FOOT & ANKLE INTERNATIONAL

Current Concept Review: Acquired Adult Flatfoot Deformity

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INTRODUCTION

Acquired adult flatfoot deformity (AAFD) is a common and often debilitating chronic foot and ankle condition. AAFD is characterized by flattening of the medial longitudinal arch and dysfunction of the posteromedial soft tissues, including the posterior tibial tendon. Key[46] described a chronic partial rupture of the posterior tibial tendon in 1953. Further descriptions of chronic posterior tibial tendon pathology began to identify an association between posterior tibial tendon dysfunction (PTTD) and flatfoot deformity.[24,45,88] Initially, the condition was called PTTD; however, more recently it has become known as AAFD in recognition that the pathology encompasses more than just the posterior tibial tendon.

The purpose of this review was to describe the key elements of AAFD and to outline treatment options based on the peer-reviewed literature. In keeping with the trend toward evidence-based practice, the level of evidence supporting each clinical research study has been reviewed. The idea behind assessing the level of evidence is that while each study constitutes evidence, some studies by virtue of their design are more persuasive than others.[89] Recently, the AAOS developed the Level of Evidence table and Grades of Recommendation for the evaluation of studies and providing recommendations (Table 1).[4–9]

ANATOMY AND BIOMECHANICS

The posterior tibial tendon, spring ligament, and portions of deltoid ligament are commonly abnormal in patients with AAFD. The posterior tibial muscle or tendon originates from the posterior tibia, fibula, and interosseous membrane. The tendon runs posterior to the medial malleolus, arches plantarward and inserts into the navicular tuberosity with multiple insertions spread out across the plantar aspect of the midfoot.[2] The malleolus serves to change the direction of pull on the tendon. The main blood supply to the tendon originates from the posterior tibial artery. Anatomic dissections have demonstrated a hypovascular zone in the retromalleolar region,[65] with fibrocartilagenous changes in tendon structure identified in the retromalleolar region.[64] In patients with AAFD, the abnormal tendon region demonstrated marked changes in both the molecular composition of matrix collagens and the structural organization of the tendon.[25] Tendon changes are thought to occur because the tendon is subjected to repetitive microtrauma and has a compromised repair response because of the limited vascularity.

The spring ligament (calcaneonavicular ligament) cradles the plantar medial aspect of the talar head extending from the anterior margin of the sustentaculum tali to the plantar medial aspect of the navicular. The spring ligament is composed of two main components, the superomedial and the inferior calcaneonavicular ligament. The superomedial calcaneonavicular ligament lies medial to the talar head and blends with the deltoid ligament.[16] This component of the spring ligament is commonly noted to have attenuation or gross tears in patients with AAFD.[22] The deltoid ligament has multiple components, with the distal aspect of this ligament blending into the spring ligament and talonavicular joint capsule. These structures are subjected to repetitive stress during midstance. Complex interplay exists between the ankle joint, the subtalar joint, and the transverse tarsal joint, which is composed of the talonavicular joint and the calcaneocuboid joint. The subtalar joint and the transverse tarsal joints move as a unit and together form the triple joint complex.

The major phases of gait are stance and swing. The stance phase includes heel strike, midstance, heel-rise, and toe-off. A complex interplay exists between heel position and relative rigidity of the transverse tarsal joint to allow physiologic advantageous positions that allow the foot to accommodate most any terrain. During the heel strike phase, the hindfoot is inverted. From heel strike to midstance, the posterior tibial

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Table 1: Level of Evidence and Grades of Recommendation

Level of evidence
- Level I: High quality prospective randomized clinical trial
- Level II: Prospective comparative study
- Level III: Retrospective case control study
- Level IV: Case series
- Level V: Expert Opinion

