ADULT ACQUIRED FLATFOOT DEFORMITY: A REVIEW AND GAIT ANALYSIS OF POSTERIOR TIBIAL TENDON DYSFUNCTION

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Abstract

The posterior tibial tendon plays a key role in positioning the foot for effective propulsion during gait. Posterior Tibial Tendon Dysfunction (PTTD) is a common tendinopathy that leads to flatfoot deformity with a profound decrease in gait function. Single leg heel rise (SLHR) is a key functional test for assessing PTTD as its successful completion requires dynamic stabilization of the hindfoot by the PTT. Numerous studies have investigated differences in gait kinematics and kinetics between patients with PTTD and healthy controls, but there is limited data comparing the stability during SLHR between these groups. The purpose of this study was to: (1) summarize the previous findings of gait alterations with PTTD in order to obtain a comprehensive understanding of the differences between PTTD patients and healthy control, and (2) to extend these findings by comparing differences in frontal plane foot kinematics and kinetics during gait and SLHR. The results from this study showed conclusive changes in foot kinematics and kinetics of PTTD patients during both gait and SLHR. These changes may be associated with the pathological process of PTTD and provide a more comprehensive understanding in optimizing treatment and standardization of care of PTTD.

Introduction

1.1 Anatomy and Biomechanics

The posterior tibial tendon's (PTT) primary purpose is to elevate the medial longitudinal arch into the hindfoot and assist in inverting the midfoot across the transverse tarsal joint (the talonavicular and calcaneocuboid joints)[1]. During midstance of gait, the medial longitudinal arch is elevated and the hindfoot is inverted, effectively transforming the foot from a shock absorber into a rigid lever that is capable of transferring force to the ground for push-off. PTTD is a tendinopathy which often leads to the more common clinical diagnosis of Adult Acquired Flatfoot Deformity (AAFD). When left untreated AAFD can lead to severe impairments in gait function. The Tibialis Posterior is a deep muscle, originating from the posterior tibia, fibula, and interosseous membrane running along the posterior side of the lower leg into the gastrocnemius-soleus complex involved in plantarflexion of the foot. The tendon inserts posterior to the medial malleolus and into the navicular tuberosity with multiple insertions out across plantar aspect of the foot. The main blood supply to the posterior tibial tendon is the posterior tibial artery. Many PTTD patients have displayed hypovascularity to the retromalleolar tendon, most likely limiting the individual's recuperative ability against repetitive microtrauma during gait.

The role of the Tibialis Posterior and the PTT changes throughout gait to allow the foot to dynamically relax as stance phase begins and stabilize to propel the foot into swing phase. In normal, healthy gait the foot begins inverted during the heel strike phase. As the foot transfers from heel strike to midstance, the PTT relaxes, which allows the hindfoot to reach a valgus position and the midfoot to abduct through the transverse tarsal joints [3]. When the heel is in valgus and the foot is in full contact with the surface, it is positioned to absorb the force of the stride during midstance. As late-stance begins, the Tibialis Posterior contracts to activate the PTT to invert the hindfoot and lock the talonavicular and calcaneocuboid joints along divergent axes. The hindfoot is now positioned into varus and the entire foot is acting as a rigid lever for the gastrocnemius-soleus complex to execute toe-off phase by propelling the foot forward into swing phase. The inability of PTTD patients to achieve the rigid lever and minimize hindfoot valgus prior to heel-rise leads to impaired gait and reduced force transfer to the ground. With the

PTT acting as the primary support of the medial longitudinal arch of the foot, any injury to the tendon can impinge on normal gait tasks, ankle strength, and mobility that, if left untreated, can negatively impact a patient's quality of life.

