Color Doppler ultrasound findings in patellar tendinopathy (jumper's knee)

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ABSTRACT

Background: Recent studies have revealed structural changes with neovessels in patients with jumper's knee and Achilles tendinopathy, and treatment with sclerosing injections have shown promising clinical results.

Purpose: To study the prevalence of neovascularisation and structural tendon changes on color Doppler ultrasound examination in elite athletes with clinical symptoms of jumper's knee, and to examine the ultrasound characteristics of the tendon after sclerosing injection treatment with polidocanol.

Study Design: Cohort study Level of Evidence 3.

Methods: We recruited patients among elite athletes with a clinical diagnosis of jumper's knee who participated in a previous randomized clinical trial. The patients recorded knee function using VISA score. Patients were examined by color Doppler ultrasound at baseline and, for patients with structural changes and neovascularisation who received sclerosing treatment, also post-treatment.

Results: We included 63 patients (11 women and 52 men) with 79 symptomatic tendons. The ultrasound examination revealed that neovascularisation was present in 48 of the 79 tendons (60%). Of 33 patients (43 tendons) who received sclerosing injections, 29 patients (37 tendons, 86%) were examined 37 (19 to 53) weeks after their final sclerosing injection. Of these, 7 tendons (18.9%) had no change in neovascularisation after treatment, 21 tendons (56.8%) had less neovascularisation, and 9 tendons (24.3%) had more visible neovascularisation after treatment. There were no significant differences in the change in VISA score between patients who had less, more or unchanged neovascularisation after treatment (ANOVA, p=0.9).

Conclusion: About 2/3 of patients with jumper's knee can be expected to have structural tendon changes with neovascularisation. There was no relationship between changes in ultrasound characteristic and knee function after sclerosing treatment.

Key terms: Patellar tendinopathy –color Doppler ultrasound – neovascularisation – sclerosing therapy – tendon changes

INTRODUCTION

In two early studies, Weinberg et al.[20] and Terslev et al.[18], using color Doppler ultrasound examination, showed that neovessels can be demonstrated in some patients with jumper's knee. In a study on the midportion Achilles tendon, color Doppler ultrasound revealed neovascularisation in the painful area with structural changes in tendons with chronic tendinosis, but not in pain-free normal tendons.[15] We later showed that an eccentric exercise program stopped the flow in these vessels, and that the patients became symptom-free.[11,12] In another study, on active jumping athletes, the presence of neovascularisation in abnormal patellar tendons was associated with more tendon pain compared to abnormal tendons without neovascularisation.[5] Taken together, these findings led to the hypothesis that the vessels, and possibly also the nerves accompanying the vessels, were involved in the pain mechanism in chronic painful Achilles tendinosis. This hypothesis led to a pilot study of a new treatment method with sclerosing of these vessels,[13,14] showing promising results in midportion Achilles tendinosis patients. In a recent randomized controlled trial on 42 patellar tendons with tendinopathy, injection treatment with the sclerosing agent polidocanol showed convincing clinical results in this group, as well.[6] The area with neovascularisation/increased blood flow is the target area for sclerosing injection treatment. However, it is not known, in tendons clinically diagnosed with jumper's knee, how many display neovascularisation and could be suitable for treatment. Also, it is of interest to follow the ultrasound characteristics of the patellar tendon after sclerosing treatment with polidocanol, since it is not known how such treatment affects tendon structure and vessels.

Therefore, we wanted to study a number of elite athletes with clinical symptoms of jumper's knee to see how many display neovascularisation, and to study the post-treatment color Doppler ultrasound characteristics of the tendons included in the randomized trial[6] after sclerosing treatment.