Grades of recommendation given to various treatment option based on level of evidence supporting that treatment
- Grade A: Treatment options are supported by strong evidence (consistent with level I or II studies)
- Grade B: Treatment options are supported by fair evidence (consistently positive level III or IV studies)
- Grade C: Treatment options are supported by conflicting evidence (level IV studies)
- Grade I: Insufficient evidence exists to make a recommendation

muscle is relaxed allowing the hindfoot to reach a valgus position and the midfoot to abduct through the transverse tarsal joints. As the heel moves into a valgus position, the axes of the talonavicular and calcaneocuboid joints become parallel, allowing motion through the transverse tarsal joint.[63] The valgus position helps the foot absorb shock during midstance. To create a rigid lever before the heel-rise phase of gait, the posterior tibial muscle contracts. This contraction helps reduce the abduction of the midfoot on the hindfoot and moves the heel out of valgus and into a neutral or inverted position. In doing so, divergent axes are created in the talonavicular and calcaneocuboid joints thereby locking the transverse tarsal joint. With the foot now functioning as a rigid lever, the gastrocsoleus can act to move the body forward. Patients with AAFD and posterior tibial tendon insufficiency are unable to reduce the hindfoot valgus and midfoot abduction. Therefore, they cannot obtain a rigid lever before the heel-rise phase of gait. Subsequent gastrocnemius-soleus contraction further weakens the posteromedial soft-tissue structures, exacerbating the pes planovalgus deformity.

In patients with a flatfoot deformity, the heel is in increased valgus and the midfoot is excessively abducted on the hindfoot. This creates an increased load on the posteromedial structures including the posterior tibial tendon. With time, and repetitive loading these structures can degenerate and eventually decompensate, leading to the classic symptoms of AAFD. The altered hindfoot and midfoot joint relationship secondary to soft-tissue incompetency ultimately brings the patient's symptoms to require medical help.

A few theories exist as to why certain people have flatfeet and others do not. One suggestion has been that excess motion of the medial column leads to forefoot varus and produces a compensatory hindfoot valgus.[36,54] The heel, the plantar medial forefoot, and the plantar lateral forefoot are the three major weightbearing regions of the foot during midstance.[15] The medial column consists of the talus, navicular, cuneiforms, and the first metatarsal. Extra motion in the medial column allows for an increased dorsal position of the first ray. This extra medial column motion is thought to contribute to flattening of the medial longitudinal arch and a compensatory hindfoot valgus. Another theory is that a short Achilles tendon or contracted gastrocnemius muscle causes a compensatory flattening of the medial longitudinal arch.[31] Maximal dorsiflexion of the foot is required at the heel rise phase of gait. If a short Achilles tendon or tight gastrocnemius muscle does not allow the foot to reach neutral dorsiflexion with the talonavicular joint reduced, this joint will remain subluxated, placing increased stress on the posteromedial soft-tissue structures.

CLINICAL MANIFESTATIONS

Patients with AAFD typically give a history of a flatfoot deformity that has become painful and more pronounced. Patients commonly present with a gradual onset of medial hindfoot pain. An acute, relatively nontraumatic precipitating episode is reported by some patients. Pain is typically localized to the posteromedial hindfoot distal to the medial malleolus. Pain secondary to lateral impingement of the fibula on the calcaneus also may be present in the lateral hindfoot near the sinus tarsi.[49] Symptoms usually are aggravated by standing and walking. In addition to pain, patients often note dysfunction in their gait. They are unable to run and note difficulty taking a longer stride as well as the inability to push off and raise their heel. This push-off weakness occurs with altered foot biomechanics secondary to the foot's inability to lock the transverse tarsal joints and achieve a rigid foot lever arm.

A history of inflammatory arthritides should be ruled out as a possible risk factor for AAFD.[57] Excessive weight also appears to increase the rate of developing AAFD most likely because of increased repetitive load absorbed by the posteromedial soft tissues.[37] A family history of flatfeet also may be a risk factor.

The physical examination is characterized by a flatfoot deformity that is more pronounced on the affected side. This deformity consists of flattening of the medial longitudinal arch, hindfoot valgus, and abduction of the midfoot on the hindfoot. This abduction allows relatively more toes to be seen when standing behind the patient leading to the "too many toes" sign which is characteristic of this condition.[41,42] Gait assessment typically reveals a somewhat antalgic gait pattern with a decreased stride length. Patients have a flatfooted heel-toe progression and a poor or absent heel rise.