1.2 Etiology

Individuals with congenital flatfoot, rheumatoid arthritis, or hindfoot trauma (e.g., PTT rupture) are more likely to develop PTTD [5-10]. The chronic insufficiency of the PTT can lead to severe foot deformity and abnormal articulation between the four-foot segments (tibia, hindfoot, forefoot, and hallux). Often, the end-result of this chronic insufficiency is a severe decrease in ambulatory function. It has also been identified by numerous investigators that many PTTD patients (60%) who have displayed ruptures of the PTT also present with multiple co-morbidities or a family history of hypertension, obesity and diabetes. Prior procedures and steroidal injections to the medial aspect of the foot have also been linked to PTTD [19]. The failure of the tendon most likely arises from the repetitive microtrauma of normal daily tasks in addition to intrinsic abnormalities of the medial foot anatomy rather than acute or extrinsic injury. Due to the origination of the PTT just distal to the medial malleolus, the small region of hypovascularity affects the tendon more than other medial foot soft-tissue structures. Frey et al. showed that the PTT has a zone of hypovascularity that begins 1 to 1.5 centimeters distal to the medial malleolus and extends 1 centimeter farther distally. This area of hypovascularity is often where degenerative changes in the tendon caused by inflammatory disorders occur [20]. The degenerative changes identified in the medial foot aspect have been highly associated with the obvious flatfoot deformity and its pathological gait of the midfoot and hindfoot structures.

1.3 Diagnosis and Classification

Many patients with AAFD often report a gradual onset of pain in the medial ankle and heel, along with worsening flatfoot deformity. As the patient's pain progresses, there seems to be a marked increase in ankle swelling and a shift from reporting pain medially to the lateral aspect of the ankle. With a complete rupture of the PTT obvious flat-foot deformity presents in fixed valgus of the hindfoot and the patient is unable to complete a SLHR. Fatigue during gait and other normal, daily tasks along with pain

associated with the patient's typical footwear are also common symptoms. Upon examination, the patient's foot is viewed from many angles, including from above and behind to assess hindfoot valgus deformity and angulation. Classic presentation of AAFD typically involves the "too-many-toes" sign, caused by the forefoot abduction which pushes the toes laterally which become visible when viewed from behind (Figure 1). In terms of dynamic stability, clinicians often use the single leg heel rise (SLHR) test to determine the strength and function of the PTT. A healthy individual will be able to rise up onto the ball of their foot without the assistance of the other leg and display an inverted hindfoot, stabilized by a healthy PTT. A patient with an injured or diseased PTT would display inability to rise up onto one foot, or have their hindfoot remain in valgus even in complete heel rise. Along with dynamic movement being used as a diagnostic tool, the physician will provide manual resistance against a plantarflexed and inverted foot position to assess the integrity of the spring and deltoid ligaments. Palpation for tenderness allows for assessment of mobility along the subtalar and ankle joints in conjunction with shortening of the gastrocnemius-soleus complex.

Classifying the severity of the tendon dysfunction and flatfoot deformity along with reported pain and mobility upon examination is placed on a four-stage spectrum. The degree of flatfoot deformity, especially the amount of supination in the forefoot and rigidity of the subtalar joint, may affect the treatment selected to improve valgus deformity if possible [1]. In addition to physical examination, anteroposterior and lateral weight-bearing radiographs are useful for confirming physical exam findings and can provide objective severity of midfoot and hindfoot joint misalignment. Ultrasound has also been investigated as a useful tool in assessing the integrity of the PTT in an inverted and plantarflexed position. Each stage of PTTD is characterized by a progression of reported symptoms and worsening deformity. In Stage I, pain and swelling are often localized to the medial aspect of the ankle, but no significant increase in deformity has been identified, although tendinitis may be associated with signs of degeneration. Stage II involves a partial or complete rupture of the tendon and secondary to increased pain is a worsening flatfoot deformity, with midfoot pronation and forefoot abduction being most notable upon examination. In stage II, the patient notes significant decrease in foot strength and stability and is unable to perform

SLHR. A severe flatfoot deformity and decreased midfoot and hindfoot mobility places the tendinopathy into Stage III. Lastly, Stage IV, which was recently added by Myerson, is characterized by valgus angulation and early degeneration of the ankle joint [1].

