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## Correlation between power Doppler ultrasonography and clinical severity in Achilles tendinopathy

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**Abstract** Twenty-five patients with chronic Achilles tendinopathy were clinically and ultrasonographically evaluated. A positive correlation existed between power Doppler ultrasonography (PDU) and tendon thickness ( $r=0.63$ ,  $p<0.001$ ) and patient's age ( $r=0.40$ ,  $p<0.05$ ). A negative correlation existed between PDU and a functional test (number of toe raises to pain) ( $r=-0.57$ ,  $p<0.005$ ) and one recorded item of the Victorian Institute of Sport Assessment Achilles score (VISA-A questionnaire, item 6: jumping capability) ( $r=-0.46$ ,  $p<0.05$ ). Three patients had no detectable blood flow on PDU. PDU of Achilles tendons does not seem to be strictly related to symptoms but rather to functionality and chronicity of tendinopathy as indicated by toe-raises testing, jumping capability, patient age and tendon thickening.

**Résumé** Vingt-cinq malades avec une tendinopathie chronique du tendon d'Achille ont été évalué d'une manière clinique et ultrasonographique. Une corrélation positive existait entre l'amplitude au doppler, l'épaisseur du tendon ( $r=0.63$ ,  $p<0.001$ ) et l'âge du malade ( $r=0.40$ ,  $p<0.05$ ). Une corrélation négative existait entre le doppler, un test fonctionnel et une article du 'Victorian Institute of Sport Assessment Achilles' questionnaire (VISA-A article 6: la capacité de saut) ( $r=-0.46$ ,  $p<0.05$ ). Trois malades n'avaient aucun courant sanguin détectable au doppler. L'amplitude ultrasonographique au niveau du tendon d'Achille ne paraissait pas en rapport avec les symptômes mais plutôt avec la fonction et la chronicité de la tendinopathie comme indiqué par les différents test, l'âge du patient et l'épaississement du tendon.

### Introduction

Chronic Achilles tendinopathy is a frequent overuse problem in athletes, which is difficult to treat. Its aetiology remains in part unexplained, although histological and biochemical studies have demonstrated a non-inflammatory tendinosis as underlying pathology [8]. Several studies indicate that Achilles tendinosis is accompanied by neovascularisation or hypervascularity [8, 11]. The exact role of neovascularisation or angioblastic hyperplasia in chronic tendinopathy remains an issue of discussion, which has recently received new attention. Tendons represent in general a comparatively poorly vascularised tissue that relies heavily on synovial fluid diffusion [7]. The concept that Achilles tendinopathy at least in part is caused by an impoverished vascular supply to the afflicted region of the Achilles tendon has been commonly accepted. In apparently complete contrast to this is the hypervascularity demonstrated on biopsies from patients with Achilles tendinopathy [1]. These findings of hypervascularity are in accordance with the laser Doppler flow measurements of Astrom et al. who concluded that a local deficiency in tendon blood supply does not initiate the lesion, nor does it explain why the condition persists [2]. Öhberg and Alfredson added recently the assumption that vessels, and possibly nerves accompanying the vessels, are involved in the pain mechanism in chronic painful Achilles tendinosis [10]. Thus, neovascularisation in tendinopathy is not thought to be associated with an attempt of tendon repair but rather with tendon degeneration. Moreover, it has been suggested that the invasion and proliferation of new blood vessels may be a contributory factor to the pain and chronicity of the disease [5].

Ultrasonography has been established as an accurate and cost-effective method of Achilles tendon evaluation [4, 12]. Doppler ultrasonography enables an estimation of tissue vascularity. In contrast to conventional colour Doppler ultrasonography, power Doppler ultrasonography (PDU) imaging is independent of the angle of incident beam [9]. Colour Doppler and PDU provide the cli-

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nician with the possibility of detecting neovascularisation in chronic tendinosis problems [11, 13]. This may, in future, have therapeutic implications. In a pilot study of ten patients, the injection of a sclerosing agent against neovessels appeared effective in reducing pain in chronic Achilles tendinopathy [10]. While this approach undoubtedly needs further scientific confirmation, the clinician is left with the dilemma of whether to promote or counter blood flow to the Achilles tendon in the therapeutic approach for chronic Achilles tendinopathy. A first step towards a solution would be an explanation of the relationship between symptoms and disease severity and tendon neovascularisation.