Palpation usually reveals tenderness along the distal aspect of the posterior tibial tendon from the medial malleolus to the navicular tuberosity; however, tenderness to palpation proximally along the musculotendinous junction of the posterior tibial muscle also may be present. Additional areas of tenderness may be noted in the sinus tarsi and subfibular region because of impingement, particularly late in the clinical course, and may be more annoying to the patient than posteromedial symptoms.[49] In addition, findings suggestive of plantar fasciitis and tarsal tunnel syndrome may be present.[47]

Range of motion should be assessed carefully. In the earlier stages of the condition, the subtalar and transverse tarsal joints are supple with a full range of motion. As the condition progresses, these joints can lose motion and may eventually become fixed. In addition, the forefoot position should be assessed relative to the neutrally positioned hindfoot. A flexible or fixed forefoot varus will lead to a flatfoot deformity with a compensatory hindfoot valgus.

Assessment of ankle dorsiflexion typically demonstrates an equinus contracture when measured using the technique described by Bordelon.[3] He emphasized reducing the talonavicular joint and keeping the knee straight to reproduce the foot position at the heel-rise phase of gait. If the hindfoot is maintained in a deformed valgus alignment with a contracture of Achilles tendon, a false impression of dorsiflexion beyond neutral will be possible. However, correction of hindfoot to neutral position will allow the true extent of equinus contracture become apparent. A neurovascular examination should also be performed. Sensory and vascular status usually is normal and symmetric to the contralateral side.

Manual muscle testing of the posterior tibial tendon, even with plantarflexion and inversion, may not demonstrate the full deficit of the posterior tibial tendon secondary to some contribution of anterior tibial tendon to inversion. However, weakness of the posterior tibial muscle is characteristic of AAFD. This manifests in the patient's inability to perform a single-leg heel rise. The single-heel rise test is a critical clinical test in evaluating AAFD. The inability to stand on one foot and lift the heel fully off the ground with inversion of the hindfoot is indicative of posterior tibial tendon insufficiency. Another subtle sign of AAFD is the hindfoot remaining in valgus even when the patient can perform a heel rise.

IMAGING STUDIES

An accurate diagnosis of AAFD with posterior tibial tendon insufficiency can be made based entirely upon the clinical examination. However, full weightbearing radiographs of the foot are helpful in determining the severity of the condition by demonstrating the extent of alignment abnormalities and assessing evidence of hindfoot arthritis. Nonweightbearing radiographs fail to provide any valid information on the alignment status of the foot. The extent of the deformity can be assessed by reviewing a weightbearing anteroposterior and lateral radiograph. On the lateral view, the angle formed by the intersection of the long axis of the talus and the first metatarsal is known as the lateral talometatarsal angle or Meary's angle. In a neutral foot these two lines are essentially parallel. With loss of the medial longitudinal arch and abduction of the midfoot the lateral talometatarsal angle increases.[26] On the weightbearing anteroposterior radiographs, the talonavicular coverage angle can be used to assess the extent of midfoot abduction.[69] This angle is formed by drawing a line from the edges of the articular surface of the talar head and another line from the edges of the articular surface of the navicular. An angle of more than 10 degrees represents increased midfoot abduction. Other imaging modalities can be helpful if a specific clinical question needs to be answered. A weightbearing ankle series is essential if tibiotalar asymmetry or ankle arthritis is suspected.

Advanced radiographic studies, such as MRI, CT, and ultrasonography can provide extra and sometimes helpful information. However, in typical cases of AAFD, an accurate diagnosis can be made based on clinical examination and weightbearing radiographs of the foot. MRI provides an accurate assessment of the status of the soft tissues, including the posterior tibial tendon, the spring ligament, deltoid ligament, and even the functional status of a muscle (i.e. fatty replacement versus normal muscle signal).[83] However, because an MRI provides a detailed assessment of bone and soft-tissues within the foot, abnormal findings often are identified that are not clinically relevant. A CT scan can provide accurate information on the status of the bones and joints allowing tarsal coalitions to be diagnosed and evidence of hindfoot arthritis to be assessed. In addition, some studies have shown that ultrasound can provide an accurate assessment of the status of the posterior tibial tendon.[23] While advanced radiographic studies can provide additional information, their routine clinical use has not been advocated in the diagnosis of AAFD. CT, particularly in the coronal plane, is indicated if middle facet tarsal coalition is suspected as the etiology of the planovalgus deformity.

