Ultrasonography as a tool for diagnosis, guidance of local steroid injection and, together with pressure algometry, monitoring of the treatment of athletes with chronic jumper's knee and Achilles tendinitis: a randomized, double-blind, placebo-controlled study

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Background: The diagnosis of Achilles and patella tendinitis has until recently been based on clinical examination, and treatment with local steroid injection has been given blindly. This is the first randomized, double blind, placebo-controlled study of local steroid injection in athletes with chronic tendinitis, which used ultrasonography to increase diagnostic accuracy, to guide the correct placement of local steroid and, conjunctively with pressure algometry, to objectify and monitor the results of treatment.
Method: Forty-eight athletes each with severe symptomatic tendinitis of a patellar (24) or Achilles tendon (24) for more than 6 months, whose conditions were confirmed ultrasonographically, and who all failed conservative treatment (rehabilitation) were included in this double-blind, placebo-controlled study and treated with three ultrasonographically guided peritendinous injections of steroid or placebo.
Results: The conditions of only one-third of the referred athletes with clinically suspected tendinitis were confirmed by ultrasonographic examination. The ultrasonographically guided peritendinous injection of steroid had a significant effect in reducing pain and thickening of tendons.
Conclusion: Ultrasonography should be used in the future to assure precise diagnosis and to guide the peritendinous injection of steroid in chronic Achilles and patella tendinitis. Ultrasonography and pressure algometry are recommended as objective methods for monitoring the effect of treatment. Ultrasonographically guided injection of long-acting steroid can normalize the ultrasonographic pathological lesions in the Achilles and patellar tendons, and has a dramatic clinical effect but when combined with aggressive rehabilitation with running after a few days, many will have relapse of symptoms within 6 months.

Chronic tendinitis of the Achilles and patellar tendons is among the most frequent injuries in sport. Treatment includes active rest, eccentric training of the calf muscle (1–3), non-steroidal anti-inflammatory drugs (NSAIDs) (4), local steroid injection (5–7), and surgery (8–11). The injuries will often, however, bring the athlete's sports activity to an end.

The typical symptom pattern of chronic tendinitis expressed by athletes is one that occurs in progressive stages, as outlined by Blazina (12) and modified by Lian et al (13): Stages 1, 2, 3A (permanent pain but able to train and play), 3B (permanent pain that causes impairment of the athletic performance), and Stage 4 (total rupture of tendon). However, more than half of the total ruptures occur without any warning symptoms, and degenerative changes have been found in nearly all of the ruptured tendons (14). The spontaneous rupture rate in patients with chronic achillodynia is unknown, due to a lack of systematic examination, but frequencies of 3.5–8% have been reported (15, 16). One study (17) reported that among athletes with chronic achillodynia and ultrasonographically confirmed swelling of the Achilles tendons, spontaneous rupture occurred in 28% after a follow-up of 48 months.

In sports injuries, the use of local injections of steroid has been associated with many dogmas, and treatment has been controversial. There is, however, no scientific data to support that peritendinous injection of steroid would harm the tendons if correct indication and correct technique are used (6, 7, 16).

Usually the injections are given blindly. However, earlier studies have shown that the effect of steroid injections depends on correct placement in the bursa

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(18) and joint (19), but there have been no studies of tendons without tendon sheath. Both the Achilles and patellar tendon are without tendon sheaths. They are the two biggest tendons on the lower legs, and are often injured by running and jumping, although two different tendons, they have many things in common.

In earlier publications local steroid injections were carried out solely on the clinical diagnosis of tendinitis. Many of the tendinitis cases were incorrectly diagnosed by only clinical examination, however, and were in some cases examples of total rupture (20, 21). In cases of partial or total rupture local steroid injection would naturally not be useful.

The present study is the first randomized, double-blind, placebo-controlled study of local steroid injection in athletes with chronic tendinitis, in which ultrasonography was used in the diagnosis and guidance of injection, and in which NRS-score (numeric rating scale) and soreness, as measured by pressure algometry, were conjunctively employed to objectify and monitor the results of treatment.

