Morphological and functional outcomes of operatively treated Achilles tendon ruptures

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ABSTRACT

Background: Achilles' tendon (AT) rupture leads to functional impairments and these may be underpinned by morphological changes in the muscle-tendon unit.

Hypothesis: Functional performance of the injured limb will be impaired regardless of time since surgery and these impairments occur alongside changes in muscle-tendon morphology.

Study Design: Descriptive laboratory study.

Methods: Following operative treatment of AT rupture (mean, 4.4 years postoperative; range, 6-36 months) and short term immobilisation, 12 patients completed the Achilles' tendon rupture score(ATRS), tests of ankle and hip range of motion (ROM) and ultrasound measurements of muscle-tendon architecture. Data from isokinetic (30 °/s, 60 °/s) plantar flexion strength, jumping performance and walking-running were collected. Differences were expressed relative to the non-injured limb.

Results: The repaired limb showed a shorter muscle fascicle length (12.1-19.6% diff), increased fascicle pennation (18.0% diff) and reduced muscle thickness (9.1-20.1% diff) in the Gastrocnemius and/or Soleus along with greater tendon cross-sectional area (46.7% diff). Functionally, impairments were found for countermovement jump height (-12.6% diff) and drop jump contact times (+5.5% diff). Also, the repaired limb showed reduced hip internal-external ROM (6.3% diff) but no differences existed between limbs for plantar flexion ROM and strength or gait characteristics. Good ATRS outcomes were reported (mean 87.9) which related to time since surgery (r=0.79) but individual ATRS items did not correlate with corresponding objective measures.

Conclusion: Plantar flexor atrophy following surgically treated AT rupture is partially compensated for by remodelling of the fascicles however, impairments may still persist many years into the postoperative period although these may be more pronounced in high-velocity activities.

Clinical Relevance: Morphological changes will have functional implications in high-velocity movements and these may persist many years post-surgery.

Key Terms: isokinetic, ATRS, muscle morphology, gait

What is known about the subject: AT rupture is known to result in long-term reductions in ROM, strength and joint function in the injured leg.

What this study adds to existing knowledge: Despite morphologically remodelling of the triceps surae complex following AT rupture, these compensatory mechanisms may be insufficient to prevent long-term impairments in high-velocity functional performance.

INTRODUCTION

The Achilles Tendon (AT) is the strongest tendon in the human body with a crucial role in transmitting mechanical energy generated by the triceps surae complex. Incidence of AT rupture has been estimated at between 6-21.5 per 100,000 (Maffulli et al. 1999; Lantto et al. 2015) with notable increases being reported in recent decades (Aujla et al. 2018). Although non-operative treatment of AT ruptures avoids many of the complications associated with surgery (e.g. infection), decreased rates of re-rupture, reduced tendon lengthening and less atrophy have been reported with operative management (Cetti et al. 1993; Calder et al. 2005; Wilkins & Bisson, 2012).

Impaired functional outcomes shown for individuals with a history of AT rupture include reduced ankle proprioception, decreased plantar flexor strength and asymmetric performance in activities requiring propulsion (Mortensen et al. 1999; Don et al 2007; Wang et al. 2013). Despite the prevalence of AT ruptures, research into post-operative deficits has generally focussed on relatively short follow-up periods of 6-36 months (Naim et al., 2005; Willits et al., 2010). Recent research has provided evidence of long-term deficits (>10 years) post-surgery (Horstmann et al., 2012; Barfod et al., 2017) which include reduced ROM, strength and joint function in the injured leg. Whilst information on the long-term outcomes is important, it is unclear whether the patients underwent homogenous surgical treatment and postoperative rehabilitation strategies, which are known to impact on patient outcomes (De la Fuente et al., 2016; Valeo et al. 2017).

Apart from functional deficits, AT rupture results in alterations of the neuromechanical properties of the muscle-tendon unit including tendon elongation, triceps surae (TS) atrophy and compensatory changes in muscle activation (Horstmann et al. 2012; Heikkinen et al. 2017; Peng, 2017). Manegold and co-workers (2018) showed that AT elongation and TS atrophy are related to the deficits in strength and ROM that are often seen in the ankle joint. This remains one of few studies that has sought to understand the mechanisms underpinning deficits in functional outcomes. In terms of muscle morphology, recent studies (Peng et al. 2017; Baxter et al. 2018) have suggested medial gastrocnemius remodelling following AT rupture as a compensatory mechanism to restore resting tendon tension. However, such changes (e.g. larger pennation, reduced volume are known to be related to impairments in activities requiring force transmission and muscle excursion (e.g. heel raise; Peng et al. 2017). Although these studies provide important information about morphological remodelling,

they have not integrated a wide range of functional tasks (e.g. jumping) alongside the morphological measures nor have investigated post-surgery periods longer than 2.5 years.

