Posterior Tibial Tendon Rupture: A Refined Classification System

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Posterior tibial tendon rupture (PTTR) is commonly implicated in the adult acquired flatfoot deformity [1]. Johnson and Strom [2] described three stages of PTT dysfunction in 1989. The diagnosis and treatment of PTTR has been organized around this staging scheme [3]. In stage I disease, patients have tenderness, fullness, and paratenonitis (with or without tendinosis) of the PTT without deformity. Treatment is typically conservative but may include debridement, tenosynovectomy, and possibly calcaneal osteotomy. In stage II disease, patients have a dynamic, or flexible, flatfoot deformity with a flattened arch, forefoot abduction, and hindfoot valgus malalignment. Surgical treatment usually involves some form of reconstruction of the PTT, commonly using the flexor digitorum longus (FDL) tendon, combined with an osteotomy, such as a lateral column lengthening or medial displacement calcaneal osteotomy. Resulting forefoot varus and loss of dorsiflexion may be corrected with medial cuneiform osteotomy and tendo Achilles lengthening (TAL), respectively. Additional procedures, such as spring ligament reconstruction with peroneus longus tendon [4] and augmentation of the FDL transfer with peroneus brevis tendon [5], have also been described. Stage III disease is characterized by fixed hindfoot valgus deformity, often accompanied by fixed compensatory forefoot varus. Because the hindfoot is typically irreducible in stage III disease, surgical treatment generally consists of a triple arthrodesis. Myerson [3] has described
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**Abbreviations:** HF, hind foot; FDL transf., flexor digitorum longus transfer; med. displ. calc. osteot., medial displacement calcaneal osteotomy; NSAID, nonsteroidal anti-inflammatory drugs; PTT, posterior tibial tendon; TAL, tendo Achilles lengthening; TMT, tarsometatarsal joint; TTC, tibiotalocalcaneal.
a fourth stage that involves valgus tilting of the ankle joint within the mortise and associated deltoid ligament insufficiency with or without lateral tibiotalar arthritis. Treatment for stage IV disease is determined by the presence of ankle arthritis and the ability to reduce the deformity. Treatment of stage IV generally consists of arthrodesis [1,6,7] or a foot realignment with deltoid reconstruction [8–10].

Since Johnson and Strom’s initial 1989 classification, an increasingly complex array of deformities of the foot has been recognized in association with PTTR. This wide spectrum of deformity is not completely addressed by the current classification system. Further, the existing system does not leave sufficient room for variation within a given treatment stage. For example, there are a myriad of treatment options for stage II disease. This reflects the wide variation in medial ray hypermobility, forefoot abduction, and forefoot varus seen in patients grouped together as having a “flexible flatfoot.”

We believe that a more comprehensive classification system that addresses the diversity of the adult acquired flatfoot deformity is necessary. This scheme should take into account ankle and hindfoot valgus, forefoot supination, forefoot abduction, and medial column instability. Accordingly, we now present and discuss a new classification for PTTR based on the clinical experiences of the senior author (MSM). It is not the authors’ intention to propose the ideal surgical correction for each deformity encountered. It is our hope, however, that this revised classification assists the foot and ankle surgeon in planning treatment for the varied, complex problem of adult acquired flatfoot deformity. Although we suggest treatment alternatives based on these stages and its subcategories, a range of surgical alternatives given the exact stage of deformity is available, many of which we have limited experience with and we therefore do not discuss. We maintain the existing Myerson modification to the Johnston and Strom scheme and use it as a general outline into which we have subdivided a more descriptive and comprehensive system. Table 1 summarizes the refined classification, including pertinent findings and suggestions for treatment for each of the described stages.

Refined classification and treatment recommendations

Stage I: tenosynovitis without deformity

In stage I disease, the paratenon is inflamed or the tendon partially ruptured. This may or may not be accompanied by systemic inflammatory disease. In either case, there is no (or minimal) deformity, and the overall continuity of the tendon is maintained. Tendon continuity is confirmed on physical examination by direct palpation with the foot in plantarflexion and inversion [11] and by an intact single leg heel rise and good resisted
foot inversion strength with the foot plantarflexed. Stage I is subdivided into three categories:

A. **Inflammatory disease.** PTT inflammation or rupture secondary to systemic disease, such as rheumatoid arthritis and the other inflammatory arthritides, is recognized as a separate entity [12]. In stage I-A, hindfoot anatomy is maintained and the foot alignment is normal. Treatment consists of conservative nonoperative care [13] or tenosynovectomy [14].

