Current Concepts in the Management of Adult-Acquired Flatfoot Deformity

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Abstract: Adult-acquired flatfoot deformity refers to the progressive collapse of the medial longitudinal arch of the foot. It is most commonly associated with Posterior Tibial Tendon Dysfunction, and is known to cause significant deformity and debilitation if left untreated. The purpose of this article is to review current concepts on the conservative and surgical management of adult-acquired flatfoot, as well as provide recommendations on treatment of the deformity based on clinical staging.

Key words: Flatfoot, Adult-Acquired Flatfoot, PTTD, Flexible Flatfoot, Equinus,

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Introduction

The term adult-acquired flatfoot deformity (AAFD) is defined as progressive collapse of the medical longitudinal arch, and gradual loss in strength of the Posterior Tibial Tendon typically seen after skeletal maturity is reached [1]. It is a deformity with multifaceted pathology involving posterior tibial tendon insufficiency combined with failure of the capsular and ligamentous structures of the foot. Posterior Tibial Tendon Dysfunction (PTTD) is the most common culprit, leading to progressive deformity and significant disability if left untreated.

Adult-Acquired Flatfoot is most commonly caused by dysfunction of the PT tendon attributed to degenerative and inflammatory causes. AAFD can also result from fractures and dislocations of the medial column, Lisfranc joints, and trauma to vital soft tissue structures such as the spring ligament complex and the plantar fascia. Tarsal coalitions, neurologic weakness, chronic steroid exposure, and degenerative arthritides such as Rheumatoid Arthritis are also
sources of flatfoot. Obesity, hypertension, and diabetes are also risk factors.[3]

Anatomy

The Posterior Tibial Tendon originates at the posterior aspect of the tibia, fibula, and interosseous membrane. The tendon courses to the medial aspect of the posterior talus, medial aspect of the talar neck, and the inferior surface of the spring ligament. The tendon has numerous insertions, with slips inserting at the navicular tuberosity, the inferior surface of the naviculo-cuneiform joint, the plantar aspect of the intermediate cuneiform, and the plantar aspect of the bases of the second, third, and 4th metatarsals.[3] The Posterior Tibial Artery is the main blood supply to the tendon, with variable supply from branches of the dorsalis pedis artery. According to Buchanann, there is a small area of hypovascularity 1 to 1.5cm distal to the medial malleolus, distally extending 14mm.[4] This may explain mechanical causes for tendon rupture and non-traumatic tears in patients with vascular compromise. The PT tendon is the most powerful inverter of the foot, and the most frequently affected dynamic stabilizer in adult acquired flatfoot deformity.[2] The Posterior Tibial tendon functions to adduct and plantarflex the navicular on the talar head, preventing medial longitudinal arch collapse. The tendon is also considered a stance phase muscle, and acts to invert the hindfoot, opposing the Peroneus Brevis Tendon. During midstance, the posterior tibial muscle produces subtalar joint inversion, creating a rigid lever for propulsion.[3] It also functions in ankle planarflexion and forefoot supination/adduction.

The triceps-surae complex includes the gastrocnemius, soleus, and plantaris muscles. All three muscles insert on the posterior one-third of the calcaneus, forming the Achilles Tendon. The gastrocnemius muscle is the most superficial muscle of the complex and originates the femoral condyles and the popliteal surface of the femur, and inserts on the middle one-third of the posterior surface of the calcaneus. The soleus muscle originates on the medial tibia and the posterior surface of the fibula, and inserts on the middle one-third of the posterior surface of the calcaneus, creating a conjoined tendon with the gastrocnemius muscle. The plantaris muscle is a very small structure deep to the lateral head of the gastrocnemius, whose tendon travels intermediate to the gastrocnemius and soleus muscles. It originates at the lateral femur, and inserts on the medial edge of the posterior one-third of the calcaneus, medial to the Achilles tendon. The triceps-surae complex functions as the primary plantarflexor of the ankle joint. The muscles are innervated by the tibial nerve, and receive its arterial supply from the sural artery, posterior tibial artery, and the peroneal artery.[3]