Therefore, despite the wealth of research investigating the outcomes of operatively treated AT ruptures, there is a need to investigate morphological and biomechanical outcomes alongside a broad range of functional activities over a long follow-up period whilst standardising confounding factors relating to the surgical treatment and rehabilitation strategies. This will permit a clearer understanding of the morphological factors underpinning deficits in functional tasks which impact on daily living allowing rehabilitation strategies to be optimised. Consequently, this study aimed to retrospectively examine the morphological and functional outcomes of operatively treated AT ruptures.

METHODS

Twelve patients participated in the current study (ten men, two women; age [mean ± SD]: 43.3 ± 13.6 years; stature: 1.74 ± 0.09 m; body mass: 80.2 ± 10.5 kg). They had all previously suffered unilateral AT rupture and had undergone operative treatment, the time since surgery of the cohort was 4.4 ± 2.6 years. Following a medical screening assessment, participants provided informed consent and completed the Achilles Tendon Rupture Score (ATRS) to assess patient reported outcomes (Nilsson-Helander et al., 2007). The study received approval from the university research ethical committee and was in accordance with the Declaration of Helsinki.

Operative treatment

All participants underwent identical operative treatment performed by the same surgeon. Each patient was positioned prone and a 5cm posteromedial curvilinear incision was made centred over the Achilles tendon rupture. The frayed ruptured ends were debrided and Bunnell type sutures were inserted into each end of the ruptured tendon ends using number 1 absorbable Vicryl plus sutures. The foot was placed in maximum plantar flexion and the ends of the ruptured tendon approximated and tensioned with 2 knots medially and laterally. The leg was initially immobilised in full plantar flexion in plaster for 2 weeks, following suture removal the patients were placed in a semi-equine weight bearing cast for a further 2 weeks. At 4 weeks the plaster was removed and patients were placed in a walker boot in a plantigrade position for another 4 weeks during which time fully weight bearing was permitted along with range of motion exercises. At 8 weeks post-surgery patients started to mobilise out of the boot and commenced formal departmental physiotherapy.

Testing protocol

Jumping performance

Participants performed unilateral countermovement jumps (CMJ) and bilateral 30 cm drop jumps (DJ) onto two synchronised force platforms (Kistler; Switzerland) sampling at 2,000 Hz. Following familiarisation, participants performed five maximal jumps per condition with hands akimbo and they were urged to keep contact time as minimum as possible. The average values for jump height, contact time, reactive strength index (RSI) and for peak landing force (DJ only) were calculated. Jump height was calculated from flight time ($d = v_i t * ½ at^2$) whilst and RSI by dividing the jump height in the DJ by the contact time; for the CMJ, contact time was replaced by time to take-off in order to calculate RSImod (Ebben and Petushek, 2010).

Gait assessment

The gait assessment was carried out using a treadmill (h/p/cosmos, Gaitway; Germany) instrumented with two embedded force platforms. To replicate normal and running gait, participants walked at 1.11 $m \cdot s^{-1}$ for several minutes before 30 s of data collection whilst - the same protocol was replicated for running at 2.22 $m \cdot s^{-1}$. Step length and contact time were obtained for both limbs during both conditions. Weight acceptance and push off rates were assessed to quantify the efficacy of the plantar flexor complex in accepting and producing force during locomotion.