1.4 Treatment

Early treatment of PTTD is often targeted as non-operative, symptomatic management. Non-steroidal anti-inflammatory medication, orthotics, and bracing are often combined with physical therapy in the first 6 to 12 weeks of treatment. PTTD patients with stage-I disease are able to perform SLHR with some pain, as well as resist manual pressure in an inverted position. Immobilization and anti-inflammatory medications typically provide positive results for these patients. Orthotics are useful for targeting the flatfoot deformity with a medial heel-and-sole wedge to remove the hindfoot from valgus and into inversion. Stage II-III PTTD may respond well to non-operative treatment, however there is a risk of progressive deformity and worsening symptoms without more direct intervention.

After non-operative treatment has been determined unsuccessful, operative treatment is planned based on classification of the patient's symptoms and deformity. Minimally invasive procedures, such as a tenosynovectomy, are suggested first. More invasive procedures such as calcaneal osteotomy or ankle joint arthrodesis are used with more severe deformity or failure of previous procedures.

1.5 Kinematic and Kinetic changes

The changes in gait kinematics and kinetics associated with PTTD have been studied by a larger number of investigators, however the etiology of PTTD and the development of AAFD is uncertain. Treatment of PTTD is determined by classification and progression of the tendinopathy, however there is no correction to the biomechanics of the foot during gait that prevents re-injury or further exacerbation of the symptoms. Kinematic changes observed in multiple studies with PTTD patients have displayed a decrease in ankle sagittal plane power during the stance phase of gait [7, 21]. Abnormal changes in gait have been found in patients with PTTD that can aggravate the symptoms already present and lead to development of the tendon dysfunction. It has been reported that everted hindfoot is a common clinical observation in patients with PTTD. Hindfoot eversion is considered another contributor to the unlocking

of the bones in a flexible foot. A previous study found that an everted hindfoot resulted in more forefoot mobility in a cadaver model [14]. More specifically, this study found increased forefoot range of motion in the sagittal plane when the hindfoot was everted. Therefore, it appears that greater hindfoot eversion accounts for decreased ability to form a rigid foot, which may lead to decreased energy transfer during the late-stance [13]. Current understanding of surgical and non-surgical treatment of PTTD tends to focus on correcting the symptoms and physical presentation, however the underlying cause of the tendinopathy has yet to be identified through kinetic and kinematic parameters during gait.

A variety of studies have applied multi-segment foot models to evaluate the complex 3D movement of the foot segments in PTTD [4, 11-20]. These studies have quantified differences in multi-segmental foot kinematics between patients with PTTD and healthy controls, uncovering some of the pathomechanics of PTTD. For example, a few studies have found that patients with PTTD have increased dorsiflexion and abduction of forefoot [4, 11-13, 15], as well as increased plantarflexion and eversion of the hindfoot [4, 11, 13-16, 19] as compared to healthy controls. Other studies have found that these kinematic changes appeared to result in a medial shift of the center of pressure [17, 24-26] and a decrease in ankle sagittal plane power [4, 20] during the stance phase of gait. Despite these being significant findings in abnormal foot kinematics from the progression of PTTD, the generalizability of these studies is limited due to the small number of patients.

PTTD patients in later stages of tendinopathy present during the physical exam with obvious deformity and valgus of the hindfoot. Gait in healthy control patients show a natural progression of eversion during heel-strike to inversion with late-stance. PTTD patients display increased eversion throughout their gait cycle, transferring the center of pressure medially. Frontal plane kinematics and kinetics during gait analysis and during double/single-leg heel-rise should expect to show increase in hindfoot and tibial joint moments and increased valgus/eversion at the ankle. The purpose of this study is to provide an encompassing literature review of gait alterations associated with PTTD as well as examining frontal and sagittal plane kinematics and kinetics during gait and SLHR.

2. Methods

2.1 Subjects

All motion capture collections were performed at the University of Arizona Human Movement Biomechanics Lab (HMBL). A total of 10 stage II PTTD individuals and 10 healthy controls were recruited for this study. All PTTD patients were age and gender-matched. The 10 PTTD patients were assessed by a fellowship-trained orthopaedic surgeon for a physical examination and their ability to perform SLHR. Anthropometric and demographic information was collected and compared across study participants.

