ORIGINAL ARTICLE

Vascularity and pain in the patellar tendon of adult jumping athletes: a 5 month longitudinal study

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Background: This study investigated changes in tendon vascularity in 102 (67 men and 35 women) volleyball players over a 6 month competitive season.

Methods: Athletes were examined with both grey scale ultrasound and standardised colour Doppler settings. Vessel length and pain were measured each month on five separate occasions. Vascular tendons were divided into (i) those that were vascular on all occasions (persistent vascularity) and (ii) those that were vascular on more than two but less than five occasions (intermittent vascularity).

Results: A total of 41 of the 133 abnormal tendons were vascular on two or more occasions. Of these, 16 had persistent vascularity and 25 had intermittent vascularity. There was no significant difference in the prevalence of vascularity between men and women. None of the tendons had a pattern of vascularity over the season that could be clearly interpreted as the onset or resolution of vascularity. Subjects with changes in both tendons were more likely to have persistent vascularity ($p = 0.045$). Vessels were longer in tendons with persistent vascularity ($p < 0.000$) and pain was significantly greater ($p = 0.043$) than in tendons with intermittent vascularity. Tendons with intermittent vascularity had similar pain scores on all days, whether or not they had detectable blood flow.

Conclusions: These data suggest that the presence of blood vessels is more likely to be the source of pain than the blood flow in them.

Abnormal tendon vascularity, detected with Doppler ultrasound, has been shown to be an important source of tendon pain in both the Achilles[1] and patellar tendons.[2] Previous studies have only examined tendons at a single point in time, thus the nature of abnormal tendon vascularity over time in both men and women is unclear.

In athletes such as volleyball players, pain from patellar tendinopathy can vary from one week to the next due to the amount of athletic activity,[3] but clinically it is often observed that tendon pain can change in severity for little reason. Changes in tendon vascularity (tendon blood vessels) may be one explanation for pain variation, although investigations of the relationship between tendon vascularity and pain are required to confirm if this is the case.

The aim of this study was to investigate changes in the vascularity and pain in the patellar tendons of male and female volleyball athletes over a full indoor competitive season. In particular, we examined the changes in vascularity and pain in these athletes by measuring the length of the abnormal tendon vessels, the presence of blood flow, and the intensity of pain.

METHODS

A total of 102 volleyball players (67 men and 35 women) who played weekly in three grades from elite to domestic competition participated in the study. Athletes with and without knee pain were included in the study. Ethics approval was granted by the La Trobe University Human Ethics Committee and all subjects provided informed consent.

The patellar tendons in 102 participants (204 tendons) were examined using ultrasound by an experienced musculoskeletal sonographer (JDL) blind to the participant pain scores. The participants were imaged each month for 5 consecutive months from the start to the end of the regular volleyball season. On average, 80% of players were imaged once each month. Those that missed measurement sessions were either not playing or unable to attend for imaging due to other injury (sprained ankle, back pain, etc) or family/social reasons. In no case was the player missing due to patellar tendon pain. All participants were imaged a minimum of three times during the study.

Pain and imaging were recorded prior to competition whenever possible, however for practical reasons 32% of pain measurements and 38% of imaging was done at variable times after the game. Close examination of the data revealed no pattern of pain and vascularity that appeared to be affected by this variation and so all data were considered together.

Ultrasound protocol

Tendon abnormalities in both grey scale and Doppler were imaged on an ultrasound machine with a 13.5 MHz linear transducer (Siemens Acuson CV70, Siemens, Erlangen, Germany). Colour Doppler settings were standardised with a gain of 68 dB, sensitivity of 8 cm/s, and pulse repetition frequency of 1250 Hz. Doppler examination was conducted in all tendons regardless of grey scale status. Both normal and abnormal tendons were examined.

Tendons were designated as vascular if they demonstrated a vessel in the sagittal plane scan that was estimated to be greater than 1 mm in length. If the tendons demonstrated vascularity, a standard 15 mm sampling box was placed on the sagittal image with the greatest vascularity, centred over the proximal aspect of the tendon. To standardise the area measured, only vessels within this box were measured or estimated for all vascularity measures. All images were then recorded to compact disc to allow accurate measurement of vascular length. The vessel lengths on the stored images were measured in millimetres in the sagittal plane using a software package (Photoshop, Adobe version 7). This measurement technique has demonstrated excellent test-retest and inter-tester reliability.[4]

Table 1 Number of tendons and type of vascular changes in men and women

Gender	n	Abnormal (grey scale)	Vascular (colour Doppler)		
			Total	Persistent*	Intermittent*
Male	134	96	33 (34%)†	15	18
Female	70	37	8 (22%)†	1	7

*Fisher exact test, p = 0.08 between men and women; †$\chi^2 = 1.1$, p<1.

