A Clinical Approach to Diagnose Flatfoot Deformity

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Abstract

Flatfoot is a complex disorder combining multiple static and dynamic deformities, associated with a collapsing medial longitudinal arch. The etiology of flatfoot is multifactorial and can be divided into two main groups; congenital and acquired groups. History, physical examination, and radiographs of the foot are used to establish and confirm the diagnosis. The staging system for flatfoot demonstrates the deformity and guides the appropriate treatment. This article will focus on etiologies, pathophysiology, and clinical approach to diagnose flatfoot deformity.

Keywords: Acquired adult flatfoot, Flatfoot, Pediatric flatfoot, Pes planus, Posterior tibial tendon dysfunction, Staging.

Journal of Foot and Ankle Surgery (Asia Pacific) (2021): 10.5005/jp-journals-10040-1149

Background

A flat foot (Pes planus) is a complex deformity illustrated by a flattening medial longitudinal arch of the foot.[1,2] In the pediatric population, two types of flatfoot deformity have been noted, i.e., flexible and rigid flatfoot. Flexible flatfoot deformity accounts for approximately 95% of cases and is considered a physiologic condition.[3] On the contrary, rigid flatfoot deformity is defined by significant restriction of subtalar joint motion and this constitutes a non-physiologic condition often associated with pain and a more serious underlying pathology, such as, tarsal coalition or neuromuscular disorder.[3,4] In adult populations, flatfoot deformity was known as posterior tibialis tendon dysfunction (PTTD). However, it recently has become known as adult-acquired flatfoot deformity (AAFD) in recognition that the pathology is not only caused by posterior tibialis tendon insufficiency but also by the failure of the arch supportive ligament, including calcaneonavicular ligament, talonavicular capsule, and deltoid ligament.[5] The clinical manifestation varies, based on its stage classified by Johnson and Storm and later modified by Myerson.[6,7] The characteristics of the deformities are failure of the medial longitudinal arch, abduction of the forefoot and hindfoot valgus. The deformity spectrum is copious, ranging from flexible deformity with normal alignment to severe rigid arthritis of the ankle joint with severe deformity of the hindfoot. The peak incidence of this complex deformity is among elderly adults especially among females older than 55 years.[7,8] Moreover, the insufficiency or the failure of the structures is related to patient demographic factors, genetics, and some medical diseases.[9–19] Early detection and early aggressive conservative treatment should be firstly considered. Then, surgical treatment to maintain flexibility and alignment and prevent worsening deformity is considered after failure of conservative management.

Etiology

The etiology of flatfoot can be divided into two main groups; congenital and acquired groups (Table 1).

Congenital Group

Humans can be born with flat feet. The arch develops at approximately 2 years old and matures at 8 years old.[3,20–22] Flexible deformity is mostly resolved after the first decade of life, by the development of the medial longitudinal arch while a minority of children fails to develop their arch. Pediatric flatfoot is common among children under 10 years old. Pfeiffer et al. reported flexible flatfeet in 44% of children 3–6 years old and <1% of pathological rigid flatfeet.[23] The flat arch remains throughout their adolescence which is likely to be found among overweight children.[20,23–25] The clinical features of flexible flatfoot are painless and flexible, and they can perform the full function of the foot. A normal arch can be observed in the non-weight-bearing position while a flat arch is observed in the full weight-bearing position.[3,26,27] Unlike the flexible type, the arch in rigid flatfoot is flattened in all positions with joint stiffness and pain. Although rigid flatfoot is uncommon, the underlying causes must be defined, e.g., tarsal coalition, the accessory navicular bone, congenital vertical talus, or other forms of congenital hindfoot pathology.[4,27,28]

Acquired Group

The most common cause of AAFD is mainly caused by secondary PTTD. Studies have revealed that the most common factor for AAFD is a loss of elasticity due to the degenerative process of the posterior tibial tendon (PTT) leading to being unable to support the foot.[9,29–31] The intrinsic factor, e.g., matrix metalloproteinase (MMP) polymorphism; MMP-1, MMP-8, and MMP-13 have also been discovered to be linked to tendinopathy of the PTT.[17–19] Several risk factors are involved including advanced age, obesity, abnormal foot alignment, equinus contracture, ligamentous laxity, trauma, systemic inflammatory conditions, and steroid use.[6,9–16] Additionally, in neglected or improper posttraumatic injury, such as, Lisfranc joint injury or fracture-dislocation of the

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How to cite this article: Rungprai C, Maneeprasopchoke P. A Clinical Approach to Diagnose Flatfoot Deformity. J Foot Ankle Surg (Asia Pacific) 2021;8(2):48–54.

