Clinical Outcomes and Tendon Structure at 3- to 4-Year Follow-up After Exercise-Based Treatment of Patellar Tendinopathy

A Prospective Study

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Background: The long-term recovery (in years) of patellar tendinopathy treated with loading-based rehabilitation remains largely unknown.

Purpose: To examine the clinical outcome and tendon structure years after exercise-based treatment of chronic patellar tendinopathy.

Study Design: Case series; Level of evidence, 4.

Methods: This was a 3- to 4-year follow-up evaluation of participants (N = 28) from a previous randomized clinical trial by the author group. All participants received loading-based rehabilitation for 12 weeks with either moderate-slow resistance (55% of 1 repetition maximum) or heavy-slow resistance (up to 90% of 1 repetition maximum). Both groups showed similar improvements after 3 and 12 months and were therefore collapsed in the present analysis. Function and symptoms (the Victorian Institute of Sport Assessment–Patella [VISA-P] questionnaire), tendon pain (numeric rating scale [NRS] during activity and during a single-leg decline squat test), and tendon structure (tendon vascularization and thickness on ultrasound) were assessed.

Results: The mean follow-up was 3.6 ± 0.4 years after the baseline assessment in the original clinical trial. The VISA-P score was 83.9 ± 11.9 (95% CI, 79.3-88.5) at the latest follow-up and did not differ from the 1-year follow-up score ($P = .54$). Similarly, NRS score during preferred sport (1.6 ± 1.7; 95% CI, 0.9-2.2) and single-leg decline squat (1.0 ± 1.8; 95% CI, 0.3-1.7) did not differ from the 1-year values and remained elevated. Power Doppler area and tendon thickness decreased significantly from 1 year to latest follow-up ($P < .0001$ and $P = .02$, respectively), but power Doppler area >1 mm^2 was still present in 43% of the participants after 3 to 4 years, and the tendon thickness was still mildly elevated (6.4 ± 1.8 mm; 95% CI, 5.7-7.1 mm). Sports participation after 3 to 4 years (3.9 ± 2.7 h/wk; 95% CI, 2.9-7.1 h/wk) was significantly lower compared with preinjury levels ($P < .0001$).

Conclusion: Clinical symptoms remained even years after loading-based treatment for patellar tendinopathy, whereas some but not all tendon structures normalized in this longer term follow-up.

Keywords: patellar tendon; tendinopathy; long-term follow-up

Patellar tendinopathy is a common sports-related injury in both elite and recreational athletes[11,39] and is characterized by activity-related pain, localized tenderness upon palpation, impaired physical performance, and structural changes, such as swelling and hypervascularization.[20] In most cases, the injury is associated with overuse,[30] but the exact cause is likely multifactorial.[27] Although it is well recognized that patellar tendinopathy often causes long-term (months to years) reductions in

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performance,[19] the majority of studies that investigate the effect of a treatment have a follow-up of only 12 to 26 weeks.[9]

Loading-based interventions have become the prevailing conservative treatment strategy for patellar tendinopathy.[22,24] Loading-based interventions have resulted in short-term clinical improvements with varying effectiveness,[3,8,22,24,31] but only a few clinical studies[3,6,36] have included a 1-year follow-up, and these show that the patients did not fully recover. This is not surprising given that tendon tissue has a slow turnover in general,[16] although it can adapt to loading-based treatment.[7,23,35] To our knowledge, only 1 study[19] has evaluated the long-term prognosis for symptoms related to patellar tendinopathy, and it showed more subjective symptoms and functional limitation than healthy controls at a 15-year follow-up. Collectively, this underscores the importance of longer term follow-up evaluations in this patient population.

To what extent structural changes in the tendon relate to the clinical picture has been debated.[12] Although clinical imaging modalities such as ultrasound can be used to document tendon neovascularization and thickness, only low-to-moderate correlations between clinical outcome (pain and function) and ultrasound findings have been reported.[12,13,29] Moreover, it remains unknown whether structural data can provide additional useful information when evaluating the long-term recovery after patellar tendinopathy since it has been reported only once as an outcome with a short-term follow-up[29] and once with a 1-year follow-up after exercise-based treatment.[3]

The aim of the present study was to evaluate the 3- to 4-year (considered long term for this patient population) clinical and structural outcome for patients with patellar tendinopathy previously treated with loading-based exercise.

METHODS

This was a prospective follow-up of participants originally included in a randomized clinical trial published in 2021[1,3] that investigated to what extent load magnitude (high and moderate) influences the clinical outcome, tendon structure, and mechanical function in patients with patellar tendinopathy. All participants received oral and written information and provided written informed consent to participate. Ethical approval was obtained by our regional ethics committee, and the original study was registered at ClinicalTrials.gov (NCT03096067).