MATERIALS AND METHODS

Patient recruitment

We recruited patients by contacting clubs and players in the elite division in basketball, team handball and volleyball for both male and female players in the southern part of Norway with an invitation to take part in a clinical examination. The coach and the club were informed of the purposes and procedures of the study by letter (mid-March 2004), and we visited each of the clubs to inform the players of the purposes and procedures of the study towards the end of the competitive season at a time convenient to them. After an oral presentation, the players were asked to take part in a clinical screening exam, where they were required to complete a questionnaire detailing their anthropometric details, history of their knee pain, any treatment received, sporting profile and activity level. Patients who fulfilled the inclusion criteria were invited to the ultrasound screening. In addition, a press release about the study led to coverage in a major newspaper, where elite athletes from all sports were asked to contact the investigators for a clinical and ultrasound exam in the same way as for the team sports.

The study was approved by the Regional Committee for Medical Research Ethics and written consent was obtained from each subject.

Inclusion criteria

The inclusion of patients to the baseline ultrasound examination was based on the clinical examination alone. The following diagnostic criteria were used to identify patients with jumper's knee:[9]

- History of pain in the patellar tendon or the patellar tendon insertion into the tip of the patella in connection with training or competition
- Tenderness to palpation corresponding to the painful tendon area
- Symptoms from the patellar tendon for a minimum of three months
- VISA (Victorian Institute of Sport Assessment) score (0 to 100 points) of less than 75 points
- Both knees were included if the patient had bilateral problems.

Subjects were excluded if they had a history of knee problems caused by patellofemoral pain syndrome, inflammatory joint conditions or degenerative conditions. Subjects had to be between 18 and 40 years old. These age limits exclude the diagnoses of Osgood-Schlatter and Sinding-Larsen-Johansson disease in the adolescent athlete, and significant osteoarthritis in the older athlete.

Subjects also had to be residents of Norway and understand oral and written Norwegian.

Sclerosing treatment and follow-up

The baseline ultrasound examinations were performed in May – June 2005, one to six weeks after the clinical screening exam. All patients with neovascularisation/increased blood flow corresponding to the painful area were invited to be included in a randomized clinical trial to test the effect of sclerosing treatment with polidocanol. Patients without neovascularisation were not offered further treatment or follow-up. The design, methods and clinical results of the clinical trial have been described in detail in a separate paper.[6] Polidocanol (Aethoxysklerol [10 mg/ml], Inverdia AB, Sweden) was used as the sclerosing agent. Patients were randomized to a treatment group, who received polidocanol injections against the area with vessels entering the patellar tendon from the dorsal side of the tendon, or a control group, who initially received similar injections with lidocaine and epinephrine (Xylocaine-Adrenalin [5 mg/mL + 5 µg/mL], AstraZeneca, Oslo, Norway). After 4 months of treatment, the control group was also given active treatment with polidocanol. The patients in both groups received from one up to a maximum of five injections at 4-6 week intervals depending on their pain level and whether there was visible neovascularisation after last treatment session. In other words, patients in both groups received polidocanol injections until they had an acceptable clinical result. The post-treatment ultrasound examinations were performed in June through August 2006, i.e. 12-15 months after the baseline ultrasound examination.

Ultrasound examination and evaluation

Both examinations were performed by the same, experienced ultrasonographer (LÖ), using high-resolution grey-scale ultrasound with the aid of color Doppler (Philips EnVisor HD, Vingmed as). A linear multifrequency (8-13 MHz) probe (type L12-3) was used, and the pathological changes in the painful thickened patellar tendon were registered on a standard form. Color Doppler was used to diagnose vascularity/increased blood flow.

We classified the main ultrasound findings in three categories:

- Normal tendons: Tendons with a regular and smooth fiber structure without hypoechoic areas or vascular flow (Fig. 1a).

- Abnormal tendons with structural changes: Tendons with a localized widening, including irregular structure and hypoechoic areas (Fig. 1b).
- Abnormal tendons with neovascularisation/increased blood flow: Tendons with a localized widening, including irregular structure, hypoechoic areas and vascular flow (one or several vessels inside the area with structural changes) (Fig. 1c).

(Figure 1 near here)

We also measured the thickness of the tendon before and after sclerosing treatment at the apex, as well as 0.5 cm and 1.0 cm from the apex.