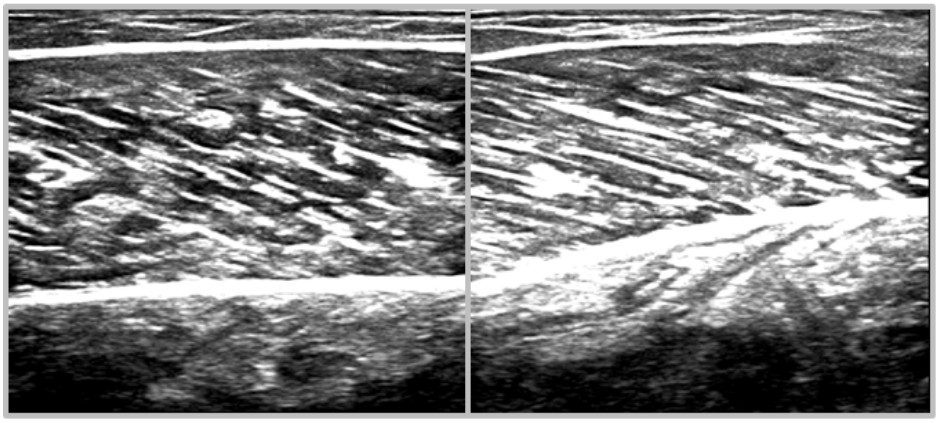
Range of motion assessments

A knee-to-wall test was carried out by progressively moving the foot away from a wall and moving the knee forward until it made contact with the wall. The test was then terminated when the knee could no longer reach the wall, with the best successful attempt being taken as the measurement. This measurement was taken at the toe and heel (to compensate for potential differences in foot length). Maximal ROM of internal and external rotation at the hip joint was taken in prone, supine and seated positions with a goniometer (E-Z Read; Greendale, USA). Each measurement was repeated three times to ensure repeatability.

Morphological measurements of the muscle-tendon complex (MTC)

Static ultrasound measurements (figure 1) of the Triceps surae MTC were taken with the participants laying prone on a physiotherapy bench and the ankle joint in a neutral position. Longitudinal images were taken of the muscle belly of *m.* Gastrocnemius medialis (GM), *m.* Gastrocnemius lateralis (GL) and *m.* Soleus (Sol) in both legs using B-mode ultrasound with a 50 mm linear array probe (5 to 12 mHz; Acuson P300, Siemens; Munich). Bilateral transverse plane images of the AT for the measurement of the cross-sectional area (CSA) were taken 40 mm proximal to the calcaneal insertion, while longitudinal images of the AT were taken 30, 40

and 50 mm proximal to the calcaneal insertion using a 40 mm linear array probe (6 to 18 mHz). These sites

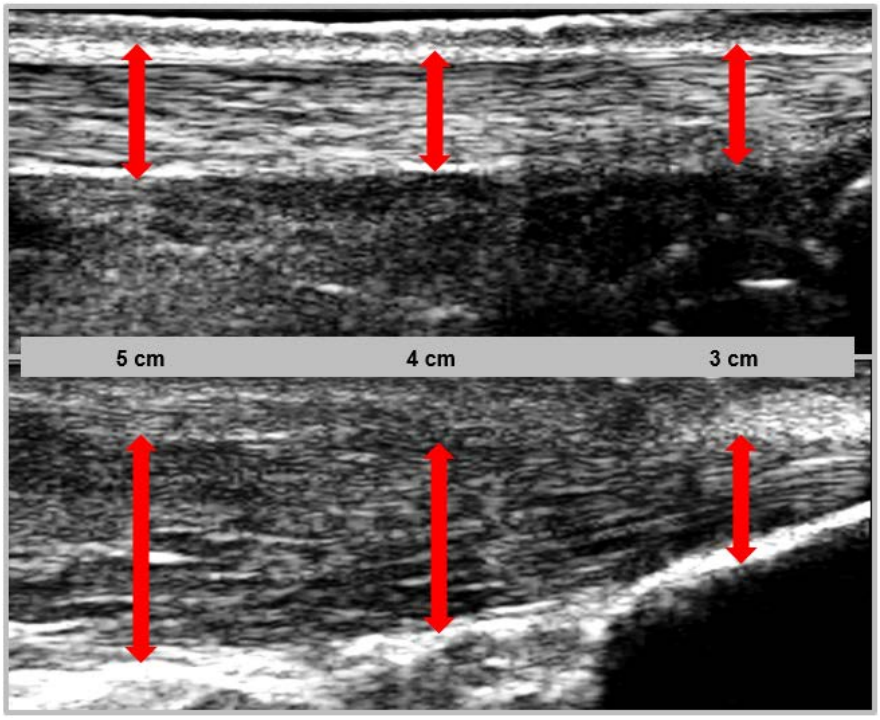


were chosen since the region 20-60 mm proximal to the calcaneal insertion is the most commonly ruptured region (Hess, 2010).