Participants and Treatment in the Original Study

The original study[1,3] included male athletes aged 20 to 45 years with patellar tendon pain for >3 months who were clinically diagnosed with unilateral or bilateral patellar tendinopathy by a sports medicine physical therapist or physician, based on defined clinical findings and confirmed by ultrasonography. The exclusion criteria were corticosteroid injection for patellar tendinopathy, previous knee surgery, arthritis, diabetes, any confounding diagnoses to the knee joint, smoking, and patellar tendinopathy for >12 months. The participants were recreational athletes who participated in sport 8 ± 4 h/wk.

In the original study, participants were treated over a 12-week rehabilitation training period with heavy-slow resistance (HSR) or moderate-slow resistance (MSR) with the total training volume matched between groups. Outcomes in the original study were function and symptoms by the Victorian Institute of Sport Assessment–Patellar questionnaire (VISA-P), pain during activity on an 11-point numeric rating scale (NRS), tendon vascularization (Doppler area), and thickness by ultrasound. All outcomes were obtained at baseline (0 weeks), midintervention (6 weeks), postintervention (12 weeks), and 1-year follow-up (52 weeks). The exercise program details and study results have been published previously.[1,3]

Participants in the Present Study

Enrollment in the present study occurred between May 2021 and October 2021. Of the 44 participants included in the original study, 40 had agreed to be contacted for longer term follow-up evaluations. Three of the invited participants did not respond. Further, 9 participants declined to participate because of meniscal surgery (n = 1), lack of time and feasibility (n = 5), or having moved to another part of the country or long travel distance (n = 3). Among the 28 included participants, the group distribution from the original study was almost equal (HSR, n = 15; MSR, n = 13). All of the present follow-up analyses are collapsed values across the HSR and MSR groups because there were no significant differences between the groups in the original study for any of the measured outcomes.[1,3]

Patient Evaluation

The patient evaluation for the current study consisted of a single test day and included completion of patient-reported outcome measures, functional evaluation, and ultrasonography. These were identical with the 1-year follow-up

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evaluation in the original study.[3] The investigators conducting the clinical and functional follow-up tests (A.A.) and ultrasound evaluation (N.M.M.) were the same as in the original study. The examination order was identical for all follow-up evaluation, starting with ultrasound examination followed by a 5-minute warm-up on a bicycle ergometer before functional testing and further examination.

Patient-Reported Outcome Measures. Self-reported perceptions of symptoms, function, and ability to participate in sports were evaluated with the VISA-P questionnaire. The VISA-P consists of 8 questions, with a maximum score of 100 indicating that the person is asymptomatic with full sports performance and lower scores indicating more symptoms and limitations of function and activity.[34] Maximal tendon pain during running and during the patient's preferred sport was evaluated on an 11-point NRS, with 10 being the worst imaginable pain and 0 denoting no pain. Furthermore, the number of hours of sport participation during the preceding week was reported. All self-reported outcomes were evaluated using an electronic questionnaire with no investigator assistance. Participants were interviewed by the primary investigator about the presence of new comorbidities with relevance for their patellar tendinopathy, whether they had tried other treatment modalities, their fear of return of pain and symptoms, presence of morning stiffness, and the "warm-up phenomenon" with reduced pain after repeated loading but reappearing pain the day after plyometric activities.

Functional Evaluation. Pain during function was examined using a reliable patellar tendon pain provocation test, the single-leg decline squat (SLDS).[38] The test was performed as previously described.[3] In brief, participants performed a single-leg squat until reaching 50° of knee flexion on a 25° decline board and reported their experienced pain upon completion using the NRS. The patients performed 1 practice trial followed by 2 actual tests, with a 1-minute rest period between the tests. The NRS score was collected after each test, and the mean of the 2 scores was used for further analysis.

Ultrasound Evaluation. Ultrasonography (HI VISION Ascendus; Hitachi Medical Systems) was used for B-mode gray scale (GS) and power Doppler (PD) assessment of the patellar tendon as described in the original study, with GS and PD settings identical to the ones used for all examinations in the original study.[3] In brief, participants were instructed to avoid strenuous physical activity 24 hours before the examination, and the GS and PD settings were identical to those used for all examinations in the original study. The GS examination was performed using the linear 10.5 transducer (EUP-L53L; Hitachi Medical Systems), with the participants in a seated position at 90° of hip and knee flexion. Two images were obtained, with the transducer removed from the skin between the 2 recordings. The PD examination was performed using the linear 18.5 transducer (EUP-L75; Hitachi Medical Systems), with the participants lying supine with a fully extended and relaxed knee. Two 4-second sine loops were recorded.

