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Original research

Are pain coping strategies and neuropathic pain associated with a worse outcome after conservative treatment for Achilles tendinopathy? A prospective cohort study

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ABSTRACT

Objectives: To analyse whether (1) passive or active pain coping strategies and (2) presence of neuropathic pain component influences the change of Achilles tendinopathy (AT) symptoms over a course of 24 weeks in conservatively-treated patients.
Design: Prospective cohort study.
Methods: Patients with clinically-diagnosed chronic midportion AT were conservatively treated. At baseline, the Pain Coping Inventory (PCI) was used to determine scores of coping, which consisted of two domains, active and passive (score ranging from 0 to 1; the higher, the more active or passive). Presence of neuropathic pain (PainDETECT questionnaire, −1 to 38 points) was categorized as (a) unlikely (≤12 points), (b) unclear (13–18 points) and (c) likely (≥19 points). The symptom severity was determined with the validated Victorian Institute of Sports Assessment-Achilles (VISA-A) questionnaire (0–100) at baseline, 6, 12 and 24 weeks. We analysed the correlation between (1) PCI and (2) PainDETECT baseline scores with change in VISA-A score using an adjusted Generalized Estimating Equations model.
Results: Of 80 included patients, 76 (95%) completed the 24-weeks follow-up. The mean VISA-A score (standard deviation) increased from 43 (16) points at baseline to 63 (23) points at 24 weeks. Patients had a mean (standard deviation) active coping score of 0.53 (0.13) and a passive score of 0.43 (0.10). Twelve patients (15%) had a likely neuropathic pain component. Active and passive coping mechanisms and presence of neuropathic pain did not influence the change in AT symptoms ($p = 0.459$, $p = 0.478$ and $p = 0.420$, respectively).
Conclusions: Contrary to widespread belief, coping strategy and presence of neuropathic pain are not associated with a worse clinical outcome in this homogeneous group of patients with clinically diagnosed AT.

Practical implications

- An active or passive coping strategy does not lead to a different course of AT symptoms.
- Patients with a likely neuropathic pain component have more severe AT symptoms, both at baseline and follow-up.
- Patients with a likely, unclear or unlikely neuropathic pain component have a comparable improvement in their AT symptoms over time.

1. Introduction

query>Pain is a pesky and often persistent symptom of Achilles tendinopathy (AT), with swelling and impaired performance completing the triad of AT symptoms.[1] Despite best available management consisting of exercise therapy, 23–60% of patients remain symptomatic 5–10 years after diagnosis and treatment of AT.[2–4] Coping strategies and a neuropathic pain component might influence the course of AT symptoms. Therefore, coping strategies and assessing the type of pain might be relevant features in treating patients with longstanding AT.

Pain coping is defined as cognitive and behavioural attempts to manage or tolerate pain and its effects.[5] It can be classified into

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an active and a passive coping strategy. Active coping consists of the patient's attempt to control the pain or to function in spite of the pain, for example 'I distract myself by undertaking a physical activity'. Active coping could influence AT symptoms as patients could overload their tendon due to distraction from the pain; however, this has not been researched yet. Passive coping consists of helplessness and strategies that externalize pain control to other resources, for example 'I do not exert myself physically'.[6] Systematic reviews of musculoskeletal conditions associated with chronic pain, such as osteoarthritis, fibromyalgia and rheumatoid arthritis, demonstrate that a passive coping mechanism may be associated with increased pain and disability.[7,8] As AT is also associated with chronic pain, its course could be influenced by the type of coping strategy.

Neuropathic pain might also play a role in the chronicity of AT symptoms.[9–12] Neuropathic pain can consist of both peripheral and central sensitization. Peripheral sensitization is an increased responsiveness and reduced threshold to afferent nerve stimuli.[9,13] After an injury or cell damage to the area, an unrealistic flare response is created after release of many neuropeptides by nociceptors.[14,15] Central sensitization is similar, with an increased responsiveness in the central nervous system, which is associated with a low pain threshold.[13] Sensitivity of pain transmission neurons is increased for various peripheral stimuli, including mechanical pressure.[12,16] There appears to be an association between neuropathic pain and chronic tendinopathies,[10,12] however, this has not been researched in AT.