Source of support: Nil

Conflict of interest: None

Table 1: Etiology of flatfoot deformity

Congenital group	*Acquired group*
Flexible flatfeet	Posterior tibial tendon dysfunction
Rigid flatfoot	Matrix metalloproteinase polymorphism
• Tarsal coalition	Post-traumatic
• Congenital vertical talus	Osteoarthritis
• Accessory navicular bone	Inflammatory
• Congenital hindfoot pathology	• Rheumatoid arthritis • Seronegative spondyloarthropathies
	Neuropathic
	• Diabetic mellitus
	• Charcot neuroarthropathy
	Tight gastrocnemius–soleus complex iatrogenic

medial column, the bone develops a malunion and eventually AAFD.[32–34] Furthermore, injury to soft tissue, e.g., the spring ligament or plantar fascia can also cause AAFD by a progressive collapse of the medial longitudinal arch.[5,35,36] Although PTTD is a common cause of AAFD, deltoid ligament insufficiency is usually a primary pathology of posttraumatic flat foot.[32] Moreover, both inflammatory and degenerative arthritis are possible causes of AAFD. An inflammatory joint such as found in rheumatoid arthritis, seronegative spondyloarthropathies, and gout can cause deformity with progressive collapse of the medial longitudinal arch and this depends on disease control and activities.[37] In addition, the patient with neuropathic pes planus loses sensation due to neuropathy. Examples of this category include diabetic mellitus and Charcot neuroarthropathy. The neuroarthropathic foot is responsible for the collapse of the midfoot by repetitive mechanical trauma to the bony architecture and failure in supportive mechanisms. This deformity is the so-called rocker-bottom foot.[38,39] A tightness of the gastrocnemius–soleus complex can also induce AAFD by passing ankle dorsiflexion force from the ankle to the transverse subtalar joint and midfoot joint. For this pathology, the force direction is relatively lateral to the subtalar joint leading to midfoot collapses and the hindfoot valgus causes lateral peritalar subluxation of the navicular and subfibular impingement.[40–42] Finally, an iatrogenic AAFD was once hypothesized especially in the surgical treatment of the cavovarus deformity or foot drop by transferring the PTT; however, Pecheva et al. reported that these were not significantly related.[43] Only a few patients undergoing PTT transfer develop spring ligament sprain with a normal radiograph. Despite efforts to identify underlying causative factors among patients with AAFD, some patients do not exhibit any known predisposing factors.

Pathophysiology

The PTT serves as the arch dynamic stabilizer which originated from the posterior tibia, fibular, and interosseous membrane to the midfoot. The course lies posteriorly to the medial malleolus.[1,40] PTT provides the Herculean plantar flexion, inversion, and supination for the ankle, hindfoot, and forefoot, respectively. As well, it strongly supports the medial longitudinal arch at the level of the navicular tuberosity. The three anatomical parts for the PPT are described below.[44] First is the anterior part, the largest one. Its course ends at the navicular tuberosity, inferior capsule of the medial naviculocuneiform joint, and inferior aspect of the medial cuneiform. The second is the middle part, which attaches to the middle, lateral cuneiforms, cuboid, and bases of the second to fifth metatarsals. The third is the posterior part, which inserts on the sustentaculum tali of the calcaneus. The PTT is well nourished by the branches of the posterior tibialis artery. In contrast, a relative hypovascularity area is observed for it involving a lack of intratendinous anastomoses. This insufficiency leads to frequent devastating ruptures of the tendon. The area is located about 1.5 cm distal to the medial malleolus.[45]