2.2 Motion Analysis testing protocol

The multi-segment Oxford foot model was used to collect motion capture data for PTTD and healthy control participants (Figure 2). The HMBL consists of a 10m walkway with a single forceplate in the middle of the floor. Kinematic data was obtained using a 9-camera Vicon Nexus Motion Analysis System (Vicon, Los Angeles, CA). Kinetic data was collected at 120Hz using a single AMTI Kistler force plate (AMTI, Watertown, MA). Reflective markers were placed with respect to anatomical bony landmarks corresponding to the gait model for biomechanical assessment. The Oxford foot model was used to define the four segments of the affected foot within the sagittal, frontal, and transverse planes during gait and SLHR (Figure 3).

After marker placement was confirmed, static and dynamic trials were conducted. Static trials were collected with the participants standing on the force plate in relaxed, anatomical position to provide neutral reference of the pelvis and lower extremities. Dynamic trials consisted of walking at a self-selected pace with the affected limb striking the force plate during gait (Figure 4). Participants were allowed practice attempts to ensure their gait was as natural as possible and their affected limb was completely striking the force plate throughout the entirety of the stance phase. A minimum of three trials for each dynamic condition were collected. Single leg and double leg heel rise, as well as balance during single leg stance and double leg stance, was also collected if the PTTD participant was able to complete these conditions without exacerbation of their pain (Figure 5).

3. Discussion

While data collection and processing has been completed, further analysis of the data in MATLAB is required for a final assessment to be made on the efficacy of the pilot study. Upon analysis, the completion of the study should give us a further objective explanation of the differences in frontal and sagittal plane kinematics and kinetics between patients with PTTD and healthy individuals. It is expected that the gait and motion of PTTD patients would display much more compensatory strategies for reducing impact on the affected foot and ankle. This, however, can be seen in the clinical setting by the physician, however quantifying these compensatory strategies as well as comparing them to the progression of the PTTD would allow for standardization of treatment for PTTD patients. If it can be proven that early stage PTTD patients who use orthotics and bracing, combined with a course of physical therapy, can avoid development of abnormal foot kinematics then the understanding of treatment for PTTD would be further improved.

The study had limitations in both sample size, with only twenty total participants, as well as differences in the ability to perform all tasks. There were multiple PTTD patients who were unable to complete the single leg stance balance or the SLHR at the baseline data collection. One patient also required an assistive walking device for the gait trials, which may have disrupted their natural gait pattern while taking larger steps to cross the force plate. While inability to perform these daily tasks does not affect the analysis of the data, ensuring a complete dataset between baseline and follow-up can be challenging. The results from this study would be particularly useful in understanding the efficacy of certain non-operative treatment in reducing overall stress and impact at the ankle joint especially during gait and SLHR. Combining the results of this study with patient-reported outcome measures would also be of interest in assessing lifestyle choices and improvement or decline of functional outcomes in physical therapy or conservative use of bracing and orthotics to treat PTTD. Additional research on the efficacy of treatment in early-stage PTTD patients in the prevention of abnormal foot kinematics is of critical importance in the decision-making process since non-operative treatment often fails in PTTD patients as their gait and overall foot function rapidly declines.