The Spring Ligament (Calcaneonavicular Ligament) complex consisting of superstriormedial and inferior bands that originates from the calcaneal middle facet and the sustentatulum tali. The ligament inserts on the plantar navicular tuberosity and the supero-medial side of the navicular articular margin.[4] The superomediaband and the inferomedial bands of the spring ligament are most commonly affected in PTTD. This ligament functions to provide support to the talar head, medial longitudinal arch, and the talonavicular joint. According to Deland, it is the most frequently affected static stabilizer in symptomatic adult-acquired flatfoot deformity.[5]

The Talo-navicular joint is an articulation of the talar head and the concave surface of the navicular with strong ligamentous support. The joint receives its blood supply from the dorsalis pedis artery, and the medial plantar artery. The TNJ is an important stabilizer of the medial longitudinal arch, as well as an important static stabilizer in adult acquired flatfoot deformity. According to Buchanan, The TNJ is often compared to the femoral head and acetabular articulation, and is referred to as the “acetabulum pedis”.[4]

Pathophysiology

Loss of Posterior Tibial Tendon function due to stretching or rupture of the PT tendon leads to loss of inversion strength, resulting in overpowering of the Peroneus Longus and Peroneus Brevis tendons. This leads to flattening of the medial longitudinal arch, forefoot abduction, and hindfoot valgus. According to Hansen, the primary mechanism of failure is the loss of dynamic arch support, followed by tension failure of static ligaments.[6] Biomechanically, the medial longitudinal arch progressively flattens, the ankle moves in equinus, with resultant forefoot abduction and heel valgus.

Forefoot supinatus is also common sequelae of AAFD. It is defined as a soft tissue adaptation of the
forefoot where the forefoot is inverted on the rearfoot. This occurs due to excessive calcaneal eversion, excessive inversion of the foot around the longitudinal midtarsal joint axis, and ankle equinus. According to Evans and Catanzariti, the level of forefoot supinatus depends on the amount of calcaneal eversion, the degree of adaptive muscle and osseous changes, and the time at which the problem has been present. Forefoot supinatus can imitate a forefoot varus, however the former is considered an acquired deformity, and the latter is defined as a congenital deformity.

Classification/Staging of Flatfoot Deformity

The Johnson and Strom classification system is the most commonly used classification scheme in the clinical diagnosis of PTTD. This classification combines clinical presentation and radiographic findings that serve as a guideline for surgical management. The classification has 4 stages and is summarized below:

**Stage 1—Tenosynovitis without Deformity**

The Posterior Tibial Tendon is inflamed or partially ruptured. The overall continuity of the tendon is maintained. No hindfoot valgus is appreciated, the “too many toes” sign is not present, and the patient is able to perform a single heel raise with evidence of hindfoot inversion. Clinically, patients may complain of medial ankle pain caused by tenosynovitis, and have pain to palpation of the tendon distal to the medial malleolus.

**Stage 2—Partially Ruptures PT Tendon with Flexible Flatfoot**

This stage is defined by the presence of PT tendon rupture with elongation and degeneration with hindfoot valgus. When the heel is reduced from valgus to neutral, there is evidence of forefoot supinatus. Patients are unable to perform a single heel raise test, and the “too many toes” sign”, is present. At this stage, the deformity remains flexible, and is reducible through passive inversion at the TN joint and hindfoot. Deland et. al added subsections to this stage as follows:

- Stage 2a—mild to moderate flexible deformity with minimal abduction through the TN joint with less than 30% talar head uncovering on AP radiographs.
- Stage 2b—Flexible deformity with greater than 30% talar head uncovering.

**Stage 3—Rigid Hindfoot Valgus**

Stage 3 is associated with advanced tendon rupture and deformity. There is rigid hindfoot valgus and rigid forefoot abduction not passively correctible beyond neutral. Midfoot abduction is also seen. Rigidity is present at the TN joint, the subtalar joint, and calcaneocuboid joint. Patients are unable to perform single and double heel rise tests, and the “too many toes” sign is present. Patients complain of lateral subfibular pain.