Evaluating and recognizing specific pain coping strategies and specific subtypes of pain may have important clinical implications for treatment of patients with AT. Identification of patient subgroups with altered pain coping strategy or neuropathic pain components that do not respond to regular treatments would impact on clinical decision-making. However, until now it is unknown whether these subgroups are present in an AT population and whether these subgroups have altered outcomes after conservative treatment.

We conducted this study with the primary aim to analyse whether the level of active coping strategy influences change of AT symptoms over a course of 24 weeks. We also analysed the level of passive coping strategy and its influence on AT symptoms over a course of 24 weeks. We hypothesized that a more active coping strategy would have a positive influence on the change of AT symptoms over the course of 24 weeks, as the treatment of AT stimulates the use of the active domains distraction and transformation. Our secondary aims were to analyse the influence of (1) the difference between an active and passive coping strategy on the course of AT symptoms; (2) the presence of a neuropathic pain component on the severity of AT symptoms at baseline, 6, 12 and 24 weeks; and (3) the influence of a neuropathic pain component on the course of AT symptoms over 24 weeks. We hypothesized that a larger discrepancy between an active and passive coping strategy had a positive influence on the course on AT symptoms. We also hypothesized that the presence of a neuropathic pain component increased the severity of AT symptoms and that it flattened the course of AT symptoms over 24 weeks.

2. Methods

This study is part of a randomized clinical trial, the High-volume image-guided injections in chronic midportion Achilles Tendinopathy (HAT).[17] The RCT was approved by the Medical Research Ethics Committee Southwest Holland, Leiden, The Netherlands (MEC-14-100). Extensive description of the materials and methods are registered at clinicaltrials.gov (identifier: NCT02996409).

The HAT study evaluated the effect of high-volume image guided injection compared to a placebo injection in addition to eccentric exercises in patients with chronic midportion AT.[17] Written informed consent was acquired from all patients before inclusion. Patients were recruited in a large district hospital (Haaglanden Medical Center, The Hague, The Netherlands) between December 2016 and January 2019. Patients were examined by a sports physician, and had to comply to the following inclusion criteria: (1) 18–70 years old, (2) painful swelling of the Achilles tendon, 2–7 cm proximal of the calcaneal insertion, (3) symptoms for more than 2 months, (4) non-responsive to a minimum of 6-weeks of exercise therapy and (5) neovascularisations on Power Doppler Ultrasonography. Main exclusion criteria were clinical suspicion of Achilles tendon rupture, clinical suspicion of insertional tendinopathy and inability to participate in an active exercise program.

For the randomized clinical trial, patients were randomized into either intervention group (high-volume injection) or placebo group (low-volume image-guided injection). An independent secretary of the trial performed the randomization, which consisted of using a computer-generated randomization list using blocks, varying from 4 to 10 patients. A sports medicine physician administered the intervention, which consisted of a 50cc (40cc 0.9% sodium chloride and 10cc 1% lidocaine solution) injection in the peritendinous area were most Doppler flow was seen. The placebo group was injected using the same procedure with a 2cc solution (1.6cc 0.9% sodium chloride and 0.4cc 1% lidocaine solution). Patients were blinded for the allocated treatment. After injection, both groups performed a progressive exercise training program for 6 to 24 weeks instructed by a blinded outcome assessor, depending on their personal goals and progress. As there were no between-group differences in patient-reported outcomes and patients were not able to predict whether they received the intervention or placebo treatment, we considered the included patients as a cohort. Length of follow-up was similar in both groups.

