An important cause of pes planus: the posterior tibial tendon dysfunction

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Abstract

Posterior tibial tendon dysfunction (PTTD) is an important cause of acquired pes planus that frequently observed in adults. Factors that play a role in the development of PTTD such as age-related tendon degeneration, inflammatory arthritis, hypertension, diabetes mellitus, obesity, peritendinous injections and more rarely acute traumatic rupture of the tendon. PTT is the primary dynamic stabilizer of medial arch of the foot. Plantar flexion and inversion of the foot occurs with contraction of tibialis posterior tendon, and arch of the foot becomes elaveted while midtarsal joints are locked and midfoot-hindfoot sets as rigid. Thus, during the walk gastrocnemius muscle works more efficiently. If the PTT does not work in the order, other foot ligaments and joint capsule would be increasingly weak and than pes planus occurs. We present a 10-year-old female patient diagnosed as PTTD and conservative treatment with review of the current literature.

Introduction

Frequently observed in adults, posterior tibial tendon dysfunction (PTTD) is an important reason of acquired pes planus. Its etiology involves age-related tendon degeneration, inflammatory arthritis, hypertension, diabetes mellitus, obesity, paratendinous injections and acute traumatic tendon rupture in rarer cases. Tibialis posterior tendon is the primary dynamic stabilizer of the medial arc of foot. Other ligaments of the foot and the joint capsule gradually weaken in case of posterior tibial tendon dysfunction and pes planus develops. Conservative treatment is indicated for all cases in PTTD, which is clinically observed in three stages. Presence of acute inflammation and flexibility/rigidity of foot deformity are key factors in treatment disposition. Here, we present a 10-year-old female case that we diagnosed with PTTD and her conservative treatment along with the recent literature.

Case Report

A 10-year-old girl applied with a complaint of pain in her left foot. She had a history of outward sprain in her left foot about one and a half years ago. She had foot pain increasing with walking since a year and she was limping since two months. No rash, swelling or temperature increase was detected at the ankle or foot joints during physical examination. Pes planus and calcaneovalgus were apparent at the left foot. Jack's test of toe rising performed by passive pollex extension showed no arch flattening. Talotibial joint movements were free and with minimal pain. She was able to full range of motion for dorsiflexion. Subtalar joint passive supination was limited in the last part. No active supination could be performed in the left foot. Stiffness or shortening were not detected for the calf muscle. Left foot pronation was observed in the walk analysis performed. Plantar flexion could not be performed when patient's forefoot was in outward rotation. Too many toe sign and hind foot valgus were observed in the standing rear examination (Figure 1). Besides of non-inflammatory characterized joint pain, juvenile rheumatoid arthritis and such inflammatory conditions were ruled out with detailed blood samples (complete blood count, erythrocyte sedimentation rate, C-reactive protein and rheumatoid factor were normal).

Her joint hypermobility was assessed with Marshall Test and scored grade 3.

Clinical findings of the patient were considered to conform to PTTD and ultrasound and magnetic resonance (MR) imaging were carried out. However, no pathology was observed in both assessments and MR imaging was repeated as fat suppressed sequenced included and fatty degeneration in tibialis posterior tendon and a decrease in tendon diameter as against the healthy side were observed (Figures 2 and 3). The patient diagnosed with PTTD did not approve surgical therapy, thus she was treated and followed up conservatively. Alternating toe - heel walking, walking exercises stepping on lateral side of the foot, foot and ankle Thera-band augmentation, and stretching exercises for Achilles and hamstring muscles were given in the conservative treatment. Patient's foot was supported with medial heel wedge. Fourth week controls revealed no marked improvement in patient's deformities whereas significant decrease in pain was recorded.

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Key words: posterior tibial tendon, dysfunction, pes planus, treatment, child.

Conflict of interests: the authors declare that they have no conflict of interests.

Contributions: KE, AYK, UK, research concept and design; AYK, LT, MŞ, EK, collection and/or assembly of data, data analysis and interpretation; KE, AYK, BO, LT, MŞ, EK, writing the article; KE, AYK, EK, critical revision of the article; KE, AYK, UK, BO, LT, MŞ, EK, final approval of article.

Received for publication: 21 August 2014.
Revision received: 18 October 2014.
Accepted for publication: 12 November 2014.

Clinics and Practice 2015; 5:699
doi:10.4081/cp.2015.699

Discussion and Conclusions

Reason of PTTD is a controversial subject. Its etiology is often multifactorial. These include spontaneous rupture, progression of congenital flexible pes planus, trauma (especially in medial malleolar fractures), recurring micro traumas, collagen diseases, vascular reasons and accessory navicular bone.[1,2] The pathology responsible for PTT dysfunction is not clear in rheumatoid arthritis. Rheumatic disease cause pan planus in several ways.[2,3] Valgus deformity puts stress on PTT with joint destruction resulting in hind foot valgus, ligament complex destruction of subtalar and talonavicular joints etc. and this contributes to pes planus progression. Correlation between PTTD and obesity, hypertension, diabetes and steroid use was found. All of these conditions directly or indirectly influence the blood circulation of PTT.[1-3] On medial malleolar level, direct contact of tendon to bone and compression of flexor retinaculum to tendon are to blame for blood build-up insufficiency in this area of the tendon. Collagen content and type and orientation of fibers are the factors affecting on PTTD. Collagen structure begins to

change with aging. Increasing mucin content and myxoid degeneration disrupts the normal linear orientation of the collagen bundle inside the tendon. Thus, tensile strength of the tendon decreases.[1-3]

