Ultrasonographic Study of Achilles Tendon and Plantar Fascia in Chondrocalcinosis

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ABSTRACT. ***Objective.*** To investigate by high frequency grey-scale ultrasonography (US) and power Doppler sonography (PDS) the modality and frequency of involvement of the Achilles tendon and plantar fascia in chondrocalcinosis (CC), and to correlate these findings with clinical complaints and radiographic evidence.

Methods. The heels of 57 consecutive patients with CC were evaluated by US, PDS, and radiography. One control group of 50 consecutive patients with osteoarthritis (OA) without signs of CC was studied in the same way. A second control group of 50 healthy subjects underwent only US/PDS examination. All subjects also underwent clinical assessment.

Results. US revealed Achilles tendon calcifications in 57.9% of those with CC, but none in the control groups. Plantar fascia calcifications were observed in 15.8% of CC and in 2% of OA cases, but not in healthy controls. US showed no significant difference in postero-inferior and inferior calcaneal enthesophytosis between subjects with CC (59.6% and 61.4%, respectively) and those with OA (46% and 44%, respectively). Such alterations were also present, in lower percentages, in the healthy controls. Posterior and inferior calcaneal erosions were absent in all groups. Achilles enthesopathy was found in 22.8% of patients with CC (14.9% of heels, with vascular signals in 11.4% of heels on PDS). Deep retrocalcaneal bursitis was found in 10.5% of patients with CC (7% of heels, with vascular signals in 5.2% of heels on PDS). Plantar fasciitis was found in 40.3% of patients with CC (36% of heels, with vascular signals in 2.6% of heels on PDS) and in 14% of OA patients, but not in healthy controls. No significant correlation was found between talalgia or sex of patients and presence of calcifications. A significant correlation was observed between talalgia and Achilles enthesopathy ($r = 0.78$, $p < 0.0001$), deep retrocalcaneal bursitis ($r = 0.7$, $p < 0.0001$), and plantar fasciitis ($r = 0.31$, $p < 0.001$). A significant correlation between talalgia and vascular signals on PDS was observed in Achilles enthesopathy ($r = 0.91$, $p < 0.0001$) and deep retrocalcaneal bursitis ($r = 0.65$, $p < 0.0001$). The presence of vascular signals on PDS was significantly associated with the presence of tendinous and bursal grey-scale US alterations. Achilles tendon calcifications were 39% sensitive, 100% specific, and 77% accurate for the presence of CC, whereas plantar fascia calcifications were 15% sensitive, 98% specific, and 54% accurate. Excellent agreement was found between US and radiography in detecting Achilles tendon calcifications ($k = 0.86$), plantar fascia calcifications ($k = 0.77$), postero-inferior enthesophytosis ($k = 0.90$), and inferior enthesophytosis ($k = 0.83$).

Conclusion. Calcaneal tendon calcifications are frequent and asymptomatic findings in patients with CC, and they have a high specificity for this disease. US shows high agreement with radiography in depicting calcifications and enthesophytosis. Inflammatory changes of the calcaneal soft tissues are frequently observed by US and PDS in patients with chondrocalcinosis. (J Rheumatol 2004;31:2242–50)

Key Indexing Terms:
CHONDROCALCINOSIS
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ACHILLES TENDON
PLANTAR FASCIA

Chondrocalcinosis (CC) is an arthropathy generally caused by deposit of calcium pyrophosphate-dihydrate (CPPD) microcrystals in the joints and in the tendons[1,2]. In CPPD crystal deposition disease typical linear calcifications can be found in fibrocartilage, especially in the knee menisci, carpus triangular ligament, symphysis pubis, and in hyaline articular cartilage, especially in the knee[1,2].

Calcifications in tendons have been described in previous radiological reports[3-9]: Achilles tendon, gastrocnemius tendon, hip adductors, and quadriceps tendon were the sites primarily involved.