We classified the neovascularisation before and after treatment in four categories ranked according to the degree of visible neovascularisation, were 0 represents no visible blood vessels, 1 represents 1-2 blood vessels (low blood flow), 2 represents some blood vessels (some blood flow), and 3 represents many blood vessels (strong blood flow).

Symptom evaluation

Knee function was assessed using the VISA (Victorian Institute of Sport Assessment) score and a visual analog scale for pain. The VISA score was designed specifically to quantify knee function in subjects with patellar tendinopathy and has also been shown to be a reliable and valid measure.[4,7,18] The patients self-recorded VISA score separately for each knee during the clinical screening exam and before the ultrasound exams. At the time of the ultrasound exams, the patients also self-recorded their knee pain during training the previous two weeks on a 10-cm continuous line marked “no pain” on one end and “worst pain” on the other. The 10-cm visual analog scale has been shown to be a reliable and sensitive scale for pain and has been used extensively in orthopedic investigations.[8,16]

Before the ultrasound exams, the patients were asked to perform 15 squats on a 25° decline board on the affected leg at a frequency of 15 squats per minute. They were instructed to use five seconds for the eccentric part of the squat, and three seconds for the concentric part, and to stop if it was too painful to continue. These squats were done to evaluate their level of patellar tendon pain during the exercises, and this pain level was also self-recorded on a visual analog scale.

Statistics

Subject characteristics were compared for differences in baseline anthropometry and injury severity. To test the principal null hypothesis, that there was no group difference in VISA scores and pain score in relation to the ultrasound findings, these were compared using ANOVA and paired t-tests.

We have used a significance level of 5%, and unless otherwise noted, the results are presented as the mean ± standard deviation.

RESULTS

Patient characteristics

We included 63 patients (11 women and 52 men) with 79 tendons having a clinical diagnosis of jumper's knee, i.e. 16 patients with bilateral problems (Table 1). The patients primarily represented handball (n=24), basketball (n=10), volleyball (n=9) and football (n=8), but some were also included from ice hockey (n=1), orienteering (n=2), taekwondo (n=1), boxing (n=1), aerobic fitness (n=1), tennis (n=1) and athletics (n=4). The self-reported VISA score at the time of the clinical screening exam for the 79 symptomatic tendons was 57±15 (range 15 to 89).

Baseline ultrasound characteristics

The ultrasound examination revealed that structural tendon changes (thickening with hypoechoic areas) and neovascularisation was present in 48 of the 79 tendons (60%), while 20 of the tendons had structural changes without neovascularisation, and 11 were without structural changes. Of the 20 tendons with structural changes without neovascularisation, 14 displayed thickening and hypoechoic areas, 5 had hypoechoic areas only, and 1 was classified as thicker than normal.

There were no differences in age, height, body mass or training history between players with normal tendons, tendons with structural changes and tendons with neovascularisation (Table 1). In addition, there was no significant difference in the duration of symptoms between the groups (tendons with neovascularisation: 34±45 months; structural changes: 43±39 months; normal tendons: 27±23 months; ANOVA, p=0.5).

(Table 1 near here)

However, the self-reported VISA score at the time of the ultrasound exam was lower for tendons with neovascularisation than for tendons with structural changes without neovascularisation or tendons with normal ultrasound findings (ANOVA, p=0.001) (Fig. 2).

(Figure 2 near here)

For the athletes who had the clinical screening exam during the competitive season and the ultrasound examination after the end of the season with an interval of more than 3 weeks between the two examinations (n=50 tendons), the change in VISA score was significantly greater for tendons with structural changes only compared with tendons with structural changes and neovascularisation (ANOVA, p= 0.001). For tendons with structural changes only (n=17), VISA score improved by 15±3 (SE) points from the clinical screening exam until the ultrasound exam, while there was no change for tendons with structural changes and neovascularisation (-2±2, n=27) or normal tendons (4±4, n=6).