CLASSIFICATION

AAFD has been classified into four stages. Johnson and Strom[42] described stages 1–3, with Myerson[53] adding a fourth stage. Stage I consists of painful tenosynovitis of the posterior tibial tendon; however, the tendon itself is of normal length and function. Stage II consists of a flatfoot deformity with pain and dysfunction of the posterior tibial tendon. Patients have normal hindfoot motion but are unable to perform a single-leg heel rise. Stage III also includes dysfunction of the posterior tibial tendon. However, in this stage the hindfoot joints are stiff and may be arthritic. Stage IV consists of a stage III deformity with evidence

of associated tibiotalar asymmetry because of the prolonged hindfoot valgus deformity.

This classification system provides an organized way to think about the spectrum of pathology in AAFD. However, it has not been subjected to interobserver or intraobserver reliability testing. Problems underlying the reliability of this classification system include difficulty clearly differentiating between the various stages. For example, is a markedly flattened medial longitudinal arch with functional posterior tibial tendon a stage I or a stage II deformity? Alternatively, does a small amount of stiffness in the hindfoot joint mean that a patient has a stage III deformity? The difficulties with this classification system's reliability create a problem because many research studies assess patients with a certain stage deformity. For example, a number of studies look at the treatment options for stage II deformities, yet we have no way of knowing whether each study group of stage II patients is the same from one study to another.[10,20,28,56,68,84]

NONOPERATIVE MANAGEMENT

Nonoperative management focuses on improving a patient's symptoms, usually by attempting to decrease the forces going through the posteromedial hindfoot. This can be done by encouraging weight loss, improving footwear, decreasing repetitive loading, and other activity modifications. The use of nonsteroidal anti-inflammatory medication may help provide some symptomatic relief of pain. Initially, patients with Stage I or II AAFD may be immobilized with short-leg cast or CAM walker, followed by a specific orthosis.

Orthoses have been recommended in an effort to improve symptoms in patients with painful flatfeet.[1,87] The goal of orthoses in stage II AAFD is to support the medial longitudinal arch and decrease the hindfoot valgus. Custom orthoses with the hindfoot casted in a neutral position have been recommended for flexible flatfoot deformities. The University of California Biomechanics Laboratory (UCBL) semirigid orthosis was designed to control the major elements of the flatfoot deformity by supporting the medial longitudinal arch and minimizing the hindfoot valgus.[52] Imhauser et al.,[38] in a biomechanical cadaver study, assessed the efficacy of various orthotic devices in controlling flatfoot deformity. The UCBL orthoses has demonstrated the ability to partially correct both the arch and the hindfoot deformity.[32,38] However, the force required to control the hindfoot position is considerable. Therefore, some patients may find a rigid orthosis to be uncomfortable and prefer softer orthoses. For patients with a fixed foot deformity in-situ positioning of the foot is recommended when casting for an orthosis.[87] Custom-molded AFOs such as the Arizona brace are designed to provide increased ankle stability and have been used to help manage AAFD.[1] However, ankle bracing does not restore the angular orientation of the hindfoot.[38] Other ankle braces, such as a double upright brace with an inside (medial) T-strap, may function in a similar manner by sharing some of the load that would otherwise be transferred to the posteromedial structures.

OPERATIVE TREATMENT

A wide variety of operative treatments have been reported for AAFD with PTTD. Stage I PTTD, which rarely requires operative management, has been treated with debridement and immobilization. Good results have been reported in two retrospective reviews (level IV evidence).[51,75] At this time, it is not possible to make a specific treatment recommendation for or against the operative management of stage I AAFD based on the published literature (Grade I). Debridement of the posterior tibial tendon is not likely to be successful when a pronounced deformity is present. However, tenosynovectomy does offer the advantage of a relatively short recovery time in patients with stage I AAFD in whom nonoperative treatment has failed and can provide potentially marked pain relief in patients with inflammatory arthritides.