Figure 1. Example ultrasound image of the Medial Gastrocnemius in a non-injured (left) and repaired (right).

Ultrasound images were analysed using ImageJ (Java, National institution of health). AT thickness was measured from the superficial to deep edges of the tendon with muscle thickness measured from the deep to superficial aponeuroses. An average of three images was used for all variables. The angle between the deep aponeuroses and a fascicle that met the deep aponeuroses was defined as the pennation angle. Fascicle length was established through trigonometry using the pennation angle and muscle thickness.

Figure 2. Typical example of Achilles tendon thickness measurements in a non-injured (top) and repaired (bottom).



Strength assessment

Participants underwent strength assessments of the TS using an isokinetic dynamometer (System 4 Pro, Biodex Medical Systems; NY, USA) in two testing positions: a) seated with a hip angle of approximately 65° and a fully extended knee joint, b) same hip angle with the knee in 50° of flexion. Following familiarisation, the concentric torque was measured for the injured and non-injured leg at two angular velocities (30 and 60°·s^{-1}) over three maximal trials.

Data analysis

Statistical analysis was carried out using the Statistical Package for the Social Sciences (SPSS, version 24.0). Between-limb differences (injured vs. non-injured limb) were analysed using a Paired-samples t-test. Percentage differences were calculated between the two limbs as well as the limb symmetry index ([LSI] injured limb value / non-injured limb vale x 100). Potential relationships between variables and time since surgery were tested using Pearson's correlation coefficient. For all statistical tests, homogeneity of data was confirmed and significance levels were set to $p < 0.05$.

RESULTS

ATRS Outcomes

Table 1 Descriptive statistics (mean [standard deviation]) for the 10 items in the ATRS along with the total score. *r* values depict the relationship between each ATRS item and months since surgery.

ATRS Outcomes	Score	*r*
1	8.67 (2.06)	0.76
2	9.17 (0.83)	0.87
3	7.50 (1.98)	0.74
4	9.42 (1.00)	0.58
5	9.42 (1.51)	0.58
6	9.33 (1.61)	0.61
7	9.42 (1.38)	0.62
8	8.42 (2.84)	0.70
9	8.17 (2.92)	0.76
10	8.42 (2.57)	0.59
Total	87.92 (16.22)	0.79

For the responses to each ATRS item, there was a lower mean score for item 3 with the highest mean scores being reported for items 4, 5 and 7 (table 1). Strong positive correlations (r = 0.70-0.87) were observed between a

number of ATRS outcomes and the months since surgery with the strongest correlation being observed for ATRS item 2.

Strength Outcomes

Despite a mean difference in PT (straight leg) at 30°/s and 60°/s for the two limbs, ankle plantar flexion PT was not statistically different between the two sides at either testing speed (table 2) mainly due to the variability of scores. The PT limb symmetry values showed an improvement when the test was performed with the knee flexed.

Table 2 Peak torque (N m) for the ankle plantar flexor muscles for the injured and non-injured limb. *r* values depict the relationship between strength outcomes and months since surgery.

Isokinetic Outcomes	Injured side	Non-injured side	% diff	*r*
Peak Flexor Torque (knee straight)				
30 °/s	82.67 (33.89)	91.67 (27.70)	-11.9 (16.0)	-0.07
60 °/s	68.43 (26.22)	75.78 (21.51)	-11.7 (24.6)	0.06
Peak Flexor Torque (knee flexed)				
30 °/s	86.80 (33.84)	90.46 (35.87)	-1.3 (34.4)	0.05
60 °/s	75.27 (30.14)	79.53 (26.11)	-7.5 (26.9)	0.19

Functional Outcomes

Regarding functional performance, CMJ height was significantly lower (p=0.02) on the injured compared to the non-injured limb (LSI: 87.4 ± 15.7%) with significant differences (p=0.01) also being observed in RSImod between the two limbs (LSI: 85.8 ± 17.6%) (table 3). Similar functional deficits were observed in the DJ with a significantly longer contact time (p=0.01) for the injured side (LSI: 94.5 ± 5.7%) but there were no significant differences in landing forces (LSI: 88.4 ± 19.7%).