B. **Partial PTT tear with normal hindfoot anatomy.** Although the etiology is separate from stage I-A, similar treatment with conservative nonoperative care or tenosynovectomy is recommended.

C. **Partial PTT tear with minor hindfoot valgus.** There is slight (5° or less) hindfoot valgus deformity to distinguish this from stage II disease. Although the authors still begin with conservative treatment of anti-inflammatory medications and immobilization in a cast, walking boot, or custom brace, stage I-C may represent incipient rupture and should be monitored closely. If surgery is performed, in addition to a tenosynovectomy a medial translational osteotomy of the calcaneus should be considered [15].

**Stage II: ruptured posterior tibial tendon, flexible flatfoot**

The presence of stage II disease implies significant PTT attenuation or frank rupture as evidenced on physical examination by a clinically apparent flatfoot deformity, inversion weakness of the plantarflexed foot, and inability to perform a single or repetitive heel rise. Stage II disease is subdivided into three categories (with the first further subdivided into two subcategories), depending on the most salient feature present. Because some patients exhibit several of the following features, some degree of overlap may exist.

A. **Hindfoot valgus.** In stage II-A, the salient feature is valgus deformity of the hindfoot (Fig. 1A, B). Once the heel is reduced from valgus to neutral, there are varying degrees of residual forefoot supination. This supination is caused by accommodative changes that allow the medial and lateral columns of the forefoot to remain in contact with the ground in the setting of hindfoot valgus. The forefoot supination may be minimal or reducible (stage II-A-1) or fixed (stage II-A-2). Nonoperative treatment typically involves an inverted, medially posted foot orthosis or a custom brace [13,16]. The authors’ preferred operative treatment for stage II-A consists of a medial displacement calcaneal osteotomy and FDL tendon transfer to the navicular (Fig. 1C, D) [17,18]. As an alternative to calcaneus osteotomy an arthroereisis may be considered. Treatment does not end here, however; the deformity may have pulled the forefoot into varus, and this needs to be assessed and dealt with as follows:

1. Flexible forefoot varus. In stage II-A-1, reducing the hindfoot from valgus to neutral results in forefoot varus (Fig. 2). The forefoot
deformity is flexible, however; if the ankle is plantarflexed to relax the gastrocnemius and remove the hindfoot valgus, the forefoot varus is corrected. After medial calcaneal slide and FDL transfer, the forefoot typically moves into a varus position; intraoperative assessment is made as to whether this varus corrects to neutral. If the deformity corrects without plantarflexion, no further operative treatment is required. If the forefoot varus corrects only with ankle plantarflexion, a gastrocnemius recession or a percutaneous Achilles tendon lengthening is added.

2. Fixed forefoot varus. In stage II-A-2 adaptive changes occur in the frontal plane of the forefoot because of the longstanding hindfoot valgus. Although the hindfoot deformity is supple and reducible to neutral, the forefoot deformity thus remains fixed in varus, even with the ankle plantarflexed to relax the gastrocnemius. After the medial calcaneal slide and FDL transfer, a dorsal opening wedge medial cuneiform osteotomy in the manner of Cotton [19] is then performed. This latter procedure plantarflexes the medial column to correct the forefoot

Fig. 1. Radiographic stage II-A, or flexible flatfoot with hindfoot valgus. Preoperative lateral (A) and AP (B) views. Lateral (C) and AP (D) views of the same patient after medial displacement calcaneal osteotomy and flexor digitorum longus to navicular transfer.
supination deformity. Typically this is performed with corticocancellous allograft, and internal fixation for this stable osteotomy is rarely used.

