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

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.
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. 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 (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<.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 vascularity 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

<p>| Table 1 Number of tendons and type of vascular changes in men and women |
|---------------------------------|----------------|----------------|----------------|</p>
<table>
<thead>
<tr>
<th>Gender</th>
<th>n</th>
<th>Abnormal (grey scale)</th>
<th>Vascular (colour Doppler)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>134</td>
<td>96</td>
<td>33 (34%)†</td>
</tr>
<tr>
<td>Female</td>
<td>70</td>
<td>37</td>
<td>8 (22%)†</td>
</tr>
</tbody>
</table>

*Fisher exact test, p = 0.08 between men and women; †χ² = 1.1, p<.1.

<p>| Table 2 Vascular changes in subjects with unilateral and bilateral changes |
|---------------------------------|----------------|----------------|----------------|</p>
<table>
<thead>
<tr>
<th>Vascularity</th>
<th>n</th>
<th>Bilateral vascularity</th>
<th>Unilateral vascularity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent</td>
<td>16</td>
<td>14</td>
<td>2</td>
</tr>
<tr>
<td>Intermittent</td>
<td>25</td>
<td>14</td>
<td>11</td>
</tr>
</tbody>
</table>

Fisher exact test, p = 0.045.

<p>| Table 3 Pain scores (mm) in tendons with persistent and intermittent vascularity |
|---------------------------------|----------------|----------------|----------------|</p>
<table>
<thead>
<tr>
<th>Vascularity</th>
<th>n</th>
<th>Median</th>
<th>Interquartile range</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent vascularity*</td>
<td>71</td>
<td>21</td>
<td>45</td>
<td>0–99</td>
</tr>
<tr>
<td>Intermittent vascularity*</td>
<td>114</td>
<td>8.5</td>
<td>28.5</td>
<td>0–95</td>
</tr>
</tbody>
</table>

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

<p>| Table 4 Total vascular length (mm) in tendons with persistent and intermittent vascularity |
|---------------------------------|----------------|----------------|----------------|</p>
<table>
<thead>
<tr>
<th>Vascularity</th>
<th>n</th>
<th>Median</th>
<th>Interquartile range</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent*</td>
<td>71</td>
<td>12.2</td>
<td>12.8</td>
<td>1.5–35.0</td>
</tr>
<tr>
<td>Intermittent*</td>
<td>50</td>
<td>6.2</td>
<td>5.3</td>
<td>1.0–22.4</td>
</tr>
</tbody>
</table>

*Mann-Whitney U, z = −4.45, significant difference p<.000.
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, 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 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, but not always in adults. 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. 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) 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 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. 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, 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.

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.

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 Interquartile Range</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-vascular days</td>
<td>64</td>
<td>8</td>
<td>27</td>
</tr>
<tr>
<td>Vascular days</td>
<td>50</td>
<td>8.5</td>
<td>32.7</td>
</tr>
</tbody>
</table>

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 ingrowth 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

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