Table 3 Performance variables for the CMJ and DJ tests for the injured and non-injured limbs. *r* values depict the relationship between jump outcomes and months since surgery. *significant difference from non-injured side.

Jump Outcomes	Injured side	Non-injured side	% diff	*r*
Countermovement Jump				
Jump Height (cm)	0.10 (0.03)*	0.12 (0.04)	-12.6 (15.7)	-0.12
RSImod	0.12 (0.04)*	0.15 (0.05)	-14.2 (17.6)	-0.14
Drop Jump				
Peak Landing Force (N)	1597 (474)	1874.05 (599)	-11.6 (19.7)	0.14
Contact Time (s)	0.34 (0.08)*	0.36 (0.09)	-5.5 (5.7)	0.05

In contrast to the functional deficits observed during jump activities, there were no significant differences between limbs for any gait variable measured. Furthermore, LSI for numerous gait outcomes appeared unrelated to the time since surgery.

Table 4 Gait variables collected during running and walking for the injured and non-injured limbs. *r* values depict the relationship between gait outcomes and months since surgery.

Gait Outcomes	Injured side	Non-injured side	% diff	*r*
Weight Acceptance Rate				
Walking (N/s)	6233.40 (2260.94)	6991.95 (2558.59)	-9.1 (18.1)	0.23
Running (N/s)	17956.88 (6971.58)	17501.48 (6256.44)	2.9 (18.5)	-0.09
Push Off Rate				
Walking (N/s)	5874.15 (1265.73)	5809.76 (1252.62)	1.4 (6.2)	0.46
Running (N/s)	13155.06 (4791.33)	12283.11 (4898.21)	8.9 (16.4)	-0.23
Step Length				
Walking (cm)	70.26 (19.17)	70.30 (19.17)	-0.3 (6.3)	0.18
Running (cm)	96.52 (32.50)	96.61 (31.91)	-0.2 (5.4)	-0.23
Contact Time				
Running (s)	0.306 (0.054)	0.312 (0.055)	-1.7 (3.5)	0.23

Morphological Outcomes

The injured limb demonstrated morphological differences in the AT (figures 2 and 3) and lower-limb muscles (table 6) compared to the non-injured limb. Specifically, the AT on the injured limb displayed significantly increased thickness at 3cm (LSI: 149.7 ± 29.5%), 4cm (161.3 ± 32.2%) and 5cm (177.6 ± 41.6%) and greater CSA (46.7 ± 34.5, LSI: 146.9 ± 34.5%, p=0.001) than on the non-injured side.

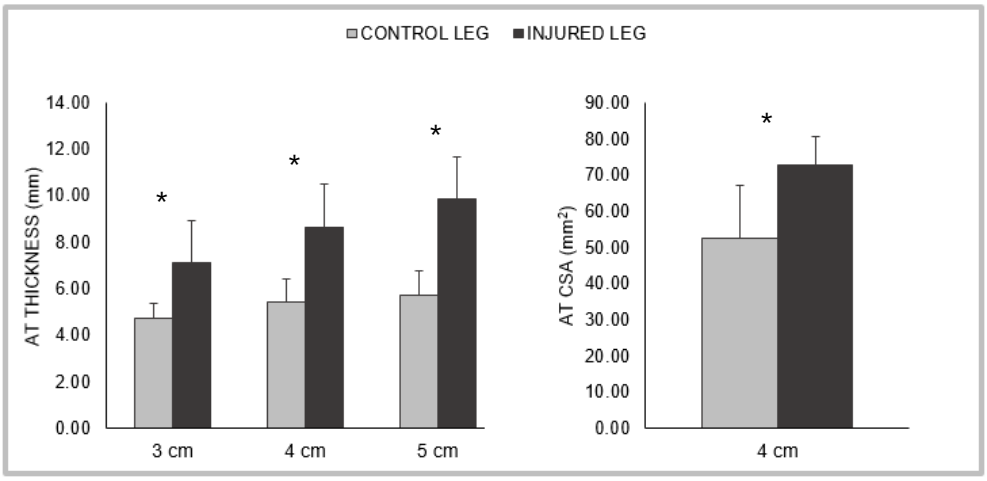


Figure 3. Achilles tendon thickness and CSA at the different measured sites. * significant difference between injured and control leg.