Materials and method

The study group comprised 48 athletes initially referred for surgery because of symptomatic unilateral tendinitis at Stage 3A or 3B [24 with jumper's knee (PAT-group) and 24 with Achilles tendinitis (ACH-group)] for >6 months. (For abbreviations used throughout, Table 1)

All had failed conservative treatment (non-medical and non-surgical) and had ultrasonographically determined thickening (>1 mm) of the symptomatic tendon in relation to the asymptomatic tendon. The athletes in the ACH-group were between 24–55 years old (mean 43.7 years) and in the PAT-group between 18–47 years (mean 28.4 years). The exclusion criteria were either partial or total rupture of the tendon, ongoing treatment with steroid, ongoing infection near the tendon, tuberculosis, earlier surgery on tendons, earlier steroid injection in the vicinity of the tendons, diabetes, and general inflammatory disease. The characteristics of the athletes included are given in Table 2.

This study was block randomized, double blind and placebo-controlled. Forty-eight athletes (24 ACH and 24 PAT) were included. Twenty-four were primarily treated with steroid injections (12 ACH-S and 12 PAT-S), and 24 were primarily treated with placebo injections (12 ACH-P and 12 PAT-P). All athletes received three injections, one each at Days 0, 7, and 21 (if they were without symptoms after the second injection, the third injection was not given). At Days 0, 7, 21, 28 and after 6 months all athletes underwent clinical examination, and each completed a questionnaire to register 'walking pain' (numeric rating scale, NRS). A questionnaire was also completed at 3 months. If any problems occurred, an extra control was performed. At all clinical controls each athlete was subjected to ultrasonographic examination, and pressure-pain detection thresholds of the damaged and undamaged tendons were measured by pressure algometry. Two years after the first steroid injection a telephone interview was conducted.

A high resolution Toshiba ECCOCEE ultrasonograph with a mechanical 7.5 MHz linear and phazed array with waterpad was used for the ultrasonographic examination. A Somedic Algometer was used for pressure algometry. Pressure diameter was 1.0 cm^2 and pressure rate was 40 kPa/sec. Pressure-pain detection thresholds, as measured by pressure algometry, increases when pain decreases.

Table 1. Abbreviations. The placebo (P) treated athletes (ACH-P and PAT-P) were all subsequently treated with steroid (S) (ACH-PS and PAT-PS). The athletes in the P-group and the PS-group were therefore the same.

Abbreviation	Explanation
ACH-S	Achilles tendons primarily treated with steroid
ACH-P	Achilles tendons primarily treated with placebo
ACH-PS	Achilles tendons primarily treated with placebo (ACH-P) and then steroid
PAT-S	Patellar ligaments primarily treated with steroid
PAT-P	Patellar ligaments primarily treated with placebo
PAT-PS	Patellar ligaments primarily treated with placebo (PAT-P) and then steroid

Table 2. Characteristics of athletes included in the Achilles tendinitis group (ACH) and jumper's knee group (PAT).

	ACH-group	PAT-group
Sex (M/F)	15/9	18/6
Age, years	43.7 (24–55)	28.4 (18–47)
Weight, kg	84.8 (61–116)	78.7 (60–95)
Height, cm	180 (162–197)	180 (162–193)
Hours used on sports every week (average)	6.4	5.4
Duration of symptoms, months	18.7 (6–60)	21.5 (6–78)
Stage [3A/3B, (12)] before treatment	7/17	13/11

Following an ultrasonographic examination to locate the lesions, the injections were performed as near to the damaged areas as possible, typically one injection longitudinal on both sides of the tendon. No injections were performed under the tendons. After each injection, 4 days of rest was recommended. After this the athletes could start graduated training, which included running, and any pain would reduce or stop the training. All were instructed in rehabilitation techniques, including training and stretching of calf muscles (ACH-groups) or thigh muscles (PAT-groups). At Day 21 before the third injection, the treatment regimen was discontinued if an athlete did not feel improvement. If the primary treatment had been placebo (ACH-P and PAT-P), the athlete was offered treatment with three steroid injections in an open study, later referred to as ACH-PS and PAT-PS. If treatment with steroid had insufficient effect, the athlete was referred to surgery. If the treatment regimen was not discontinued and the athlete still had symptoms, the third injection was given. The athletes were also allowed to discontinue the treatment whenever s/he liked after Day 21 if no significant improvement was felt.