The self-reported pain level at the time of the baseline ultrasound exam was also higher for tendons with structural changes and neovascularisation, compared to tendons with structural changes alone or normal tendons (ANOVA, p=0.001) (Fig. 3). The self-recorded pain level during the squat test showed the same trend as the recorded pain during activity; higher for tendons with structural changes and neovascularisation compared to tendons with structural changes alone or normal tendons (ANOVA, p=0.001) (Fig. 3).

(Figure 3 near here)

Post-treatment ultrasound characteristics

A total of 38 patients (5 women and 34 men) had structural changes and neovascularisation at the ultrasound examination, and therefore were eligible for inclusion in the randomized clinical trial to receive sclerosing treatment. Of these, 33 (5 women and 28 men; 43 tendons) elected to go through sclerosing treatment. Four of the patients (6 tendons) who received sclerosing treatment were not able to attend the second ultrasound examination for practical reasons, and a total of 29 patients (37 tendons) participated at the post-treatment ultrasound examinations. The follow-up ultrasound examination was done 37 (19 to 53) weeks after their final sclerosing injection.

As shown in Table 2, in 7 tendons (18.9%) there was no change in neovascularisation after sclerosing treatment compared to before treatment, 9 tendons (24.3%) had more neovascularisation, and 21 tendons (56.8%) had less neovascularisation compared to before treatment.

(Table 2 near here)

All patients had hypoechoic areas before treatment (n=37). After treatment 3 tendons (5.4%) no longer had hypoechoic areas, and 34 tendons (94.6%) still revealed hypoechoic areas. Furthermore, in 16 tendons (43.2%) there was no change in hypoechoic status, one tendon (2.7%) had greater hypoechoic areas, and 20 tendons (54.1%) had less hypoechoic areas after sclerosing treatment compared to before treatment.

The mean tendon thickness at the apex was not different after treatment compared to before treatment. However, the tendon thickness was significantly *greater* 0.5 cm (0.73 cm ± 0.03 vs. 0.87 cm ± 0.03 [SE]) and 1 cm (0.62 ± 0.05 vs. 0.74 ± 0.05) distal to the apex after treatment compared with before treatment.

Relationship between ultrasound findings and clinical function

There was a significant improvement in VISA score from 52 ± 13 (SD) at baseline to 80 ± 18 at the time of the post-treatment examination (p=0.001, n=37). There was no difference in VISA scores at follow-up between the 37 tendons that were available for ultrasound examination, and the 6 tendons we were not able to examine (87.0 ±14.6 (SD), p=0.23).

Furthermore, at the time of the follow-up investigation, there was no difference in the change in VISA score between subjects in the control group, who received polidocanol injections after 4 months of placebo treatment, and the treatment group, who received polidocanol injections without delay (p=0.72).

The improvement in self-reported VISA score after treatment compared to before treatment was 30 ± 17 for tendons with unchanged neovascularisation status (n=7), 29 ± 21 for tendons with less neovascularisation (n=21), and 25 ± 30 for tendons with more neovascularisation (n=9). There were no significant differences in the change in VISA score between tendons with less, more or unchanged neovascularisation after treatment (ANOVA, p=0.9). The relationship between change in neovascularisation status and change in VISA score is also shown in Table 2.

The increase in self-reported VISA after treatment compared with before treatment was 28 ± 18 for tendons with no change in hypoechoic areas (n=16), 29 ± 24 for tendons with less hypoechoic areas (n=20), and 1.0 for one tendon with greater hypoechoic areas. The increase in VISA score for patients who no longer had visible hypoechoic areas after treatment was 21 ± 21 (range -4 to 34), and 30 ± 23 (range -15 to 80) for patients who still had hypoechoic areas after sclerosing treatment. There were no significant differences in the change in VISA score between tendons with or without hypoechoic areas after treatment (p=0.54).

DISCUSSION

This study showed that in competitive athletes with clinical symptoms of jumper's knee, 60% of the examined tendons displayed structural tendon changes together with neovascularisation/increased blood flow, while 26% had structural changes alone, and 14% were normal. For patients with neovascularisation who went through sclerosing injection treatment with polidocanol, knee function had improved, but tendon structure was abnormal in most cases and there was no correlation between clinical improvement and structural changes as seen on the follow-up ultrasound examination 13-53 weeks after the final injection.