Each tendon with visible vascularity was given a vascularity score. This score was determined by assigning one point for each millimetre of vessel visible in the sagittal plane. Vessels estimated and subsequently measured to be less than 1 mm were not scored or measured, while vessels that were not continuous but had breaks of less than 1 mm between ends were considered to be a continuous vessel. Vessels clearly within the fat pad or superficial to the tendon were not counted, while those whose location was more difficult to determine were considered to be tendon vessels.

Pain protocol

The subjects also completed a decline squat to assess patellar tendon pain on each occasion they were imaged. This is a single leg squat on a 25° decline board and athletes were instructed to report anterior knee pain only. The level of pain was recorded by the athlete on a 100 mm visual analogue scale for each leg. This test has been shown to discriminate increases in extensor mechanism pain.[5] The area of pain was recorded on a pain map to exclude those with patellofemoral pain. Pain scores were self reported by the athlete prior to imaging.

Data analysis

All data were entered into a statistical software program (SPSS) and examined for normality. All distributions varied significantly from the normal and non-parametric tests were applied. The persistent and variable vascularity groups were compared for pain (Wilcoxon signed rank test) and vessel length (Mann-Whitney U test). The prevalence (χ^2 analysis) and type of vascularity (Fisher exact test) were compared for men and women.

RESULTS

A total of 133 of the 204 tendons were abnormal on grey scale ultrasound. Of these, 41 were vascular on more than one occasion in 27 athletes (six women, 14 bilateral, 13 unilateral). As expected, no normal tendons exhibited vascularity. Of the 41 vascular tendons, 24 (59%) were imaged all five times, 13 (32%) were imaged four times, and four (9%) were imaged on three occasions.

Sixteen of these 41 tendons had detectable vascularity on every occasion they were imaged (persistent vascularity group), while the remaining tendons had no detectable vascularity on one or more examinations (intermittent vascularity group). No tendons showed a clear pattern of vascularity that could be interpreted as the tendon developing or resolving vascularity over the study period.

Although there was no difference in the prevalence of tendon vascularity between men and women ($\chi^2 = 1.1$, p<1), men tended to have more persistent vascularity than women (Fisher exact test, p = 0.08; table 1).

There was a significant difference in the number of subjects with persistent vascularity in one or both tendons (Fisher exact test, p = 0.045). Fourteen of the 16 tendons that had persistent vascularity were in subjects who had bilateral vascular changes. In contrast, variable vascularity occurred in 11 of the 13 unilateral subjects (table 2).

Table 2 Vascular changes in subjects with unilateral and bilateral changes

Vascularity	n	Bilateral vascularity	Unilateral vascularity
Persistent	16	14	2
Intermittent	25	14	11

Fisher exact test, p = 0.045.

Table 3 Pain scores (mm) in tendons with persistent and intermittent vascularity

	n	Median	Interquartile range	Range
Persistent vascularity*	71	21	45	0–99
Intermittent vascularity*	114	8.5	28.5	0–95

*Mann-Whitney U, z = −2.025; significant difference in pain scores between groups, p = 0.043.

Table 4 Total vascular length (mm) in tendons with persistent and tendons with intermittent vascularity

Vascularity	n	Median	Interquartile range	Range
Persistent*	71	12.2	12.8	1.5–35.0
Intermittent*	50	6.2	5.3	1.0–22.4

*Mann-Whitney U, z = −4.45, significant difference p<0.000.

The amount of pain in the tendons with persistent vascularity and intermittent vascularity on every occasion was then combined and examined. There was a significant difference in the pain scores between those tendons with persistent vascularity and those with intermittent vascularity (Mann-Whitney U, z = −2.025, p = 0.043; table 3).

Persistent and intermittent vascularity

The total length of the tendon vessels was compared between tendons with persistent vascularity and tendons with intermittent vascularity. This analysis obviously excluded tendons with intermittent vascularity on the days that they had no detectable blood flow. Those with persistent vascularity had significantly longer vessels than those with intermittent vascularity (Mann-Whitney U test, z = −4.45, p<0.000; table 4).

Pain

The presence of pain in tendons with persistent and intermittent vascularity was then examined to differentiate the role of vascularity in pain, to see if the temporary loss or gain in vascularity in the intermittent vascular group impacted on pain. There was no clear difference in the presence of pain in tendons with persistent and intermittent vascularity when pain was examined over the entire study period ($\chi^2 = 0.87$, p<1; table 5).

When pain was examined in the intermittent vascularity group and compared between days where no vascularity was evident and days with demonstrable vascularity, there was no difference in pain scores, the tendons being equally painful on the non-vascular days as on the vascular days (Wilcoxon signed rank test, z = −0.268, p = 0.79) (table 6).