At all examinations, ultrasonography and pressure algometry of the Achilles and patellar tendons were carried out with the ankle and knee flexed 90°. All measurements were performed by the same investigator. The thickest point of the tendon was identified by ultrasonography. At Day 0, the distance between the thickest point of the tendon and its insertion on calcaneus or patella was measured and recorded by ultrasonography with 0.1 mm precision, enabling the measurements in the following controls to be performed in exactly the same place (Figure 2 A, B, and C, and Figure 4 A and B). The thickest point of the tendons was marked on the skin with a pen, so that pressure algometry could be made at exactly that same point.

The steroid injection contained 3.5 mL of 10 mg/mL lidocain and 0.5 mL kenalog containing 20 mg triamchinolone in a 5 mL syringe. The injection was placed peritendinously about the thickest point on both sides of the tendon. The placebo injection contained 3.5 mL of 1% lidocain and 0.5 mL 20% intralipid in a 5 mL syringe. Intralipid was added in order to make the placebo look like the milky kenalog solution. It was not possible to tell the difference between placebo and active treatment by colour or viscosity. All the injections were administered by the same investigator under blind conditions.

In both the ACH and PAT-groups, the athletes were randomized in four blocks of six athletes each (three steroid and three placebo). One person was responsible for the randomization and preparation of the injected medicine, however, the same person had nothing to do with diagnostic procedures or monitoring of effects.

Statistics

Since the difference between the two measurements in tendon thickness before and after treatment seemed to depend on the magnitude of the changes, natural logarithmic transformation was used. Instead of looking at absolute differences, we used relative changes. Unpaired *t*-tests were used to compare changes in the treatment groups. The correlation between the change in diameter of an affected tendon, as measured by ultrasonography, and age of athletes was tested by Spearman's rank correlation test. The difference between the results of the questionnaire (NRS value) in the steroid and placebo groups was tested by Fisher's exact test, 2-tailed. Breslow – Day test for homogeneity of the odds ratio for improvement was $p = 0.778$, which allowed the uniting of the two groups (ACH and PAT) when the odds ratio was calculated.

Results

Even though the athletes were referred from orthopaedic departments, only one-third of them fulfilled the inclusion criteria. The other two-thirds with clinical tendinitis had ultrasonographically confirmed normal tendons, having only minor changes in the tendons, peritendinitis, bursitis, or partial ruptures. Two of the referred athletes had a neglected total rupture of an Achilles tendon.

The 48 athletes had, on average, experienced symptoms for >1.5 years (6 – 78 months), and 16 of the athletes (33%) had also had earlier problems with their affected tendon. The ACH and PAT groups were identical in all respects except age (Table 2). The PAT group was on average 15 years younger than the ACH group. In both placebo groups (ACH-P-group and the PAT-P-group), treatment regimen was discontinued because the athletes did not feel sufficient improvement in all cases except one. Two athletes whose treatment regimens were not discontinued were treated for 6 months with placebo, still had symptoms at the 6-month control, and both accepted subsequent steroid treatment. In this way, all 24 athletes who were primarily treated with placebo (ACH-P and PAT-P) were subsequently administered steroid treatment (ACH-PS and PAT-PS).

In the ACH-S-group three (25%) discontinued the treatment regimen before the 6-month control and were referred to surgery. In the PAT-S-group six (50%) discontinued the treatment regimen before the 6-month control and were referred to surgery.

At 2-year follow-up, six of the 24 athletes (25%) in the ACH-groups (ACH-PS and ACH-S) and 14 of

the 24 athletes (58%) in the PAT-groups (PAT-PS and PAT-S) were operated on.

Ultrasonography

The athletes in the ACH-group had Achilles tendons that were spindle shaped, with the thickest point being 2–4 cm from the insertion on the calcaneus (Figure 4 A) except one who had a granuloma at the insertion on calcaneus. All athletes in the PAT-group had a granuloma (hypoechoic focal area) in the patellar tendon at its insertion on the patellae (Figure 2 A and 3 A). Five of the athletes with jumper's knee also had diffuse thickening of the whole patellar tendon.