All ultrasound recordings were imported into Fiji/ImageJ for quantitative analyses carried out by a single investigator (A.A.) The anterior-posterior patellar tendon thickness was measured exactly 0.5 cm distally from the patellar apex on the 2 GS recordings. In each of the 2 PD series, the frame containing the largest area of Doppler signal within the tendon was found and quantified by a custom macro. The maximum anterior-posterior thickness and largest Doppler area were used for further analysis.

TABLE 1
Characteristics of the Study Participants at Latest Follow-up (N = 28)[a]

Variable	Value
Age, y	33.8 ± 5.2 (27-45)
Height, cm	182.8 ± 7.5 (168-198)
Weight, kg	85.9 ± 10.2 (66-108)
Body mass index, kg/m^2	25.1 ± 2.7 (20-32)
Follow-up, y	3.6 ± 0.4 (3-4)

[a]Values are expressed as mean ± SD (range). Time to follow-up indicates years after baseline assessment in the original study.[1,3]

Statistical Analysis

Statistical analyses were carried out using GraphPad Prism (Version 9.2.0 for Mac; GraphPad Software). Descriptive statistics were used to present participant characteristics and follow-up values. Data were assessed for normality using *Q-Q* plots and Shapiro-Wilk normality tests, and results are reported as the mean ± SD unless otherwise noted. To assess changes in outcome parameters from 1-year follow-up to the latest follow-up, the paired Student *t* test was used for analysis of parametric data and the Wilcoxon signed-rank test for analysis of nonparametric data. The significance level for all tests was set to $P < .05$.

RESULTS

Participants

A total of 28 participants were included in the current study at a mean follow-up of 3.6 ± 0.4 years. At enrollment in the original study,[1,3] the included participants had a symptom duration of 6.7 ± 2.8 months and a 1-year follow-up VISA-P score of 84 ± 12, which was not significantly different from the participants who declined to participate in the current study (VISA-P score, 87 ± 10) (P = .46). Of the current participants, 54% reported that they had continued exercise-based treatment, and 36% reported receiving other treatments (knee-supporting braces, injections, acupuncture, restricted blood flow training, and osteopathy). The characteristics of the 28 participants at the latest follow-up are presented in Table 1.

Clinical Evaluation

The VISA-P did not reach the maximum score of 100 at the latest follow-up (mean ± SD, 83.9 ± 11.9; 95% CI, 79.3-

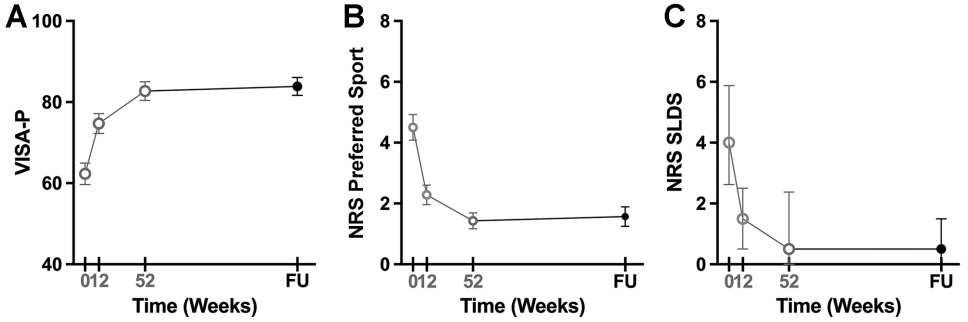

Figure 1. Changes in (A) Victorian Institute of Sports Assessment–Patella (VISA-P) scores, (B) numeric rating scale (NRS) pain scores during preferred sport, and (C) NRS for the single-leg decline squat (SLDS) test. Values are presented as mean ± SE in panels A and B and as median ± quartiles in panel C. Gray color indicates data from the original randomized clinical study from baseline (0 weeks), after the intervention (12 weeks), and at the 1-year follow-up (FU) (52 weeks).[1,3] Black color indicates data from the latest follow-up. At all time points, data are from the participants of the current study (N = 28). Paired statistics were calculated on differences from 52 weeks to latest follow-up. VISA-P (P = .54) and NRS preferred sport (P = .60) were analyzed by parametric test, and NRS SLDS was analyzed by nonparametric test (P = .25).

