Tibialis posterior dysfunction

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Tibialis Posterior Dysfunction: A Common and Treatable Cause of Adult Acquired Flatfoot

Graduate Thesis

Zagreb, 2014.

This graduate thesis was made at Department of Orthopaedic Surgery, University Hospital Center Zagreb, mentored by doc. dr. sc. Ivan Bojanić, and was submitted for evaluation during the academic year 2013/2014.

ABBREVIATIONS

PTTD	posterior tibial tendon dysfunction
MRI	magnetic resonance imaging
AFO	ankle-foot orthosis
FDL	flexor digitorum longus
NSAIDs	non-steroidal anti-inflammatory drugs
LAFO	low-articulating ankle-foot orthosis

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SUMMARY

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Posterior tibial tendon stabilizes the medial arch of the foot and has a crucial role during gait. Posterior tibial tendon dysfunction is a clinical condition characterized by hindfoot valgus, collapsed longitudinal arch, and abduction and supination of the forefoot. Over the past twenty years, the incidence of posterior tibial tendon dysfunction has been rising, most frequently occurring in middle-aged women. It is a common and treatable cause of adult acquired flatfoot. Posterior tibial tendon dysfunction is often misdiagnosed leaving the patient frustrated. The cause of posterior tibial tendon dysfunction is not completely understood since there are multiple factors that can contribute to posterior tibial tendon difficulties. There is usually swelling and tenderness in the line of tibialis posterior, at and distal to the medial malleolus. Patients often report change in the shape of the foot. Diagnosis is mainly clinical; however, history and imaging may help to support the diagnosis. Conservative care is possible in the earliest stages, whereas operative reconstruction and eventually arthrodeses become needed in the later stages. An increased awareness of posterior tibial tendon dysfunction is of importance for prompt diagnosis and correct management.

Key words: posterior tibial trendon dysfunction, flatfoot, ankle, nonoperative treatment.

1. INTRODUCTION

Posterior tibialis tendon dysfunction (PTTD) is accepted as the most common aetiology of the acquired adult flatfoot deformity. It is one of the pathologies that have increased dramatically in frequency over the past 20 years[1]. Despite increased awareness and intensive research over the past decade, PTTD is still frequently overlooked during diagnosis[2].

The aetiology of tendon dysfunction ranges from inflammatory synovitis to degenerative rupture and, occasionally, to acute trauma[3]. Myerson, Solomon and Shereff identified two groups of patients with dysfunction of the tendon. The first group consists of patients with a mean age of 30 years who may present with some form of systematic inflammatory disease. The second group is older with a mean age of 55 years, and dysfunction is caused by chronic overuse[3]. There is a strong predilection for middle-aged women, and the prevalence is known to increase with the degenerative processes of aging and often presents in the sixth decade of life[1,2]. Acute trauma is rarely the cause of injury[3].

PTTD is a slowly progressing condition that is characterized by hindfoot valgus, flattening of the longitudinal foot arch, and abduction and supination of the forefoot[2]. In its different stages, PTTD can cause rigid structural foot deformities and degenerative changes[4]. To prevent this, early diagnosis and treatment are crucial. A medical history and physical examination should be sufficient for diagnosis which can be confirmed with imaging modalities[2,5]. Nonoperative care is possible in the earliest stages, whereas surgical treatment becomes necessary in the latter stages[6]. This article will review the current concepts with regard to the pathomechanism, diagnostics, and treatment strategy of this debilitating condition.

2. ANATOMY

The posterior tibialis muscle is situated in the deep posterior compartment of the leg along with popliteus, flexor hallucis longus, and flexor digitorum longus muscles. It is the most deeply-seated muscle of this deep posterior compartment[2]. It originates from the posterior surface of the interosseous membrane, the lateral portion of the posterior surface of the body of the tibia and from the upper medial surface of the fibula[2]. The tendon forms in the distal third of the leg, runs within its sheath and passes immediately posterior to the medial malleolus where it changes direction acutely[3]. A groove, which is situated in the posteromedial aspect of the distal part of the tibia, holds the posterior tibial tendon but it is not deep enough to hold it in place after an injury, causing it to bow-string or dislocate[7]. It then passes deep to the flexor retinaculum and superficial to the deltoid ligament, and then beneath the plantar calcaneonavicular (spring) ligament inserting into different regions of the foot. In a fanlike manner, it inserts primarily into the navicular tuberosity and with other multiple extensions to the sustentaculum tali, three cuneiforms, cuboids and the bases of the second, third and fourth metatarsal bones[2,3,5].

