Symptomatic acquired flatfoot is an important orthopaedic problem, due to progressive loss of whole foot function and the increasing problem of patient disability. It is a complex entity, involving the tibialis posterior tendon, ankle joint, hindfoot and midfoot. In most cases the posterior tibial tendon (PTT) is the root cause of acquired flat foot, but there are other contributions and many different factors have an influence. The clinical picture varies depending on the stage of the deformity, as well as the treatment approach. Initially soft tissue procedures, synoviectomy and augmentation of the PTT are advised. In stage 2, lateral column lengthening and calcaneal osteotomy, with soft tissue-tendon transfers (TA, FHL, FDL) are recommended. In stage 3 subtalar, double or triplearthodesis is preferable, while in stage 4 pantalar fusion is indicated. In the article more detailed etiology, the clinical picture, diagnosis and modalities of treatment are presented.

Key words: acquired flat foot, posterior tibial tendon dysfunction, treatment

INTRODUCTION

Flat foot, especially the acquired type, is a common condition and can be seen in 10-25% of the entire population, but, as it is usually asymptomatic, it is overlooked and not treated in most cases. Moreover, in practice, it is usually misdiagnosed as ankle sprain. It can be divided into several groups: adult non-Posterior Tibial Tendon Dysfunction (PTTD), adult PTTD flatfoot, flatfoot due to coalition, post-traumatic, neuropathic-Charcot foot. The first type is progression of the juvenile form, while tarsal coalition, post-traumatic, neuropathic flatfoot is not so common. In this paper adult PTTD flatfoot is described, with concern about the other forms, which must be excluded before treatment.

Acquired flatfoot is defined as partial or total loss of the dynamic and static supportive structures of the medial arch of the hindfoot and ankle. Furthermore, in most cases it correlates with tibial posterior insufficiency, i.e. posterior or tibial tendon dysfunction is the most common cause of acquired flatfoot among adults. There are other risk factors, such as female gender, age, obesity, diabetes mellitus (DM), hypertension (HT), "overuse syndrome", inflammatory arthritis, chronic recurrent tenosynovitis, which could have an impact on expression of the acquired flatfoot.

As mentioned, besides the PTT, there are other, less common, causes of flatfoot: flatfoot due to coalition, neuroathy (due to diabetes mellitus, peripheral neuritis), and post-traumatic. Also, arthritis in the ankle, the talonavicular joint and tarsometatarsal (Lis-franc) joint, as well as inflammatory arthritis, may result in the occurrence of flatfoot.

There are statements that isolated loss of the PT tendon without ligamentous disruption will not lead to a progressive flatfoot deformity, i.e. the adult acquired flatfoot deformity cannot be reproduced experimentally by releasing the tibialis posterior tendon alone. "In conclusion, we have shown that, to create flattening of the plantar arch, there is a need to cut the medial structures, including the spring and plantar ligaments and possibly the plantar fascia." This is true. In biomechanics the dynamic supporting structures of the arch are: plantar aponeurosis, posterior tibial tendon and plantar intrinsic musculature, while static supportive structures of the arch are: the spring ligament complex, superficial deltoid ligament, long and short plantar ligaments, plantar aponeurosis. However, in practice, without the dynamic support of the PTT the other ligaments and joint capsule become weak and flatfoot develops. The circle of disturbances of the foot then completes: PTT elongation leading to spring ligament and plantar fascia problems (elongation) and del-
toid ligament insufficiency. Coupling of static and dynam- 
ic support of the arch is important, as once imbalance 
occurrs, flatfoot is mandatory. These changes happen 
through six stages and the last one is irreversible: 
1. Pre-existing flatfoot; 
2. Increased gliding resistance of the posterior tibial tendon; 
3. Increased strain on supportive ligaments and tibialis 
posterior with attenuation and rupture of the posterior tib- 
ial tendon; 
4. Sequential rupture of the spring ligament, superficial 
deltoid, and interosseous talo-calcaneal ligaments; 
5. Valgus of the heel, shortening of the Achilles tendon; 
6. Degenerative changes in the ankle joint, midfoot and 
forefoot. 
To be able to treat it adequately, one must understand 
this pathology and pathogenesi-etiolo-gy of the PTT and 
flatfoot and that will be discussed here. 