Patients filled in a baseline questionnaire, inquiring age, sex, duration of symptoms, sports participation and ankle activity score. The ankle activity score is a scoring system which ranks the patients activity level and ankle loading by their level and type of (sports) activity.[18] An ankle activity score of 4 or higher ranges from physical work and power lifting to competitive basketball.[18]

The outcome measures in this study were the Victorian Institute of Sports Assessment-Achilles (VISA-A) questionnaire, the Pain Coping Inventory (PCI) and painDETECT questionnaire. The VISA-A questionnaire was completed at baseline, 6, 12, and 24 weeks, and the PCI and painDETECT were completed only at baseline. We used the VISA-A score as primary outcome measure, which is a reliable questionnaire to determine the severity of AT symptoms (both pain and activity level) and it has been validated in Dutch language.[19,20] The patients completed the patients completed the Pain Coping Inventory (PCI) and painDETECT questionnaire were secondary outcome measures.[5,21] The PCI is a validated questionnaire to determine the level of active and passive coping.[5] Patients were asked to rate 33 items on a 4-point Likert scale ranging from 1 (hardly ever) to 4 (very often). These items were used to calculate the score of their corresponding domains. The domains transformation, distraction and reducing demands formed the score for active coping; the passive coping score was formed by retreating, worrying and resting. The sum of the domains were added up and divided by the maximum score. Both active and passive coping have a score ranging from 0 to 1, where a score closest to 1 represents a high level of active or passive coping. The painDETECT questionnaire is a validated tool to detect neuropathic pain components and has a maximum possible score of 38 points.[21,22] The scores were divided in three categories: (1) $\leq$12 points, neuropathic pain component is unlikely (<15%), (2) 13–18 points, unclear result, and (3) $\geq$19 points, neuropathic pain component is likely (>90%).[21]

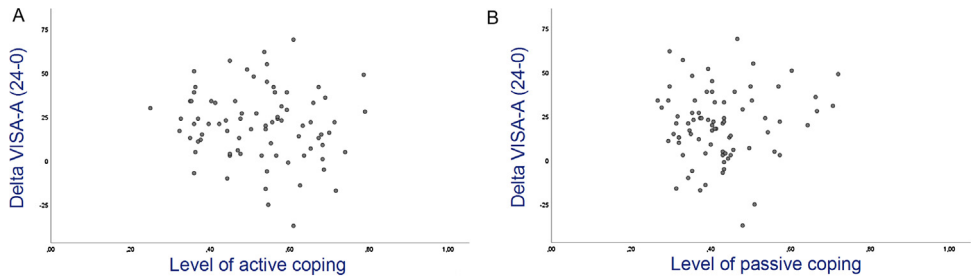

Fig. 1. (A) The influence of the level of active coping strategy on the change in VISA-A score at 24 weeks. (B) The influence of the level of passive coping strategy on the change in VISA-A score at 24 weeks.

We used the Shapiro Wilk test to determine normality of the data, where we assumed a normal distribution when $p > 0.05$. Normally distributed data were expressed as mean (standard deviation), and non-normally distributed data as median (interquartile range, IQR). To determine the association between active coping and the course of AT symptoms, expressed by the VISA-A score and measured at baseline, 6, 12 and 24 weeks, a Generalized Estimating Equation (GEE) model was used. To determine whether the course of AT symptoms was associated to the level of active coping, we added the interaction term active coping * time point. We adjusted for the predefined variables age, sex, body mass index (BMI), duration of symptoms at baseline in weeks and level of sports activity, measured with the ankle activity score.[18] This GEE model was also used to determine the association between the following variables and the course of AT symptoms: passive coping, neuropathic pain component and the difference between active and passive coping score. The difference between an active and passive coping score was calculated by the active score minus the passive score. Associations were considered significant if $p < 0.05$. As reported in the study protocol, imputation was needed if the sensitivity analysis reported ≥5% missing data of the primary outcome.[17] We used SPSS software (V.24.0.0.1; SPSS, Chicago, Illinois, USA) for statistical analysis.