The posterior tibialis tendon is innervated by the tibial nerve. The main source of the tendon's blood supply is the posterior tibial artery. Distal parts are supplied by branches of the posterior tibial and dorsalis pedis arteries[8]. The anterior part of the tendon has a significantly reduced intravascular volume in the retromalleolar region compared with that in the distal and proximal areas, leaving this zone hypovascularized and more prone to degeneration and possible rupture of the tendon under strain[8].

The posterior tibialis tendon is an important dynamic stabilizer of the arch. The healthy tendon functions as the plantar flexor of the ankle, invertor at the subtalar joint, and as adductor of the forefoot at the midtarsal joint, opposing the action of the peroneus brevis muscle[5]. During normal gait, contraction of the posterior tibialis muscle causes subtalar inversion thereby causing the calcaneocuboid and talonavicular joints (transverse tarsal joint) to lock. This locking creates a right lever for forward propulsion of the foot over the metatarsal heads caused by the gastrocsoleus complex[3].

3. AETIOLOGY

The causes of posterior tibialis tendon dysfunction are not completely understood since it is not only the tendon involved, but includes several surrounding structures as well. Hypertension, diabetes mellitus, seronegative arthropathies, steroid injections around the tendon and obesity have all been identified as risk factors[2,6,9].

Middle-aged women are most commonly affected, and the prevalence is known to increase with age[9].

Overpull of the opposing peroneus brevis muscle, overuse and injury are possible mechanical aetiologies[9].

The area of decreased vascularity in the tendon and the tendon's acute angulation as it passes posterior to the medial malleolus also contribute to the pathology[3,10].

4. DIAGNOSIS

4.1. History

In the early stages of the disease patients describe pain around the posteromedial aspect of the ankle. It is often exacerbated with activity and can radiate along the arch of the foot[7]. Pain is usually described as dull at rest, sharp with the aggravating activity, and participation in sports becomes difficult or impossible[3,6,11]. Swelling is common in the presence of tenosynovitis[3]. Symptoms may be present for months or years and patients will have consulted numerous doctors before presenting to a foot and ankle specialist[6,9]. There is often history of participation in a new sport or exercise, an increase in the intensity of physical activity and 50% of patients can recall a history of some form of trauma[6,11].

As the deformity progresses, patients report change in foot shape. A typical observation is the abnormal wear of the medial side of the shoes. They will also report pain at the lateral side of their foot secondary to calcaneofibular or lateral subtalar impingement[3,5,6]. Patients presenting later in the course of the condition can, therefore, present with medial as well as lateral pain[10,12]. The patient may also have a feeling of instability, a limp, a restricted walking distance, and an inability to walk on uneven surfaces[9].

Another reason why patients may seek medical attention is an increased awareness of other foot pathologies, such as bunions, hallux rigidus, and metatarsalgia[9].

There should be no presence of systemic symptoms[11].

Box. Symptoms suggesting PTTD[9]

- Pain and/or swelling behind the medial malleolus and along the instep
- Change in foot shape
- Decrease in walking ability and balance
- Ache on walking long distances

4.2. Physical Examination

Attentive clinical examination is crucial. Inspection should be made after legs, ankles, and feet are exposed. Gait analysis should be performed to recognize any symmetries or antalgic components[7]. Both feet should be inspected in a standing position with feet parallel, shoulder width apart from above as well as from behind the patient. Inspection includes hindfoot alignment, deformity, swelling, and muscle atrophy[2,3,11].

Forefoot abduction and valgus angulation of the hindfoot are best observed if the foot is viewed from behind[3,7]. Forefoot abduction is indicated by the so-called “too many toes” sign, which was originally described by Johnson[6]. The test is positive when more than normal (one and a half to two) toes are visible lateral to the ankle joint of the involved side than on the contralateral side, when viewed from behind. Valgus angulation of more than 10° (or valgus in bilateral comparison) is typically found in PTTD in stage II and above[2,9].

