rise; pain may involve lateral foot or sinus tarsi; difficulty walking; most will have presented by this stage; foot is still reconstructible	Increased forefoot abduction; minimal arch height; arthritic changes in tarsal joints; MRI shows partial or complete rupture of tendon or avulsion
Stage IV	End stage; rapid progression of pes planus; only option is fusion	Rapid progression of degenerative joint disease

The patient usually presents with mild to moderate pain along the course of the PT tendon, frequently between the medial malleolus and navicular. The pain is usually a chronic type of weightbearing achiness (2). The patient may report a gradual collapse of the arch of the involved foot. They rarely recall a specific traumatic incident (1).

Physical examination reveals swelling medial to the rearfoot (2). There is usually tenderness and warmth in the region between the medial malleolus and navicular. If a complete rupture is present, a defect may be palpable (1). Some authors advocate manual muscle strength testing by isolation of the PT muscle (7, 8). Johnson and Strom (3), however, feel that manual testing is essentially useless because of the difficulty in isolating the muscle, substitution of other extrinsic muscles, and the relative strength of the PT muscle. They feel the more reliable method to be the single-heel-raise test. If the patient is unable to rise up on the ball of the foot, or if the heel rises without inverting and locking, some degree of PT dysfunction is noted and is correlated with physical findings (3).

PTD is a progressive disorder and, as mentioned earlier, it can be classified into stages, depending on condition of the tendon and patient symptoms (3, 8). Stage I represents a medial aching usually induced by physical activity and relieved by rest (3, 7). There may be a fullness present to the area inferior to the medial malleolus when compared with the other foot (3). Single-heel-rise testing may reveal a mild weakness (3, 7), and the tendon itself may be inflamed, but will be normal length (3). No x-ray changes are noted in the bony architecture, but MRI may show evidence of degenerative changes in the tendon, such as longitudinal tearing or edema of the tendon sheath (3) (Table 1).

Stage I can progress to stage II. These changes evolve over several months to years. The pain is increased in severity and may even be present after rest (3). At this stage, foot deformity may be noted, with increasing valgus of the rearfoot and flattening of the medial arch (7, 8). When viewed from behind, the patient may demonstrate the "too-many-toes" sign (1–3, 6, 7). When standing, the heel goes into a relaxed everted position and the forefoot assumes an abducted position. This results in "too many toes" being visible when sighting along the lateral border of the affected foot. On x-ray, the forefoot will be abducted to the rearfoot, with the navicular being subluxed from the head of the talus and collapse of the longitudinal arch (2, 3, 6–8). MRI can show a discontinuity in the tendon or a proximal thickening of the tendon. This is the most common stage for patient presentation (3, 8) (Table 1).

Left untreated, a stage III deformity is present for even longer periods, and the pain may transfer to the lateral side of the foot or over the sinus tarsi because of the sustained pronation (3). The most prevalent change is increased abduction of the forefoot and valgus of the rearfoot. There is an absence of inversion of the rearfoot and even more diminished ability to rise onto the ball of the foot (3). X-rays show continuing changes and early degeneration of the tarsal joints from the abnormal pronatory forces (3). As the forefoot moves into a more abducted position, the angle of the calcaneocuboid joint assumes a concave angle (7). This is a good determinant of the degree of deformity present. Mueller refers to stage III as the "rupture (or functional rupture) phase." He states that the foot is reconstructible up to this point. Most patients will have presented by this stage (8) (Table 1).

Mueller's stage IV is described as "end stage," with rapid progression of pes planus and degenerative joint disease. The changes to the foot structure can be extremely debilitating. The only viable treatment option at this stage is joint fusion (8) (Table 1).

The deforming force in PTD is caused by abduction of the forefoot while the oblique midtarsal joint is attempting to supinate. The TP and ligaments of the talonavicular joint attempt to control these opposite forces (8). The ligaments become stretched and the TP muscle fatigues while trying to support the ligaments, and gradually a pes planovalgus foot results. The pes planovalgus condition causes more ligamentous stretching and tendon fatigue that worsens the pes planovalgus, and the cycle is self-propagating (8). The altered pull of the PT tendon that arises from the planovalgus foot structure also contributes to collapse of the foot (8). PTD is therefore a progressive disorder that can be prevented from worsening, and delays in treatment will only increase the problem (1).