The GM (LSI: 90.9 ± 9.4%, p=0.01) and SOL (LSI: 79.9 ± 19.0, p=0.01) displayed significantly greater thickness on the non-injured side as well as a significantly longer fibres for the GM (LSI: 80.4 ± 18.0%, p=0.00) and GL (LSI: 87.9 ± 18.9%, p=0.03) and SOL (LSI: 80.4 ± 15.3%, p=0.00). Only the fascicles of the GM showed a significantly greater pennation angle (LSI: 118.0 ± 22.1%, p=0.02) on the injured side.

Table 5 Morphological characteristics of the lower-limb muscles of the injured and non-injured limbs. *r* values depict the relationship between morphological outcomes and months since surgery.

Morphological Outcomes	**Injured side**	**Non-injured side**	**% diff**	***r***
Muscle Thickness				
GM (mm)	17.18 (2.35)*	19.03 (2.99)	-9.12 (9.39)	0.23
GL (mm)	13.51 (3.04)	14.39 (2.53)	-6.32 (14.16)	0.14
SOL (mm)	11.62 (2.78)*	15.00 (3.92)	-20.14 (19.00)	0.16
Fibre Pennation				
GM (°)	25.31 (4.72)*	21.82 (3.84)	17.99 (22.14)	0.26
GL (°)	15.99 (3.14)	14.79 (2.43)	9.99 (23.75)	-0.30
SOL (°)	24.40 (3.74)	24.44 (6.16)	4.46 (25.03)	0.34
Fibre Length				
GM (mm)	40.97 (6.37)*	52.32 (9.49)	-19.56 (17.99)	-0.11
GL (mm)	49.76 (11.46)*	57.32 (12.06)	-12.11 (18.89)	0.51
SOL (mm)	29.34 (5.71)*	37.13 (7.03)	-19.57 (15.33)	-0.10

Range of Motion Outcomes

No significant differences were observed between the two limbs for ankle ROM, furthermore the LSI for ankle ROM showed no significant relationships with time since surgery (table 5). For the hip ROM in seated position, there was markedly less internal (LSI: 89.92 ± 15.07%) and total (LSI: 93.75 ± 8.16%) ROM in the injured limb with the total ROM reaching statistical significance (p=0.02).

Table 6 Ankle and hip range of motion (internal, external and combined) for the injured and non-injured side. *r* values depict the relationship between range of motion outcomes and months since surgery.

ROM Outcomes	Injured side	Non-injured side	% diff	*r*
Ankle ROM				
Knee-to-Wall (cm)	7.96 (4.41)	8.83 (4.90)	-6.4 (39.6)	0.31
Knee-to-Wall Mod (cm)	33.41 (5.58)	34.54 (5.42)	-3.5 (10.7)	0.35
Hip External ROM				
Prone	44.11 (15.63)	44.17 (17.97)	3.7 (18.2)	0.29
Supine	37.47 (14.60)	36.64 (17.78)	8.6 (28.2)	-0.33
Seated	41.81 (14.72)	42.89 (18.30)	1.4 (19.0)	0.02
Hip Internal ROM				
Prone	46.19 (20.38)	47.72 (16.10)	-4.1 (20.6)	-0.07
Supine	42.83 (20.55)	45.44 (15.44)	-4.0 (35.0)	0.30
Seated	44.81 (17.71)	49.64 (15.34)	-10.1 (15.1)	-0.07
Hip Total ROM				
Prone	90.31 (34.56)	91.89 (32.12)	-1.6 (13.5)	0.16
Supine	80.31 (33.91)	82.08 (30.78)	-2.0 (18.1)	0.07
Seated	86.61 (30.44)	92.53 (30.66)	-6.3 (8.2)	-0.07

DISCUSSION

This study examined the morphological and functional outcomes of operatively treated AT ruptures. Significant differences were found between injured and non-injured limbs many years after surgery with individual variation in deficits despite standardised treatment and rehabilitation. In addition to the changes in AT structure following surgical repair, the muscle fibres in the TS complex display architectural changes resulting in superior muscle quality in the non-injured limb. Importantly, these alterations occur alongside deficits in functional performance, with propulsion and landing during jumping activities being impacted to a greater extent than gait activities. Finally, it was noted that patient reported symptoms do not always reflect objective measures of functional deficits despite being widely utilised.