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Only a few studies have evaluated the role of ultrasonography (US) in the detection of calcifications on hyaline cartilages of the knee[10-13] and on carpal fibrocartilages[11], with encouraging results. One recent work showed the usefulness of US to reveal signs of CC in elbow entheses with no radiographic evidence of CPPD crystal deposition disease[14]. To date US has not been applied to the study of Achilles tendon and plantar fascia in patients with CPPD crystal deposition disease.

High frequency US has proved to be an excellent technique for accurate evaluation of tendons, entheses, and cortical bone involvement in rheumatic diseases[15,16]. Moreover, US is easy to perform and is repeatable and inexpensive. Recently, power Doppler sonography (PDS) has proved to be a reliable tool for semiquantitative assessment of the vascularity of the soft tissue, estimating the presence and extent of inflammation, and its changes after therapy[17-19].

We used US and PDS to examine a large sample of heels of patients with CPPD crystal deposition disease to investigate the modality and the frequency of involvement of the Achilles tendon and plantar fascia in this disease and to correlate these findings with clinical complaints and radiographic evidence.

MATERIALS AND METHODS

Fifty-seven consecutive outpatients with CPPD crystal deposition disease according to criteria described by McCarty[2], 27 women and 30 men, mean age 69.4 years (range 44–92) and mean disease duration 7.2 years (range 2.6–6.1), were enrolled in this study (CC group). The diagnosis of CPPD crystal deposition disease was definite in all cases (radiographic evidence of calcification of fibrocartilage or hyaline cartilage and presence of crystals compatible with CPPD on polarized microscopy of fresh synovial fluid)[2]. Clinical patterns observed in the patients were pseudogout in 13 patients, pseudo-rheumatoid pattern in 8, pseudo-osteoarthritis (OA) in 15, pseudo-OA with recurrent inflammatory flares in 18, and polymyalgia-like onset in 3 patients. Two patients had hyperparathyroidism and one patient had alkaline hypophosphatasia. Two control groups of comparable age and sex were also selected. The first control group was composed of 50 consecutive patients, 29 women and 21 men, with a mean age of 66.4 years (range 50–81), who had OA without radiographic signs of CC, with a mean disease duration of 9.1 years (range 5.8–12.3) (OA group). The second control group comprised 50 healthy subjects (volunteers and personnel of the Institute), 25 women and 25 men, with a mean age of 66.3 years (range 42–73) (control group).

Clinical examination for posterior or inferior talalgia was performed by experienced rheumatologists in all the subjects. Tenderness on pressure or spontaneous talalgia at each site was recorded.

Lateral radiography of the heels was performed in all patients (CC group and OA group).

All subjects of the 3 groups underwent an ultrasound (US)/power Doppler sonography (PDS) examination of the heel regardless of the presence of signs or symptoms of inflammatory involvement. US/PDS was performed by an experienced operator (PF). The sonographer was blind to the diagnosis and the radiography results. Each examination was carried out bilaterally and symmetrically, with both longitudinal and transverse scans[16]. Anatomical structures observed in the US examination were Achilles tendon and its enthesis, retrocalcaneal deep and superficial bursae, plantar fascia and enthesis, subcalcaneal fat pad, and cortical bone of posterior and inferior aspects of the heel (Figure 1). Conventional grey-scale US and PDS examinations were carried out using a Technos MP (Esaote Biomedica, Genova, Italy) with a 10–13 linear transducer.

Calcifications of the Achilles tendon and the plantar fascia insertional tract were diagnosed at US examination when hyperechoic deposits with acoustic shadowing generally not in continuity with the bone profile were observed within the fibrillar tendon structure (Figures 1b, 1d).

Sonographic findings of Achilles enthesopathy were considered to be heterogeneous hypoechogenicity and thickening of the insertional tract of the tendon (> 5.29 mm at the postero-superior calcaneal surface), sometimes associated with enthesophytosis, erosions, and peritendinous edema (Figure 1b)[15,16,20-22].

Sonographic findings of bursal involvement were considered to be anechoic bursal space widening (> 1 mm in thickness and 7 mm maximum craniocaudal diameter; interpreted as effusion), homogeneous echoic or irregularly echoic widening (interpreted as synovial proliferation associated with effusion) (Figure 1b)[15,20,21,23].

