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REVIEW ARTICLE

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Ivan Szergyuk et al., Twisted AT microvasculature

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ABSTRACT

The Achilles tendon (AT) is reportedly the most vulnerable to rupture at the midportion, a section of relative hypovascularity. It has been postulated that the twisted structure of this tendon may constitute a critical factor contributing to increased propensity to vascular compromise, decreased regenerative capacity, and rupture in the midsection of the AT. In this review, we will give an overview of the most relevant research on AT vasculature and twist, and delve into the interplay between the two elements in the context of AT disorders. The pertinent body of research suggests a considerable variability in tendon twist among individuals, which likely constitutes a determining factor in the extent to which vessels coursing along and between AT fibers are compressed during contraction-induced elongation of the tendon. Consequently, further research is necessary to investigate the precise association between tendon torsion and blood flow within the AT.

Keywords: Achilles tendon, twist, microvasculature, hypovascularity, tendinopathy

INTRODUCTION

The Achilles tendon (AT) is a large twisted collagenous structure spanning from the musculotendinous junction at the distal portion of the triceps surae muscle (the gastrocnemius and soleus muscles), down to the calcaneal tuberosity, its site of insertion [1, 2] (Fig. 1). This tendon facilitates foot plantar flexion, undergoing elongation and further torsion during contraction of the triceps surae [3]. Despite its size and capacity to withstand loads of up to 3500 N [4, 5], AT disorders, such as rupture and tendinopathy, are common phenomena among athletes and also the general population [6–8]. Interestingly, it is less frequent among females than males [9]. Most injuries are attributed to competitive and recreational sport [10], however, non-sports-related overuse injuries are becoming increasingly more prevalent in the general public, leading to decrease of quality of life and increased economic burden [11, 12].

Reportedly, the majority (80%) of injuries occur in the midportion of the tendon [13, 14], corresponding to an area of poor vascularity [15]. Given that tendon vasculature is a critical factor influencing its mechanical and regenerative properties, many authors have concluded that inadequate or impaired tendon vascularity and resultant degenerative changes represent an important mechanism responsible for AT injury [16]. This has been widely studied in many other tendons of the body [17–19]. The midsection of the AT, located at approximately 4–7 cm from the insertion, is apparently less densely vascularized by its supplying artery, the fibular artery, than the adjacent proximal and distal regions, which receive blood supply from the posterior tibial artery [15, 20]. The relative hypoxia results in lower potential for regeneration from microinjuries due to overuse, in turn making the AT more prone to rupture in this region [15]. Radiographic, angiographic, and anatomical dissection studies have so far provided a great deal of invaluable insight into mechanisms underlying midportion AT injury, in addition to offering clues to potential therapies.

It has been postulated that the twisted structure of this tendon may constitute another critical factor contributing to increased propensity to vascular compromise and rupture in the midsection of the AT [16]. An increasing body of research on AT vascularity and twist offers insights into the relationship between the two factors. In this review, we will give a comprehensive overview of the most relevant research on this clinically important topic.

Achilles tendon vasculature

The AT was initially thought to receive equal contribution from the fibular artery medially and posterior tibial artery laterally, with an anastomotic region situated vertically along the

midline of the tendon [21, 22]. This anastomotic plane, a so-called “choke zone” [23], served as one of the primary explanations for hypovascularity in this region, leading to a decrease in the biomechanical performance of the AT [21, 22]. Since then, detailed angiographic studies have provided evidence redefining the vascular distribution of the AT. Using angiography, microdissection, and histological analyses, Chen and colleagues established that the fibular and posterior tibial arteries supply distinct transverse territories of the AT: proximal and distal sections of the AT are vascularized by the posterior tibial artery, while the midportion is supplied by the fibular artery [15]. The researchers discovered that the midsection is susceptible to hypoxia and rupture not only due to insufficiency of the fibular arterial branch, but also due to the adjacent watershed zones, areas where capillaries of the fibular and posterior tibial arteries anastomose [5, 15]. An immunohistochemical study utilizing antibodies against laminin, a component of blood vessel walls, determined that the vascular density in the middle section of the AT was 28.2 vessels/cm^2 [16]. In contrast, the distal and proximal regions were 2 and 2.6 times more densely vascularized, respectively [16].

Vessels supplying the AT course predominantly along its anterior and deep surface, subsequently branching into a thin network of vessels within the paratenon, a sheath enveloping the tendon [15]. The posterior surface, in contrast, is less densely vascularized as the vessels must traverse from the ventral side of the AT where they originate. Longitudinally oriented vessels which course superficially along the tendon and deep between the tendon fibers follow the spiraling course of the tendon fascicles in a medial to lateral direction [15]. As such, these vessels may rotate and get compressed following torsion of tendon fibers [15].

When it comes to genetic susceptibility, alterations in the expression of angiogenesis-associated signaling pathways in response to mechanical loading and injury have been linked to increased tendinopathy risk [24]. Certain isoforms of vascular endothelial growth factor A (VEGF-A) have been associated with diminished expression of the *VEGFA* gene, leading to decreased circulating VEGF-A levels, impairing vascular formation and remodeling of extracellular matrix following mechanical stress [24].