Tendon diameter (and the granulomas) as measured by ultrasonography was significantly reduced after treatment in both the ACH-S, and PAT-S groups (Figure 1). As early as 1 week after the first steroid injection, the average diameter of the affected tendon was significantly reduced in the ACH-S group from 9.8 mm to 9.0 mm ($p = 0.002$), and in the PAT-S group from 7.8 mm to 6.9 mm ($p < 0.0001$) (Figures 2 and 3). After 3 weeks, average diameter was reduced further to 8.0 mm ($p = 0.0003$) in the ACH-S group and to 6.1 mm ($p < 0.0001$) in the PAT-S group. After 6 months, tendon diameter in the ACH-S group was increased to 8.6 mm and in the PAT-S group to 6.4 mm, which was still significantly less than the first measurement ($p = 0.002$ for both groups) (Figure 4 A and B, Figure 2 A and C, Figure 3 A and B).

There was no change of diameter at any time in the placebo-treated groups (ACH-P and PAT-P). The reduction of diameter seen in the groups first treated with placebo and then steroid (ACH-PS and PAT-PS) was the same as that in the groups primarily treated with steroid (ACH-S and PAT-S), Figure 1. There was no correlation between the change in diameter of the affected tendons as measured by ultrasonography and age of athletes in either the ACH-S or PAT-S groups.

In all tendons there was reduction in pathological change (tendon thickening and inflammation/oedema of the tendon), but only in seven of the asymptomatic athletes, who believed they were well, were the tendons ultrasonographically determined completely normalized, compared to the contralateral side.

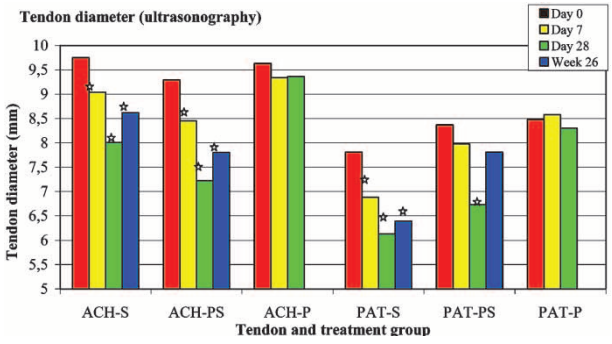
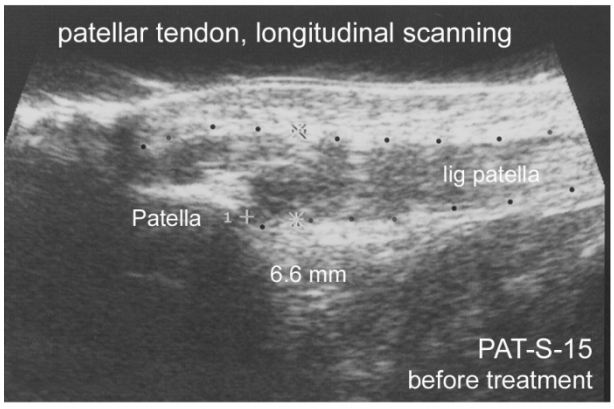


Figure 1. Tendon diameter as measured by ultrasonography before and after injection (☆ = measurement significantly different from measurement at Day 0, $p < 0.05$). The athletes were treated with injection at Days 0, 7 and 21. The values of ACH-P and PAT-P at Day 28 are not exactly the same as ACH-PS and PAT-PS at Day 0 because the treatment regimen was discontinued for some athletes later than Day 28. There were 12 athletes in all six treatment groups.

