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BIOMECHANICS OF POSTERIOR TIBIAL TENDON DYSFUNCTION

Posterior tibial tendon dysfunction (PTTD), a cause of adult acquired flatfoot deformity, is a pathological condition which is progressive and can cause significant pain and disability. Even though abnormal foot and ankle biomechanics is a common cause of PTTD, other causes of PTTD also include age-related tendon degeneration, inflammatory arthritis, and, infrequently, acute traumatic rupture (Geideman WM, Johnson JE. Posterior tibial tendon dysfunction. J Ortho Sp Phys Ther, 30(2):68-77, 2000).

Typically, the patient with PTTD complains of pain and swelling in their medial ankle area which limits their ability to walk and stand for prolonged periods. It is common for many patients to not remember any inciting event that could have led to the tendon injury that caused their PTTD. However, some patients do remember an accident, involving a sprain or sudden movement of their foot and ankle, that occurred just prior to the pain and swelling presenting within their medial ankle that led to their PTTD.

In order to best understand the biomechanics and etiology of the progression of deformity that typically occurs with PTTD, it is important to detail the function of the posterior tibial (PT) muscle and tendon during weightbearing activities. Since the PT tendon passes medial to the subtalar joint (STJ) axis, contractile activity of the PT muscle will cause an internal STJ supination moment. When a muscle, such as the PT, can generate an internal STJ supination moment, it also then has the ability to cause 1) an acceleration of STJ supination, 2) a deceleration of STJ pronation, or 3) a stabilizing effect against STJ pronation moments (Kirby KA: Conservative treatment of posterior tibial dysfunction. Podiatry Management, 19:73-82, 2000). Since the PT tendon has a long STJ supination moment arm, it is one of the strongest supinators of the STJ.

The PT tendon also passes plantar and medial to the midtarsal joint (MTJ) which allows PT contractile activity to cause both an arch-raising moment and a forefoot adduction moment. The ability of PT contractile activity to prevent medial arch flattening and forefoot abduction (think "too-many toes sign") is very important when considering the progression of deformity commonly seen in PTTD. Furthermore, due to the PT tendon being only slightly posterior to the ankle joint axis, PT muscle contractile activity can also cause a relatively weak ankle joint plantarflexion moment.

With the ability of the PT muscle to cause a strong STJ supination moment and also cause a relatively strong MTJ plantarflexion and adduction moment, the PT muscle must function without weakness in order to allow normal foot and lower extremity biomechanics. However, if even the slightest injury occurs to the PT tendon, the resultant damage to the PT tendon can cause the beginning of a cascade of foot deformities that marks the progression of stages of PTTD including flatfoot deformity, forefoot abduction on the rearfoot and medial displacement of the ankle relative to the plantar foot.

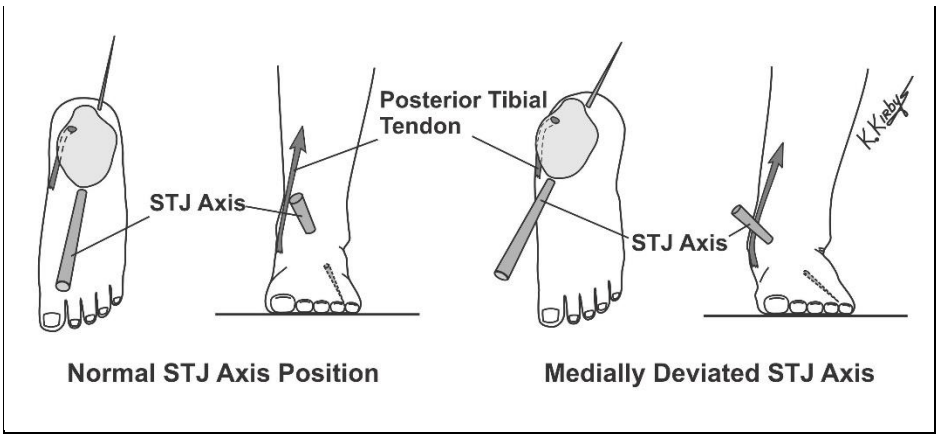

Figure 1. In a foot with a normal subtalar joint (STJ) axis location (left), the posterior tibial (PT) tendon has a relatively large moment arm (i.e., lever arm) to cause a STJ supination moment with contractile activity of the PT muscle. However, when the STJ axis is significantly medially deviated due to adduction and plantarflexion of the talus relative to the plantar foot (right), the PT tendon will lose a large proportion of its STJ supination moment arm which will not only effectively weaken the PT muscle, but will also place greater tension stress on the PT tendon, possibly leading to PTTD over time.