DYSFUNCTION OF THE TIBIALIS POSTERIOR 
TENDON 

Dysfunction of the tibialis posterior tendon is the most 
common cause of acquired flatfoot in adults. This prob- 
lem can be very easily overlooked, so this condition was 
considered in the past as very rare, but PTT dysfunction 
is currently diagnosed in 13-64% of cases of flatfoot. It 
happens mostly in females, aged over 40, as well as in yo- 
ung males for whom acute trauma is the leading cause of 
PTT damage.

In 1936, Kulowski was the first to report cases with eff- 
usion into the tendon sheath of tibialis posterior tendon. 
The first case of posterior tibial rupture was described by 
Key in 1953, but the initial publication about surgical ex- 
ploration of PTT ruptures was written by Kettelkamp and 
Alexander in 1969. In 1974, Goldner inserted either fle- 
xor digitorum longus or flexor hallucis longus on the spri- 
ging ligament instead of the ruptured PTT to replace the ti- 
bial tendon. From that time there have been many publi-
cations regarding treatment options for PTT dysfunction 
and symptomatic flatfoot.

RELEVANT ANATOMICAL DATA 

Tibialis posterior originates at the proximal one-third of 
the tibia and interosseus membrane (in the deep layer). 
The tendon (with sheath) passes posterior to the medial 
malleolus (fixed by flexor retinaculum). It has a multiple 
insertion to stabilize the medial and middle column of the 
foot. The PTT is inserted: anteriorly on the navicular tube-
rosity joint capsule N-C, 1st cuneiform; medially on the 
2nd cuneiform, 3rd cuneiform, cuboid, PL tendon, 2nd, 3rd, 
4th and 5th metatarsal bone; and posteriorly on sustenta-
culum tali. 
The complexity of PTT insertions is confirmation of its 
multiple and significant functions. 
Due to its location, tibialis posterior is considered as a 
plantar flexor as well as an invertor of the hindfoot. It is 
the primary stabilizer of the medial longitudinal arch of 
the foot.

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>CLASSIFICATION BASED ON MRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type</td>
<td>MRI</td>
</tr>
<tr>
<td>Type I</td>
<td>One or two fine longitudinal splits, without evidence of transubstance degeneration</td>
</tr>
<tr>
<td>Type II</td>
<td>The tendon is narrowed with longitudinal splits and evidence of intramural degeneration</td>
</tr>
<tr>
<td>Type III</td>
<td>Diffuse swelling of the tendon with uniform degeneration. A few strands can be intact of the tendon is completely replaced by scar tissue</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>CLASSIFICATION ACCORDING TO THE CLINICAL SIGNS -JOHNSON-STROME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage</td>
<td>Clinical Presentation</td>
</tr>
<tr>
<td>Stage I</td>
<td>Medial foot and ankle pain, swelling, mild weakness, tendon length is normal</td>
</tr>
<tr>
<td>Stage II</td>
<td>A flexible panovalgus foot. Medial or lateral pain or both. The &quot;too many toes sign&quot; and single limb heel rise are present. The tendon is elongated and functionally incompetent</td>
</tr>
<tr>
<td>Stage III</td>
<td>All the signs of stage II are present, the pes planovalgus deformity is fixed. Lateral Pain at the calcaneal -fibular contact</td>
</tr>
<tr>
<td>Stage IV</td>
<td>End stage, valgus tilt of the talus in the ankle mortise leads to lateral tibiotala degeneration, osteoarthritis of the ankle, with limited ability to walk</td>
</tr>
</tbody>
</table>

Stage IV was added to the original Johnson-Strome classification later by Mayerson.

The normal antagonist to the tibialis posterior is the pe- oneus brevis muscle. Thus, dysfunction of the tibialis 
posterior leads to valgus deformity (hindfoot eversion) 
and foot abduction (midfoot and forefoot abduction de- 
formity), due to predominance of the peroneus muscle. 