A



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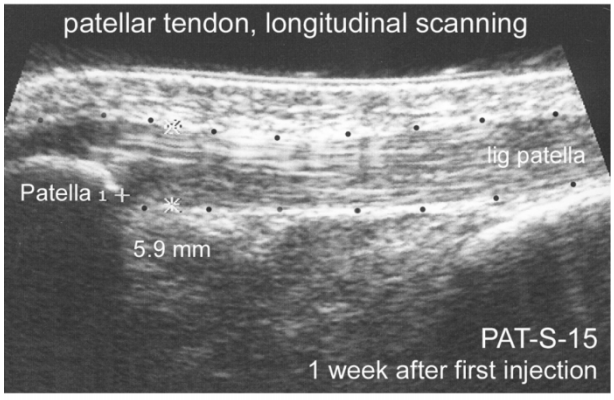

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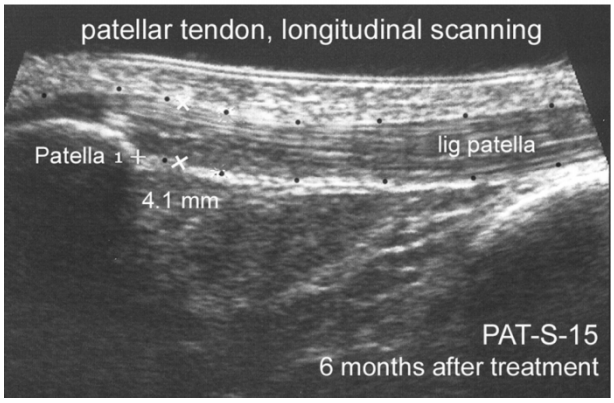

Figure 2. (A, B and C). The left patellar tendon, as viewed under longitudinal scanning in an athlete (athlete no PAT-S-15) with jumper's knee. The AP-diameter of the tendon was measured 3.6 mm from the insertion on patellae at Day 0 (6.6 mm), Day 7 (5.9 mm) and after 6 months (4.1 mm).

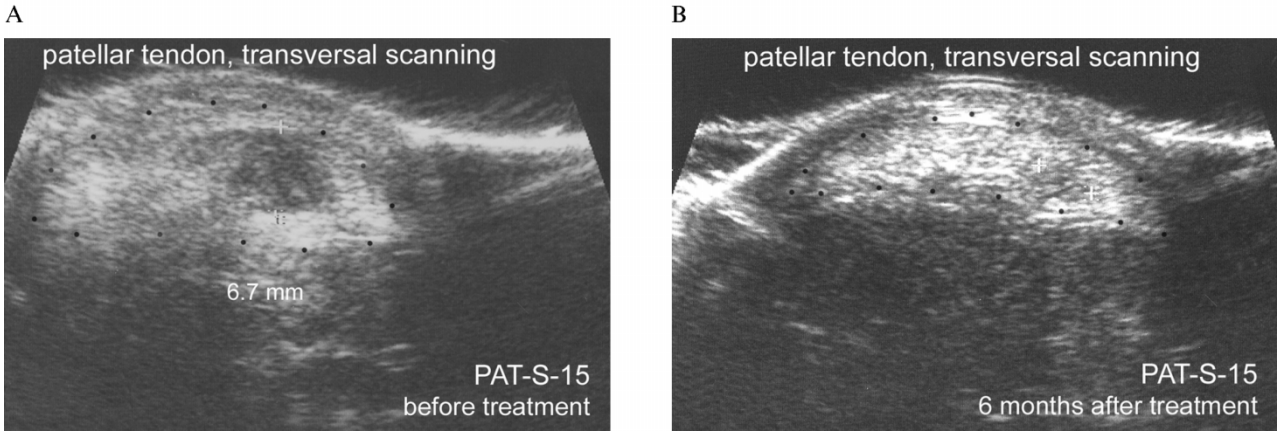

Figure 3. (A and B). The left patellar tendon as viewed under transversal scanning in an athlete (athlete no PAT-S-15) with jumper's knee. At Day 0 the granuloma in the patellar tendon measured 6.7 mm, 6 months later the granuloma had nearly disappeared.