According to the gait cycle, the PTT adducts and inverts the foot. These motions separate the axis of the transverse tarsal joint from each other during the midstance phase. Lastly, in the late stance phase, the hindfoot becomes rigid. The antagonist muscle or tendon for the PTT is the peroneus brevis which causing the foot to develop an eversion manner. Hence, when the PTT becomes insufficient, the powerful peroneus brevis pulls the hindfoot into eversion. As a result, the hindfoot and forefoot develop into valgus and abduction, consecutively.[9,46,47] Moreover, the primary static stabilizers, composing the spring ligament and interosseous talocalcaneal ligament, are stretched.[35] Additionally, the gastrocnemius–soleus complex direction is converted, causing the transverse tarsal joint and midfoot to develop more valgus deformity.[41,42,48] In the later stage of AAFD classified by Johnson and Storm along with Myerson,[6] the deformity is seen in the lateral longitudinal arch including impingement and arthritis.[42] Finally, the deltoid ligament fails or becomes insufficient in the last stage resulting in talar tilt, ankle instability, and leading to arthritic changes of the hindfoot and midfoot.[2,49]

Diagnosis

To precisely diagnose this complex deformity, AAFD, clinical manifestations history taking, thorough examination as well as radiographic evidence are required.

History

This sort of deformity is common among elderly females especially those with obesity. The presentation begins with gradual onset of pain along the medial foot which may migrate distally to the arch of the foot or proximally to the leg. It accounts for tenosynovitis or deformity. In the later stage, the pain can either resolve or progress to the lateral side according to the increasing deformity which is responsible for subtalar or subfibular impingement. Therefore, pain can occur at the lateral foot or the sinus tarsi.[42,50] Particularly, gradual onset of the midfoot planus along with valgus heel is easily observed. Not only pain and deformity can be found among patients but also ankle swelling particularly behind the medial malleolus has also been reported. A meticulous history ought to be taken. Due to the characteristics of pain, onset, timing, and severity of symptoms, the deformity must be described by the patient or relatives. Essential information including previous trauma, steroid use, orthotic use, underlying condition, e.g., hypertension and diabetes mellitus, smoking habit, and family history of inflammatory arthropathy are also to be collected. Furthermore, the last crucial information needed for making treatment decisions and counseling is body mass index.[40]

Physical Examination

First of all, the patient must be entirely exposed from knee to toe. The individual center of gravity, the genu valgus, ought

to be observed because the center of gravity moves medially. Therefore, the medial ankle especially the tendon behind it, the PTT, receives the greater load. The deformity shows in the midfoot planus and valgus heel leaving a sign called the "too many toes sign" (Fig. 1). This sign is observed at the posterior ankle of the patient and more than two toes are seen lateral to the hindfoot.[7] Additionally, it implies the examiner should note the abduction of the forefoot. However, this is not a pathognomonic sign but still supports the diagnosis. Moreover, the complete gait cycle ought to be thoroughly observed, particularly the heel inversion during the toe-off phase. Palpation along the course of the PTT should be performed to identify any tenderness, swelling, or defect. Not only the PTT but also the sinus tarsi, talar dome, and navicular tuberosity should be palpated. As well, the callus at the subluxated talar head can be detected.

The motion of the ankle and hindfoot along with muscle strength must also be assessed. First, PTT strength is evaluated by passively inverting the foot and asking the patient to resist against the examiner in the plantar flexion position. Then, its flexibility and function are firstly examined using the double leg heel raise test, then performing the single-leg heel raise test on the pathologic side. The single-leg heel raise test is performed by asking the patient to stand on only the pathologic leg, while the contralateral one is raised off from the floor. The heel of the standing foot is raised too. The positive single-leg heel raise test is addressed when the patient cannot lift the heel off or heel inversion is noted because the PTT function has disappeared.[7,29]

The range of motion of the subtalar joint and transverse tarsal joint is full in the early stage. As the condition deteriorates, the range of motion is limited because of the fixed deformity. Of rather paramount importance in ankle motion assessment is that the ankle joint range of motion ought to be measured both in knee extension and flexion along with the locked and unlocked the transverse tarsal joint. Furthermore, joint stiffness is a sign of synostosis coalition and osteoarthritis. Defect in Lisfranc joint motion and swelling at the dorsal foot are keys of a degenerative joint.