B. Forefoot abduction. Abduction of the forefoot (stage II-B) is commonly observed in conjunction with the hindfoot valgus that characterizes stage II-A deformity (Fig. 3A, B). The forefoot abduction may occur at the transverse tarsal joint (most commonly) or at the first tarsometatarsal (TMT) joint, and occasionally at both complexes. First TMT joint instability can be a primary deformity or secondary to TMT joint arthritis. The simplest way to determine this distinction is examination of the lateral foot radiograph for the presence of a gap at the plantar joint surface; this gap may be associated with primary deformity of the first TMT joint, which may then result in secondary hindfoot deformity, including rupture of the PTT. Forefoot abduction occurring through the transverse tarsal joint is easily evaluated by way of an anteroposterior (AP) foot radiograph. Abduction through this joint manifests as talar head uncovering (Fig. 3B) and can be quantified with the anteroposterior talar head uncoverage angle or by percent of the talar head uncovered [20]. Talar head uncovering of greater than 40% warrants consideration for performance of a lateral column lengthening procedure.

Surgical treatment of stage II-B deformity consists of an FDL transfer plus the addition of a lateral column lengthening to correct the forefoot abduction (Fig. 3C, D). It is recommended that the lateral column lengthening be incorporated into the surgical plan if talar head uncovering is greater than 40%. Uncoverage of lesser magnitude is corrected adequately with a medializing calcaneal osteotomy and tendon transfer. A lateral opening wedge osteotomy in the calcaneus is created 1.5 cm posterior to the calcaneocuboid joint, held open with corticocancellous allograft, and stabilized with a single 5.0-mm screw from anterior to posterior. A medializing

Fig. 2. Forefoot varus associated with acquired flatfoot deformity. (A) Appearance of forefoot with uncorrected hindfoot deformity. (B) Manual correction of the hindfoot valgus reveals residual forefoot varus. This forefoot deformity may be flexible or rigid.
calcaneal osteotomy is added to this if there is residual hindfoot valgus after completion of the FDL transfer and lateral column lengthening. Arthrodesis of the calcaneocuboid joint with lengthening is an alternative preferred by some surgeons to correct this deformity [21].

As an alternative to a double calcaneal osteotomy [22] a lateral column lengthening and arthroreisis may be performed. Arthroreisis may be a surgical option in Stage II-B when the hindfoot valgus is not corrected by FDL transfer and lateral column lengthening alone [23]. Typically a lateral column lengthening does not correct hindfoot valgus well [24].

Fig. 3. Pre- and postoperative roentgenogram views of stage II-B disease corrected with a lateral column lengthening and an opening wedge medial cuneiform osteotomy. (A) Preoperative lateral view demonstrating loss of medial arch height. Note the minimal hindfoot valgus. (B) Preoperative AP view of the same patient shows talar head uncovering of approximately 40%. (C) Postoperative lateral view shows restoration of the medial column height. Bone blocks are used to hold the lateral column and medial cuneiform osteotomies open. A fully threaded position screw gives fixation and aids in the maintenance of the lateral column lengthening. Fixation of the medial cuneiform osteotomy is usually not needed. (D) Correction of the talonavicular uncovering is evident in the postoperative anteroposterior view of the foot. In (C) and (D) the *arrowhead* demonstrates the medial cuneiform opening wedge osteotomy bone block, and the *arrow* points to the bone block in the anterior process of the calcaneus.
C. Medial ray instability. The most salient component in stage II-C disease is medial ray instability. As with the stage II-A-2 foot (fixed forefoot supination), the stage II-C foot tends to retain forefoot varus even with reduction of the heel from valgus to neutral, and even then with ankle plantarflexion. This is caused by medial column instability (Fig. 4A, B). It may arise from any component: the talonavicular, naviculocuneiform, medial cuneiform-first metatarsal joint (first TMT), or any combination thereof. After correcting the heel to neutral, the unstable medial ray tends to dorsiflex, causing the foot to pronate with weight bearing, and leads to painful subtalar impingement. Additional treatment consisting of arthrodesis of the pathologic component of the medial column may be warranted (Fig. 4C, D) provided that the first TMT joint is unstable. A medial cuneiform opening wedge osteotomy (as described under stage II-A-2) also corrects medial column instability. The decision

![Fig. 4. Stage II-C, or flexible flatfoot with medial column instability. (A) Preoperative lateral view. Gap at plantar aspect of first tarsometatarsal joint is shown by arrowhead. (B) Preoperative AP roentgenogram demonstrating forefoot abduction with talar head uncovering. (C) Postoperative lateral view showing restoration of arch height and TMT fusion to correct gapping and instability of joint. (D) Postoperative anteroposterior view showing correction of talar first metatarsal angle, first TMT fusion, and lateral column lengthening.](image-url)
as to whether a first TMT fusion or osteotomy of the medial cuneiform is performed is based on the degree of instability and presence of arthritis of the first TMT joint.