The aim of this study was to investigate in patients with Achilles tendinopathy the existence of a relationship between Achilles tendon neovascularisation measured by PDU and clinical severity parameters of Achilles tendinopathy.

## Materials and methods

Between April and September 2002, 25 patients consulting our outpatient sports clinic (University Hospitals Leuven, Belgium) for Achilles tendinopathy were selected. Inclusion criteria were pain or discomfort lasting for more than 3 months and ultrasonographically established Achilles tendinosis. Patients with other concomitant local or systemic diseases were excluded. The history and symptoms related to the Achilles tendon were taken by an experienced clinician (KP) before PDU was performed. PDU was taken by the second author (PB), an experienced musculoskeletal radiologist who was unaware of the clinical evaluation.

### Grey-scale ultrasonography

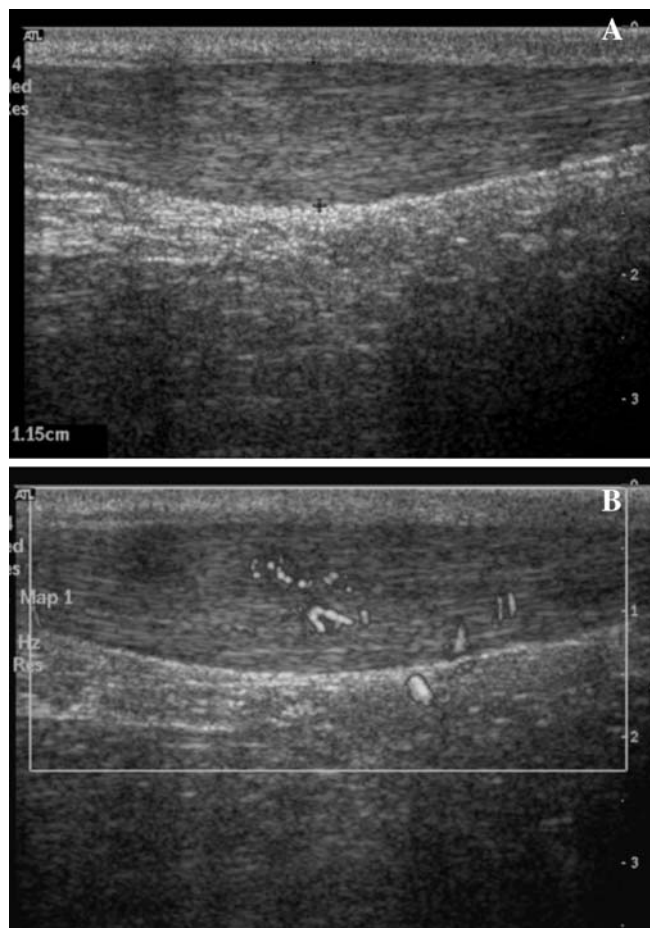
Ultrasonography was performed with the patients lying in prone position with their feet overhanging the examination table. Achilles tendons were examined with a 5 cm 5–12 MHz linear array probe (HDI 5000, ATL-Philips Ultrasound, Belgium). All tendons were examined from calcaneal insertion to the musculotendinous junction in the sagittal and axial plane. At the tendinosis site of maximal tendon thickening images were recorded. Tendon thickness was measured on axial images as the maximal anteroposterior diameter in millimetres (mm) (Fig. 1a).

### Power Doppler ultrasonography

To obtain maximal sensitivity without background noise, a pulse repetition frequency of 1,000 Hz and colour gain setting of 81% was used. Care was taken to avoid motion artefacts. After recording a set of sagittal images centred at the level of the tendinosis, the image showing maximal flow was selected. On this image, an estimation of flow quantification (millimetres<sup>2</sup>) was performed through computerised surface measurement of coloured pixels (Medical Image Computing Radiology-ESAT/PSI, Belgium). These measurements were performed by the second author who was unaware of clinical scores. Additionally, measurement stability was tested in a pilot study for intratester reliability ( $r=0.9$ ,  $p<0.001$ ) (Fig. 1b).