Finally, the equinus contracture or gastrocnemius–soleus complex tightness is cannot be ignored because it can collapse the arch and reduce the support of the midfoot. Gastrocnemius–soleus complex tightness can be examined using the Silfverskiöld test. When ankle dorsiflexion during knee extension <10° is corrected by knee flexion or ankle dorsiflexion during knee extension and flexion is >10°, the Silfverskiöld positive test result implies isolated gastrocnemius contracture.[51]

Radiography

Another source of information to diagnose AAFD is a weight-bearing plain film of the foot both anteroposterior and lateral views, standing ankle plain film, and Saltzman view. On the AP view of the foot radiograph, three angles are used for AAFD. An angle of the talo-first metatarsal appears to increase to 16° (normal foot is around 7°) when measured with the long axis of the talus and first metatarsal bone.[52] The other two angles used to detect forefoot abduction are the talonavicular coverage angle and the talar uncoverage angle.[53,54] Flatfoot is addressed when the talonavicular coverage angle is >20° (normal foot is 15–20°). The talonavicular uncoverage angle is computed as a percentage (Fig. 2). The expressed value comprises a percent of the talus surface without navicular contact particularly, of the medial part. Forefoot abduction is stated by an increased angle. A significant percentage of the value affecting treatment is 30 and is also used as the cutoff point of stage IIa and IIb.[55]

Concerning the lateral view, the three main parameters comprise two angles and a line. Meary's angle (talo-first metatarsal angle) is measure by the axis in the same fashion of the AP view showing an increase up to 20° or more than that in the flatfoot (normal foot ranges from 0 to 10°) (Fig. 3).[50,56] Another angle is the calcaneus pitch measured by the inferior inclination axis of the calcaneus to the horizontal. The normal foot shows an angle of 8° while those with flatfoot show 4°.[50,56] The length from the medial cuneiform and the base of the fifth metatarsal bone indicates the arch height. In the normal foot, the height is around 17 mm but for those with flatfoot is shorter, 6 mm.[50,56] For the standing ankle plain film, the lateral talar tilt with deltoid insufficiency and arthritis can be seen in the later stage.[6,57] The Saltzman view is used to assess the hindfoot alignment. The distance from the mid tibial axis and the most distal portion of the calcaneus represents the hindfoot moment of the arm (Fig. 4). For the normal foot, 3 mm varus is observed while those with flatfoot have 10 mm or more than found in valgus.[56,58] Another important dimension is the hindfoot alignment angle measured with the vertical axis of the tibia and

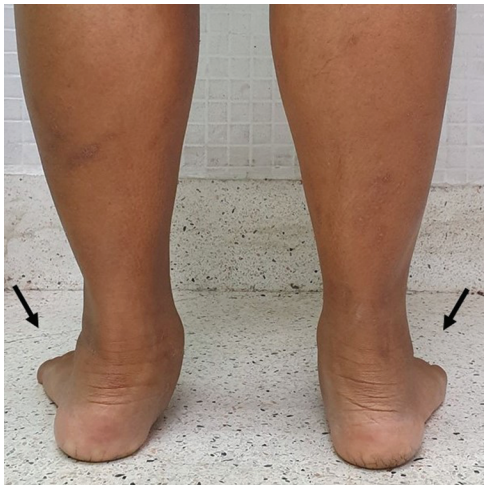


Fig. 1: "Too many toes" sign

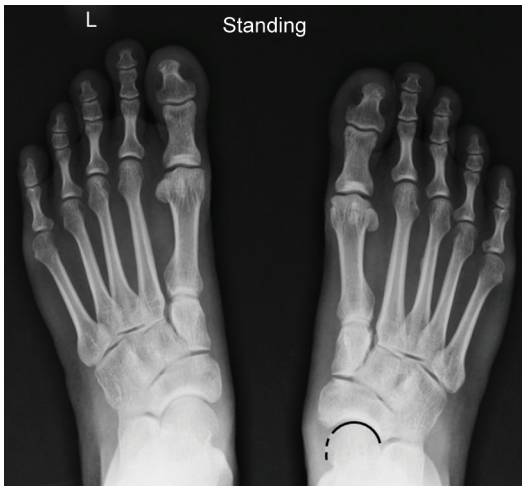


Fig. 2: Talonavicular uncoverage percentage, in which small circles represent the amount of talar head uncovered by the navicular (normal, 10–30%)