DISCUSSION

Abnormal patellar tendon vascularity was prevalent in this group of athletes who sustain regular, high patellar tendon

Table 5 Pain in tendons with persistent and intermittent vascularity

	No pain at any time in the study	Pain on one or more occasions
Persistent vascularity	3	13
Intermittent vascularity	8	17

Table 6 Tendon pain in intermittent vascular group on vascular and non-vascular days

Pain	n	Median	Interquartile range	Range
Non-vascular days	64	8	27	0–69
Vascular days	50	8.5	32.7	0–95

loads. These athletes did not clearly develop or resolve tendon vascularity over the 6 month season, suggesting that adult jumping athletes in this study have relatively stable tendon vascularity.

Other studies have suggested that adolescent athletes may develop vascularity and pain when beginning an intense training program,[6] so this may be when vascular changes are established. Adolescence in fact may be the most critical time in tendon development. Tendon pathology (evident on grey scale ultrasound) exists in elite adolescent jumping athletes at a similar prevalence to adults[7 8] and therefore must develop in the teenage years. It also appears that these changes on grey scale ultrasound are associated with a greater risk of developing pain in adolescents,[9] but not always in adults.[10] Investigations to examine strategies to prevent the onset of tendinopathy in this age group may be warranted.

It has been shown that at a single point in time tendons with detectable vascularity are more painful than either normal tendons or tendons that are abnormal but not vascular.[2] This longitudinal study demonstrates that tendons with persistent vascularity had longer vessels and a greater intensity of pain than those with intermittent vascularity. The amount of vascularity in a tendon (including both the length of the vessels and the presence of detectable flow in them) on a given day appears also to be related to the amount of pain.

By examining those tendons with intermittent vascularity on days with and without detectable blood flow, it was possible to further explore whether it is the vessels themselves or the presence of detectable blood in the vessels that causes pain. This study suggests that the existence of the vessels and the associated neural structures[11] appears to be more important than the presence of detectable blood. This is supported by the clinical observation that tendon pain ''warms up'' with exercise, at the same time that blood flow in the tendon may actually increase.[12] Clinically this implies that when imaging athletes with tendon pain, efforts should be made to examine tendons after exercise, as blood flow and the presence of abnormal vessels may be more easily detected.

This study indicates that some athletes seemed to be predisposed to abnormal patellar tendon vascularity and developed vascularity in both tendons. These athletes with bilateral tendon pathology were also more likely to have persistent vascularity than those with unilateral tendon changes. This suggests two aspects of tendon disease. First, load (but not pain) may be less important in the development of tendon pathology than some inherent individual characteristics as all athletes in this cohort had similar training and competitive loads. Second, this study increases the evidence that there may be different aetiologies in subjects with unilateral and bilateral patellar tendon pathology. Previous studies have shown that subjects with bilateral changes differ from those with unilateral changes as regards flexibility,[13] waist-hip ratio, and tibial length.[14] The reasons for this are unclear and further investigations are warranted.

Although tendon pathology has been shown to be twice as prevalent in men as in women,[8] the presence of vascularity was similar in men and women with pathological tendons in the current study. The relationship between gender and soft tissue injury remains undefined, as gender factors that increase the prevalence of tendon pathology in women do not appear to affect tendon vascularity in the same way.

What is already known on this topic

Abnormal tendon vascularity has been shown to be an important source of tendon pain, but the nature of abnormal tendon vascularity over time is unclear.

What this study adds

Vascularity is more likely to be persistent when pathological changes are present in both tendons. Tendon vascularity is associated with intensity of tendon pain which appears to be more dependent on the amount of vascularity than the volume of blood in the vessels.

CONCLUSION

Tendon vascularity in active jumping athletes was stable over the 5 months of a competitive season. When pathological changes were present in both tendons, vascularity was more likely to be persistent. Tendon vascularity was associated with the intensity of tendon pain. Nevertheless tendon pain appears to be more dependent on the amount of vascularity than the volume of blood in the vessels.

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COMMENTARY

Vascularity in tendons has gained increasing attention with the advent of colour Doppler imaging. Although early studies suggested there may be an association between pain and vascularity,[1] this was not supported in subsequent papers.[2] However, everyone took notice when Ohberg and Alfredson sclerosed neovessels and eradicated tendon pain.[3] Since then, there have been several cross sectional studies including one on high impact radiology discussing the clinical relevance of neovascularisation.[4] Alfredson published a paper entitled ''Is vasculo-neural ingrowth the cause of pain in chronic Achilles tendinosis? An investigation using ultrasonography and colour Doppler, immunohistochemistry, and diagnostic injections''.[5] So it is time for a synthesis!

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