Sonographic findings of plantar fasciitis were considered to be heterogeneous hypoechogenicity and thickening of the insertional tract of the fascia (> 4 mm where the plantar fascia crosses the anterior aspect of the inferior border of the calcaneus), sometimes associated with enthesophytosis, erosions, and perifascial edema (Figure 1d)[15,24,25].

The enthesophytosis appeared on US as a hyperechoic bony spur interrupting the cortical profile, determining the characteristic shadowing[15,16]. Erosions of the posterior and inferior aspects of the heel appeared on US as an interruption of the cortical bone profile[15,16,20].

PDS was performed after grey-scale US by longitudinal and transverse scans, selecting a region of interest that included soft tissues and underlying bone with these technical settings: pulse repetition frequency (PRF) of 750–1000 Hz, highest gain level without background noise and low filter. The presence of intratendinous and intrabursal color activity was attributed to vascularization when the following criteria were satisfied: presence of flow in orthogonal scans, presence of pulsatility of flow, permanence of flow at increase of PRF. Spectral wave analysis was performed only in a few cases because it was not an aim of this study.

The statistical analysis was performed using SPSS 11.0. The chi-square test was used for an overall approach to compare percentages among the groups. Fisher's exact test with Yates' continuity correction was used to compare percentages between 2 groups. Correlations were calculated using Spearman's rank correlation coefficient. ANOVA was used to compare the clinical and demographic characteristics of the groups. The level of statistical significance was set at a p level of 0.05.

A comparison between US and radiography was carried out. Neither imaging modality was regarded as the gold standard, so we used kappa coefficients to indicate binary agreements of these methods in detecting enthesophytosis and calcifications of Achilles tendon and plantar fascia. Values of kappa < 0.40 reflect poor agreement, values 0.40–0.75 fair to good agreement, and > 0.75 excellent agreement[15]. Moreover, the sensitivity, specificity, and accuracy of calcifications of the Achilles tendon and plantar fascia in the diagnosis of CPPD disease (compared with the group of patients with OA) were determined.

RESULTS

The clinical, sonographic, and radiographic results and some of the statistics of our study are given in Table 1.

US examination showed Achilles tendon calcifications in 57.9% (33/57 patients) of CC ($p < 0.0001$ vs OA and controls) (58/114 heels, bilateral in 25 cases); and plantar fascia calcifications in 15.8% (9/57 patients) of CC (18/114 heels, bilateral in all cases); and in 2% (1/50 patients) of OA (p not significant) and in no control ($p < 0.05$ CC vs controls). The Achilles tendon calcifications appeared in longitudinal scans as intratendinous hyperechoic linear deposits, parallel