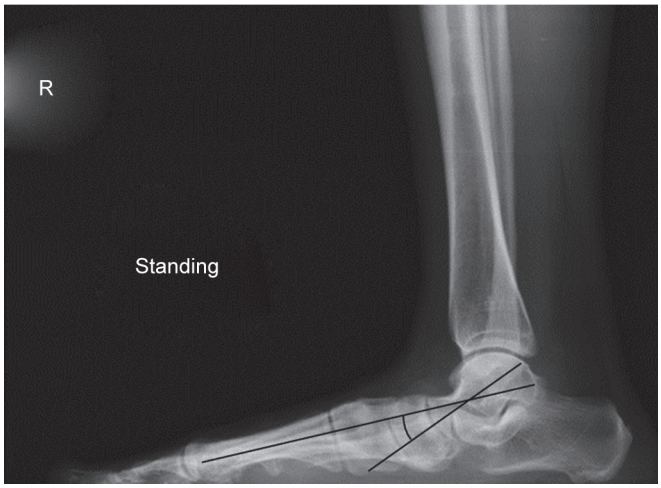


Fig. 3: Lateral talus-first metatarsal (Meary) angle, the angle between the longitudinal axes of the talus and first metatarsal (normal, 0–10°)

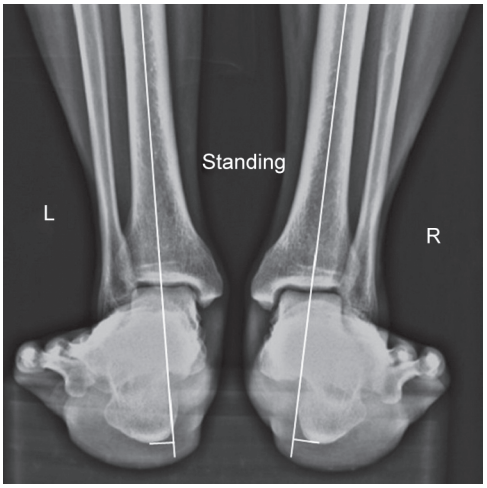


Fig. 4: Hindfoot moment arm, the shortest distance between the longitudinal axis of the tibia and the most inferior part of the calcaneus (normal averages 3.2 mm)

calcaneus tuberosity axis. The normal value is 5° while the value of 22° implies flatfoot.[53]

Although clinical examination and plain film are ample for diagnosis, both radiographs, magnetic resonance imaging (MRI) and computed tomography (CT), are crucial to confirm. MRI is unsurmountable in detecting synovial fluid and soft tissue problems, including pathology of the PTT and ligament, edema, or longitudinal split of a tendon.[43] Several studies have shown both specificities are 100%. In addition, the sensitivity of the MRI is higher than that of the CT, 95 and 90%, respectively.[59] Although the MRI is aids in the diagnosis, the weight-bearing CT is more useful in evaluating the locations of the deformity and localized impingement, joint arthritis, or severe calcaneal subluxation.[60,61]

Classification

The AAFD was classified in three stages by Johnson and Strom in 1989. Then, the fourth stage was added by Myerson[6,57] (Table 2).

Stage I represents a mild AAFD; the manifestations include pain, swelling along the PTT arising from tenosynovitis or tenodesis; but no deformity can be seen. The patient is still able to show single-heel raise. In addition, inversion and locking hindfoot are still able to be performed. Therefore, the radiographs show no abnormality while the inflammation or degenerative processes probably appear in the MRI.[62]

During stage II, the deformity can be observed and is divided into two subtypes IIa and IIb. These constitute a flexible deformity; and thus, can be corrected passively by adduction of the talonavicular joint and inversion of the subtalar joint. Inversion and locking hindfoot are limited. The radiograph reveals Meary's angle elevated resulting from ankle plantar flexion. A significant percentage of talonavicular uncoverage is observed at 30% which is the cutoff point of subtypes a and b.[55] Moreover, the interosseous ligament is insufficient due to the progression of the stage.