Prevalence of neovascularisation in patients with jumper's knee

Weinberg et al.[20] were the first to describe their experience with color Doppler sonography in the evaluation of patellar tendinosis. They found that in 20 symptomatic tendons from 14 patients, 11 had a proximal hypoechoic area, 12 had a thickened proximal patellar tendon and all but one of these had increased blood flow on color Doppler sonography. The level of sports participation, symptom intensity and duration of symptoms for these patients is not known. Next, Terslev et al.[18] examined a group of 18 elite basketball players after a match. Among these, 4 players reported symptoms of jumper's knee, which were confirmed on tendon palpation, and three of these had both hypoechoic areas and increased blood flow. The remaining results are difficult to interpret, since data were not reported separately for each knee.

In a recent cross-sectional study, Cook et al.[5] examined 111 volleyball players representing elite to domestic competition levels with ultrasound, and related the ultrasound findings to different measures of pain and knee function. The ultrasound examination revealed that of the 222 tendons examined, 124 were normal, 72 were abnormal with no vascularity and 26 were abnormal with vascularity. However, since the clinical status was not reported separately for each knee, it is not possible to estimate the prevalence of neovascularisation among athletes with clinical symptoms of jumper's knee. In a recent study, 15 elite or recreational athletes with long-standing symptoms (mean 23 months) of jumper's knee were treated with sclerosing polidocanol injections.[1] In all of these patients, ultrasonography and color Doppler examination showed structural changes with hypoechoic areas and neovascularisation corresponding to the painful area. From the above studies, it appears that neovascularisation is *not* a constant finding in patients with a clinical diagnosis of jumper's knee. This hypothesis is confirmed by the present study, which is the first to correlate the color Doppler ultrasound characteristics with clinical findings in a multisport patient group of competitive athletes having significant symptoms of jumper's knee. Our results suggest that about 2/3 of patients display neovasularisation and as such may be candidates for sclerosing therapy with polidocanol injections targeting the area with neovessels.

However, the current findings also show that patellar tendon pain is not easy to diagnose, and that the value of ultrasound examination in this group of patients may be questioned. It is possible that the 14% of patients that had normal tendon ultrasound appearance may have other causes of pain, and patellofemoral cartilage lesions and synovial plicae can give rise to clinical symptoms similar to those of jumper´s knee. Nevertheless, as discussed above it is well documented that a number of symptomatic tendons do not reveal any ultrasound changes. Also, studies have shown that a number of athletes have tendon ultrasound changes without ever having experienced symptoms of jumper's knee.[7,9] The reasons for this apparent discrepancy are not known, and the current paper provides additional evidence that the link between structural tendon changes, neovascularisation and symptoms is not clear. Other factors, as for example neuromediators, have been implicated in tendon pain. Neuropeptides, such as substance P, modulate important aspects of tendinopathy including not just vascular flow, but also pain and tissue remodeling.[17]

Relationship between neovascularisation and symptoms

Cook et al.,[5] in their cohort of symptomatic and asymptomatic volleyball players, reported significantly lower VISA scores in patients with abnormal tendons with neovascularisation than in abnormal tendons without neovascularisation. The present study also shows that there is more pain and lower function scores in patients with abnormal tendons and neovascularisation, compared with patients with abnormal tendons without neovascularisation, and patients with normal tendons.

Thus, it is possible that the percentage of patients with neovascularisation would have been even higher if they had been examined with ultrasound during the peak competitive season (when the initial screening exam was done for the team sport athletes). For the baseline ultrasound examination, most of the athletes were examined several weeks after the end of the competitive season, when training and competition load was lower, and athletes without neovascularisation reported significantly improved function and less pain than during the in-season clinical screening exam. Consequently, the apparent relationship between neovascularisation VISA score, and pain leads to the hypothesis that the degree of neovascularisation varies with activity load, and that patients in periods with less pain and load may have no visible neovascularity. Whether this means that the vessels are absent, or that blood flow is too low to be detectable on color Doppler ultrasound, is open to speculation.