Tibialis posterior distributes the body weight through 
all metatarsals, supporting the medial arch. Dysfunc-
tion of the tibialis posterior does not lead to a flat foot 
until the supporting structures (spring ligament, plantar fascia, talonavicuar, naviculocuneiform ligament and tar-
sometarsal capsule) are elongated, but this gradually hap-
pens. Later, hip and spine problems occur.

A clinically very important part in the tibialis posterior 
is the hypovascularised zone in the retromalleolar groove. 
The hypovascular zone begins 40mm proximal to the na-
vicular and extends 14 mm proximally. It is a common site for pathological features. This is the weak point of the 
PTT, responsible for PTT dysfunction and the consequen
instability of the medial and middle columnae of the foot. Clinically it presents as valgus and flat foot.

PATHOGENESIS AND PATHOLOGICAL ANATOMY

Our understanding of the pathogenesis of PTT dysfunction is still controversial. Dysfunction of the PTT leads to collapse of the medial longitudinal arch and forefoot abduction, but only in the fourth stage of PTT disorder.

AETIOLOGY

Several causes of PTT dysfunction have been proposed. Among them the most frequent are degenerative changes, which could lead to rupture. Thus, obesity, pes planus and hypovascularity have been considered as significant risk factors for the development of PPT dysfunction. Rheumatoid arthritis can lead to the valgus deformity of the forefoot, with synovial inflammation and joint cartilage destruction. This can also cause rupture of the PTT as a consequence of tendon synovitis.

Congenital equinus of the foot with overload of the PTT can lead to this lesion. Also, the role of the accessory navicular bone and talocalcaneal coalition has been discussed in the etiology of PTT dysfunction.

CLASSIFICATION

There are various classifications of PTT dysfunction, according to etiology, magnetic resonance imaging (MRI) findings or stage of disease.

Muller classified the etiological factors into four groups: Type 1- Direct injury, Type 2- Pathological rupture, Type 3- Idiopathic rupture, Type 4- Functional rupture. A new classification of PTT dysfunction has been proposed according to the MRI finding (Table 1), while one based on the clinical expression-presentation, the classical Johnson-Strome classification, is still widely employed (Table 2). The Johnson-Strome classification is useful, since it determines prognosis and treatment options.

DIAGNOSIS

Diagnosis of PTT dysfunction is based on clinical findings: unilateral progressive hindfoot valgus deformity, associated with pain and swelling behind the medial malleolus. The pain is aggravated during walking. It is more common among female patients, aged over 40 years. In stage II there is loss of function and shape of the foot. In stage III the deformities are fixed. In stage IV, according to Johnson-Strome, the pain is more pronounced on the lateral than on the medial side, which are characteristics for the initial phases. The patients may have a sense of instability, may limp and the deformities become visible. On examination, from the posterior view planovalgus (flatfoot), flattening of the medial longitudinal arch and forefoot abduction deformity are obvious, and the single rise test is positive, as well as the “too many toes sign”. The first MT head sign and Achilles tendon shortening are evident.

X-rays are helpful. Weight bearing X-rays are more sensitive. Both lateral and anterior-posterior (AP) view are mandatory. X-rays can reveal dorsolateral subluxation of the navicular bone (i.e. limited talar head coverage), in-
creased calcaneocuboid, talonavicular, talar-first metatarsal angle, decreased calcaneal pitch (normally 20-25 degrees), dorsolateral peritalar subluxation and hallux valgus deformity. The absence or presence of degenerative changes in ankle and foot joints must be assessed. Soft tissue-edema, partial or complete PTT and other tissues are best seen with MRI but ultrasound examination is easier and cheaper.

TREATMENT

This depends mainly on the stage of progression of the flatfoot and it can be non-surgical and/or surgical.

Non-surgical treatment

It is generally considered that non-surgical treatment is insufficient in the treatment of PTT and acquired flat foot, but several studies have shown that such treatment with the use of orthosis can be a reasonable alternative option to surgical treatment initially, especially in stage I and II. It is not suitable for fixed deformities and in later stages of the condition.