88.5) and did not change significantly from the 1-year follow-up to the latest follow-up (P = .54) (Figure 1A). Overall, 29% of the participants reported that they still experienced morning stiffness, 46% stated they were concerned that pain and symptoms would return, and 21% reported that pain and symptoms had not changed since the 1-year follow-up. The NRS pain score during the SLDS test (median, 0.5; interquartile range, 0.0-1.5; 95% CI, 0.3-1.7) did not reach 0, which corresponds to an asymptomatic tendon. Other NRS pain scores (mean ± SD) included scores for running (1.3 ± 1.6; 95% CI, 0.7-1.3), squat (0.6 ± 1.0; 95% CI, 0.2-1.0), and preferred sport (1.6 ± 1.7; 95% CI, 0.9-2.2). The NRS pain score did not change from the 1-year follow-up to the latest follow-up for the SLDS test (P = .25), running (P = .41), squat (P = .28), or preferred sport (P = .60) (Figure 1, B and C).

Sports participation (mean ± SE) was 3.9 ± 0.5 h/wk (95% CI, 2.9-5.0 h/wk) and did not change significantly from 1 year to the latest follow-up (mean difference ± SE, –1.0 ± 0.8 h/wk; 95% CI, –2.6 to 0.6 h/wk; P = .22). However, sports participation at the latest follow-up remained significantly lower compared with the preinjury level (mean difference ± SE, –3.1 ± 0.7 h/wk; 95% CI, –4.4 to –1.7 h/wk; P < .0001). Further, 57% of patients reported that they returned to their preinjury sport, but only 25% reported participating at the same level as preinjury. For 50% of patients, their tendon injury hampered participation in sport.

Ultrasound Evaluation

The PD area decreased significantly from the 1-year follow-up to the latest follow-up (P < .0001) (Figure 2), but PD was still present (>1 mm^2) in 43% of the participants at the latest follow-up. Tendon thickness also decreased significantly from 1 year to latest follow-up (P = .02) (Figure 2). Importantly, the mean decrease was relatively small (–0.7 ± 0.3 mm), and the tendon remained

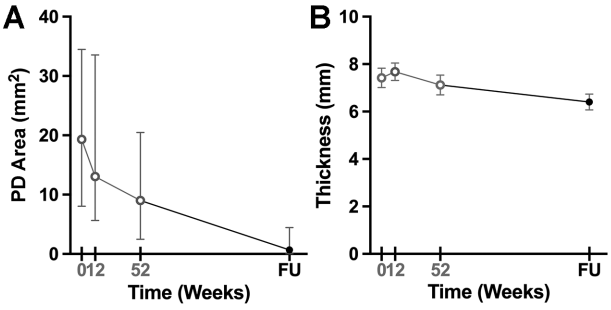

Figure 2. Changes in (A) power Doppler (PD) area and (B) tendon thickness. Values are presented as mean ± SE in panel A and as median ± quartiles in panel B. Gray color indicates data from the original clinical study from baseline (0 weeks), after the intervention (12 weeks), and the 1-year follow-up (FU) (52 weeks).[1,3] Black color indicates data from the latest follow-up. At all time points, data are from the participants of the current study (N = 28). Paired statistics were calculated on differences from 52 weeks to follow-up. PD area was analyzed by nonparametric test (P < .0001), and thickness was analyzed by parametric test (P = .02).

thickened (>6 mm) for the majority (57%) of the participants (Figure 2).

DISCUSSION

In the current study, we evaluated the clinical and structural outcomes for 28 participants at 3 to 4 years after they had undergone a 12-week loading-based treatment of patellar tendinopathy. The results showed that mild clinical symptoms of chronic patellar tendinopathy were long lasting (years) despite rigorous, supervised, loading-based treatment and that participants did not reach their preinjury activity level. Structural changes such as PD

area and tendon thickness were significantly reduced from the 1-year follow-up to the latest follow-up ($P < .0001$ and $P = .02$, respectively) but reached normal values for only 57% of the participants.