Other studies demonstrate findings that support neovessels and accompanying nerves as a possible source of pain in chronic midportion Achilles tendinosis.[3] The data in our study reveals that thickening does not occur without hypoechoic areas, and that neovascularisation does not occur without structural changes. The same pattern seems to have been observed in all of the previous studies combining gray scale and color Doppler ultrasonography.[2,5,18,20] This suggests that structural changes (thickening, irregular collagen fiber arrangement and hypoechoic areas) occur in the early stages of tendon injury, before blood vessels appear and pain symptoms worsen significantly. However, this hypothesis needs to be tested in a longitudinal study with serial ultrasound examinations and careful prospective registration of symptoms in a large cohort of jumping athletes.

Ultrasound changes after sclerosing therapy

Out of 33 patients (43 tendons) who received sclerosing treatment in this study, 4 patients could not make their appointment and did not attend the follow-up ultrasound examinations. A total of 29 patients (37 tendons, 86%) participated at the post-treatment ultrasound examinations. We are not aware of any follow-up bias, as this seemed to be more related to geographical and practical issue than clinical symptoms, as also documented by their VISA scores.

At the follow-up ultrasound examination we saw improvements in neovascularisation and hypoechoic areas, but only in some cases. All taken together, 13 of 37 tendons (35%) no longer revealed visible neovascularisation and 3 of 37 (5%) did not display hypoechoic areas. This is in contrast to results of the study by Lind et al[10] on 42 tendons from middle-aged (mean age 53 years) runners 2 years after sclerosing therapy for Achilles tendinosis. In this study, we found a considerable reduction in remaining neovessels. Color Doppler ultrasound showed no, or a few, remaining neovessels in the majority of the successfully treated tendons. There was also a significant reduction in tendon thickness at the 24 month follow-up. In the present study, the tendon thickness was not different at the apex and had *increased* 0.5cm and 1.0cm distal to the apex.

Furthermore, we could not find any relationship between the ultrasound findings and clinical function after sclerosing treatment. All patients reported improved function scores after treatment to the degree where they were playing elite sports with no or minimal symptoms, but there was no difference in VISA scores between tendons with less, unchanged or more neovascularisation and hypoechoic areas. This is in contrast to the results from the previous study on Achilles tendinopathy,[10] where patients with a good result of sclerosing treatment had no or few remaining neovessels, while all patients with a poor result had multiple remaining neovessels. One obvious potential explanation for the conflicting results between the present study and the study on Achilles tendinopathy is the time to follow-up. We did the follow-up 12 months after the first injection, and for patients who received placebo injections in the first 4 months, the follow-up was actually performed only 3-6 months after the last sclerosing injection. This may be too early to detect significant tendon remodeling with a reduction in tendon thickness and neovascularisation corresponding to the effect of sclerosing injections. Nevertheless, the present data clearly suggest that clinical improvement after sclerosing therapy is not related to the presence or absence of neovessels or hypoechoic areas within the tendon. Consequently, treatment should be based on the combination of tendon pain, structural changes and neovascularisation, and not on tendon changes and neovascularisation alone.[1]

It should also be kept in mind that the Achilles tendinopathy study included a mixed patient group consisting mainly of middle-aged, recreational athletes. In the present study we have included elite athletes with a high activity level between the sclerosing injections and after the last treatment session. This high strain may influence the tendon's potential to recover. Another explanation may be that sclerosing treatment does not result in the same ultrasound changes for insertional tendinopathies (patellar or quadriceps tendinopathy) as for midportion tendinopathy (Achilles tendon). These issues need to be examined in a larger study on patients with different tendinopathies and with longer follow-up.

