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Effect of Extra-osseous Talotarsal Stabilization on Posterior Tibial Tendon Strain in Hyperpronating Feet

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ABSTRACT

Posterior tibial tendon dysfunction is considered one of the most common causes of progressive adult acquired flatfoot deformity. The etiology leading to the dysfunction of posterior tibial tendon remains controversial. The purpose of this study was to quantify strain on the posterior tibial tendon in cadaver feet exhibiting hyperpronation caused by flexible instability of the talotarsal joint complex. We hypothesized that posterior tibial tendon strain would decrease after a minimally invasive extra-osseous talotarsal stabilization procedure. A miniature differential variable reluctance transducer was used to measure the elongation of posterior tibial tendon in 9 fresh-frozen cadaver specimens. The elongation was measured as the foot was moved from its neutral to maximally pronated position, before and after intervention with the HyProCure® extra-osseous talotarsal stabilization device. The mean elongation of the posterior tibial tendon (with respect to a fixed reference point) was found to be 6.23 ± 2.07 mm and 3.04 ± 1.85 mm, before and after intervention, respectively (N = 27; variation is ± 1 SD). The average elongation reduced by 51% and was statistically significant with $p < .001$. Strain on the posterior tibial tendon is significantly higher in hyperpronating feet. An extra-osseous talotarsal stabilization procedure reduces excessive abnormal elongation of the posterior tibial tendon by minimizing excessive abnormal pronation. This minimally invasive procedure may thus provide a possible treatment option to prevent or cure posterior tibial tendon dysfunction in patients exhibiting flexible instability of the talotarsal joint complex.

The posterior tibial tendon (PTT) plays a crucial role in the normal biomechanical functioning of the foot and ankle joint complex. The anatomy and physiology of PTT has been described in great detail in the literature (1–3). In brief, the PTT originates in the distal one third of the leg from the tibialis posterior muscle and courses posterior to the medial malleolus underneath the flexor retinaculum, that is, posterior to the axis of rotation of the ankle joint and medial to the axis of the subtalar joint (STJ). The PTT terminates in multiple insertions on the medial and plantar aspects of the tarsal and metatarsal bones, and these are important to consider from a functional standpoint (1). The PTT is a dynamic stabilizer, and it functions to support and supinate the foot during the stance phase of the gait cycle. After heel strike and foot flat/midstance phases of the normal gait cycle, the PTT initiates hindfoot inversion at the STJ complex. This causes locking of the transverse tarsal joints as the axes of the talonavicular and calcaneocuboid joints become nonparallel, converting the foot from a flexible structure to a rigid lever, allowing it to lift and propel the body to ambulate with a normal gait pattern (4–7).

Posterior tibial tendon dysfunction (PTTD) or insufficiency is a commonly diagnosed clinical condition; it is the progressive inability of the PTT to stabilize the foot during midstance and propulsion phases of the gait cycle (8–12). The signs and symptoms suggesting dysfunction of the PTT are pain and swelling along the medial foot and ankle, pain along the medial plantar arch, a decrease in walking ability and balance, early fatigue and foot ache on walking long distances, collapse of medial longitudinal arch, increased rearfoot eversion and forefoot abduction, "too many toes sign," inability to perform single-support heel rise test, and the first metatarsal rise sign (6,7,9,12–18). Several etiologic factors leading to PTTD are hypertension, obesity, diabetes mellitus, inflammatory arthropathies, steroid use, direct injury, or trauma, or it may be idiopathic (6,7,9,16,18). The presence of an accessory navicular is frequently associated with

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Conflict of Interest: Michael E. Graham is the inventor of HyProCure®. He is Founder and President of GraMedica, LLC, the company that manufactures and distributes HyProCure®. He is also Founder of Graham International Implant Institute.