**Stage 4—Ankle Valgus**

This stage, added by Myerson, is a stage 3 rigid flatfoot characterized by chronic tendon rupture and medial ankle instability. Ankle valgus results from lateral talar tilt, and deltoid ligament insufficiency is seen. Subfibular pain seen in stage 3 is now converted to ankle pain. This stage is also divided into two stages:

- Stage 4a—Hindfoot valgus with flexible ankle valgus, without significant ankle arthritis.
- Stage 4b—Hindfoot valgus with rigid or flexible ankle valgus with significant arthritis.
Diagnosis

Physical Exam

Patients with AAFD typically present with medial pain and swelling along the course of the PT tendon, starting behind the medial malleolus. This signifies tendinitis and tenosynovitis of the tendon. The medial longitudinal arch appears collapsed with significant valgus deformity of the heel. Subfibular pain with palpation at pain signifies lateral impingement.

Ankle equinus is the major deforming force in AAFD, and is present due to tight gastrocnemius/soleus muscle complex. Gastrosoleus equinus is evaluated using the Silverskold test, which distinguishes gastrocnemius equinus from gastrocnemius-soleus equinus. Patients are diagnosed with gastrocnemius equinus if they are unable to dorsiflex less than 90 degrees with the knee extended, and gastroc-soleal equinus if they are unable to dorsiflex less than 90 degrees with the knee extended and flexed.[8]

Biomechanically, patients with AAFD display forefoot varus, first ray hypermobility, and metatarsalgia with transfer lesions. Patients present with peritalar subluxation, subfibular impingement, and calcaneal valgus.[9]. Patients are unable to perform a single or double heel rise test, and many elicit “too many toes sign.”

Radiographic Evaluation

The presence of AAFD is confirmed with standard weightbearing AP, MO, and Lateral x-rays. A calcaneal axial view is also used to evaluate hindfoot position, and ankle views are used to evaluate ankle valgus in stage 4 PTTD.

On AP radiographs, the talo-calcaneal angle increases, talar head uncovering is 50% or less, forefoot adductus angle decreases, and cuboid abduction angle increases.

MO views will demonstrate any type of calcaneal-navicular joint.

On Lateral views, talar declination angle increases, calcaneal inclination angle decreases, and the talo-calcaneal angle increases. There is evidence of midfoot sag, corresponding to the navicular-cuneiform joint. There is also an increase in the Talo-1st metatarsal angle.

Calcaneal axial views and long-leg calcaneal axial views show rearfoot eversion, decreased height of the sustentaculum tali, and increased superimposition of the lesser tarsus.

Weightbearing hindfoot alignment views will show frontal and transverse plane deformity, while highlighting the degree of ankle joint and tibial deformity, giving a global view of the entire flatfoot.

AP ankle weightbearing views show integrity of the Ankle Joint, varus or valgus deformity, and the degree of arthritis present. AP ankle views are important in evaluating the patient for stage 4 deformities.

Other Imaging Modalities

MRI can be used to evaluate the integrity of the PT tendon, the deltoid ligament, and the spring ligament. On a normal MRI, The PT is usually 2.3 times the size of Flexor Digitorum Longus. When tenosynovitis is present, the tendon is hypertrophic with edema surrounding the tendon on T2 images. When the tendon is ruptured, it will appear attenuated and elongated, appearing similar sized or smaller than...
FDL. If there is a complete tear of the PT it will appear flattened on MRI.

A study performed by Deland et al stated an 87% incidence of superomedial spring ligament tears on MRI in patients with known PTTD, and 74% incidence of inferior spring ligament attenuation [10]. A study by Orr and Nunley found MRI to reliably predict spring ligament abnormality in their patients, with standard foot and ankle MRI protocol [11]. These results show that MRI is essential in diagnosing soft tissue causes of AAFD.

According to Lever and Henessey, ultrasound has been shown to have equivalent accuracy to MRI for assessing the PT tendon [12]. On ultrasound the normal PT tendon is hyperechoic, and an abnormal tendon is seen as hypoechoic.

Treatment

Treatment is divided into conservative and surgical methods. Surgical management is typically correlated with the Johnson and Strom classification.

Conservative Management

Conservative management is aimed to address the deformity, while limiting pronation. Popular conservative measures include custom foot orthoses, changes in shoegear, immobilization, activity modification, and weight loss [13]. An ankle-foot orthoses can be used in patients with severe flatfoot. Anti-inflammatory medications can also be used, and physical therapy is ideal in patients with significant PTTD. Conservative therapy is best for elderly patients, or patients with medical conditions that prohibit surgical intervention.