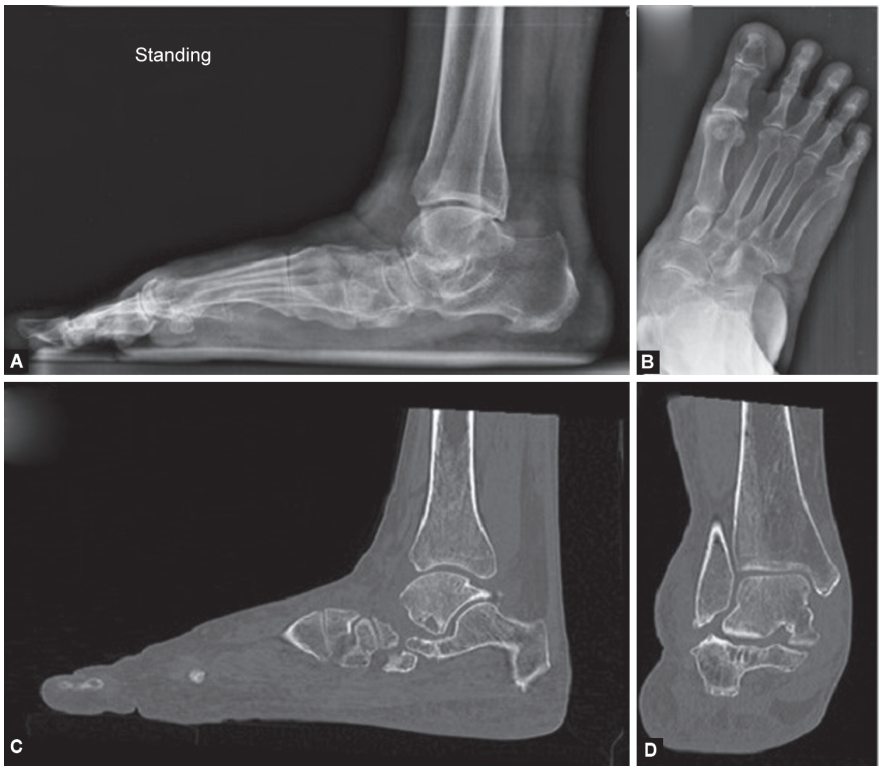
Stage III is characterized by rigid deformity which cannot be corrected by passive manner.[29] This rigidity is the degenerative change of three joints: the talonavicular, subtalar, and calcaneocuboid. Arthritis accounts for rigid hindfoot valgus and midfoot abduction (Fig. 5). During this stage, the pain subsides due to the disrupted PTT. However, for those with arthritis or impingement in this stage, the pain is on the lateral to sinus tarsi instead.[42,60]

During stage IV, the severity develops to deltoid ligament failure. Consequently, lateral talar tilt leads to the valgus ankle and ankle arthritis (Fig. 6).[6,57] Furthermore, the last stage is divided into IVa and IVb. The difference of this subtype involves the flexibility of the tibiotalar joint, that is, the flexible pes planus is classified as stage IVa. On the contrary, rigid pes planus refers to stage IVb. In addition to tibiotalar deformity, ankle arthritis may also be present.