**Stage III: rigid hindfoot valgus**

Stage III disease is generally associated with a more advanced course of tendon rupture and deformity and typically is characterized by rigid hindfoot valgus. There may also be forefoot deformity, usually consisting of rigid abduction. There is less of a role for conservative management of patients who have this stage of disease, although custom bracing may be helpful.

A. Hindfoot valgus. The consensus to date has typically been to treat rigid hindfoot valgus, especially in cases with subtalar and talonavicular joint arthritis, with triple arthrodesis, and the authors adhere to this recommendation (Fig. 5) [25].

B. Forefoot abduction. Treatment also consists of triple arthrodesis, but if the abduction is severe it may also require lateral column lengthening with a bone block arthrodesis to lengthen the calcaneocuboid joint. This serves to swing the forefoot more fully out of abduction and back to neutral [26]. Additional procedures, such as Achilles tendon lengthening, gastrocnemius recession, medial cuneiform osteotomy, first TMT fusion, and lengthening of the peroneals or anterior tibial tendon, are performed as necessary.

**Stage IV: ankle valgus**

Stage IV disease occurs in the setting of longstanding PTT rupture and is associated with deltoid ligament insufficiency and medial ankle instability, leading to ankle (tibiotalar) joint valgus deformity. It often occurs in the setting of previous triple arthrodesis (Fig. 6). Malalignment of triple arthrodeses with residual hindfoot valgus may predispose to stage IV disease [27]. The authors have seen several variants of this condition; it may be associated with or without lateral ankle instability and arthritis and a flexible or a rigid hindfoot.

A. Hindfoot valgus and flexible ankle valgus without significant tibiotalar arthritis. In this setting it is appropriate to realign the ankle joint using medial-sided ankle procedures to reconstruct the deltoid ligament [8,9] after arthrodeses or osteotomies have been performed as required to re-establish a plantigrade foot as required (Fig. 7).

B. Hindfoot valgus with rigid ankle valgus or flexible deformity with significant tibiotalar arthritis. In stage IV-B, the ankle valgus deformity is rigid and essentially irreducible. Nonoperative treatment consisting of a custom ankle orthosis should be considered only if the condition needs to be temporized before surgical reconstruction or if the patient is physiologically not able to tolerate surgery. Operative treatment must include an arthrodesis of some sort, determined by the deformity, whether it be
isolated tibiotalar arthrodesis (in the setting of prior triple arthrodesis), pantalar arthrodesis, or tibiotalocalcaneal arthrodesis (Fig. 8). Occasionally the correction is performed with appropriate hindfoot realignment (typically an arthrodesis) in conjunction with a total ankle replacement [6,28,29].
This article has taken the original Johnson and Strom [2] classification for PTT rupture and revised it into a more comprehensive and discriminating system encompassing the various presentations seen within each stage. The purpose is ultimately to make treatment decisions more rational and individualized to each patient’s particular anatomic pathology. The clinical applicability of any classification system depends to a large extent on its usefulness in planning treatment, and it is with this goal in mind that the authors have proposed this system.

The most obvious expansion is with stage II disease. From the standpoint of foot morphology seen, this has typically been the most heterogeneous group of patients, with surgical decision making being made more difficult. It has been the authors’ experience that a flexible flatfoot (stage II) typically has a combination of one or more of the following features: hindfoot valgus, forefoot abduction, forefoot varus, or medial column instability. Depending on which is the most salient feature, there may be some variability in the optimal treatment choice.