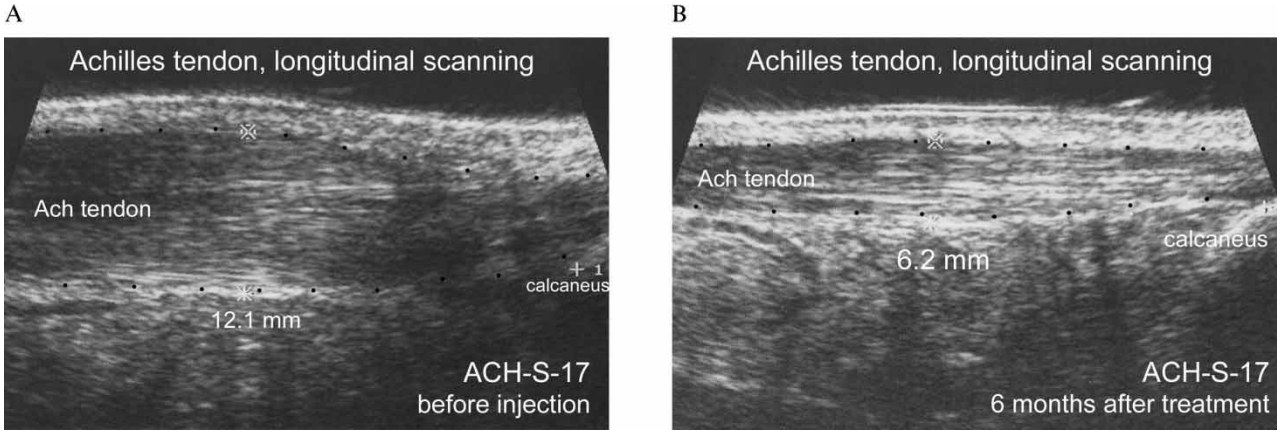

Figure 4. (A and B). The right Achilles tendon (athlete no ACH-S-17) as viewed under longitudinal scanning. The AP-diameter of the tendon was measured 24.9 mm from the insertion on calcaneus at Day 0 (12.1 mm) and after 6 months (6.2 mm), when the inflammation/oedema had nearly disappeared.

Pressure algometry

There was a significant difference in pressure-pain detection threshold between the affected tendons and the controls. One week after first injection, the pressure-pain detection threshold—as measured by pressure algometry—was higher both in the ACH-S-group, having increased from 339 kPa to 436 kPa ($p = 0.01$), and in the PAT-S-group, having increased from 430 kPa to 535 kPa ($p = 0.07$, non-significant) (Figure 5). After 3 weeks, the pressure-pain detection threshold had further increased in the ACH-S-group to 599 kPa ($p = 0.0006$), and in the PAT-S-group to 639 kPa ($p = 0.003$). After 6 months, the pressure-pain detection threshold in the ACH-S group was 487 kPa ($p = 0.02$), and 542 kPa (NS, $p = 0.07$, non-significant) in the PAT-S-group.

There was no change in the pressure-pain detection threshold of the placebo-treated groups (ACH-P and PAT-P). The results of local steroid treatment in the groups primarily treated with placebo (ACH-PS and PAT-PS) were nearly the same as in the groups primarily treated with steroid (ACH-S and PAT-S) (Figure 5).

Numeric rating scale, NRS

As early as 1 week after the first injection, walking pain measured on a numeric rating scale (NRS) of 0–10 was reduced from 3.6 to 2.1 in the ACH-S group, and from 2.9 to 1.7 in the PAT-S group (see Figure 6). After 4 weeks, the pain was further reduced to 0.7 in the ACH-S group and to 1.3 in the PAT-S group. After 6 months, walking pain in the ACH-S group was 1.9 and in the PAT-S group, 2.4. Of the 24 athletes primarily treated with steroid (ACH-S and PAT-S) 15 patients had reduced walking pain after the first injection. Of the 24 placebo-treated athletes (ACH-P and PAT-P) only six had reduced walking pain. The difference between the steroid and the placebo groups is significant ($p = 0.02$), and the estimated odds ratio for improvement was 5.00 (95% confidence interval 1.46–17.10).