Non-operative treatment consists of: rest, use of crutches, cast immobilization, orthoses, modification of footwear, short walking cast, ankle-foot-orthoses (AFO) and braces. The aim is to correct the flexible deformity. Medical treatment using NSAID suppresses pain. The goals of non-operative treatment are elimination of clinical symptoms, improvement of hindfoot alignment and prevention of progressive foot deformity. However, non-operative treatment is of limited value and is reserved for the first stage of PTT deformity, although it is advisable to start with conservative treatment.

Surgical treatment

Generally, surgical treatment is indicated when conservative treatment has failed after a period of 3 to 6 months. However, in the preoperative stage the exact etiology must be discovered, e.g. diabetes, rheumatic or neurological diseases, tarsal coalition. Also any associated deformities and contracture of the gastrocnemius muscle must be evaluated. The point of strongest pain, flexibility of the

FIGURE 2A:
PREOPERATIVE X-RAY OF THE FLAT FOOT

FIGURE 2B:
POSTOPERATIVE X-RAY AFTER THE CALCANEAL OSTEOTOMY AND MEDIAL DISPLACEMENT FIXED BY THE TWO CANULATED SCREWS - LATERAL VIEW

FIGURE 2C:
POSTOPERATIVE X-RAY AFTER THE CALCANEAL OSTEOTOMY AND MEDIAL DISPLACEMENT FIXED BY THE TWO CANULATED SCREWS- AP VIEW
foot and the integrity of the medial arch must be determined.

Surgical treatment can be divided into two main groups: soft tissue procedures (sinoviectomies, tendon repair, tendon reattachment, reinforcement, tendon transfer) and bone surgery (osteotomies, arthroeresis, arthrodeses). According to some authors, 80% of operated patients have a painless foot.

Surgical techniques

1. Synoviectomy, decompression, tenolysis
   Indicated in seronegative inflammatory disorders, without structural changes, synoviectomy can be performed in the isolated, initial phases. Besides sinoviectomy, necrotic parts of the tendon can be removed and side-to-side tenodesis with Flexor Digitorum Longus (FDL) can be added. This procedure is commonly associated with flexor digitorum longus transposition and elongation of the gastrocnemius muscle. The Flexor Hallucis Longus (FHL) can be used to transfer and repair the spring ligament. According to Kitaoka, these soft tissue procedures do not stop evolution of the disease and they are ineffective in most cases.

2. Transposition of the tendon-tendon transfer (Figure 1)
   The goal of this procedure is to counteract PTT hypofunction, if elasticity of the PTT remains normal. It can be done with tenodesis of flexor digitorum longus to the injured PTT, through a posteromedial approach, with careful exploration of the deltoid ligament, which can be re-

FIGURE 3:
SCHEMATIC PRESENTATION OF THE LATERAL COLUMN LENGTHENING WITH ILIAC CREST GRAFT, FIXED WITH THE PLATE AND SCREWS

FIGURE 4A
PREOPERATIVE X-RAY OF THE FLAT FOOT

FIGURE 4B,C:
POSTOPERATIVE X-RAY WITH THE IMPLANTED SCREW IN SINUS Tarsi-ARTHROESIS PROCEDURE
paired or substituted with the plantaris tendon. Another option is to do a partial FDL transfer to the navicular bone or medial cuneiform bone. Cobb and later Helal proposed anterior tibial tendon tendodesis in the case of ineffective PTT. Additionally, tightening of the talonavicular joint capsule is advised. Elongation of the Achilles tendon (AT) is sometimes advocated. This procedure allows normal hindfoot movement, but a disadvantage is limited correction of the deformity.

3. Medial calcaneal osteotomy with flexor transposition (Figure 2)

Compared with the above mentioned procedures, this type of surgical treatment allows deformity correction. Medial "sliding"-translation osteotomy of the calcaneus prevents valgus deformity through medicalization of the mechanical axis (Figure 2a,b,c). The approach is lateral oblique (under 45°) behind the peroneal tendons. Calcaneus must be moved medially by 1 cm and fixed with two (6.5 mm) spongious screws. In addition, cast immobilization for 6 weeks can be applied. This procedure is done in conjunction with tenosinoviectomy or/and tendon transfer, as well as eventual Achilles tendon lengthening.