Table 1
Baseline characteristics.

	Included patients *N* (%)/mean (SD)/median (IQR)
N	80
Age (years)[a]	50.0 (44;54)
Sex (% male)	39 (49%)
BMI (kg/m^2)[a]	25.7 (24.0;30.1)
Duration of symptoms (weeks)[a]	63 (40;128)
Ankle activity score (0–10)[a]	5 (5;6)
Participation in sports activity	64 (80%)
Active coping score (0–1)	0.53 (0.13)
Transformation	2.1 (0.7)
Distraction	1.9 (0.6)
Reducing demands	2.3 (0.7)
Passive coping score (0–1)	0.43 (0.10)
Retreating	1.3 (0.4)
Worrying	1.8 (0.5)
Resting	2.0 (0.6)
PainDETECT (0–35):	
1. Neuropathic pain component unlikely (≤12)	34 (43%)
Unclear result (13–18)	34 (43%)
Neuropathic pain component likely (≥19)	12 (15%)
VISA-A (0–100) at baseline	43 (16)

SD: standard deviation. IQR: interquartile range. BMI: body mass index. VISA-A: Victorian Institute of Sports Assessment-Achilles questionnaire.
[a] Non-normal distributed data, expressed in median (IQR).

3. Results

A total of 185 patients were screened for eligibility, of which 80 were included (Supplementary file 1). Included patients had a median age of 50 (interquartile range (IQR): 44;54) years, with equal sex distribution (51% female), had a median body mass index of 25.7 (IQR 24.0;30.1) kg/m^2, and 80% were physically active (ankle activity score of 4 or higher). The baseline characteristics are presented in Table 1. Only 1 patient (1%) was lost to follow-up at 24 weeks. The mean (standard deviation – SD) VISA-A score gradually increased from 43 (16) points at baseline to 63 (23) points at 24 weeks, with no difference between intervention groups.[17] The mean (SD) score for active coping was 0.53 (0.13) and 0.43 (0.10) for passive coping. A neuropathic pain component was likely in 12 patients (15%).

The interaction term active coping * time point was not statistically significantly ($p = 0.459$), with a beta (95% Wald confidence interval) at baseline of 19 (−12;50), 6 weeks of 20 (−7;47), and 12 weeks of 12 (−10;34). Similarly, the interaction term passive coping * time point was not statically significant ($p = 0.478$, with a beta (95% Wald confidence interval) at baseline of −18 (−58;21), at 6 weeks of 16 (−17;49), and at 12 weeks of 1 (−26;28). This means that both active and passive coping were not associated with the course of AT symptoms over 24 weeks, as change in VISA-A score over time does not depend on level of coping (Fig. 1).

When analyzing the difference between active and passive coping score, the interaction terms active-passive coping * time point was not statistically significant ($p = 0.158$). This means that we found no association between the course of AT symptoms over 24 weeks and the difference in level of active and passive coping.

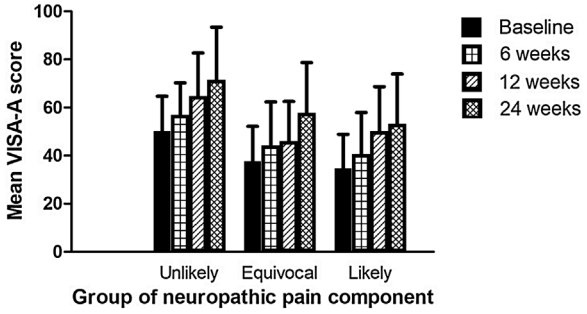

Fig. 2. The mean VISA-A score per time point divided by the three subcategories of the painDETECT score. Error bars denote standard deviations.