Stage 1—According to Hockenbury, cast immobilization or CAM walker is recommended for tenosynovitis. Patients should be immobilized for 6-8 weeks, then re-evaluated. During the casting period, the patients can weightbear as tolerated [1].

Stage 2—Flexible deformities can be managed with custom orthotics, preferably UBCL orthotic, or ankle-foot orthosis. The UBCL orthotic will allow for triplanar control of the hindfoot, while providing stability to the midfoot and stability to the midtarsal joint [1].

Stage 3—Treatment for patients in this stage of AAFD is aimed at improving mobility, and decelerating the advancement of deformity. Patients with rigid hindfoot valgus are best benefitted with bracing using an AFO or Patellar Tendon Bearing Brace (PTB).

Stage 4—Treatment is similar to stage 3, and targets stabilization of rearfoot and hindfoot joints. Conservative measures include AFO or PTB bracing. Non-operative treatment is often unsuccessful in stage 4 deformities, and surgical intervention is usually required.

Surgical Management

Surgical management of AAFD is indicated when conservative measures have failed, or there is a progression of deformity. Surgical management can be divided into two categories: Treatment of flexible AAFD, and treatment of PTTD.

Surgical Management in Flexible AAFD—Treatment of flexible AAFD is dependent on clinical and radiographic findings, activity level, and degree of deformity. Procedures include soft tissue modification, displacement osteotomies, and arthroeresis. These procedures are typically selected based on planal dominance.

Soft Tissue Procedures—Patients with flexible flatfoot without deformity may benefit from soft tissue procedures such as the Kidner, Young Tenosuspension, Flexor Digitorum Longus transfer, and Spring Ligament repair. Since Equinus is one of the major deforming forces in AAFD, it is important to address gastroc-soleus contractures intraoperatively.

The Kidner procedure is primarily used in transverse plane deformities, and entails removal of an accessory navicular with advancement of the PT tendon plantarly into the navicular. The Kidner is used to correct deformities in the talo-navicular or naviculocuneiform joints, as well as forefoot supinatus.
The Young procedure is used in patients with flexible flatfoot with forefoot supinatus. This procedure routes the Tibialis Anterior Tendon through a keyhole in the navicular. The PT tendon is also advanced to the plantar aspect of the navicular.

According to Walters and Mendicino, FDL tendon transfer is used in patients with flexible flatfoot deformity with greater than 15 degrees of subtalar joint inversion. This procedure is an “in phase transfer” of the FDL tendon to the distal aspect PT tendon, and is adequate for augmentation of a diseased PT tendon. This procedure is usually not indicated as a single procedure, and is used in combination with the medial calcaneal displacement osteotomy. The transfer is not indicated in rigid flatfoot deformities, ruptured PT tendon, or in patients with symptomatic arthritis of the midfoot and hindfoot joints.

It is common practice to incorporate repair of the spring ligament in reconstructive efforts to correct the flexible flatfoot. The Peroneus Longus, Peroneus Brevis, and the Tibialis Anterior Tendon have been used to augment the ligament. In recent years, the Arthrex Internal Brace system has been used to repair the spring ligament in conjunction with other procedures such as the medial calcaneal displacement osteotomy and the opening wedge cuneiform osteotomy. The Internal Brace System utilizes Fiber Tape for the augmentation repair, and is comparable to tendon allograft repair of the spring ligament. This should not be performed as a single procedure.

**Calcaneal Osteotomies**—Kousogiannis discussed the first medial calcaneal displacement osteotomy (MCDO) in 1971 for treatment of valgus heel deformities. This procedure is a frontal plane osteotomy between the posterior subtalar joint and the Achilles Tendon, which allows for triplanar correction. The osteotomy is shifted 1cm medially underneath the weightbearing axis of the leg. The MCDO aids in decreasing valgus deformity of the heel, while increasing the supinatory arm of the Achilles tendon.

The Evans calcaneal osteotomy, described in 1975, is a form of lateral column lengthening used for correction of forefoot abduction in transverse plane deformity. The procedure entails an through and through lateral to medial osteotomy 1.5cm proximal to the calcaneocuboid joint posterior to the anterior process of the calcaneus. The cut is made perpendicular to the later wall of the calcaneus and weightbearing surface, then parallel to the calcaneocuboid joint. A tricortical trapezoidal wedge allograft is inserted into the osteotomy, which serves to lengthen the lateral column and adduct the forefoot. In the original article, Evans did not fixate the osteotomy, however, it is commonly fixated with a plate to provide stability to the graft, and prevent displacement.