The operative management of stage II AAFD has changed dramatically in the past decade. Before 1980, many painful foot deformities were treated with a triple arthrodesis after nonoperative management failed. Beginning around 1980, operative procedures were developed to augment or substitute for an incompetent posterior tibial tendon, most frequently using the flexor digitorum longus muscle-tendon unit.[21,41,42,50] These treatments provided initial good results. However, these early procedures involving tendon transfer augmentation without bony realignment failed within a few years because the underlying deforming forces that had caused the original breakdown had not been treated.[62] Failure of the soft tissue was often treated with a triple arthrodesis.[39,44,48,86] Throughout the 1990s, joint sparing procedures that attempted to treat the underlying bony deformity while still preserving the hindfoot joints became increasingly popular.[10,20,28,56,68,84] Today, operative management of AAFD rarely involves an isolated procedure but rather some combination of bony and soft-tissue procedures done during a single procedure (Table 2A and B).[33]

A variety of soft-tissue procedures have been described as components of a flatfoot reconstructive procedure. Augmentation of the incompetent posterior tibial tendon with the flexor digitorum longus (FDL) commonly is performed. The goal of this augmentation is to restore the dynamic function of the posterior tibial tendon. MRI studies suggest that recovery of posterior tibial muscle function can occur after augmentation. Postoperatively, while fatty degeneration of the posterior tibial tendon was found in all patients, the fatty degeneration was decreased with increasing strength and muscular size on postoperative MRI.[80] Some surgeons prefer to use the flexor hallucis longus because of its increased strength relative to the flexor digitorum longus.[68] However,

Table 2A: Soft-Tissue Procedures

Procedure	Rationale
FDL augmentation of PTT	FDL provides dynamic function similar to the PTT
FHL augmentation of PTT	Similar to FDL but with a stronger tendon
P. Brevis augmentation of PTT	May be used in place of FDL when this tendon is too weak or damaged
Spring ligament repair/reconstruction	Tightens up attenuated superomedial band of the spring ligament
Deltoid ligament repair	Distal aspect of the deltoid ligament blends into the spring ligament and is often attenuated
Gastrocnemius recession	Corrects an equinus contraction that is isolated to the gastrocnemius muscle
Percutanteous Achilles tendon lengthening	Corrects an equinus contracture that involves both the gastrocnemius and soleus
Sinus tarsi implant (subtalar arthroereises)	Physically blocks eversion of the calcaneus in the sinus tarsi

Table 2B: Bony Procedures

Procedures	Rationale
Medial displacement calcaneal osteotomy	Helps correct hindfoot valgus and medializes the pull of the Achilles tendon
Lateral column lengthening	
Calcaneocuboid distraction arthrodesis	Lengthens the lateral column of the foot producing increased adduction of the mid and forefoot
Evans procedure (Anterior calcaneal osteotomy)	Lengthens the lateral column while preserving the function of the calcaneocuboid joint
Medial column procedures	
Medial cuneiform osteotomy	Corrects a flexible or fixed forefoot varus deformity
Navicular-cuneiform arthrodesis and/or 1st tarsometatarsal arthrodesis	Helps stabilize medial column hypermobility that can lead to a flexible forefoot varus deformity
Hindfoot joint-sacrificing procedures	
Subtalar arthrodesis	Stabilizes hindfoot, helps correct hindfoot valgus, reduces pain from arthritic subtalar joint
Double arthrodesis	Permanent reduction and stabilization of the transverse tarsal joint while retaining some subtalar motion
Triple arthrodesis	Permanent reduction and stabilization of hindfoot and midfoot position

concerns that it crosses the neurovascular bundle may limit its popularity. Peroneus brevis augmentation also has been described as a viable option when the flexor digitorum longus tendon is incompetent or too small.[73] Repair or reconstruction of the spring ligament must be done in an attempt to restore the static constraint of this often attenuated ligament.[22] Correction of an equinus contracture with either a percutaneous Achilles tendon lengthening or gastrocnemius recession commonly is performed.[33] A gastrocnemius recession is chosen if the equinus contracture is isolated to the gastrocnemius. A sinus tarsi implant (subtalar arthroereisis) has been used to limit the motion of the subtalar joint by blocking rotation of the anterolateral talar body into the sinus tarsi.[81,82] This can lead to improved foot position without sacrificing any of the hindfoot joints or performing a major bony procedure.