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occurrence of PTTD as it interrupts the normal pattern of tendon insertion (7,19). A zone of hypovascularity or the so-called watershed area has been identified in the PTT approximately 10 to 15 mm distal to the medial malleolus. It was hypothesized that because of lack of blood supply, this region of the tendon was most likely prone to damage (20). It is also believed that PTTD may be caused by an impingement mechanism of the tendon behind the flexor retinaculum in the fibro-osseous groove posterior to the medial malleolus (18,21).

PTTD is considered as a primary cause of adult-acquired flatfoot deformity (AAFD) (4,7,13,17,18). PTTD is progressive in nature and leads to 4 different stages of AAFD (15,17,22). Johnson and Strom (15), and Myerson (17), classified the progression of dysfunction of the PTT into 4 clinical stages. In the initial or early stages (stages I and II) the acquired flatfoot deformity is flexible and can be treated either by a structured nonoperative management protocol or by minimally invasive surgeries to repair the damaged tendon and to correct the underlying flatfoot deformity (16,18,23–26). However, as PTTD progresses to its later stages (stages III and IV), the acquired flatfoot deformity becomes rigid, requiring complete surgical correction using multiple arthrodesis procedures along with soft tissue procedures for tendon repair (13,16–18,24,26). If left untreated in its early diagnostic stages, acquired flatfoot deformity caused by PTTD may progress from a flexible and reducible flatfoot deformity (stages I and II) to a rigid and irreducible flatfoot deformity (stages III and IV) (15,17). On the contrary, it is also believed that PTTD is a result of pre-existing flexible flatfoot, and many authors suggest that a congenital or pre-existing flexible flatfoot may lead to chronic mechanical overload, microtrauma, and degenerative tendinosis of the PTT (2,5,10,15,17,27–29). Researchers also propose that mild pes planus leads to abnormal forces that may result in greater mechanical demands on the PTT as compared with a normal foot (6,28,30,31). However, the series of events that initiate a pre-existing flatfoot or mild pes planus to become symptomatic and progressively deform remains to be determined (2,6,10,18,21,28,29,31).

Hyperpronation is often associated with PTTD and AAFD (12,32). It is considered as a consequence of PTTD rather than its cause (31,33); however, this remains controversial (12,32). There is a debate regarding the etiology of these co-findings, that is, does a pathologic tendon (PTTD) lead to a hypermobile flatfoot (hyperpronation) or does a hypermobile flatfoot lead to a pathologic tendon (28). We speculate that hyperpronation caused by flexible instability of the talotarsal joint complex is the key event that initiates dysfunction of the PTT, and hence an acquired flatfoot deformity. As the talotarsal joint hyperpronates, there is subluxation (partial dislocation) of the talus on the calcaneus and the navicular; the talus adducts and plantarflexes, causing retrograde eversion of the calcaneus (2). Excessive forces are placed on the PTT and surrounding soft tissue support structures with each weight-bearing step; these structures eventually become weak because of cyclical mechanical overload. As the foot continues to hyperpronate, dysfunction of the PTT progresses, which may eventually result in a rigid flatfoot deformity. It has been advocated that treatment of PTTD in its early stages of development should focus on correcting the root cause of hyperpronation, that is, stabilizing the talotarsal joint complex (32). We believe that it is possible to have a hyperpronating or pes planus foot without PTTD, but it is not possible to have a PTTD with a stable talotarsal joint complex. An example of this would be a case in which the flexible flatfoot deformity is caused by ligamentous laxity such as in Ehlers-Danlos syndrome (34).

The goal of this study was to quantify elongation (strain) of the PTT in human adult cadaver specimens exhibiting hyperpronation (partial dislocation or subluxation of talus on calcaneus and navicular, that is, flexible instability of the talotarsal joint complex), before and after intervention using an extra-osseous talotarsal stabilization (EOTTS) device. HyProCure® (GraMedica, Macomb, MI) is an EOTTS device that eliminates excessive abnormal pronation while still allowing the normal range of hindfoot motion to occur. To the best of our knowledge, we found no studies in the literature that report measurement of the elongation or strain of the posterior tibial tendon, with or without any intervention. However, the literature states that a significant amount of mechanical strain is placed on the PTT during hyperpronation and treatments should focus on minimizing this strain (7,32,35,36). We hypothesized that strain on the PTT in hyperpronating feet would reduce significantly after the placement of the aforementioned EOTTS device.