Another test that is a sensitive indicator of PTTD is a single limb heel rise test[6]. The patient is asked to attempt to rise on to the ball of one foot while the other is lifted off the floor. As the patient rises off the floor, the posterior tibial tendon inverts and stabilizes the hindfoot. In patients with PTTD the heel fails to invert and remains in valgus[2,6]. Incomplete heel inversion, difficulty or inability to raise the heel, or sensations of weakness or pain while performing this test are highly indicative of PTTD[7]. If unable to complete this test, the patient should perform a bilateral heel-rise in which the patient’s ability to rise on to the balls of his/her feet and the absence/presence of foot inversion is assessed again. In late stage, PTTD patients will frequently not be able to complete either test[2].

The first metatarsal rise sign was described by Hintermann and Gachter. With the patient standing, the shin of the affected side is grasped with one hand and externally rotated where the heel of the affected foot is passively aligned into a varus position. The head of the first metatarsal remains on the ground in normal function of the tendon but is lifted in the presence of PTTD[3,6]. This test can identify ligamentous attenuation and resultant forefoot deformity which must be considered when planning surgical interventions[1].

With the patient seated on the edge of the exam table with both feet hanging in the air, the integrity of the posterior tibial tendon and the site of maximum tenderness should be assessed[3]. This should be done by palpating the tendon from above the medial malleolus to its insertion onto the navicular[6]. Since lateral impingement occurs in the later stages of disease, the tip of the lateral malleolus should also be palpated for possible tenderness[7].

The strength of the posterior tibial tendon should be evaluated by asking the patient to bring the foot into an inverted and plantarflexed position from an everted and dorsiflexed position against the examiner's resistance[3]. This should minimize the contribution of the anterior tibial muscle to invert the foot and allow the strength of the tendon to be assessed more accurately. The subtalar and ankle joint are then assessed for mobility[1,3].

Assessment of the Achilles tendon for contractures is most important[3]. Once heel valgus exceeds the normal range, the Achilles tendon moves lateral to the axis of the subtalar joint, the gastrocnemius group shortens and contractures of the Achilles tendon can develop[3,7]. If the subtalar joint is mobile, Achilles tendon contractures should be evaluated by dorsiflexing the ankle with knees both flexed and extended while passively correcting any valgus angulation of the heel[7]. The hindfoot angle should be measured in a relaxed position and with a corrected, neutral position of the subtalar joint. This provides an assessment of the relative flexibility and reducibility of the deformity, which can be important parameters in the choice of surgical procedures or in the selection of appropriate ankle foot orthosis for correction[1].

4.3. Imaging

The diagnosis of PTTD is essentially clinical; however, plain radiographs of the foot and ankle are useful for assessing the degree of deformity and to confirm the presence or absence of degenerative changes in the subtalar and ankle articulations[9]. Conventional radiologic imaging consists of anteroposterior and lateral weight-bearing radiographs of both feet and mortise views of both ankles[2,3].

In the early stages of the disease, radiographs may reveal normal findings or show minimal changes of angular deformity[6]. With increasing deformity, the AP view shows an increase in the talar-first metatarsal angle (normal angle, 0 to 10 degrees) and lateral subluxation of the talonavicular joint with the navicular sliding laterally on the talus, uncovering the talar head[2]. The lateral view reveals an increase in the talar-first metatarsal angle, a decrease in the lateral talocalcaneal angle, flattening of the longitudinal arch and subtalar arthritis[6,10]. Anteroposterior radiographs of the ankles with the patient standing may reveal talar tilt due to deltoid dysfunction, degeneration and impingement between the calcaneus and the fibula[2,10].

Further imaging should be considered if the patient fails to respond to conservative management, the diagnosis remains unclear, or there is pain disproportionate to the injury[11].

Ultrasonography has had an increasing role in the evaluation of pathology within the posterior tibial tendon[10]. A swollen tendon, an irregular contour, longitudinal splits, heterogeneous echogenicity, and an empty tibial groove at the level of the medial malleolus in cases of rupture may all be revealed by ultrasound[3].