### Clinical evaluation

Clinically, patients were evaluated using the Victorian Institute of Sport Assessment Achilles (VISA-A) questionnaire [14]. This questionnaire, specifically designed to serve as an index of severi-



**Fig. 1** **a** Grey-scale ultrasonography of Achilles tendinosis resulting in a distinct widening of the tendon (11.5 mm). **b** Power Doppler ultrasonography of the same Achilles tendinosis with increased blood flow (neovascularisation) intratendinously and anteriorly of the tendon

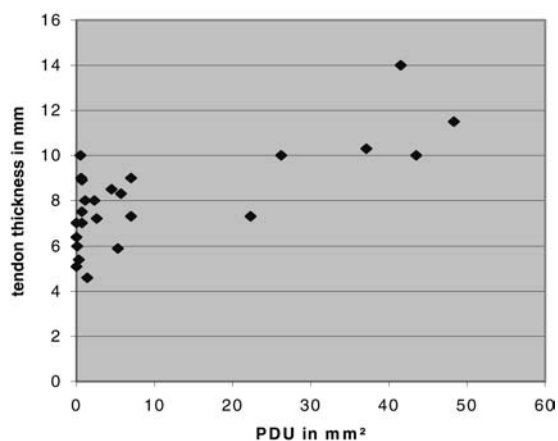
ty of Achilles tendinopathy, contains eight questions measuring the domains of pain, function in daily living and sporting activity. Scores range from 0 to 100 points, with 100 points representing a perfect score. The first six items were also taken into account separately as Visual Analogue Scores (VAS). The first question (VAS-1) quantifies morning stiffness in the Achilles tendon, the second (VAS-2) stretch pain, the third (VAS-3) pain after walking, the fourth (VAS-4) pain walking down stairs, the fifth (VAS-5) pain after heel raises and the sixth (VAS-6) jumping capability. All relevant elements of history related to Achilles tendinopathy were recorded and particular attention paid to symptom duration as well as patient characteristics.

### Statistical methodology

Spearman correlations were used to evaluate the degree of association between power Doppler flow and a list of other variables (continuous, count or ordinal variable). The alpha level was set at 5%. Due to the exploratory character of the study, no corrections were made for multiple testing. All analyses were performed with the statistical package SAS (version 8.1).

## Results

Eight women and 17 men aged 20–67 (mean 42) years were evaluated. Symptom duration ranged from 3 to 48 (mean 17) months. Participation in sport varied from 0 to 12 h per week. Three patients did not answer this question, as they were unable to estimate their average weekly sporting-activity. On average, patients recorded local pain on palpation as  $6\pm 2$  (range 2–10) points. Two patients found it too difficult to quantify pressure pain. VISA-A score ranged from 12 to 92 (mean  $54\pm 23$ ). Separate VAS scores on the first six questions of the VISA-A score averaged  $7\pm 3$  (range 0–10) for VAS-1, VAS-2, VAS-4 and VAS-5,  $8\pm 3$  (range 2–10) for VAS-3,  $5\pm 4$  (range 0–10) for VAS-6. Ultrasonographic measurement of tendon thickness ranged from 5 to 14 mm (mean  $8\pm 2$  mm). Computerised surface measurement of neovascularisation on PDU averaged  $10\pm 16$  mm<sup>2</sup> (range 0–48 mm<sup>2</sup>). Twelve per cent ( $n=3$ ) of the examined Achilles tendons had no PDU measurable neovascularisation. Calculated Spearman correlation coefficients are summarised in Table 1. A positive correlation was detected between PDU and tendon thickness (Fig. 2) and



**Fig. 2** Scatter plot of ultrasonographic tendon thickness and power Doppler tendon vascularity quantified on computerised surface measurement ( $r=0.63$ ,  $p=0.0007$ )

patient age. A negative correlation existed between PDU and the functional test (number toe raises to pain), and also one VAS-score (VISA-A questionnaire, item 6).

## Discussion

The usefulness of Doppler ultrasonography in evaluating tendinopathy has been recently explored. In patellar and Achilles tendinopathy, colour Doppler ultrasonography could depict neovascularisation, and this increased blood flow was predominantly present in symptomatic tendons [11, 15, 16]. Richards et al. show that PDU can depict vessel proliferation in Achilles tendinopathy [13]. However, a distinct relationship between symptoms or severity of disease and tendon neovascularisation had not been investigated to date.