Wolff et al. argued that the available research describing AT vasculature lacked methodological validity, owing to drawbacks of each individual technique used to analyze AT vasculature [25]. As such, the findings of poor vascularity were rather due to challenges in visualizing the smallest vessels or difficulties in interpreting the exact localization of the vessels in a three-dimensional plane [25]. Combining both detailed angiography and anatomic dissection of the same specimens, the researchers observed, that contrary to prior

observations, the alleged avascular midsection receives ample supply from a dense network of arterioles, including branches from the anterior tibial and posterior tibial arteries [25]. As such, they suggested that tendon biomechanics and localized stress are the predominant culprits of tendinopathy. Notwithstanding, vessels coursing along and through the AT are likely subject to torsion and compression during contraction-induced elongation of the tendon, potentially threatening the strength and viability of the tendon over time, regardless of its ample blood supply.

Tendon twist

When viewed from above, the AT is internally twisted, with clockwise rotation on the left and counterclockwise on the right (Fig. 2) [26–28]. Rotation begins approximately 12 to 15 cm proximal to the insertion point, reaching maximal twist in the distal 5 to 6 cm of the tendon [9]. Throughout its course toward the calcaneus, the fibers of the tendon may rotate by as much as 211.17° [1], although this degree varies to a large extent depending on the individual and the subtendon in question. Many previous studies have reported on the variable degree of AT twist and how it determines which fascicles contributing to the AT attach at the calcaneal insertion. Cummins at al. originally classified the AT into three types depending on the degree of torsion [29]. Specimens with the gastrocnemius subtendons occupying the lateral two-thirds of the posterior (superficial) layer and soleus the medial two-thirds of the anterior (deep) layer were classified as Type I (least twist), and observed in 52%. Type II (moderate twist), with the gastrocnemius subtendons occupying the lateral one-half of posterior layer and soleus the medial one-half of the anterior layer, was observed in 35%. Finally, Type III (extreme twist), with the gastrocnemius subtendons occupying the lateral one-third of posterior layer and soleus the medial one-third of anterior layer, was observed in 13%. Szaro et al., expanded on this classification, distinguishing between insertion sites of the medial and lateral heads of the gastrocnemius (MG and LG, respectively), as well as the soleus [26]. They demonstrated that subtendons from the MG give rise to posterior (surface) and lateral AT fibers, while fascicles from the LG to anterior (deep) AT fibers [26]. Subtendons from the soleus muscle constitute the anteromedial part of the AT [26]. In their effort to elucidate inconsistencies in reporting of the degree of twist, chiefly by studying a larger amount of specimens and separating each fascicle more finely, Edema and colleagues reported a slightly different classification of the “twist”, which was based on attachment to the anterior (deep) layer of the calcaneal tuberosity of each muscle tendon [30]. In specimens with type I twist,

the soleus occupied the entire anterior (deep) portion of calcaneal tuberosity insertion site, while in those with type III twist, the anterior layer of this tuberosity comprised entirely of the lateral gastrocnemius subtendons [30]. As such, an even greater degree of torsion appeared to exist in individuals with the extreme twist type, something which was not previously seen by Szaro et al. and earlier studies. In this classification, type I twist was observed in 50%, Type II in 43%, and Type III in 7% [30]. In a subsequent analysis of fetal Achilles tendons, Edama et al. reported the AT as Type I in 13%, Type II in 77%, and Type III in 10% [27]. The exact angle of torsion of each subtendon was further analyzed by Pękala and colleagues [1]. When classified according to the three torsion types, the twist angle was highest in specimens classified as type III, intermediate in type II and lowest with torsion type I (Table 1) [1]. This corroborated the findings of Edama et al. that MG fascicles course down with the least degree of twist no matter which torsion type. Interestingly, by discovering an association between the torsion of the AT and femur, Prosenz et al. proposed that the degree of AT twist may change over time as a result of age-related changes in torsion of the lower limb [28, 31].

Twisting of the tendon fibers is believed to be responsible for its elastic properties [9, 32], allowing it to stretch up to 4% of its length without failure [33, 34], and is necessary for reducing stress by balancing the distribution of strain within the tendon [35, 36]. In spite of this, an area approximately 2–5 cm from the insertion site is subject to a significant focus of stress, as a consequence of the twist and the shearing forces between overlapping tendon fascicles [5, 37, 38]. This has an important implication with regard to the vessels which course within the tendon. Although there is high variability, tendon fascicles appear to be more twisted in the midportion of the AT, resulting in greater compression of vasculature in this region, particularly when mechanical strain is applied during contraction-induced elongation of the tendon [20, 28]. Vascular compression is likely proportional to the extent of tendon fascicle twisting, such that individuals with more extreme twist (Type III), and hence stress concentration, may be at an enhanced risk for vascular compromise. Avascularity may in turn lead to reduction in tendon strength and the potential for regeneration, especially in individuals with prolonged isometric contraction of the soleus and gastrocnemius [39].