Magnetic resonance imaging (MRI) has an advantage for assessment of soft-tissue pathology and may also aid in surgical planning[5]. MRI may show variable amounts of tendon degeneration, tenosynovitis, splits, ruptures and arthritic changes in the talonavicular, subtalar, and tibiotalar joints[3,10].

5. CLASSIFICATION SYSTEM

PTTD ranges from tenosynovitis to fixed deformity[2]. In 1989 Johnson and Strom described a widely accepted classification system, which was later modified by Myerson et al. The classification describes the progression of PTTD and serves as a guide to management[2,3,7]. (Table)

Table. Classification and treatment recommendation for PTTD[2,7]

	Stage I	**Stage II**	**Stage III**	**Stage IV**
Tendon Pathology	Peritendinitis and/or tendon degeneration	Elongation and marked tendon degeneration	Elongation and marked tendon degeneration	Marked tendon degeneration
Deformity	Mobile hindfoot, normal alignment	Mobile hindfoot, valgus alignment	Fixed deformity, valgus position	Additional angulation of the talus and early degeneration of the ankle joint
Pain	Medial:focal, mild to moderate	Medial: along posterior tibial tendon, moderate	Medial: possibly lateral, moderate	Medial and lateral, distinct pain
Single limb heel rise	Normal inversion of hindfoot	No or reduced inversion of hindfoot	Unable to perform test, no inversion	Unable to perform test, no inversion
Too many toes sign	-	+	+	+

Nonoperative treatment	Medial heel + sole wedge Period of immobilization Therapy	Orthotic support (molded, articulated AFO)	Rigid AFO	Rigid AFO
Operative treatment	Tenosynovectomy Repair	FDL tendon transfer Calcaneal osteotomy Lateral column lengthening Heel cord lengthening	Triple arthrodesis	Tibiotalocalcaneal arthrodesis Triple arthrodesis with total ankle arthroplasty (experimental)

AFO, ankle-foot orthosis; FDL, flexor digitorum longus

6. TREATMENT

Treatment is based on an accurate staging of the disease with both nonoperative and operative treatments intended to correct the abnormalities particular to that stage of disease and to prevent progression to the next stage[6]. Treatment is determined by the age, weight, and level of activity of the patient, along with the severity of the deformity[13].

6.1. Nonoperative

In the majority of cases, for the initial management of symptoms and disability a nonoperative treatment would be preferable[1]. The objectives of nonoperative treatment comprise the elimination of clinical symptoms, improvement of hindfoot alignment, and the prevention of progressive foot deformity[6]. Patients are encouraged to lose weight, improve footwear and decrease repetitive loading[14]. Three point force systems applied above and below the ankle-hindfoot complex as provided by ankle-foot-orthoses (AFO's) are required for the successful treatment of intermediate to advanced stages of PTTD[1]. Nielsen et al. reported on 64 consecutive patients with a diagnosis of posterior tibial tendon dysfunction treated with physical therapy modalities; medications, such as non-steroidal anti-inflammatory drugs (NSAIDs); oral administration or local infiltration of corticosteroids; and orthotics or bracing, such as a foot orthoses, an arch and ankle brace, a low-articulating ankle-foot orthosis (LAFO) or similar AFO, cast-boot (**"**cam**"** walker), or shoe modifications. The authors reported an 87% success rate (defined as not requiring further surgical treatment). Similar results have been reported by other authors evaluating nonoperative management of PTTD with success rates ranging from 67%**–**90%[15].

Stage I

In Stage I, there has not been apparent structural change in the foot, and the patients can still raise their heel without difficulty[1]. This stage is characterized by tenderness over the posterior tibial tendon with a variable amount of oedema and warmth, consistent with tendinosis or tenosynovitis[7]. Before the chronic aspect of the

condition is treated, any acute inflammation surrounding the sheath of the posterior tibial tendon should be dealt with first[9]. The treatment depends on the level of symptoms reported by the patient[7].