Numerous bony procedures have been described to treat the underlying flatfoot deformity and protect the soft-tissue reconstruction (Table 2, B). AAFD can include a wide spectrum of pathologic conditions. Unfortunately, a single procedure cannot properly treat all deformities, and the surgeon must have an understanding of all variations and treatment options to properly treat all facets based upon clinical examinations and radiographs. A medializing calcaneal osteotomy is commonly performed in an attempt to reduce the hindfoot valgus.[10,20,28,56,68,84] A lateral column lengthening, using a tricortical graft in the anterior calcaneus (Evans procedure) or by a calcaneocuboid distraction arthrodesis, has been shown to help correct the abduction of the midfoot and forefoot.[18]

Stabilizing procedures of the medial column, such as a plantarflexion opening wedge medial cuneiform osteotomy or a naviculocuneiform or first tarsometatarsal arthrodesis, have been described.[11,15,35,36] The goal of these procedures is

to correct the residual forefoot varus deformity and compensatory hindfoot valgus by plantarflexion realignment of the medial column of the forefoot. Hindfoot joint-sacrificing procedures, such as a subtalar arthrodesis[13,40] and double or triple arthodesis,[39,44,48,86] are still done for stage II flatfoot deformity by some surgeons, but, in general, these procedures are used as salvage operations.

Medial Calcaneal Osteotomy and Posterior Tibial Tendon Augmentation

A medializing calcaneal osteotomy with tendon transfer augmentation of the incompetent posterior tibial tendon is the most common combination of procedures used to treat stage II AAFD.[33] Variations on this combination of procedures are common and include adding a spring ligament repair, using another tendon for the augmentation, and adding a procedure to correct an equinus contracture. A medializing calcaneal osteotomy is commonly done in an attempt to reduce the hindfoot valgus,[10,20,28,56,68,84] and is designed to protect the tendon transfer by correcting the hindfoot valgus, reducing the arch strain, and improving the inverting capacity of the gastrocsoleus.[59,61,74]

Brodsky et al.[10] performed a prospective gait analysis on 12 patients undergoing reconstructive surgery using a medial displacement calcaneal osteotomy, flexor digitorum longus transfer to the navicular tuberosity, and spring ligament repair.[10] They found a statistically significant improvement in cadence, stride length, and ankle push-off (level II evidence). Myerson et al.,[53] Fayzi et al.,[20] Wacker et al.,[84] Guyton et al.[28], and Sammarco and Hockenbury each published retrospective reviews of this procedure demonstrating a high rate of successful results with short to intermediate follow-up.[20,28,56,68,84] These consistently positive level IV reviews constitute fair evidence (grade B) to support recommending a medial displacement calcaneal osteotomy and a posterior tibial tendon augmentation in patients with stage II AAFD in whom nonoperative management has failed.

The benefits of a medializing calcaneal osteotomy in correcting AAFD are accepted. However, questions do exist regarding the effect of this procedure on lateral foot pain and plantar pressures. Cadaver studies have demonstrated the usefulness of medializing calcaneal osteotomy in off-loading the plantar surface of the first and second metatarsal heads but with a concomitant increase in peak pressure over the lateral forefoot and heel.[29,30] Superior translation of the Achilles insertion may relieve this problem.

Lateral Column Lengthening and Posterior Tibial Tendon Augmentation

Lateral column lengthening using a tricortical graft was originally described as a treatment for flatfoot deformity in pediatric patients.[19] The lengthening is performed either by a calcaneocuboid arthrodesis or an anterior calcaneal osteotomy. Lengthening the lateral column corrects the deformity by adducting and plantarflexing the midfoot around the talar head.[18] Recent level IV studies suggest that lateral column lengthening combined with tendon transfer to augment the incompetent posterior tibial tendon can treat stage II AAFD with satisfactory results.[34,79] However, complications were relatively high, including abnormal gait (forefoot varus with the patient walking on the lateral border of foot), lateral overload, graft failure, nonunion of the structural graft, and painful hardware.[14,76,78,79] Based on the existing literature, insufficient evidence exists to make a specific treatment recommendation (grade I) for or against the use of lateral column lengthening in the surgical management of stage II AAFD.