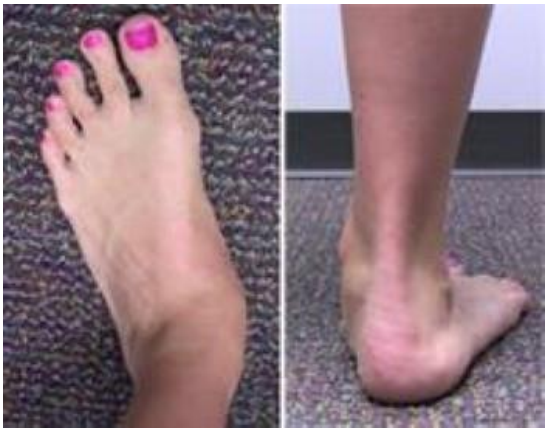

Figure 1. "Too Many Toes" Sign during physical examination

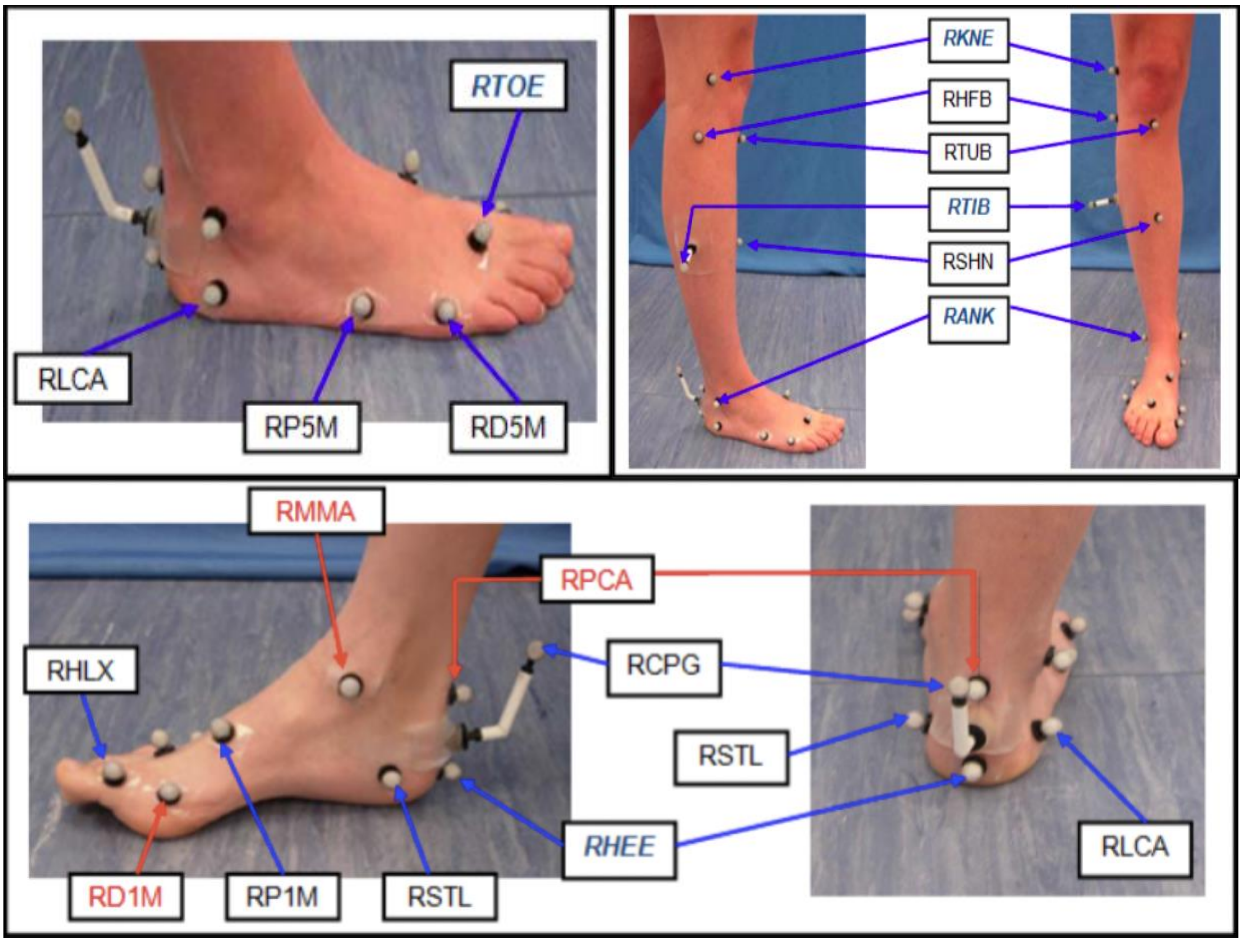

Figure 2. Multi-segment Oxford Foot Model

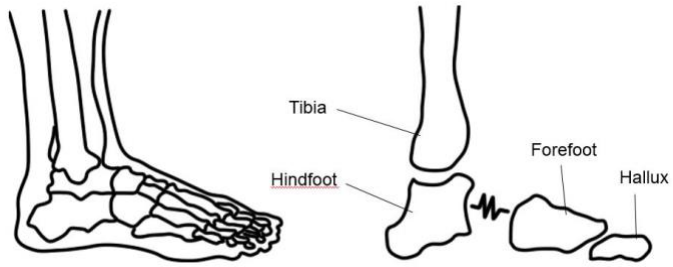

Figure 3. Four-foot segments defined by Oxford Foot Model