The significant reductions in muscle thickness in the medial gastrocnemius and soleus agree with previous studies but the current findings highlight the fact that plantar flexor atrophy may be chronic in nature (Horstmann et al., 2012; Heikkinen et al. 2017) and therefore represents an ongoing challenge during rehabilitation. Interestingly the lateral gastrocnemius did not demonstrate the same magnitude of atrophy as the other plantar flexors. It is plausible the differing responses of the plantar flexors reflect their contrasting contributions to different activities (Riemann et al. 2011; Vieria et al. 2013) or that regional differences in muscle atrophy were not detected by the single region uniplanar ultrasonography despite ultrasound

corresponding well with gold-standard imaging techniques (Franchi et al., 2018). Whilst chronic atrophy of TS complex theoretically indicates a reduced capacity for force generation, the significant increases in AT thickness and CSA more likely reflect the healing process rather than improved mechanical properties of the tendon (Freedman et al. 2014). Although increased AT thickness has been consistently observed post rupture (Eliasson, 2018), the findings underline that asymmetries in thickness may be chronic in nature and are more pronounced at more proximal regions.

Although the changes in muscle and tendon thickness post rupture were somewhat expected, the significant increases in fascicle pennation and decreases in fascicle length in the injured limb demonstrate the multi-faceted nature of muscle-tendon function and highlight the need to consider tissue quality as well as tissue quantity. Skeletal muscle has been shown to remodel is response to alterations in surrounding structures in animal models (Burkholder & Lieber, 1998; Koh & Herzog, 1998) and whilst changes in plantar flexor architecture have been recently observed in humans following AT rupture (Peng, 2017; Baxter et al. 2018), this study highlights the long-lasting nature of these changes. It seems likely that the changes in fascicle pennation and length may have occurred as one of many compensatory mechanisms to the loss in muscle size. Specifically, the observed increases in fibre pennation may have facilitated increased fibre density (Kearns et al. 2000) and tendon tension whilst decreases in fascicle length may have served to restore resting tension caused by tendon elongation (Peng, 2017). Although the consequences of greater pennation cannot be fully appreciated through two-dimensional ultrasonography, the trends towards tendon elongation in the injured limb and associations between AT length and fibre length asymmetries are supportive of such outcomes (Kangas et al., 2007; Eliasson, 2018).

Functionally, the chronic atrophy and changes in tendon characteristics did not affect ankle plantar flexion isokinetic strength suggesting that compensatory remodelling of the triceps surae is successful at limiting reductions in ankle joint function. Imaging asymmetries have been observed to exist without impaired functional performance (Khan et al. 2003), something that necessitates the inclusion of function assessment during rehabilitation. Obviously, the success of the compensatory remodelling mechanisms presumes a given level of neural activation during the strength assessment, so increased neural activation could be another mechanism for the recovering leg as the effect of enhanced neural drive on maximum contractions is well established (Trezise et al., 2016). Whilst the isokinetic assessments in the current study were strengthened by the use of differing knee positions to discern between gastrocnemius and soleus contribution, variation was evident in the magnitude of strength asymmetries with some individuals displaying LSI values of below 85% years after surgery. Indeed, previous investigations (Horstmann et al. 2012; Bäcker et al., 2019) have proved controversial when attempting to link morphological and strength changes following AT ruptures and although

isokinetic testing provides a reliable means of isolating the joint over a range of motion, time-dependent variables such as angular work may be more sensitive to changes that take place following AT rupture (Mullaney et al. 2006).

The fact that no strength impairments were observed in the injured limb was also consistent with the absence of gait asymmetries despite some individuals reporting perceived impairments in locomotion during every day activities. Baxter et al. (2018) suggested that gait analysis is a poor test of patient function following an AT rupture since stereotypical gait patterns have been previously observed in patients with severely compromised plantar flexors (Fukunaga et al., 2001). Whilst the lack of differences in gait variables may be population sample related, it is also plausible that mechanisms such as elastic energy transfer and/or optimisation of muscle activation have compensated over time for the gait asymmetries (Horstmann et al., 2012).