As in all foot problems, patient's shoes should be in observed carefully in the medical examination. First, lower extremities should be monitored from front, rear and sides when standing.[1-4] Walking should be monitored. Pes planus deformity, swelling in PTT, distension in medial part of ankle, lateral rhytides, too-many toes sign and hind foot valgus may be observed in the inspection of the patient with PTTD. All findings should be examined with patient standing on her foot.[4,5] Calluses may be observed in plantar and talar distal areas. Tenderness with palpation may be felt in the medial malleolar and middle foot insertion areas. PTT may be markedly sensitive in the active inflammation period, while being painless in the late-stage. Also, temperature increase and swelling are points to take into consideration during examination. Ranges of motion of ankle, subtalar and midfoot joints should be assessed bilaterally. Heel raising and direct force examinations are specific tests. Heel rising is performed bilaterally and physician stands behind the patient.[4,5] Hind foot of the patient symmetrically passes from valgus to mild varus. Asymmetry indicates the inability of PTT to bring subtalar joint to inversion. Primary pathology in subtalar or talonavicular joint can show similar results and should be excluded. One-foot heel raising test is performed for verification. PTT is the heel raise-initiating muscle, while gastro-soleus muscle carries the raising on. PTTD patient moves his/her body's center of gravity to start the heel raise. Posterior tibial muscle strength is assessed with patient at sitting position. Isolated PTT is assessed keeping the ankle in plantar flexion and eversion. Physician places his/her hand on the medial part of the first metatarsus and asks patients to perform inversion against the resistance. Muscle strength is compared with the other side. Pain against resistance is recorded, if any.[5,6]

Treatment options for the patients in symptomatic flexible pes planus are various activity modifications, orthosis and physiotherapy. Non-steroid anti-inflammatory drugs can be prescribed in some cases.[1,5,6] Comorbidities like obesity, hyper-laxity and proximal extremity problems may be present in patients. These should be identified and controlled. Orthosis and observation is adequate if symptoms are relieved with non-operative approach.[1,5]

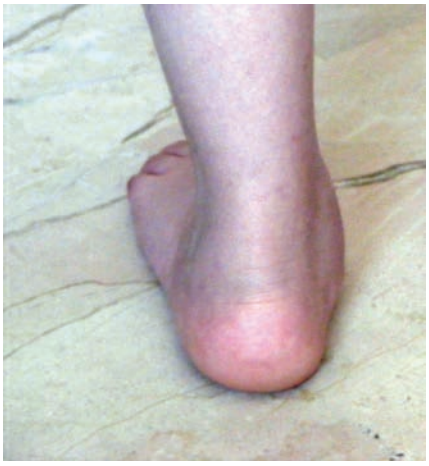

Figure 1. ***Too many*** **toe sign.**

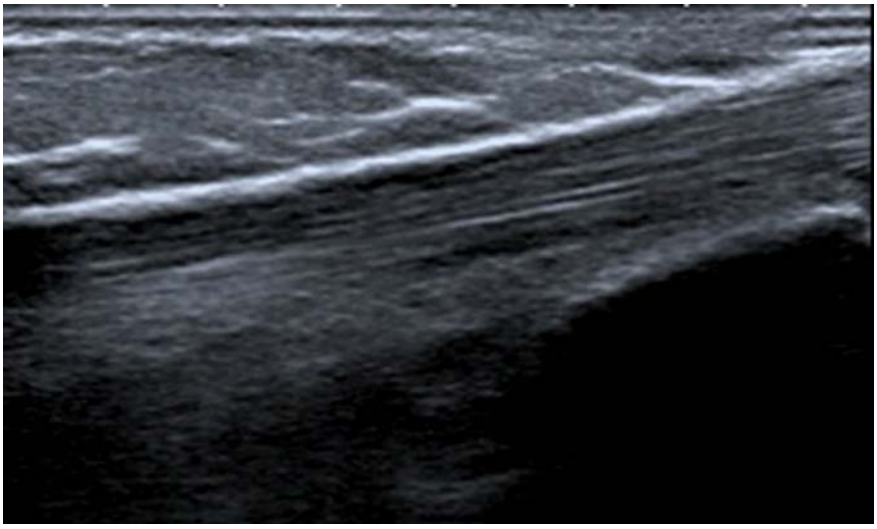

Figure 2. Ultrasound image demonstrates the normal appearance of left tibialis posterior tendon.