The explanation for the apparent discrepancy between clinical changes and post-treatment ultrasound appearance is not known. However, the speed with which the tendon pain is reduced suggests that the injections may be interfering with the local nerve supply, essentially creating a partial denervation of the tendon. The treatment also results in an acute increase in tendon blood flow, possibly from alternate collateral circulation.[1] Thus, the effect of this treatment on neuropeptide distribution and neurovascular regulation warrants further investigation.

Conclusion

Among elite athletes with a clinical diagnosis of jumper's knee, 60% had tendon changes together with neovascularisation, making them candidates for sclerosing therapy. Athletes with neovascularisation reported more pain than patients without neovascularisation. Although sclerosing treatment lead to significant improvements in function, to the degree that all patients were competing with no or minimal pain, the majority of tendons still revealed significant neovascularisation and hypoechoic areas 3-12 months after the final injection. Also, there was no relationship between changes in function scores and changes in ultrasound status.

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FIGURE LEGENDS

Fig. 1. Typical ultrasound appearance: a) Normal tendon with regular and smooth fiber structure, no hypoechoic areas or vascular flow. b) Abnormal tendon with localized widening, including irregular structure and hypoechoic areas. c) Abnormal tendon with localized widening, including irregular structure, hypoechoic areas and vascular flow (several vessels inside the area with structural changes).

Fig. 2. VISA score. The self-reported VISA score before the ultrasound exam for tendons with neovascularisation, tendons with structural changes without neovascularisation and tendons with normal ultrasound findings.

Fig. 3. Pain level during training for the previous two weeks and self-recorded pain level during the squat test showed for tendons with neovascularisation, tendons with structural changes without neovascularisation, and tendons with normal ultrasound findings.

TABLES

Table 1. Subject characteristics (n=63 patients) in three different groups, categorized according to the baseline examination. Patients with two normal tendons were categorized in the normal tendon group, patients with neovascularization on at least one side in the neovascularization group, and patients with structural changes on at least one side in the structural changes group.

	Normal tendon (n=10)	Structural changes (n=16)	Neovascularisation (n=37)
Age (years)	23.8 ± 5.6 (17 - 42)	25.9 ± 5.2 (19 - 36)	24.6 ± 6.0 (17 - 42)
Height (cm)	172.8 ± 5.6 (17 - 42)	186.9 ± 7.1 (172 - 202)	182.5 ± 8.7 (168 - 201)
Body mass (kg)	72.6 ± 11.3 (55 - 95)	86.1 ± 10.2 (62 - 102)	81.1 ± 9.7 (62 - 96)
Sports-specific training (h/week)	7.1 ± 3.1 (2.0 - 12.0)	11.2 ± 2.7 (6.0 - 15.0)	10.1 ± 3.2 (3.0 - 15.0)
Weight training (h/week)	2.7 ± 2.4 (1.0 - 8.0)	3.9 ± 1.3 (1.0 - 6.0)	3.5 ± 2.1 (1.0 - 8.0)
Jump training (h/week)	1.0 ± 0.0 (1.0 - 3.0)	1.3 ± 0.5 (1.0 - 2.0)	1.6 ± 2.4 (0.0 - 9.0)
Total amount of training (h/week)	11.2 ± 4.8 (2.0 - 18.0)	14.4 ± 5.2 (5.0 - 22.0)	14.5 ± 5.2 (5.0 - 28.5)
Duration of symptoms (months)	27.3 ± 23.9 (5.0 - 72.0)	43.1 ± 39.9 (5.0 - 120.0)	34.0 ± 44.7 (4.0 - 240.0)

Table 2. Change in VISA score in relation to change in neovascularisation status from before to 12-15 months after sclerosing treatment (n=37).

	Neovascularisation grading after treatment			
Neovascularisation grading before treatment	0	1	2	3
1	35 (25 to 44) n=6	22 (10 to 33) n=2	30 (-4 to 59) n=4	62 (-) n=1
2	21 (-15 to 35) n=4	26 (8 to 45) n=5	33 (7 to 58) n=5	11 (-14 to 43) n=4
3	53 (31 to 80) n=3	22 (1 to 42) n=2	-9 (-) n=1	- n=0