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

J L Cook, P Malliaras, J De Luca, R Ptasznik, M Morris


ABSTRACT

Abnormal tendon vascularity, detected with Doppler ultrasound, has been shown to be an important source of tendon pain in both the Achilles and patellar tendons. 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, 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. 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 vascularity. 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-reader reliability.

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.
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 in extensor mechanism pain.5 The area of pain was recorded on a pain map to exclude those with patellofemoral pain protocol.

**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 (χ² 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 (χ² = 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).

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 (χ² = 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 vascularity.
Table 5  Pain in tendons with persistent and intermittent vascularity

<table>
<thead>
<tr>
<th></th>
<th>No pain at any time in the study</th>
<th>Pain on one or more occasions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent vascularity</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>Intermittent vascularity</td>
<td>8</td>
<td>17</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Pain</th>
<th>n</th>
<th>Median</th>
<th>Interquartile range</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-vascular days</td>
<td>64</td>
<td>8</td>
<td>27</td>
<td>0–69</td>
</tr>
<tr>
<td>Vascular days</td>
<td>50</td>
<td>8.5</td>
<td>32.7</td>
<td>0–93</td>
</tr>
</tbody>
</table>

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.

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, waist-hip ratio, and tibial length. 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, 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.

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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REFERENCES

Vascularity and pain in the patellar tendon

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, this was not supported in subsequent papers. However, everyone took notice when Ohberg and Alfredson sclerosed neovessels and eradicated tendon pain. Since then, there have been several cross sectional studies including one on high impact radiology discussing the clinical relevance of neovascularisation. Alfredson published a paper entitled “Is vasculo-neural ingrowtih the cause of pain in chronic Achilles tendinosis? An investigation using ultrasonography and colour Doppler, immunohistochemistry, and diagnostic injections”. So it is time for a synthesis!

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REFERENCES

Announcement

8th International Congress of the Society for Tennis Medicine and Science
14–15 January 2006, Melbourne, Australia

Join Australian and international medical practitioners, including sports physicians, orthopaedic surgeons, and sports physiotherapists at this premier tennis medical conference. To be held immediately prior to the 2006 Australian Open tennis championships, the conference will combine presentations from international and Australian experts, including Professors Tim Noakes, Bruce Elliott, and Mark Hargraves to stimulate discussion on topical tennis science and medicine issues. There will also be an engaging and entertaining social programme. Of course there is also the opportunity to attend the prestigious Australian Open and visit many of the wonderful sites Australia has to offer.

For further details please email stms2006@meetingplanners.com.au or visit www.stms2006.com.au
Convenor: Dr Timothy Wood