Materials and Methods

Nine fresh-frozen human adult cadaver specimens (5 left-footed and 4 right-footed, all female) were used for posterior tibial tendon strain measurements. Each specimen consisted of the foot, ankle, and distal segment of the leg (~20 cm proximal to the ankle joint). All specimens were inspected for their range of motion at the ankle joint complex. Clinical examination revealed that each specimen used in this study exhibited hyperpronation, indicating flexible instability of the talotarsal joint complex. Exclusion criteria for the specimens were previous operative intervention, fracture, or pathologic conditions in the ankle-hindfoot complex such as tarsal coalition or arthritic degeneration of midfoot and hindfoot joints. The specimens were adequately thawed to room temperature before testing. Each specimen was dissected free of soft tissue at the proximal tibial and fibular segment. The proximal segment of leg was potted with polymethylmethacryalte for mounting in the testing fixture. Care was taken to avoid damage to the soft tissue structures of the foot and ankle joint complex.

A differential variable reluctance transducer (DVRT) (Microstrain, Williston, VT) with a 9-mm linear stroke range and a resolution of 1.5 μm was used to measure strain. The output voltage of the DVRT was amplified with a DEMOD-DVRT signal processor (Microstrain) and recorded with MB-SMT 4 motherboard with data acquisition software (Microstrain). Using the calibration equation provided by the manufacturer, the output voltage (in volts) was converted to displacement (in millimeters). The PTT was exposed by dissecting the skin and tissues overlying the area of the tendon within the "watershed" area (Fig. 1). The potted specimen was mounted on a material testing system (MTS Bionix 858, Eden Prairie, MN) solely for support purposes. Because we used below-knee cadaver specimens, the tibialis posterior muscle was held in constant tension at the most proximal segment of the foot with an Alice clamp. This was done to avoid buckling of the PTT, which could have caused measurement errors. With the foot in neutral position, the body of the DVRT was held in a fixed (immovable) position at the level of the medial malleolus with a needle pin (permanently clamped to the body, Fig. 2). Similarly, a barbed pin clamped to the core of the DVRT was inserted into the PTT distal to the medial malleolus. This was the movable end of the DVRT, that is, as the tendon elongated, the core would move relative to the fixed DVRT body (Fig. 2). With the foot held in neutral position, the DVRT output voltage was recorded to calculate the length between the needle and the barbed pin; this was denoted as the reference or the

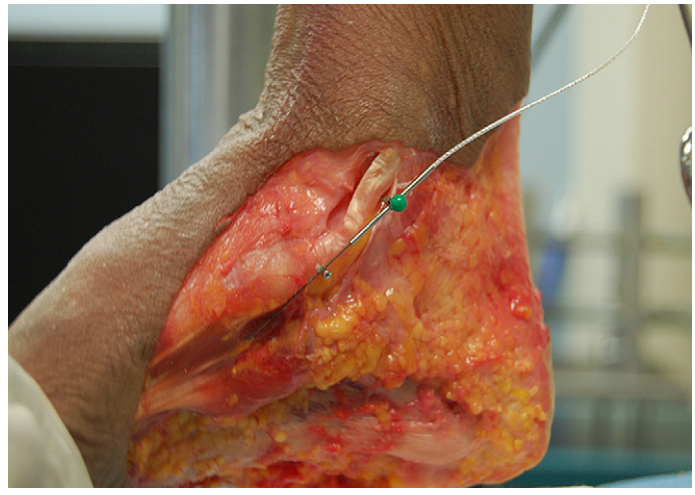

Fig. 1. Exposed posterior tibial tendon in a right-footed specimen in the so-called watershed region, that is, in the zone of hypovascularity. The 2 ends of the DVRT were fixed along the most linear segment of the tendon for obtaining strain measurements.