Treatment

Most authors agree that conservative treatment of PTD has a limited success rate (1–3, 5). The most often used forms are shoe modifications, orthoses, immobilization, nonsteroidal anti-inflammatory medications, and local corticosteroid injections (1, 2, 4–6). Incidents of complete rupture of tendons following local steroid injections have been reported, and for this reason they should be avoided (1, 2, 7). Jahss (4) notes that conservative treatment generally gives no relief or may even allow the condition to worsen. Banks and McGlamry (1) believe that conservative treatment only delays the inevitable breakdown of the foot structure and should therefore be reserved for patients who are not surgical candidates or those who present with minimal initial deformity and symptoms.

Surgical treatment of PTD generally focuses on repair of the tendon itself, or joint fusions in advanced degenerative cases. The most common treatment described involves bridging the PT rupture with a side-to-side anastomosis to the flexor digitorum longus (FDL) tendon (1, 2, 3–5). Johnson (2) initially described treatments that involved reattachment of the tendon to its navicular insertion in cases of avulsion, end-to-end suturing in simple ruptures, FDL anastomosis in complex ruptures, and arthrodesis in degenerative cases. Later, Johnson and Strom (3) advocated synovectomy of the PT sheath and tendon debridement in stage I cases, FDL bridging or substitution in stage II cases, and subtalar arthrodesis in stage III cases (3). Helal (6) described the Cobb procedure used in eight cases of PT rupture. This involves splitting the tibialis anterior tendon, leaving the insertion intact and passing the split portion through a drill hole in the first cuneiform, running the tendon behind the medial malleolus, and attaching it to the proximal stump of the TP. Helal (6) noted that, of 8 feet treated, 5 were restored to normal. Banks and McGlamry (1) believe that no improvement in basic foot architecture is accomplished with any of the aforemen-

tioned soft-tissue procedures and for the treatment to be successful, the deforming force must be addressed. Mann and Specht (5) also felt that inadequate correction was obtained with soft-tissue procedures, and osseous procedures may be necessary for complete correction.

The osseous procedures most commonly mentioned involve arthrodesis of the various tarsal joints. Again, arthrodesis is reserved for advanced cases with degenerative arthritic changes present. Johnson and Strom (3) feel that subtalar fusion is sufficient enough to realign the calcaneus beneath the talus. This, however, is based on their assumption that most of the deformity is located in the frontal plane (1). Conversely, examination will reveal that transverse plane deformity occurring at the midtarsal joint is the primary deformity (1). For this reason, Banks and McGlamry (1) feel that triple arthrodesis should be the fusion procedure of choice to realign properly the forefoot, as well as the rearfoot. The patients they report on, however, are older, with a long-standing deformity and sedentary life-styles necessitated by their condition (1).

It seems that the references cited herein have missed one specific group of patients in their treatment regimens who would not be properly served by tendon repair alone, or by arthrodesis. This specific patient group is one of a relatively young age and fairly active life-style with a recent onset of injury. Most of these patients also have a history of bilateral pathological pes planus. Clearly, a soft-tissue procedure alone would not eliminate the deforming force that likely initiated the tendon injury. A soft-tissue procedure would provide temporary relief if any, and because the abnormal structural element has not been addressed, the pathology is likely to return (1, 5, 7). Their lack of degenerative joint disease or of rigid, long-standing deformity would indicate that arthrodesis procedures are not warranted (7). Other disadvantages of arthrodesis include joint degeneration proximal or distal to the fusion site, malpositioning, nonunion, and more difficult rehabilitation (7). Activity levels will certainly never be the same after arthrodesis (7). For these patients, the authors suggest double calcaneal osteotomy to realign loading forces in the foot. Combined with necessary soft-tissue reconstructive procedures, this allows restoration of foot structure while preserving joint motion.

Hall (7) indicates that the orthopedic community has recently begun investigating the use of calcaneal osteotomy for the treatment of PTD. They feel that this may be a better way of dealing with the resultant pes planovalgus deformity while maintaining foot motion and providing a more cosmetically pleasing result (7). To his knowledge, he notes that there are no published reports of calcaneal osteotomy being used to correct PTD in the literature, but osteotomies being investigated include lateral column lengthening (Evans procedure) and osteotomy of the body of the calcaneus (Gleich/Koutsogiannis). These are performed in an attempt to correct both frontal and transverse plane foot deformities (7), and when soft tissue procedures provide less-than-desirable results (1, 4, 7).