In this study, we have shown that in chronic Achilles tendinopathy tendon blood flow measured by PDU does not firmly relate to different methods of pain or symptom evaluation. Only one item of the VISA-A questionnaire showed to be slightly, though significantly, inversely related to the power Doppler signal. On the other hand, power Doppler ultrasonography was positively related to the dimensions of tendinosis and the patient's age. This may indicate that via PDU measured neovascularisation is an expression of histological deterioration or tendinosis but not necessarily an indication of the degree of pain. Taken together, these results corroborate the hypothesis mentioned earlier that neovascularisation in chronic Achilles tendinopathy is associated with tendon degeneration or a failed healing response rather than an attempt of tissue repair. It can only be speculated why in this study the absence of a consistent link between neovascularisation and pain was demonstrated. In general in overuse injuries, structural or anatomopathological changes do not immediately or consistently relate to pain or symptoms. A subclinical phase often exists and frequently patients return to full sport activities before structural changes resolve. Intrinsic and extrinsic risk factors always interfere with clinical symptoms in overuse injuries. It has been proven that stretch through ankle dorsiflexion diminishes Achilles tendon blood flow [3].

**Table 1** The Spearman correlation between different variables and power Doppler flow

Variable	Spearman correlation	<i>p</i> -value	No.
VAS-1	-0.20693	0.3210	25
VAS-2	0.00435	0.9835	25
VAS-3	-0.12617	0.5479	25
VAS-4	0.09067	0.6664	25
VAS-5	-0.22239	0.2853	25
VAS-6	-0.46195	0.0201	25
VISA-A	-0.12493	0.5518	25
Tendon thickness	0.63168	0.0007	25
Age	0.40262	0.0460	25
Duration of symptoms	0.32618	0.1116	25
Sport (hours/week)	-0.20402	0.3624	22
Pain on palpation	0.01602	0.9422	23
Number of toe raises to pain	-0.56753	0.0031	25
Number of physiotherapy sessions	-0.18943	0.3645	25

VAS-1 to VAS-6 represent items 1–6 of VISA-A questionnaire

For this reason, all patients were examined in the same relaxed, unstretched position. Nevertheless, it remains possible that confounding factors that were not assessed or measured may have, to a certain extent, influenced the results.

Our study demonstrates that symptomatic Achilles tendinopathy can exist with or without neovascularisation detectable on PDU. In our study, three out of 25 (12%) patients with clinical Achilles tendinopathy did not have detectable neovascularisation on PDU. This is in contrast to the findings of Alfredson et al. who recorded detectable colour Doppler flow in all symptomatic tendons [11]. It is possible that neovascularisation was present in all cases in our study; however, a flow slower than 4–6 mm/sec is not detectable on Doppler ultrasonography. The absence of a clear link between pain and PDU does not contradict the possible importance of neovascularisation and the development of symptomatic Achilles tendinosis. Neovascularisation, as part of the degenerative process, may represent an additional and easily obtainable indication of an increased risk for – but not a sign of – pain in Achilles tendinopathy. In a longitudinal study of Danish football players, a pre-season ultrasonographic positive screening for Achilles tendinosis represented a manifest increased risk for developing symptoms during the following year [6]. Likewise, neovascularisation of Achilles tendinosis may represent an additional risk increase. If pain in some patients with Achilles tendinopathy does originate from nerve endings accompanying blood vessels, it seems logical that proof of neovascularisation does increase the possibility of symptomatic tendinopathy.

In conclusion, in Achilles tendinopathy patients, PDU often but not always reveals neovascularisation. Tendon flow on PDU seems positively related to tendon degeneration and functionality indicated by tendon size measurement, patient age and functional question or test. PDU does not strictly relate to pain or symptoms but possibly represents an additional risk adding to the clinical information on Achilles tendinopathy. In order to establish unambiguously the role and value of PDU in Achilles tendinopathy, the relative risk of neovascularisation in asymptomatic Achilles tendinosis and PDU effects of different treatments also need to be established in longitudinal studies.

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