Interestingly, Clement et al. postulated that, in runners who overpronate, simultaneous ankle pronation and knee extension during push-off produces concurrent internal and external rotatory force on the tibia, which as a result exerts a wringing effect on the AT and its vasculature [40]. This conclusion was drawn from the observation of varus foot alignment

accompanied by compensatory overpronation at the subtalar joint in 56% of patients with injury of the AT [40]. The presence of tight calf muscles, noted in 38% of the cohort, represented a further etiological factor for straining of the AT tendon during running [40]. In an attempt to compensate for impaired dorsiflexion, these individuals may overpronate the foot during push-off. As such, these functional deformities may aggravate the avascularity of the midsection of the AT. Another study, however, observed that overpronation was not associated with AT injuries when compared to controls [41]. Instead, an association was present for underpronation, which was linked to poor shock absorption. As such, a delicate balance seems to exist with regard to foot alignment, outside of which injuries may occur, as exemplified with the wringing effect exacerbating relative avascularity of the AT in overpronation, and impaired shock absorption of the tendon in underpronation.

In summary, the regional vascularization pattern of the AT is a critical factor influencing function, resilience, and regenerative capacity of the tendon. AT torsion is highly variable among individuals, and likely constitutes an important factor in determining to what extent local blood flow is compromised during contraction-induced elongation of the AT. As such, further research is necessary to investigate the association between tendon twist and blood flow within the AT.

Article information and declarations

Author contributions

Ivan Szergyuk: Conception, review and interpretation of the literature, writing of the paper, revision and approval of the final article.

Alicia del Carmen Yika: Conception, review and interpretation of the literature, writing of the paper, revision and approval of the final article.

Jerzy A. Walocha: Supervision, consultations and approval of the final article.

Przemysław Pękala: Conception, supervision, revision and approval of the final article.

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Table 1. The torsion angle for each subtendon of the Achilles tendon in the different torsion types

	Type I	**Type II**	**Type III**
MG	16.67°	35.31°	67.67°
LG	107.39°	156.53°	211.17°
SOL	104.69°	144.73°	199.67°

MG — medial head of gastrocnemius; LG — lateral head of gastrocnemius; SOL — soleus.

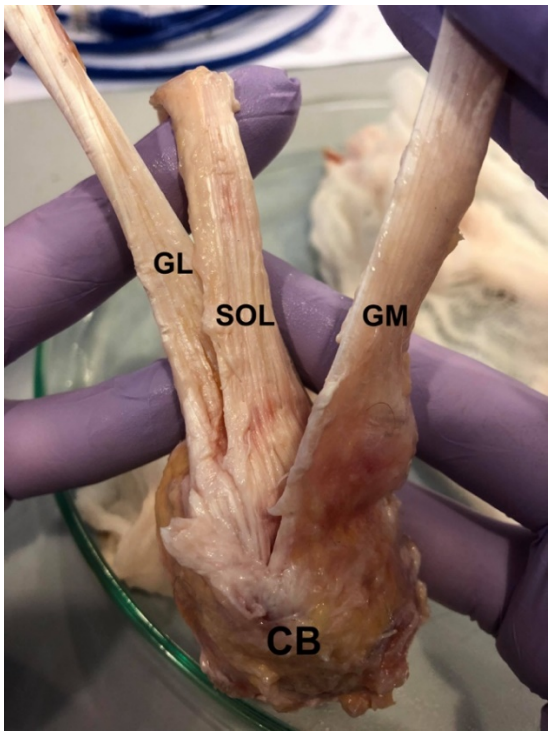

Figure 1. Dissected subtendons of the Achilles tendon — posterior view. CB — calcaneal bone; MG — medial head of gastrocnemius; LG — lateral head of gastrocnemius; SOL — soleus.

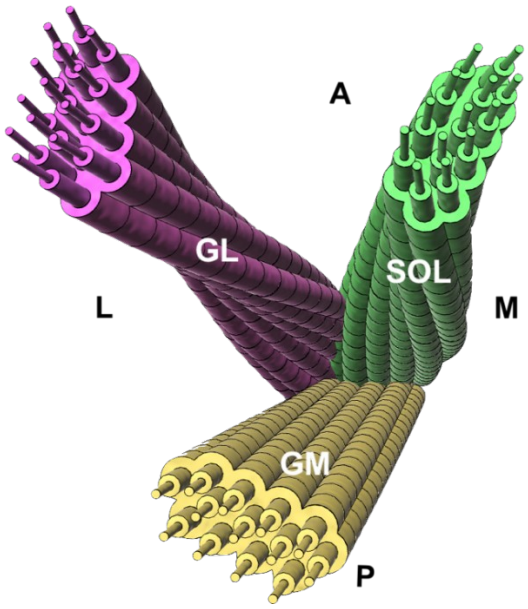

Figure 2. Schematic illustration of the subtendons of the Achilles tendon. MG — medial head of gastrocnemius; LG — lateral head of gastrocnemius; SOL — soleus; A — anterior; L — lateral; M — medial; P — posterior.