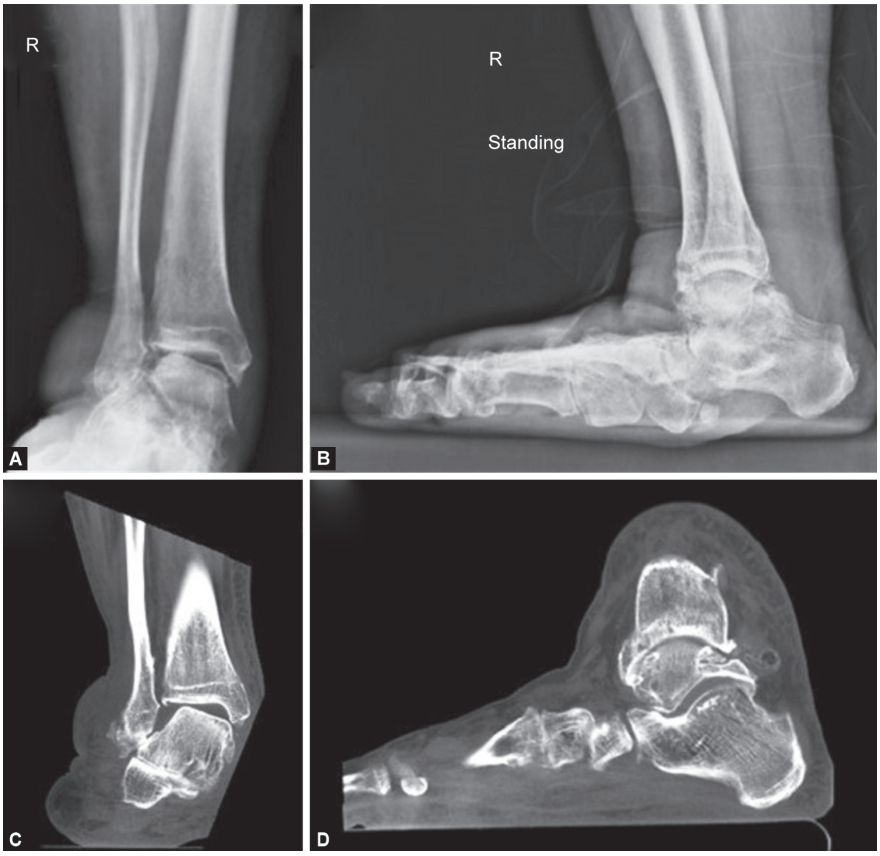
Table 2: Myerson modification of Johnson and Strom classification of adult-acquired flatfoot deformity

Stage	*Clinical findings*	*Imaging*
I	Medial pain and swelling No deformity Tenosynovitis on pathology with normal tendon length	Normal radiographs
II		
IIa	Medial pain and swelling Flexible hindfoot deformity Elongated tendon with longitudinal tears	Talonavicular uncoverage <30%
IIb	Severe flexible hindfoot deformity Forefoot abduction	Talonavicular uncoverage >30%
III	Fixed hindfoot deformity Disruption of PTT Subfibular impingement	Degenerative changes in the triple joint complex
IV		
IVa	Fixed hindfoot deformity Flexible ankle valgus without severe arthritis	Lateral talar tilt
IVb	Fixed hindfoot deformity Fixed ankle valgus with or without arthritis	Lateral talar tilt ± ankle arthritis

Adapted from Johnson and Strom,[7] Myerson,[6] and Deland et al.[55]



Figs 5A to D: Radiograph and weight-bearing CT of stage III AAFD showing evidence of talonavicular, subtalar, and calcaneocuboid arthritis: (A) Lateral view of the foot; (B) Anteroposterior view of the foot; (C) A sagittal plane of the weight-bearing CT; (D) A coronal plane of weight-bearing CT



Figs 6A to D: Radiograph and weight-bearing CT of stage IV AAFD showing evidence of tibiotalar joint valgus angulation associated with the flatfoot deformity: (A) Anteroposterior view of the ankle; (B) Lateral view of the foot; (C) A coronal plane of weight-bearing CT; (D) A sagittal plane of the weight-bearing CT

Conclusion

Flatfoot is a common condition with a large clinical spectrum. The typically different types of flatfoot deformity include congenital or acquired, adult or pediatric, and flexible or rigid. Recognizing the biomechanics of the foot and the relations of the forefoot to midfoot to hindfoot are also important to identify and treat the underlying cause properly. An AAFD is the most commonly related to PTT insufficiency. Although the etiology remains unclear today, the study revealed many possible causes including underlying medical comorbidities, hypovascularity, genetics, foot morphology, or trauma. Clinical manifestations and radiographic findings are used to establish and confirm the diagnosis. The staging system for flatfoot describes the deformity and guides the proper treatment.

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