After nearly four decades of treating hundreds of patients with PTTD, and examining the STJ axis location in these patients, I have concluded, using known biomechanical principles, that most patients who develop PTTD originally had pre-existing pronated feet with abnormal medial deviation of their STJ axis (Kirby KA: Biomechanics of the normal

and abnormal foot. JAPMA, 90:30-34, 2000). In all the patients with unilateral PTTD I have examined over this time, I have yet to find a patient who had a normal STJ axis location and normal arch height in their unaffected, contralateral foot. This clinical observation leads me to suspect that nearly all PTTD pathologies that began without major trauma initially had a pronated foot with a significantly medially deviated STJ axis in their affected foot that caused the PT tendon injury.

Why would an individual with a medially deviated STJ axis tend to be more likely to develop an injury to the PT tendon and, ultimately, develop the pathology of PTTD? There are multiple biomechanical reasons for PT tendon injury to be more likely in individuals with a medially deviated STJ axis, with the main reason being that a medially deviated STJ axis will shorten the moment arm (i.e., lever arm) for the PT tendon to cause a supination moment across the STJ. Since moment, or torque, at a joint is equal to the mathematical product of the magnitude of tension force within the muscle-tendon unit and length of the moment arm (i.e., perpendicular distance from the muscle-tendon tension force to the joint axis), then any decrease in moment arm length will also weaken the moment that the muscle can generate with its contractile activity.

For example, in a foot with a normal STJ axis location and arch height that stands close to STJ neutral position, the PT tendon moment arm length is given to be equal to 3.0 cm. However, if the STJ is pronated sufficiently so that the STJ axis has now adducted and medially translated 2.0 cm relative to the PT tendon, the PT tendon will have a reduced moment arm length equal to only 1.0 cm. Even though this 2.0 cm loss in PT tendon moment arm length seems like a relatively small shift in the STJ axis location relative to the PT tendon, this "small" 2.0 cm medial shift of the STJ axis will now result in the PT muscle-tendon unit only having 33% of the muscle power to generate STJ supination moment that it originally had (Fig. 1).

In addition, not only will any abnormal STJ axis medial deviation significantly weaken the ability of the PT muscle-tendon unit to generate sufficient STJ supination moments to allow normal foot and lower function during weightbearing activities, it will also cause an large increase in the magnitude of PT tendon tension force and tension stress which can increase the risk of PTTD developing in the individual. When the central nervous system (CNS) of the individual determines that increased magnitudes of STJ supination moment are necessary to correctly perform a certain weightbearing activity, it will increase the contractile activity of its most powerful STJ supinator, the PT muscle, in order to perform that weightbearing activity.

If the CNS determines that it needs, for example, a STJ supination moment of 3.0 Newton-meters (Nm) in order to optimally perform a certain weightbearing activity, if the individual has a PT tendon supination moment arm equaling 3.0 cm, then the PT tendon would only need to generate 100 N (22.4 lbs) of tension force to meet the demands for STJ supination. If, however, the STJ axis becomes significantly medially deviated and the PT supination moment arm is decreased to only 1.0 cm due to medial STJ axis deviation, now the PT muscle-tendon unit will need to generate 300 N (67.4 lbs) of tension force to meet the demands for STJ supination. In other words, this three-fold increase in PT tendon tension caused by abnormal medial deviation of the STJ axis will cause increased tension stress within the fibers of the PT tendon and, as a result, may lead to an increased likelihood of PT tendon injury and PTTD developing over time.

Therefore, medial deviation of the STJ axis in an individual's foot is a likely precursor to spontaneous development of PTTD due to the biomechanical fact that medial STJ axis deviation increases the PT tendon tension force and tension stress during weightbearing activities. Likewise, this plastic deformation of the PT tendon (e.g., partial tear, stretching or complete tear of the tendon), due to chronic increased tendon stress, will lead to further PT muscle-tendon unit weakness which, over time, will lead to the classic progression of deformity typically seen in patients with PTTD including medial arch flattening, STJ pronation and forefoot abduction. The next ProLab newsletter will discuss proper conservative treatment of PTTD and the specific custom foot orthosis modifications recommended for the treatment of patients with this disabling pathology.

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