Double Calcaneal Osteotomy and Posterior Tibial Tendon Augmentation

Double calcaneal osteotomies have evolved to treat all aspects of the deformities of stage II AAFD. The double osteotomy includes a medial displacement calcaneal osteotomy and an anterior calcaneal osteotomy using a tricortical graft.[55,66] The medial displacement calcaneal osteotomy treats the hindfoot valgus and the lateral column lengthening provides improved correction of the abduction deformity. This combination of procedures creates a more anatomic correction of the deformity than an isolated medial calcaneal osteotomy. This may further decrease the load through the posteromedial structures, including the augmented posterior tibial tendon. One retrospective review (level IV evidence) demonstrated a high patient satisfaction rate with intermediate-term follow-up and distinctive improvements in radiographic parameters, particularly with respect to the position of the midfoot and forefoot.[55] Complications included the need to remove painful hardware, evidence of calcaneocuboid arthritis, and lateral foot overload.[78] Based on the present literature, insufficient evidence exists to make a specific treatment recommendation (grade I) for or against the use of double calcaneal osteotomies in the operative management of stage II AAFD.

Subtalar Arthroereisis

The use of a sinus tarsi plug or an implant to restrict eversion of the subtalar joint is known as subtalar arthroeresis.[58,82] This type of procedure was originally developed as an isolated pediatric procedure, which is a relatively straightforward way to treat the flatfoot deformity while preserving the hindfoot joints. The sinus tarsi implant itself does not treat the etiology of the flatfoot deformity but rather serves to block or redirect the anterolateral talar body thereby limiting hindfoot eversion. Most clinical studies assessing subtalar arthroereisis have been done in children for a variety of different flatfoot conditions, including neuromuscular disorders and flexible flatfoot deformities.[71,81] A sinus tarsi implant combined with repair of the posterior tibial tendon has been used to treat adults with stage II AAFD (level IV study).[82] Satisfactory preliminary results were reported in this small retrospective review. Complications have been reported with sinus tarsi implants including persistent sinus tarsi pain, foreign body reaction, implant failure,

and osteonecrosis of the talus.[70,71] The limited research on subtalar arthroereisis in adult patients with AAFD means that there is insufficient evidence (grade I) to make a recommendation for or against this treatment approach.

Hindfoot Arthrodeses

Joint-sacrificing procedures such as subtalar arthrodesis, double arthrodesis, and triple arthrodesis are still used by some surgeons as their primary procedure to correct a stage II AAFD.[13,33,40] Hindfoot arthrodeses limit motion and force the remaining joint or joints to absorb more force. In the case of a triple arthrodesis, this leads to increased wear in the ankle joint and a higher rate of degenerative ankle arthritis.[67] For this reason, hindfoot arthrodeses in patients with AAFD are typically reserved for salvage operations (failed soft tissue procedure) and patients who have evidence of hindfoot arthritis (stage III deformities).[12] However, some surgeons may choose to use a subtalar fusion with PTT augmentation or triple arthrodesis in patients who have an inflammatory arthritis with poor soft tissue integrity, nonreconstructable deformities, such as massive spring ligament tears, or are excessively overweight or elderly, believing that hindfoot arthrodesis offers a more predictable short-term result.

One critical issue is the relative nonunion rate of different joints (talonavicular, calcaneocuboid, and subtalar joint) in a triple arthrodesis. Because of the high rate of nonunion, the use of specific adjuncts (i.e. bone graft) to high-risk arthrodesis with meticulous technique has been emphasized.[77]

STAGE IV TIBIOTALAR TILT

Long-standing AAFD with attenuation of the deltoid ligament leads to tibiotalar tilt asymmetry that can be identified on a weightbearing ankle series. Traditional operative treatment of a stage IV AAFD is a tibiotalocalcaneal or pantalar arthrodesis. Correction of the bony alignment abnormality combined with a deltoid ligament reconstruction has been described.[17] However, to date all operative treatments of stage IV AAFD have insufficient evidence (grade I) to make specific treatment recommendations.