Although recent research (Peng et al., 2017; Baxter et al., 2018) correlated architectural remodelling with reductions in functional daily activities (i.e. heel raise measurements), the present findings go one step further by highlighting the architectural remodelling that occurs alongside the high-velocity functional activities that are performed in many sports. Whilst the aforementioned architectural changes would appear to minimise impairments in the injured limb, it is likely that these changes may also result in decreases in muscle force transmission due to fascicle angulation (Kawakami et al. 1998) and reductions in peak power at higher angular velocities due to shorter length of fibres (Lichwark & Wilson, 2008). The significant reductions in jump height, increased take-off times and reduced RSImod scores in the unilateral CMJ are indicative of impaired concentric propulsion capabilities of the plantar flexors (Hubley & Wells, 1983). Moreover, it would appear that patients placed a greater reliance on the non-injured limb during the bilateral DJ as suggested by the significantly longer ground contact times. Whilst individuals may adopt an altered landing strategy to protect the injured side during propulsive and landing phases of jumping, sustained chronic loading of the non-injured side may itself pose a greater risk of further injury. Therefore, there is a strong rationale to develop rehabilitation programmes which integrate functional jumping/landing and targeted resistance training aimed at restoring fascicle morphology (So & Pollard, 1997; Franchi et al. 2016).

The reduced hip external and internal rotation range of motion of the injured limb was a novel finding of the present investigation. That being said, previous investigations into running mechanics following AT ruptures have observed increased knee joint loads (Willy et al., 2017) and internal knee abduction moments (Jandacka et al. 2018) in the injured limb. It is not possible to identify if impairments in the range of motion in the current study are the result of post-operation movement management or they existed prior to the AT rupture.

However, as previous research has linked femoral mechanics with lower limb compensatory mechanisms and loading of AT, there may be a need to consider hip function in clinical assessments aimed at preventing AT rupture or re-rupture (So & Pollard, 1997; Schepsis et al. 2002; Hess, 2010).

Long lasting differences between the injured and non-injured limbs have been observed previously (Horstmann et al., 2012; Barfod et al., 2017) in individuals who have undergone surgical repair of an AT rupture. Whilst some individuals showed limited or no impairments in the injured limb, notable (i.e. LSI <85%) long-lasting differences manifested themselves for a number of individuals in the functional, morphological and self-reported measures. This further supports the previous suggestion that there are limited meaningful improvements as the time since surgery increases (Olsson et al. 2011). Only the self-reported patient outcomes showed significant correlations with time since surgery indicating reduced perceived impairments as time goes by. The lack of association between objective patient outcomes and time since surgery may be indicative of the variation seen between individuals in the magnitude of asymmetries. This variation was evident despite patients having followed the same rehabilitation pathway after undergoing identical surgical procedures. This further highlights the multi-faceted nature of AT rupture outcomes, a factor which healthcare practitioners should consider when managing patient expectations. The lack of association between objective outcomes and time since surgery should be interpreted with caution given the small sample size of the current study. Indeed, a number of individuals displayed marked impairments in the injured limb a number of years following surgery which may highlight the higher sensitivity of biomechanical markers over subjective markers for monitoring the time-course of recovery. This is supported by the fact that the perceived impairments scored on ATRS items 1, 3, 8 and 9 did not closely reflect objective assessments of strength, stiffness, running and jumping.

This study was affected by several limitations. The study is retrospective, meaning pre-injury levels of asymmetry are not known. The sample size was restricted and heterogeneous for age and time post-surgery. Whilst this may have resulted in some variability, the standardisation of surgical methods, surgeon and rehabilitation pathway permitted the control of important confounding variables. As such, the study provides novel information on self-reported, morphological and functional outcomes following AT ruptures treated in a homogenous manner.

CONCLUSION

This study confirmed our hypothesis that the TS complex undergoes architectural and structural changes following surgical repair of AT rupture. These chronic changes appear to partially compensate for plantar flexor atrophy and tendon elongation in the postoperative period. Nevertheless, functional impairments in the

repaired limb may persist which may be more pronounced in high-velocity activities. Further work is required to optimise morphological rehabilitation strategies in the postoperative period with the aim of minimising long-term changes in the structure and architecture of the muscle-tendon unit. From a monitoring perspective, it would appear that patient reported symptoms do not always closely reflect objective measures of functional outcomes. Finally, individual variation in patient outcomes exists despite standardised treatment and rehabilitation routes.

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