The Cotton osteotomy is an opening wedge osteotomy through the medial cuneiform used to plantarflex the medial column. This procedure is used in conjunction with the other displacement osteotomies to correct forefoot supinatus. A structural allograft or autograft is used to pack the osteotomy and enhance correction. Fixation options include kirshner wires, screw, staple, or plate.

**Arthroeresis**—Subtalar joint arthroeresis is a minimally invasive procedure used in flexible flatfoot deformities with the intent to block subtalar joint pronation, and anterior rotation at the lateral talus process. The Maxwell-Brancheau arthroeresis is the most widely used implant, and is considered a self-locking wedge implant. It is a titanium implant with variable diameters measuring from 6mm to 12mm. The implant is a cannulated system inserted over a guide pin through the sins tarsi. The correctly sized implant should allow for 2-4 degrees of subtalar joint eversion. Post-operatively, the patient is weightbearing in a CAM walker for 2 to 3 weeks. This procedure is reversible, and can be removed at a later date if the patient experiences subtalar joint pain. Subtalar arthroeresis is not typically used as an isolated procedure, and there is controversy regarding its use in the adult population. A study performed by Zhu et al in 2015 endorses the use of subtalar arthroeresis as a treatment option for stage IIA and IIC AAFD. The study states that the procedure can be used alone to correct mild hindfoot valgus, and it can also be performed with a calcaneal osteotomy to gain more correction in severe stage II AAFD. A study by Needleman et al in 2006 used the MBA implant in 23 patients who had adult flexible flatfoot of various etiologies with an average follow-up of 44 months. He concluded that arthroeresis procedure to treat
adult acquired flexible flatfoot compares satisfactorily with joint-sparing hindfoot osseous procedures; the most commonly observed complication was sinus tarsi pain [19].

Surgical Management of Posterior Tibial Tendon Dysfunction—Surgical management of PTTD is warranted if the patient has failed all conservative treatment options. Procedure selection is dependent on the stage of PTTD and the degree of deformity.

Stage 1—Procedures involving direct repair of the tendon are indicated in stage 1 PTTD. Options include repair of the PT tendon sheath, tenosynovectomy, tendon debridement, and repair of longitudinal tears. Postoperatively, patients are placed in a short-leg cast for 3 weeks, and are casted for custom orthotics. Pomeroy et al states that FDL transfer should be considered when changes in the substance of the tendon are present, in order to supplement the function of the PT tendon. Tendo-Achilles lengthening or gastrocnemius recession should be considered if the patient has equinus [20]. According to Myerson, tendosynovectomy combined with a MCDO should be considered patients with stage 1 deformity who exhibit a partial PT tendon tear with 5 degrees or less of hindfoot valgus [8].

Stage 2—Surgical management of stage 2 deformities initially address equinus contracture, before addressing hindfoot and medial arch deformity. A gastrocnemius recession or Tendo-Achilles lengthening is performed based on the level of equinus determined by the Silverskold test. According to Cottom, addressing equinus contracture places the calcaneus in rectus position to enhance flatfoot reconstruction [21]. Once equinus is addressed, procedures are determined based on the degree of deformity and planal dominance.

In stage 2a deformities, MCDO combined with an FDL tendon transfer is the most commonly accepted combination of procedures. Chang states the MCDO effectively alters the biomechanical axis of the lower limb, reducing valgus influence on the hindfoot. The osteotomy also redirects the pull of the gastrocnemius-soleus muscle group medial to the axis of the subtalar joint. As a result, the Achilles tendon is positioned medially, increasing its varus pull on the hindfoot [22]. If forefoot supinatus is present, a cotton opening wedge osteotomy may be used to plantarflex the first ray if the first metatarsal cuneiform joint is stable and no hypermobility is present. If the first metatarsal cuneiform joint is arthritic or hypermobile, a lapdus arthrodesis can be used to correct forefoot supinatus.