COMPLICATIONS

Complications in patients undergoing operative reconstruction of an AAFD are common. These include general complications, such as infection, deep vein thrombosis (DVT), nonunion, painful hardware, wound healing problems, and neurologic injury. Procedure-specific complications may include over or under-correction of the deformity and failure of tendon transfer or soft-tissue repair.

Infection rates vary, depending on the extent of the surgery and the presence of patient risk factors such as diabetes and previous surgery. Timely preoperative prophylactic antibiotics, skin preparation with an alcohol-based solution, and meticulous operative technique may reduce the overall infection rates.[43,60] Deep vein thrombosis and pulmonary embolism are relatively uncommon in patients undergoing foot surgery.[72,85] Risk factors for developing thromboembolic disease may include previous deep vein thrombosis, prolonged immobilization, older age, smoking, obesity, birth control pill use, air flight, and a positive family history of blood clotting. In patients with known risk factors, the surgeon should consider whether treatment with prophylactic anticoagulants is warranted. Nonunions are relatively uncommon in medial displacement calcaneal osteotomies but are more common in anterior calcaneal lengthening osteotomies.[14,76] Patients who smoke, take steroids, or bear weight too early postoperatively have a higher rate of nonunion. Appropriate fixation techniques and adequate postoperative immobilization are necessary to optimize the rate of bony union. Painful hardware is a common complaint and many patients eventually require hardware removal.[55] Wound healing problems have been reported in up one third of patients undergoing a major flatfoot reconstruction.[12] These wound healing problems appear to be more prevalent in the anterolateral hindfoot. The anterolateral incision used for a lateral column lengthening, subtalar arthrodesis, or sinus tarsi procedure often is under tension after correction of a flatfoot deformity. Neurologic structures are at risk during flatfoot reconstruction. Injuries to the sural nerve can occur during a calcaneal osteotomy, lateral column lengthening, Achilles tendon lengthening, or gastrocnemius recession. Medial displacement osteotomy may place the lateral calcaneal branch of the sural nerve, branches of the lateral plantar nerve, and branches of the posterior tibial artery at risk.[27] Care with soft-tissue dissection and completing osteotomies with an osteotome in a controlled manner may decrease the rate of neurologic injury.

SUMMARY

AAFD has clinical manifestations ranging from a flexible flatfoot with a painful but functional posterior tibial tendinopathy to a complete rupture with an associated fixed painful deformity and evidence of ankle arthritis. Treatment options are varied and depend on the severity of the condition. Treatment options published in the peer-reviewed literature are mostly retrospective reviews constituting level IV evidence. Concepts that appear to be guiding the approach to treating AAFD include:

1. Pathology in AAFD is more than isolated dysfunction of the posterior tibial tendon.
2. Soft-tissues such as the posterior tibial tendon fail from degeneration likely because of excess repetitive loading.

3. Once the posterior tibial tendon has failed, nonoperative treatment may be helpful in improving a patient's symptoms but is unlikely to return the patient to presymptomatic clinical condition.
4. Isolated soft-tissue procedures designed to compensate for a dysfunctional posterior tibial tendon have a high incidence of recurrent deformity if they are not combined with a bony procedure that fundamentally changes the alignment of the foot to protect the new reconstruction.
5. Reconstructive surgery should attempt to preserve normal hindfoot motion to protect the surrounding joints from increased load.
6. Multiple osteotomies can improve alignment of the foot when compared to an isolated medial calcaneal osteotomy. However, the more extensive surgery must be balanced against a potentially higher complication rate.
7. Hindfoot fusions (subtalar or triple) in a patient with AAFD are typically reserved for salvage operation and patients who have evidence of hindfoot arthritis. Other relative indications include inflammatory arthritis, significant soft-tissue damage, obesity, and older age.
8. Recovery from AAFD reconstructive surgery is prolonged and an eventual return to asymptomatic unrestricted activities is unpredictable.

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