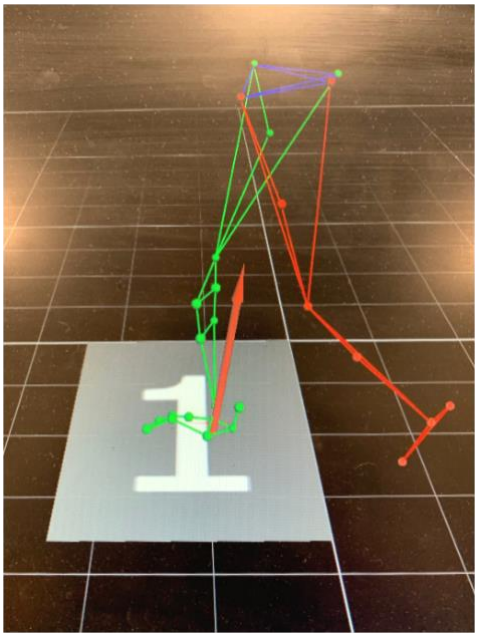

Figure 4. Dynamic gait trials with Vicon Motion Capture System.

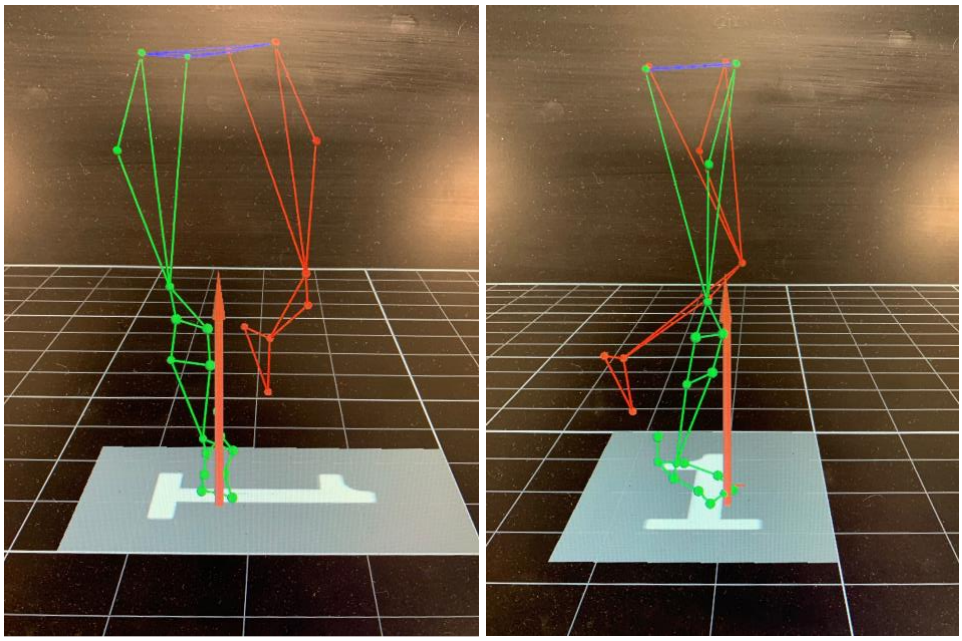

Figure 5. Single Leg Heel Rise Dynamic Trial, front and side views.

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