There was a difference in severity of AT symptoms, measured with the VISA-A score, between the 3 categories of the painDETECT score at baseline ($p < 0.001$), 6 ($p = 0.002$), 12 ($p < 0.001$) and 24 weeks ($p = 0.011$) (Fig. 2). While the difference in severity of AT symptoms can be explained by the presence of a neuropathic pain component, it is also influenced by two other variables: a longer duration of symptoms negatively influenced the severity of AT symptoms ($p = 0.032$), while a higher ankle activity score had a positive influence on the severity of AT symptoms ($p = 0.001$).

The interaction term neuropathic pain category * time point was not statistically significant ($p = 0.420$), indicating that there was no association between likeliness of a neuropathic pain component and the course of AT symptoms over 24 weeks (Fig. 2).

4. Discussion

This is the first study to analyse the association between pain coping strategy, presence of a neuropathic pain component and the course of AT symptoms. Contrary to widespread belief, we found no association between level of active or passive coping and the course of AT symptoms. Although patients with a neuropathic pain component had more severe AT symptoms at every time point, the course of AT symptoms was similar between patients with an unlikely, unclear and likely neuropathic pain component.

Degenerative joint diseases (e.g. osteoarthritis) and tendinopathy have multiple similarities: the main feature in both pathologies is extracellular matrix degeneration, they both respond well to mechanotherapy and they have similar risk factors.[23] Osteoarthritis and tendinopathy are both associated with a chronic pain component.[23] In osteoarthritis, a higher level of passive coping leads to more chronic pain.[7,8,24] Due to the similarities between osteoarthritis and tendinopathy, we wondered if passive coping is also associated with more severe symptoms in AT. Patients with AT had a similar score in all subscales of the passive coping score (AT versus osteoarthritis patients: retreating 2.1 versus 1.7, worrying 1.8 versus 2.0 and resting 2.0 versus 2.5).[24] We found no association between severity of AT symptoms and level of passive coping strategy. Rest of the Achilles tendon will result in an immediate decrease of pain, but it will also decrease the load tolerance.[25] Furthermore, a previous study proved no difference in outcome between patients who were treated with relative rest and patients who continued tendon-loading activities.[26] This might explain why a passive coping strategy is not beneficial, but also not detrimental, for patients with AT.

The level of active coping was also not associated with the course of AT symptoms. Although an active coping strategy was hypothesized to be beneficial,[5] results of multiple studies in osteoarthritis patients showed no conclusive results.[7,24] Patients with AT had similar scores in subscales of active coping score as patients with osteoarthritis: transformation 2.1 versus 1.9, distraction 1.9 versus 1.9 and reducing demands 2.3 versus 2.6.[24] In patients with AT, there is a delicate balance between underloading and overloading of a recovering tendon.[27] There might be two subgroups of patients within the group with a high level of active coping: a group of patients that uses the coping strategies transformation and distraction to function in daily life and to rehabilitate, and a group of patients who use these coping strategies to continue their activities on their previous level, which may lead to overloading of the tendon and therefore might hamper recovery. This is supported by Smith et al.[28], who researched coping in patients with rheumatoid arthritis and found that active coping strategies are more context sensitive: they suggest that active coping might be harmful as patients ignore pain signals.[28] An active coping strategy might be beneficial when it facilitates adequate rehabilitation, but disadvantageous when patients continue to overuse their tendon. These delicate differences within the active coping strategy are unfortunately not detected by the PCI questionnaire.

The general hypothesis is that patients with a neuropathic pain component have abnormal pain processing, which causes an abnormal response to regular stimuli.[10–12,29] We confirmed that patients with a neuropathic pain component started and ended a conservative treatment program with more severe AT symptoms than patients without a neuropathic pain component. One other study evaluated the prevalence of a neuropathic pain component with the painDETECT questionnaire in patients with tendinopathies, amongst which insertional ($n = 36$) and midportion AT ($n = 31$).[29] They found a prevalence of neuropathic pain in 28% of patients with insertional AT and 26% of patients with midportion AT, but neuropathic pain was not associated with a worse outcome.[29] Interestingly, we also found that all three groups (unlikely, unclear and likely neuropathic component) had a similar improvement in symptom severity at every time point. One explanation might be that patients with a neuropathic pain component respond similar to conservative treatment of AT as patients without a neuropathic pain component. However, as the neuropathic pain component is still present and untreated, they experience more severe symptoms.[30] As the treatment of neuropathic pain is centred around pharmacological intervention and non-pharmacological treatments like cognitive behavioural therapy,[30] addition of these therapies might be beneficial to patients with a high likeliness of having a neuropathic pain component.