Short-term immobilization (2-6 weeks) with a rigid walking boot or pneumatic walking boot will usually decrease the severity of the symptoms[1]. Weightbearing is allowed, and non-steroidal anti-inflammatory drugs are used to relieve the pain. After a 4-6 week period of immobilization, patients are progressed in a comfortable shoe with an orthotic with arch support and a medial heel post to correct hindfoot valgus[7,15]. Strengthening, stretching, proprioceptive and muscular re-education of the tibialis posterior, peroneous longus, and gastrocnemius-soleus has proven to be effective. The combination of these exercises has been shown to be more effective than orthoses alone.significantly diminish the extent of hindfoot pronation more than orthotics alone[4,16,17].

Treatment is continued for 3 to 6 months prior to considering operative intervention[7].

Stage II

In Stage II, there has been attenuation of the posterior tibial tendon, accompanied by noticeable change in foot structure alignment with loss of one or more critical ligaments in the hindfoot; however, deformity in this stage is still flexible and reduceable[1]. The treatment includes a supramalleolar orthotic or molded articulated ankle-foot orthosis (AFO), which may serve to control the deformity and reduce symptoms. Study by Lin et al. demonstrated a 70% success rate at a 7-10 year follow-up for nonoperative treatment of stage II PTTD using a double upright AFO for an average bracing period of 14 months[7].

Stage III and IV

In Stage III, the deformity is now more rigid and arthritic changes become visible on radiographs[1]. Over time, without operative correction or treatment to prevent progression, patients with stage III will progress to stage IV, which is

characterized by arthrosis involving the ankle joint as well[7]. Whether the patient demonstrates the Stage III or Stage IV findings, the nonoperative treatment should be more aggressive in terms of restricting ankle and hindfoot motion[1]. A more rigid AFO, such as an Arizona-type brace or an articulated AFO is recommended. At this point, the goal is to prevent progression by accommodating the deformity, correcting any correctable deformity, and controlling pain. If patients do not obtain relief within 3 to 6 months, or if they cannot tolerate bracing, operative intervention should be considered[7].

6.2. Operative

Once conservative treatment options fail, operative intervention is the next step. When surgery is the treatment of choice, there are several options. The choice on which sort of procedure should be completed depends on the stage of deformity that the patient is in.

Stage I

Operative treatment includes tenosynovectomy, in addition to debridement or repair of degenerative areas or tears. This can be done by both open and tendoscopic techniques. An open procedure allows an easier identification and inspection of the local anatomy[7]. Ankle tendoscopy is a newer technique and its advantages over open techniques include low postoperative morbidity, less blood loss, shorter hospital stay and a faster recovery[18]. If hypertrophic tenosynovium or a necrotic portion of the tendon is encountered, as evidenced by discoloration, attenuation, and longitudinal rents, they may be excised and the remaining part of the tendon repaired in an end-to-end fashion[6]. Postoperatively, there is a 3-week immobilization followed by wearing a boot with controlled ankle movement for another three weeks and physiotherapy. Six weeks after operation a stirrup ankle brace may be worn six weeks after the operation[3].

Stage II

Today, operative treatment of Stage II rarely involves an isolated procedure but rather a combination of bony and soft-tissue procedures done during a single procedure[14,19] The explanation behind this approach is that the osteotomy is required to correct the bony architecture of the foot in order to optimize the biomechanics of the reconstructed tibialis posterior tendon[9]. The reducible stage II can be approached with calcaneal osteotomy (medializing or lateral column lengthening) and/or medial column stabilization with posterior tibial tendon augmentation as well as spring ligament repair[1]. The treatment at this stage is most controversial, with some physicians performing lateral column lengthening and other not, instead resorting to fusions or performing less correction of alignment. Whatever the treatment, the surgeon should seek to obtain good alignment and retain as much function as possible[12].

Flexor digitorum longus (FDL) tendon transfer and transfer of a split anterior tibial tendon are the two recommended tendon reconstruction techniques (Cobb procedure). Attaching a part of the anterior tibial tendon, split proximally near the belly, through a drill hole to the plantar aspect of the medial cuneiform allows the posterior tibial tendon to pull at its physiological insertion site. Another benefit of the Cobb procedure is that it decreases the tension of the anterior tibial tendon, thus in some degree preventing anterior tibial tendon ruptures[2].