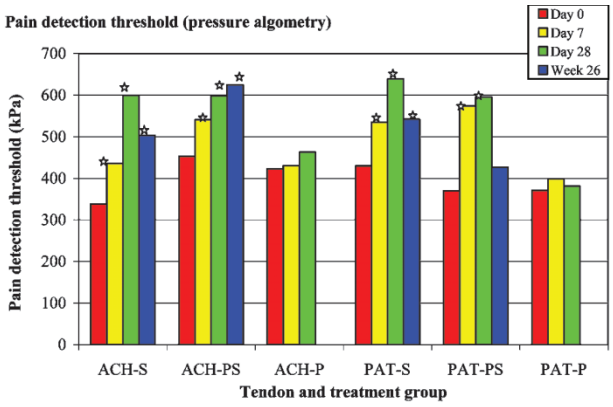

Figure 5. Pressure pain detection threshold before and after injection. (☆ = measurement significantly different from measurement at Day 0, $p < 0.05$). The pressure-pain test was performed with Somedic equipment. Higher pressure (score) means less soreness. The values of ACH-P and PAT-P at Day 28 are not exactly the same as ACH-PS and PAT-PS at Day 0, because the treatment regimen was discontinued for some athletes later than Day 28.

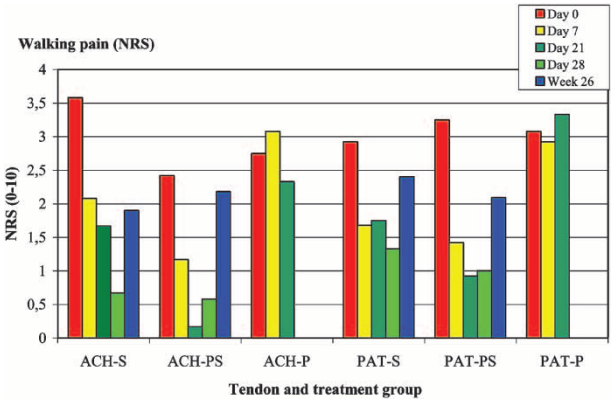

Figure 6. Walking pain before and after injection. The values of ACH-P and PAT-P at Day 28 are not exactly the same as ACH-PS and PAT-PS at Day 0, because the treatment regimen was discontinued for some athletes later than Day 28. The difference between the steroid (S) and the placebo (P) groups is significant ($p = 0.02$)

All of the 24 athletes in the ACH-S and ACH-PS groups were at Stage 3B or 3A before treatment with steroid (Table 3). Three months after the first steroid injection, 10 (42%) of the athletes were at Stage 0 and nine (38%) at Stage I. Of the 23 non-operated athletes who completed the questionnaire 6 months after steroid treatment, only three (13%) were at Stage 0 and six (26%) were at Stage 1.

All of the 24 athletes in the PAT-S and PAT-PS groups were at Stage 3B or 3A before treatment with steroid (Table 3). Three months after the first steroid injection, 14 (58%) of the athletes were at Stage 0 and three (13%) were at Stage I. Of the 23 non-operated athletes who completed the questionnaire 6 months after steroid treatment, only seven (30%) were at Stage 0 and four (17%) were at Stage 1.

Complications

There were no side effects in the placebo groups. Reversible atrophy was a frequent side effect of the peritendinous injection. It was seen in 11 of the 24 steroid-treated in the ACH-groups, and in nine of the 24 steroid-treated in the PAT-groups. In nearly half of the athletes the changes disappeared before the 6-month control. In none of the athletes did the atrophy give any problems. One of the athletes in the ACH-S-group with symptoms deriving from the left Achilles tendon insertion at the calcaneus in the last 5 years was injected twice. Ultrasonography showed a granuloma at the insertion, but the tendon was normal 3 cm from the distal insertion. She discontinued the treatment regimen after two injections of steroid and was referred for surgery. During the following 2 months of waiting for surgery, she was symptom-free for the first time in 5 years. Without preliminary training, she participated in a handball match, where she developed a total rupture of the Achilles tendon 3 cm from the distal insertion. The tendon was operated on and 6 months after surgery, she could walk without problems, but had yet to start sport activities again.