4. Lateral column lengthening

This type of procedure was first described by Evans (opening wedge osteotomy of the calcaneus with insertion of bone graft). It is indicated in the second stage of the disease, when the deformity is flexible and the PTT is free and insufficient. It can be done through a lateral incision over the calcaneocuboid joint (Figure 3).

Nowadays, percutaneous placement of a device for enlargement of sinus tarsi is popular in correction of flatfoot deformity (Figure 4a,b,c), while arthroeresis indicated for the 2nd stage of flatfoot disorder.

5. Single, double and triple arthrodeses

These procedures are used in rigid deformities with associated inflammatory and degenerative arthritis, i.e. in stage III deformity (Figure 5).

Single arthrodesis could be: subtalar, talonavicular or calcaneocuboid.

Double or triple arthrodesis is recommended for stage IV of flatfoot deformity. Double arthrodesis includes: subtalar + talonavicular or talonavicular + calcaneocuboid.

Triple arthrodesis involves: subtalar + talonavicular + calcaneocuboid (Figure 6a,b,c,d)

Triple arthrodesis is done through two incisions:
- a medial approach presents the talonavicular joint, between tibialis anterior and posterior tendons and arthrodesis is fixed with a compression screw.
- the "olierv" incision allows approach to the calcaneocuboid joint, subtalar joint and lateral part of the talonavicular joint (with meticulous preservation of the sural nerve and superficial peroneal nerve branches). The tendon of extensor digitorum brevis must be moved distally, the sinus tarsi should be cleaned with a drill, the subtalar
and calcaneocuboidal joints must be fixed with screws. Rarely bone grafts must be used.

Treatment according to the stages of the condition. The nature of the deformity determines the type of surgical procedure:

In stage I conservative measures, tendon debridement, synovietomies, (and sometimes calcaneal osteotomy could be done)\(^2\). In stage II tendon transfer is performed in combination with corrective osteotomies. The mechanical deformity should be corrected by calcaneal osteotomy and be augmented by arthroereisis or lateral lengthening. PTT is augmented by splitting the anterior tibial tendon or FDL\(^2\). Achilles tendon lengthening or gastrocnemius resection could be done\(^2\).

In stage III correction is obtained through subtalar, calcaneocuboid and talonavicular articulations\(^2\).

Stage IV. Due to the degenerative changes in the ankle joint, the salvage procedure is pantalar arthrodesis (ankle, subtalar, calcaneocuboid and talonavicular) or total ankle replacement, supramalleolar osteotomy\(^2\).

**CONCLUSION**

Adult acquired flatfoot is a condition represented by a cascade of soft and articular tissue failures leading to dysfunction of the foot and ankle during everyday activities. The main cause of this condition is PTT dysfunction and underlaying comorbidities. Intervention with ankle foot orthoses has become the usual standard of treatment before surgical intervention. In later stages with a painful foot surgical treatment re-mains the gold standard for treatment and the type of pro-cedure depends on the stage of disease.

**SAŽETAK**

Simptomatsko stečeno ravno stopalo predstavlja važan ortopedski problem, zbog progresivnog gubitka funkcije stopala i pogoršanja pacijentovog funkcionalnog stanja. Ovo je kompleksan entitet, uključuje tetivu tibialis posterior, skočni zglog, zadnji i srednji deo stopala. U najvećem broju slučajeva tetiva tibialis posterior je uzrok stečenog ravnog stopala, ali postoje i drugi uzroci i uticaj ostalih faktora. Klinička slika varira u zavisnosti od stepena deformiteta, kao i pristup lečenju. Inicijalno se savetuju mekotkivne procedure, sinoviektomije, ojačanje tetive m. tibialis posterior. U II stadijumu se savetuje produženje lateralne kolumne i kalkanealne osteotomija, sa tetivnim transpozicijama (TA, FHL, FDL). U stadijumu III se savetuje subtalaruma, dvostruka ili tripila artrodeza, dok je u stadijumu IV indikovana pantalaruma artrodeza. U ovom radu su prikazani etiologija, klinička slika, dijagnoza i načini lečenja stečenog ravnog stopala usled lezije tetive m. tibialis posteriora.

Ključne reči: stečeno ravno stopalo, disfunkcija m.tibialis posterior, lečenje

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