Another method is augmentation of the incompetent posterior tibial tendon with the FDL[14]. The FDL tendon is detached distally, just proximal of the juncture with the flexor hallucis longus (Henry's knot). The periosteum over the navicular is then dissected and FDL tendon is passed from plantar to dorsal through a drill hole made in the tuberosity through a drill hole from the dorsal to plantar aspects. The tendon is sutured side-to-side to the posterior tibial tendon and passed through the drill hole from plantar to dorsal[2]. The objective of this augmentation is to restore the dynamic function of the posterior tibial tendon. Some surgeons prefer to use the flexor hallucis longus on the basis of its increased strength relative to the flexor digitorum longus[14].

However, concerns that it crosses the neurovascular bundle and its important role in the push-off phase of the foot may limit its popularity[2,14].

Intraoperative exploration of the spring ligament in PTTD is compulsory, because of its frequent concomitant degeneration of rupture. If found ruptured, reconstruction has to consider both components of the spring ligament complex. If medial ankle instability is suspected upon clinical examination, an ankle arthroscopy needs to be performed to rule out deltoid ligament involvement[2].

Correction of an equinus contracture with either a percutaneous Achilles tendon lengthening or gastrocnemius recession is commonly performed. A gastrocnemius recession is prefered if the equinus contracture is isolated to the gastrocnemius[14].

The pathological bony alignment can be corrected by various osteotomies of the calcaneus[2]. Unfortunately, a single procedure will not 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[14].

If a hindfoot valgus deformity has occured, a medial calcaneal slide osteotomy may be necessary and it should be performed prior to fixation of the tendon. A sharp incision is made down through the skin over the lateral aspect of the calcaneus just posterior to the peroneal tendon. It is carried down through subcutaneous tissues while care is taken to avoid injury to the sural nerve. Dissection is carried down along the lateral aspect of the calcaneus[7]. An oblique transverse osteotomy is made in the calcaneus parallel to the incision following relaxed skin tension line with use of an oscillating saw. The cut is made perpendicular to the lateral border of the calcaneus and is inclined posteriorly at an angle of approximately 45 degrees. The posterior fragment of the calcaneal tuberosity is translated medially ten or more milliimeters and is secured with a cannulated headless compression screws[2].

In PTTD with pronounced hindfoot valgus and distinct forefoot abduction, a lateral calcaneus lengthening osteotomy is recommended. Lengthening the lateral column restores the medial longitudinal arch secondarily to the induced adduction movement of the forefoot that supinates the foot at the subtalar and talonavicular

joint[2]. There are two techniques for lengthening the lateral column, the “classis” Evans method, which is performed 1.5 cm proximal to the calcaneocuboid joint, and the calcaneocuboid joint distraction arthrodesis. For the classic Evans procedure a transverse incision of approximately 4cm in length is made over the lateral aspect of the anterior calcaneal tuberosity. With an oscillating saw the osteotomy is performed parallel to the calcaneocuboid joint, taking care not to transect the medial cortex. Using a laminar spreader, the osteotomy is distracted[3]. Depending on the desired lengthening, a tricortical graft or alternatively allograft bone is inserted into the distracted osteotomy until it is in the level and resting within the lateral surface of the calcaneum. The graft is fixed with one 3.5 mm cortical screw. Care is taken not to penetrate the calcaneocuboidal joint[2,3]. For the calcaneocuboid distraction arthrodesis a transverse incision is made over the calcaneocuboidal joint. Extensor digitorum brevis is split and the calcaneocuboid joint is exposed. After distracting the calcanecuboid joint, appropriately-sized bone graft is inserted. The arthrodesis is either fixed by crossed cortical lag screws or an AO H-plate[3].

Stage III

Arthodesis of one or more joints of the hindfoot are favored in this stage where rigidity and arthritis are key findings[1]. The aim of operative treatment is correction of the deformity and pain relief[2]. Most commonly, a subtalar or triple arthrodesis of the subtalar, calcaneocuboid, and talonavicular articulations are performed. This correction of alignment should be achieved without overcorrection. The talonavicular joint should be fused in neutral position without excessive abduction, but also without supination. The metatarsal heads should be even to the floor with the heel in neutral position. Calcaneocuboid arthrodesis can be omitted to sustain residual motion of the lateral column[2,12].