Stage 2b deformities are treated with double calcaneal osteotomies combining the MCDO and Evans calcaneal osteotomy. FDL transfer and medial column procedures are also added if necessary. Vulcano and Deland also cite the addition of spring ligament repair to the double osteotomy in patients having lateral column lengthening, who do not gain efficient correction with addition of graft alone [2].

Postoperative management in stage 2 deformities consists of 2 weeks of non-weightbearing in a posterior splint, followed by a cast or removable boot for at least 6 weeks. Patient can begin range of motion exercises 2 weeks post op. Full weightbearing is achieved at 8-10 weeks post op upon evidence of radiographic healing [2].
Stage 3—Fixed deformity is treated with medial double arthrodesis of the talonavicular joint and the subtalar joint. Indications for the medial double arthrodesis include severe and non-reducible end-stage AAFD. Catanzariti and Adeleke also cite this procedure as an alternative to triple arthrodesis in patients with diabetes, rheumatoid arthritis and long-term steroid use. Advantages to a medial double arthrodesis include reduction in lateral wound complications, decreased incidence of non-union, and sparing of the calcaneocuboid joint. \(^{[23]}\) Adjunct procedures include gastrocnemius recession, and medial column stabilization as described above.

Triple arthrodesis is considered in stage 3 deformities with end-stage arthritis, and non-reducible deformity. The triple arthrodesis aims to establish a well-plantigrade foot and hindfoot realignment \(^{[24]}\). According to Vulcano and Deland, the triple arthrodesis is performed through a two medial and lateral incisions \(^{[2]}\). Myerson cites a single medial incision to access all 3 joints \(^{[8]}\). Typically the heel is fused in less than 5 degrees of valgus with the forefoot in neutral. Catanzariti et al recommends a MCDO if heel valgus is present after achieving a neutral forefoot and triple-joint complex \(^{[23,24]}\).

Medical column procedures such as the cotton osteotomy or lapidus arthrodesis may be added in patients with first ray elevatus. Postoperatively, patients are non-weight bearing for 6 to 8 weeks depending on rate of osseous fusion, followed by progressive weightbearing in a removable boot for 4 weeks \(^{[24]}\).

Stage 4—Deltoid ligament repair and ankle joint stabilization are key highlights in management of stage 4 PTTD. Treatment can be divided into ankle joint sparing versus destructive procedures.

Stage 4a deformities may be treated with ankle joint sparing procedures that correct ankle valgus with emphasis on deltoid ligament repair. According to Peterson and Hyer, flatfoot reconstructive procedures, such as medial double or triple...
arthrodesis should be achieved before repairing the deltoid ligament to ensure a stable plantigrade foot. Both the superficial and deep deltoid ligament complex must be repaired for adequate correction. Autograft or allograft reconstruction is cited in current literature as the standard for deltoid ligament repair, and can be accomplished using Achilles tendon, hamstring, or semitendinosis allografts. 

Deland et al describes repair using a peroneus longus autograft, which is passed through a tunnel in the talus and the medial malleolus to mimic fibers of the deep deltoid. Myerson describes deltoid repair using a hamstring allograft, with reconstruction technique similar to lateral ankle stabilization, and use of interference screws.

Stage 4b deformities are unable to be corrected with deltoid ligament repair alone, and are more amenable to joint destructive procedures. Deland et al states that total ankle replacements with discrectional deltoid ligament repair are indicated in patients who are non-diabetic with intact sensation, older than 60 years of age, and have less than 15 degrees of ankle valgus. Patients who are not candidates for total ankle replacement can be treated with ankle fusion or pantalar fusion, although pantalar fusions are considered a procedure of last resort. Deland states that the flatfoot can be corrected with either joint-sparing or joint destructive procedures based on the degree of flatfoot deformity.

Conclusion
AAFD is a complex deformity with multiple etiologies that is difficult to manage conservatively at the end stages. Prompt diagnosis and proper procedure selection will prevent the progression of deformity. Overall, treatment is selected based on staging, patient expectations, and flexible versus rigid deformities. Conservative management is ideal in patients with low demand and absence of significant deformity. Surgical management is warranted in patients with failed conservative treatment, with limitations in activities of daily living. The goals of treating AAFD should aim to correct valgus deformity and create a stable, plantigrade foot.

References


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