Fig. 6. Stage IV-A hindfoot valgus secondary to under-corrected triple arthrodesis with flexible tibiotalar valgus tilting. Preoperative lateral foot Roentgenogram of the foot (A) demonstrates under-corrected medial longitudinal arch from a prior triple arthrodesis. An AP view of the ankle (B) shows severe tibiotalar valgus tilting without substantial erosive changes of the joint. A revision triple arthrodesis was performed with reconstruction of the ruptured deltoid ligament. Postoperative lateral (C) Roentgenogram of the foot demonstrates restoration of the arch. Tibiotalar joint correction is shown in (D). Although the use of soft tissue anchors to aid in reconstruction of the deltoid is shown the authors have found the use of allograft for such reconstructions to be more durable.
Fig. 7. Stage IV-A rigid hindfoot valgus with flexible tibiotalar valgus tilting. (A) Preoperative lateral. (B) Preoperative AP. (C) Preoperative AP ankle. (D) Preoperative fluoroscopy showing passive correction of valgus tibiotalar tilt. (E) Postoperative lateral foot. (F) Postoperative AP foot. (G) Postoperative AP ankle. Allograft tendon with soft tissue interference screws was used for deltoid reconstruction in this patient.
Stage IV in particular is an interesting problem in its etiology. Some patients who have stage IV disease have had a previous triple arthrodesis. Fitzgibbons addressed the question of whether the ankle deformity is secondary to increased valgus moment produced by the triple arthrodesis or to natural progression of disease [30]. At least two groups have performed experiments demonstrating increased strain in the deltoid ligament complex of posterior tibial tendon deficient cadavers that had undergone triple arthrodesis [5,31].

Although some investigators have reported that the rigid tibiotalar valgus of stage IV is more frequently encountered than the correctable form, the incidences of these two substages may depend on the timing of patient presentation and referral patterns to the foot and ankle surgeon. Tibiotalar
valgus arising from conditions other than those directly attributable to posterior tibial tendon pathology does occur. Conditions that fall into this category should be neither classified nor treated according to any PTTR grading scheme [5].

Pell and colleagues [25] reported on 111 patients who underwent 132 triple arthrodeses with a mean 5.7-year follow-up. The diagnosis in most patients was PTTR. Although the investigators noted a significant increase in the severity of ankle arthritis (from 7% to 60%), there was no correlation between patient satisfaction and the presence or absence of ankle arthritis. The proper treatment of stage IV is also a matter of some discussion. With the flexible IV-A patient, it may be appropriate to perform deltoid reconstruction and triple arthrodesis. There is scant information available on deltoid reconstruction in the setting of stage IV posterior tibial tendon deformity. Kelly and Nunley [7] mention reconstruction of the deltoid complex in patients who have stage IV disease, but they do not provide details on how to accomplish this or provide any clinical examples of reconstructions. Bohay and Anderson [6] suggest autograft flexor hallucis longus (FHL) for correction of tibiotalar valgus tilting of stage IV disease. Kitaoka and colleagues [32] have reported using a cadaver flatfoot model that deltoid reconstruction was superior to FDL reconstruction in restoring arch height, metatarsal–talar position, talocalcaneal position, and tibiotalar position. Their reconstruction was specifically done to restore arch height, however, not to address tibiotalar deformity. Bluman and colleagues have reported a minimally invasive allograft method for anatomic reconstruction of the deltoid ligament [8,9]. Early results have been promising in using this technique for tibiotalar joint-sparing reconstruction of stage IV-A deformity.

With the rigid IV-B patient, pantalar or tibiotalocalcaneal arthrodesis is recommended. Papa and Myerson [33] described the results of 21 patients who had a mean age of 45 years undergoing unilateral pantalar (8 patients) or tibiotalocalcaneal (13 patients) arthrodesis. Although the indications were for post-traumatic arthritis and not stage IV posterior tibial tendon rupture, the study does shed light on the long-term results of hindfoot arthrodesis. At a mean follow-up of 32 months, there were three nonunions, two wound complications, and an 81% satisfaction rate. Compared with patients undergoing pantalar arthrodesis, those undergoing tibiotalocalcaneal arthrodesis had higher mobility and function. Many if not all patients who have stage IV-B disease who undergo such fusion procedures have increased function and decreased pain compared with those who are treated nonoperatively.

Summary

With time we have become aware of a greater variety of presentations of PTT pathology and thus have recognized the deficiencies associated with the initial generic three-stage PTTR classification. This newly proposed
classification system should not only improve the clinician’s awareness and discrimination of the spectrum of disorders associated with PTT rupture, but also help in planning the proper treatment.

References