Stage IV

If flexible deformities in the foot are present, they should be corrected as described in Stage II. On the other hand, correction of the fixed deformity is performed as described in Stage III[12]. Medial displacement calcaneal osteotomy, deltoid ligament reconstruction, or a combination of the two can also be considered, but long-term results are not available. If the ankle joint is irreducible, options include arthrodesis or arthroplasty. Arthrodesis can be performed using external fixation, internal fixation, or retrograde intramedullary nailing. A variety of implants are available for ankle arthroplasty, but the procedure is still not widely accepted[7].

7. CONCLUSION

PTTD is a common but often overlooked condition, one from which patients may suffer for years without a correct diagnosis. It is a common cause of adult acquired flatfoot. Since PTTD can result in significant disability, it is very important to recognize the condition early. In this review, several clinical examination maneuvers for easier evaluation and detection of this condition have been described. As previously mentioned, early diagnosis and staging are paramount in order to determine the course of and to begin proper treatment. Aside from the clinical examination, the patient's history and imaging aid in establishing the diagnosis. Depending on the stage, treatment can be either operative or nonoperative, and there are numerous options for either approach as described in this review. If it is determined that operative treatment is required, the surgeon should propose the best surgical procedure for the patient after having reviewed all the clinical, radiographic, and imaging tests. As the diagnosis is mostly clinical, it is especially important for clinicians to be aware of the specific signs, symptoms and risk factors associated with PTTD; a combination of proper anamnesis and the use of medical imaging tools can allow for early diagnosis, providing the patient with the best possible prognosis and limiting the amount of disability they may experience. Ultimately, the goals of the operative procedure are to alleviate the patient's symptoms and pain, restore normal foot alignment, and limit the loss of foot and ankle function, without causing any complications. It is important to correct, but not over correct, the deformity. Achieving the result of a well-aligned foot, without excessive stiffness, is a fundamental goal.[5,12]

ACKNOWLEDGMENTS

I am most thankful to my mentor doc. dr. sc. Ivan Bojanić for all the given material, for his patience, assistance and guidance in the preparation of this thesis.

I would also like to thank doc. dr. sc. Tomislav Smoljanović for all of his help and kindness.

I am also thankful to my colleagues Joseph Patten and Benjamin Aksungur for their unselfish assistance with the technical details of this graduate thesis.

And finally, special thanks go to my mother Mira, for all the sacrifices she made for me to become what I am today, and to my sisters and numerous friends that have supported me throughout all the years of schooling.

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BIOGRAPHY

Birth, Education

I was born on August 27th, 1989 in Zagreb, Croatia. After finishing my primary education and first grade of high school in Zagreb, I moved to the United States where I continued my education and graduate from high school in 2007. Soon after, I enrolled in college where I took pre-medical classes. In 2008, I started my first year of medical school at the Medical School of Zagreb, Croatia. I am currently at the end of the 6th year, and I am on course to graduate in July this year, 2014.

Work Experience

During the 6 years of my tertiary education I have kept myself busy not only with studying but also with part time jobs for Bayer Healthcare Pharmaceuticals, such as vitamins promotion, office and warehouse work. On the academic side, I was mentoring lower years on the Internal wards. In 2014, I had an incredible experience doing a one-month clinical rotation at the Department of Orthopaedic Surgery at University Hospital of North Norway in Harstad, Norway.

Activities

Up until 2010, I played indoor and beach volleyball actively. I played for both primary and secondary school in both Croatia and USA. I also played for a Croatian team which was part of the Croatian Second League. In 2006, my beach volleyball partner and I wont first place at the tournament U-18 ‘’Sportske Igre Mladih’’. In 2013, I participated at 13th Zagreb International Medical Summit where I presented a poster, made by three colleagues of mine and I, on the topic of ‘’Dislocation of Humeroscapular Joint – Iatrogenic Injury of Brachial Artery’’.

Interests

Asides from medicine, my interests include sports, travelling and languages. I would like to specialize in Orthopaedic Surgery because it is a field that interests and fascinates me the most, partly because of my love for sports, and partly because it deals with mobility and improves patient’s quality of life.