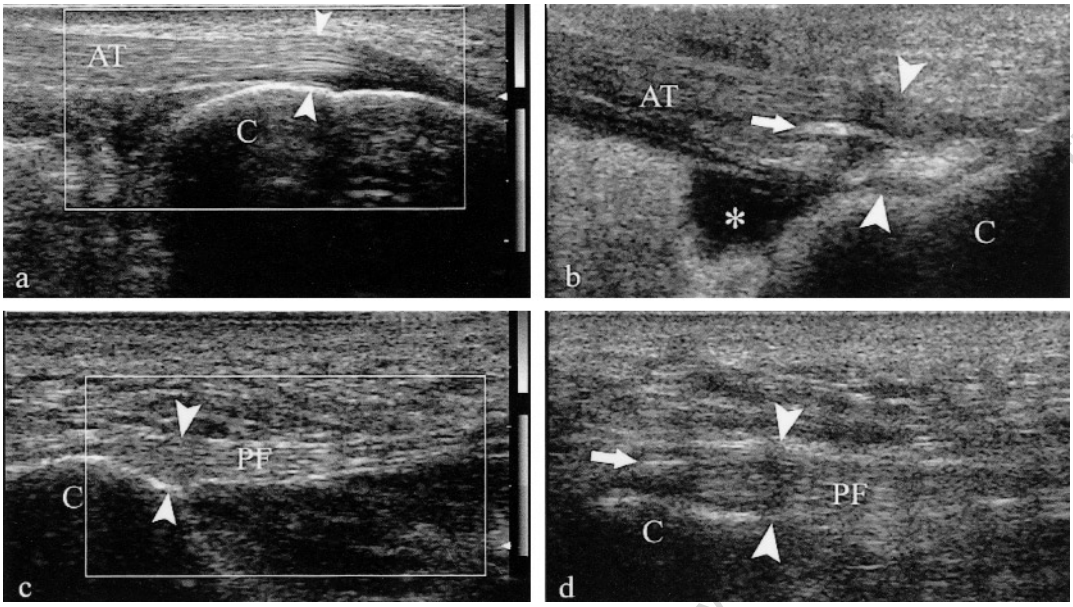

Figure 1. Site of measurement at the Achilles tendon and the plantar fascia insertional tract in normal (panels a and c) and pathologic (b, d) condition. Longitudinal scans with a 10–13 MHz linear transducer at the posterior aspect (a, b) and the inferior aspect (c, d) of the heel. AT: Achilles tendon, PF: plantar fascia, C: calcaneus. Panel a: Normal Achilles insertional tract. Antero-posterior diameter of the Achilles insertional tract is measured at the postero-superior calcaneal surface (between arrowheads). The normal fibrillar pattern of the tendon and the regular cortical bone surface are clearly visible. No vascular signal is observed within or near the tendon. Panel b: Achilles enthesopathy and retrocalcaneal bursitis in a patient with chondrocalcinosis. The Achilles insertional tract appears thickened (between arrowheads) and heterogeneously hypoechoic. A calcification (arrow) is evident within the distal portion of the tendon. The retrocalcaneal bursa is enlarged by fluid collection (asterisk). Panel c: Normal plantar fascia insertional tract. The thickness of the plantar fascia insertional tract is measured where it crosses the antero-inferior border of the calcaneus (between arrowheads). The normal fibrillar pattern of the plantar fascia and the regular cortical bone surface are clearly visible. No vascular signal is observed within or near the plantar fascia. Panel d: Plantar fasciitis in a patient with chondrocalcinosis. The plantar fascia insertional tract is thickened (between arrowheads) and heterogeneously hypoechoic. A thin linear calcification (arrow) is visible in the proximal portion of the plantar fascia insertion.

to the tendon fibrillar structure, apparently not in continuity with the bone profile[11,14-16]. Transverse scans showed hyperechoic single or multiple deposits within the anisotrophy of the tendon. An acoustic shadowing of these calcifications was generally more evident in the transverse scans.

The sonographic patterns of these calcifications were multiple thin linear bands in 72.4% of cases (42/58; Figure 2a, 2b), single fine linear bands in 17.2% (10/58; Figure 2c, 2d), and thick solid bands in 10.3% (6/58; Figure 2e, 2f). No homogenous rounded hyperechoic deposit was observed.

Calcifications of the plantar fascia appeared in all cases as a single fine linear echoic band located in the superficial region of the insertional tract of the fascia, apparently not in continuity with the cortical bone (Figure 2g, 2h).

US examination showed no significant difference in postero-inferior and inferior calcaneal enthesophytosis between cases with CC (59.6% and 61.4%, respectively) and with OA (46% and 44%). Such alterations were also present in controls (postero-inferior enthesophytosis in 24%, inferior enthesophytosis in 18%), although these percentages were significantly lower. Posterior and inferior calcaneal erosions were absent in all groups.

Achilles enthesopathy was found in 22.8% of patients with CC (14.9% heels), but it was absent in OA and in the controls ($p < 0.001$). PDS showed vascular signals in 11.4% of the Achilles insertional tracts of patients with CC.

Deep retrocalcaneal bursitis was found in 10.5% of patients with CC (7% heels), but it was absent in OA and in the controls ($p < 0.05$). PDS showed vascular signals in 5.2% of the deep retrocalcaneal bursae of patients with CC.