This study has several strengths, which ensures the integrity of this data. First, this study is based on the robust study protocol of a pre-registered randomized clinical trial.[17] Second, our data was prospectively collected and corrected for baseline characteristics, which limits the influence of bias. Third, this study had a very low lost to follow-up rate, which ensures that the data was not biased by non-responders. Last, we used valid outcome measures which increases the reliability of our data and the validity of our conclusions.

A limitation of this study is the interpretation of active and passive pain coping strategy. There are no known thresholds to indicate that a patient has a 'high' active pain coping strategy. The ability to dichotomize the data would make it easier to analyse whether absence or presence of an active pain coping strategy influences the severity of AT symptoms. Another possible limitation could be that the pain coping questionnaire has not yet been validated for patients with (Achilles) tendinopathy. Although it has been validated for other musculoskeletal conditions with a chronic pain component, it can be possible that the pain coping questionnaire does not cover the complexity (e.g. coping strategy in patients with warm-up phenomenon versus increasing pain during sports activities) of Achilles tendinopathy. Furthermore, the fact that neuropathic pain is a clinical diagnosis and we did not physically examine patients on presence of neuropathic pain could also be a limitation.[30] While the painDETECT score is proven to be a valid and reliable tool to screen for neuropathic pain, it has not been validated in patients with AT and it is not a substitute for its diagnosis.[21] As the use of a questionnaire as screening tool for sports medicine healthcare providers is more feasible, we think that this choice has better practical implications. Another limitation might be that patients might adopt a different coping strategy when being treated with an intervention. Last limitation is that all patients received a painful injection at baseline, after completing the baseline questionnaires. This intervention might have had influence on the pain perception and consequently it could affect these study results. Last, it might be possible that we failed to detect an association while there might be a true association (type I error) due to an insufficient sample size. Selection bias might be another reason why we failed to find an association. One of the diagnostic criteria for AT is localized pain with pain on local palpation, which is opposing to the criteria for neuropathic pain (which includes widespread pain).

A subgroup of AT patients has a higher likelihood of having a neuropathic pain component. As these patients ended with more severe AT symptoms after conservative treatment, it might be interesting to treat this subgroup of patients for their neuropathic pain component in a future trial.[30] A randomized clinical trial is needed to analyse whether standard neuropathic pain treatment in addition to AT treatment has a more beneficial effect on symptoms

in this specific subgroup. Furthermore, it would be interesting to analyse whether coping strategy changes over time. This could be analysed by calculating the Pain Coping Inventory at baseline and after delivery of patient education.

5. Conclusion

The course of AT symptoms after conservative treatment is not altered by the employment of a more active or more passive coping strategy in patients who received exercise therapy and an injection. We were able to identify a subgroup of patients with a neuropathic pain component having more severe AT symptoms before and after conservative treatment. However, the presence of a neuropathic pain component does not influence the course of AT symptoms in patients with the clinical diagnosis, which includes having localized pain. Future intervention trials might focus on this subgroup with the aim to further improve patient-reported outcomes.

Ethical statement

The HAT study was approved by the Medical Research Ethics Committee of Southwest Holland, the Netherlands (MEC-14-100). Trial registration number (The Netherlands Trial Register): ID number: NL4686, clinicaltrails.gov (identifier: NCT02996409)

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Declaration of interests

None.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.jsams.2021.04.001.

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