Discussion

In our study only one-third of the athletes referred with clinically suspected tendinitis had the diagnosis

Table 3. Stages [Blazina (12), as modified by Lian et al (13)], of Achilles tendinitis (ACH) and patellar tendinitis (PAT) before and after treatment.

	ACH			PAT		
Stage	Before treatment	3 months after treatment	6 months after treatment	Before treatment	3 months after treatment	6 months after treatment
Stage 4	0	1	1 *	0	0	0
Stage 3B	17	1	7	11	2	6
Stage 3A	7	1	3	13	2	3
Stage 2	0	2	3	0	3	3
Stage 1	0	9	6	0	3	4
Stage 0	0	10	3	0	14	7

*Operated and postoperative in Stage 3B.

confirmed by ultrasonographic assessment of tendon thickness. In two-thirds of athletes with clinical Achilles tendinitis and jumper's knee the clinical diagnosis was not confirmed by ultrasonography. Clinical Achilles and patellar tendinitis are obviously heterogeneous groups. This observation can explain the variation in both the conservative and surgical treatment of tendinitis. If ultrasonography were obligatory in the diagnosis of patella and Achilles tendinitis in future studies the heterogeneity of the included patients will be reduced.

Ultrasonographic guided peritendinous injection of steroid is very effective in reducing the pain and thickness of Achilles and patella tendons in athletes with chronic tendinitis. As early as 1 week after the first injection, ultrasonographic examination revealed that there was significant reduction in the thickness of tendons, as well as a significant reduction in pain. The pain-relieving effect is in good agreement with previous publications on the subject (16, 22). With ultrasonography, the steroid can be injected as close to the tendon as possible without risk of being intratendinous. One should be aware that it is difficult to sense an intratendinous injection in a thick and inflamed tendon.

There are morphological changes in the tendons after steroid injections, which can be detected by ultrasonography and pressure algometry, and the measurements can be used in monitoring the effect of treatment for chronic Achilles and patella tendinitis. This technique is recommended as an objective monitoring device of the effect of treatment in future studies.

An explanation for the high frequency of relapse of symptoms could be an aggressive training programme, which includes running a few days after injection. Steroid can ultrasonographically reduce inflammation and oedema of a tendon (23), but cannot repair degenerative changes. Although there is only sparse histological documentation of inflammatory cells in biopsies from the damaged tendons (24–26), the significant reduction in oedema and thickness of the steroid-treated tendons is most likely due to an influence on inflammatory process. When intense training is started soon after an injection, degenerative changes of the tendon will cause a recurrence of the inflammation, which could explain the high relapse frequency in this study.

The present study shows that change in a tendon is potentially reversible, i.e. 'degenerated' tissue has the potential to regenerate to clinically and ultrasonographically normal tissue. This should be kept in mind before removing degenerative tissue by surgery. The term tendinosis (degeneration of the tendon without clinical and histological signs of inflammation in the tendon) and tendinitis (inflammation within the tendon) are still controversial, and it is possible that in most of the cases, change in a tendon is both degenerative and inflammatory.

In athletes followed with weekly ultrasonographic examinations of a tendon, the tendon diameter was reduced the first month after a steroid injection. This indicates that injection with a long-acting steroid should be given with at least a 4-week interval if more than one injection is required.

With correct, graduated rehabilitation (increased training with respect to the pain) over several months, there seems to be only minor risk associated with ultrasonographically guided peritendinous steroid injection. The greatest 'risk' of steroid injection is that an athlete with chronic pain, after an injury period of several months, will be pain free after a few weeks. If an athlete, without a period of several months of increased training to strengthen the tendon, suddenly participates in sport without moderation, s/he can experience a rupture in the tendon due to the sudden extreme load. We now recommend 3–6 months of rehabilitation after local steroid injections for chronic Achilles and patellar tendinitis. Further, we recommend that ultrasonographically guided steroid injection be attempted for patients with chronic tendinitis before surgery. The treatment seems to reduce the demand for surgery for $>50\%$ of athletes.

The results of the treatment of Achilles and patellar tendons in this study are identical. The results are most likely the same in other tendons (without tendon sheaths) but further studies are needed to throw light on this hypothesis.

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