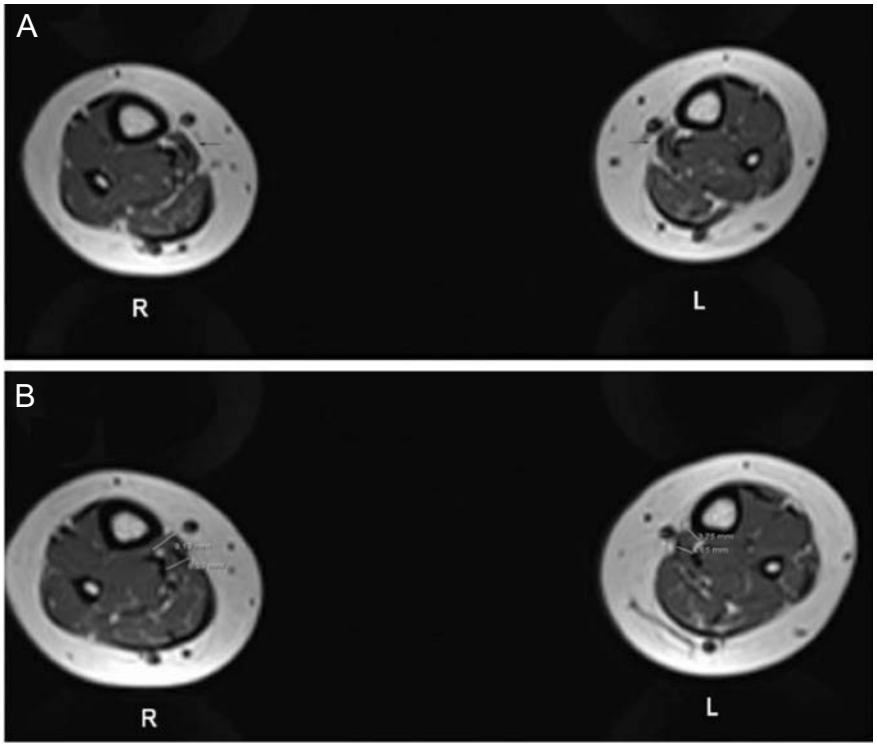

Figure 3. A) Transvers T1 weighted image demonstrates normal muscle bulk medial to the tendon on the right but on the left no muscle bulk replaced by fat tissue is seen; B) Transvers T1 weighted image demonstrates the difference of muscle bulk thicknesses.

Reduction of normal pronation, metatarsal arch supporting and enabling shock absorption is aimed with an orthosis adapted to the contours of the foot.[1,5]

Increased plantar flexion angle and momentum in the stepping on phase existing in pes planus can be fixed with appropriate orthosis use. Orthosis have a significant effect for ankle, while no important effect for knee or wait has been demonstrated. Moreover, foot orthosis reduce patient complaints of pain with proprioceptive mechanisms related to muscle activity of lower extremities.[6-8]

No difference in effect is demonstrated between orthosis wore and supports. Increase of varus in the knee with supports in the form of medial heel wedge is demonstrated. However, there is no consensus about the long-term effect of supports. In summary, orthotic shoes and supports have positive effect on ankle motions in the sagittal plane, while they have no effect on forces and motions in the frontal plane.[2-5] It is seen that orthotic devices have corrective effect on pronation in pes planus.

A final diagnosis must be established before prescribing a support. Use of supports aggravates the symptoms in flexible pes planus with short Achilles triceps tendon and in rigid pes planus. Talus does not go to dorsiflexion in flexible pes planus with short Achilles triceps tendon. Therefore, an orthosis with subtalar joint designed in inversion will raise the front part of talus and cause a resistance, and pressure will increase under the head of talus. Increasing pressure will cause pain. Shape of rigid pes planus does not change with orthosis or any non-operative approach.[2-5]

Foot exercises are done in pes planus treatment. Exercise programs aim to decrease the tension in ligaments, transfer the body weight towards the exterior part of the foot, strengthen the inverter muscles and plantar flexors, relax dorsiflexor and evertor muscles and to relief the strain of Achilles tendon, if any. For this purpose, exercises such as collecting objects like pen and paper off the ground using toes, folding towels with toes, alternating toe-heel walking, walking with stepping on lateral side of the foot and foot exercises against resistance can be done.[1-6]

Wearing shoes cause weak muscle tonus and arch position due to lack of exposure to external proprioceptive stimulants. Therefore, exercises stimulate arch development with proprioceptive stimulation of the foot. Heel stretching exercises can be attempted to change symptomatic FDT-KAT into asymptomatic flexible pes planus. There are no long-tern studies evaluating the efficacy of this program. Heel stretching exercises should be performed with knee in extension and subtalar joint in mild inversion.[1,5]

Surgical treatment may be carried out in case of conservative treatment failure, prevention of patient's normal activity, advanced deformity and excessive number of problems related to shoes. Surgical approach can target bones or soft tissues or both. Soft tissue approaches are rarely affective when performed exclusively. Bone procedures include forefoot, middle foot and hindfoot osteotomies, lateral colon stretching and calcaneus medial displacement osteotomies. These can be combined with Achilles tendon stretching and medial plication.[6-8]

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