The Gleich and Koutsogiannis calcaneal osteotomies, as reported by Hall (2), are through and through osteotomies in the body of the calcaneus, with medial transposition of the posterior fragment (tuberosity). A laterally performed osteotomy paralleling the peroneal tendons is described, along with cannulated screw fixation (7). From previous surgical experience, the authors have found that by transpositionalizing the tuberosity medially, plantarly, and in a varus frontal plane rotation, this increases the calcaneal inclination angle, increases intrinsic calcaneal varus, and increases medial pull of the gastrocnemius tendon, thereby exerting a more varus influence on the rearfoot. This will have the effect of placing the calcaneus in a more anatomical position beneath the talus.

The Evans osteotomy was originally described for reduction of overcorrection of clubfoot deformity as a lateral column lengthening procedure. Evans (10) performed the osteotomy at the anterior lateral aspect of the calcaneus, 1.5 cm. proximal to the calcaneocuboid joint and parallel to it. The osteotomy was distracted and plugged with a cortical bone graft taken from the tibia (10). No fixation was used. Evans noted that the lateral column was the foundation of the foot and that by lengthening the lateral column in this manner, the heel was forced into a more varus position (10). After publishing results from 56 procedures, he felt that the procedure was valuable as an alternative to triple arthrodesis in select cases, including flexible and rigid flatfoot (10). The procedure was initially performed in adolescent individuals (10). Powell and Cantab (11) reported on the Evans osteotomy used by itself and combined with the Dwyer calcaneal osteotomy to correct valgus hindfoot in a group of children and adults. They noted that posterior calcaneal osteotomy markedly improved the shape of the rearfoot and reduced symptoms. They believed that these procedures were not always applicable to the adult, but were much less destructive than triple arthrodesis (11).

Hall (2) currently uses a combination of Gleich/Koutsogiannis-type calcaneal osteotomy with either primary PT tendon repair for avulsion or minimal gapping, or FDL anastomosis for more extensive ruptures with significant gapping. He believes this combination will better restore the medial arch and decrease heel valgus, and provide a more cosmetically appealing foot (7). His criteria for osteotomy with tendon repair are forefoot varus <7 to ten degrees (fixed or flexible), hindfoot

valgus of <10 degrees, and no evidence of sinus tarsi or lateral impingement pain. If any of these parameters are exceeded, consideration should be given to arthrodesis. If sinus tarsi pain is present along with forefoot varus of <7 to 10 degrees and patient weight >200 lb., subtalar fusion should be performed. If forefoot varus is >7 to 10 degrees, triple arthrodesis should be performed (7).

The senior authors have been using double calcaneal osteotomy in the treatment of structural flatfoot for many years with high success rates, and believe these procedures are applicable to PTD with calcaneal valgus and dislocation of the talonavicular joint when no degenerative joint disease is present. The osteotomies are combined with the tendon repair of choice. The combination of double calcaneal osteotomy with primary tendon repair was selected for the authors' patient for the following reasons. The patient was in her middle 40s and led a fairly active life-style. Her injury was of relatively recent onset, and joint arthrosis was not present. Her pes planus foot structure eliminated the option of a soft-tissue procedure alone, but it was felt that performing an arthrodesis was not justified and would have been premature. At the patient's current stage, she has resumed her preinjury life-style, and the option of arthrodesis is still available if the deformity returns or joint degeneration develops.

Conclusion

PTD can be a very destructive pathology. It can have a sudden or gradual onset, and can be difficult to diagnose. If recognized in the early stages, the condition is correctable, and foot structure can be salvaged before degenerative arthritis develops. Conservative treatment generally is not effective in any except the most mild cases. Surgical treatment therefore is most often utilized. By grouping surgical treatments into soft-tissue reconstruction or arthrodesis, an entire group of patients may be undertreated or subjected to unnecessary joint destructive procedures. By utilizing soft-tissue reconstructive procedures along with calcaneal osteotomy, these select patients with recent injuries, pes planus, and minimal joint arthrosis can be spared recurring deformity or the uncertainties of fusion, while still leaving the option of fusion available for a later date, if necessary. The authors believe double calcaneal osteotomy is indicated in the treatment of PTD, with talonavicular dislocation and calcaneal valgus in the proper group of patients. The authors have used double calcaneal osteotomy successfully in the treatment of flatfoot and believe it can be applied to PTD in the proper circumstances. With only one patient at an early stage of follow-up, results do seem favorable and very encouraging.

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