Studies have reported incomplete recovery after a chronic patellar tendinopathy even 1 year after a loading-based treatment.[3,6,36] In the original intervention study, participants had a good treatment response, and 1-year follow-up VISA-P values were comparable to those previously reported in patients with chronic patellar tendinopathy treated with exercise.[6,36] However, the present data showed that the VISA-P score did not further improve after the 1-year follow-up. That the participants did not reach a VISA-P score of 100 (indicating that the person is asymptomatic and fully performing) may be related in part to the inadequate measurement properties of the questionnaire.[2] However, the proportions of participants who did not attain zero pain for the different functions were 54% for the SLDS, 64% for running, 40% for squatting, and 68% for preferred sport, which clearly indicate a sustained symptom burden. Moreover, weekly hours of sports participation at the 3- to 4-year follow-up were significantly lower than the preinjury level, and even though 57% of the participants reported that they returned to their preinjury sports at the 3- to 4-year follow-up, only 25% of them reported participating at the same level as before injury. This further corroborates the lack of full clinical recovery even years after loading-based treatment. Finally, the results are in line with a recent study[19] showing that clinical symptoms are long-lasting (15 years). Another possible component of the incomplete clinical recovery could be fear avoidance (ie, psychological attributes that can be associated with outcomes from tendinopathy), a topic that has received little attention in the literature.[25] In the present study, 46% of the participants stated that they feared a return of pain and symptoms, which indirectly supports that lack of full recovery in addition to persistent pain could be related to fear of exacerbating the pain.

Structural changes such as tendon thickness and increased neovascularization are well-known characteristics of a tendinopathic tendon.[5] Nevertheless, only a few studies[3,28] have investigated the structural response to loading-based treatment at 1 year, and these showed a minimal effect from postintervention at 12-week to 1-year follow-up in PD area and tendon thickness. These findings contrast with the present data, which showed that thickness and PD area decreased significantly after 1 year. It could be argued that the decrease may be the result of a reduction in activity level. Interestingly, the present data showed that hours of weekly sport participation were unchanged from 1-year to 3- to 4-year follow-up and therefore cannot explain the decrease in tendon tissue alteration. However, the protracted recovery might be due to the almost complete absence of tissue turnover that has been shown in the core of adults' healthy Achilles[16] and patellar[37] tendons. In fact, it has been suggested that the turnover increases in response to tendinopathy,[17,28] and it might be reasonable to assume that some recovery of tendon structure is ongoing during the recovery phase, albeit a protracted process.

To our knowledge, this is the first study to investigate both structural and clinical outcomes years after loading-based treatment of patellar tendinopathy. Kongsgaard et al[21] showed a decrease in both Doppler activity and tendon thickness and improved pain and function in response to 12 weeks of HSR training. However, more recent studies[3,28] have reported an absence of normalization in Doppler activity and tendon thickness at 12 weeks and 1 year, despite improved clinical outcomes. The divergence between studies regarding the short-term and 1-year results might be due to variations in ultrasound protocol and equipment and the technological improvement in imaging quality and sensitivity in the past decade.[32]

PD activity was still present in 43% of the participants at the 3- to 4-year follow-up in the present study. This could suggest that ingrown vessels, a well-known structural change in tendinopathy,[26] do not disappear within years but rather decrease in size, making them less visible with currently available ultrasound protocols. Indeed, nerve ingrowth and the increased neovascularization related to tendinopathy have been suggested to cause the pain in tendinopathy.[4,26] However, other studies have shown that PD activity can also be present on ultrasound examination of pain-free tendons,[10,14] which does not support this assumption. It is noteworthy that the results from the present study demonstrated that tendon thickness at 3- to 4-year follow-up was approximately 6 mm, which is above the previously reported values of approximately 4 mm for healthy tendons.[15,18,33] It is well established that the tendinopathic tendon contains increased content of proteoglycans and glycosaminoglycans, which increases water content and may contribute to the measured thickening of the tendon with ultrasound.[23] Thus, it could be speculated that this increased pressure rather than the nerve ingrowth might be the cause of the persistent pain. The fact that tendon thickness decreased by only a small amount (<1 mm) from the 1-year follow-up to the 3- to 4-year follow-up, in contrast to the relatively large decrease in PD area, further supports this notion.

Limitations

This study has inherent limitations. We did not systematically record what physical activity the participants had engaged in or details on other forms of treatment. The study inclusion was based on voluntary participation; therefore, it cannot be ruled out that the patients represent a group with persistent symptoms. However, the 1-year VISA-P score for the included participants was not significantly different from those who declined to participate.

CONCLUSION

The results of this study showed that despite a 12-week loading-based treatment for chronic patellar tendinopathy, a deficit remained in clinical symptoms years after the injury without any additional improvement beyond the first year. Moreover, tendon thickness and PD area decreased from the 1-year follow-up to the 3- to 4-year

follow-up. However, Doppler activity was still present in 43% of the participants, some tendon thickening remained, and participants did not reach their preinjury activity level. Collectively, the clinical and structural picture and activity level were not fully restored even years after loading-based treatment for patellar tendinopathy.

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