Plantar fasciitis was found in 40.3% of patients with CC (36% of heels) and in 14% of patients with OA ($p < 0.01$), but it was not found in any controls ($p < 0.0001$ CC vs controls, $p < 0.05$ OA vs controls). PDS showed vascular signals in 2.6% of the plantar fascia insertional tracts of patients with CC.

The presence of vascular signals on PDS was significantly associated with the presence of grey-scale US alterations of Achilles insertional tract ($p < 0.0001$), plantar fascia insertional tract ($p < 0.05$), and deep retrocalcaneal bursae ($p < 0.0001$).

Only 11 patients with CC (15 heels) had posterior talalgia and 6 patients (6 heels) had inferior talalgia.

Eleven patients with posterior talalgia had Achilles

Table 1. Results and statistics. Data are expressed as the number of subjects with the alteration studied (p) and as number of heels with the alteration studied (h); relative prevalence (%) is indicated in parentheses.

Clinical, Sonographic, and Radiographic Findings	CC (57 p, 114 h)	OA (50 p, 100 h)	Controls (50 p, 100 h)
Achilles tendon calcifications			
Clinical examination	NE	NE	NE
Sonographic examination	33 p (57.9)$^{****\#\#\#\#}$	0	0
	58 h (50.8)		
Radiographic examination	30 p (52.6)	0	0
	50 h (43.8)		
Postero-inferior enthesophytosis			
Clinical examination	NE	NE	NE
Sonographic examination	34 p (59.6)$^{\#\#\#\#}$	23 p (46)$^{\#\#}$	12 p (24)
	63 h (55.2)	45 h (45)	16 h (16)
Radiographic examination	33 p (57.8)	22 p (44)	12 p (24)
	62 h (54.3)	44 h (44)	16 h (16)
Achilles tendon enthesopathy			
Clinical examination	9 p (15.7)	1 p (2)	0
	11 h (9.6)	1 h (1)	
Sonographic examination	13 p (22.8)$^{***\#\#\#}$	0	0
	17 h (14.9)		
Radiographic examination	NE	NE	NE
Deep retrocalcaneal bursitis			
Clinical examination	4 p (7)	0	0
	4 h (3.5)		
Sonographic examination	6 p (10.5)$^{*\#}$	0	0
	8h (7)		
Radiographic examination	NE	NE	NE
Posterior erosions			
Clinical examination	NE	NE	NE
Sonographic examination	0	0	0
Radiographic examination	0	0	0
Plantar fascia calcifications			
Clinical examination	NE	NE	NE
Sonographic examination	9 p (15.8)$^{\#}$	1 p (2)	0
	18 h (15.8)	1 h (1)	
Radiographic examination	9 p (15.8)	1 p (2)	0
	17 h (14.9)	1 h (1)	
Inferior enthesophytosis			
Clinical examination	NE	NE	NE
Sonographic examination	35 p (61.4)$^{\#\#\#\#}$	22 p (44)$^{\#\#}$	9 p (18)
	70 h (61.4)	44 h (44)	12 h (12)
Radiographic examination	35 p (61.4)	21 p (42)	9 p (18)
	69 h (60.5)	42 h (42)	12 h (12)
Plantar fasciitis			
Clinical examination	6 p (10.5)	1 p (2)	0
	6 h (5.2)	1 h (1)	
Sonographic examination	23 p (40.3)$^{**\#\#\#\#}$	7 p (14) $^{\#}$	0
	41 h (35.9)	7 h (7)	
Radiographic examination	NE	NE	NE
Inferior erosions			
Clinical examination	NE	NE	NE
Sonographic examination	0	0	0
Radiographic examination	0	0	0
Vascular signal in Achilles tendon insertional tract on PDS	11 p (19.2)$^{**\#\#}$	0	0
	13 h (11.4)		
With talalgia	9 p (15.7) 11 h (9.6)	0	0
Vascular signal in deep retrocalcaneal bursa on PDS	6 p (10.5)	0	0
	6 h (5.2)		
With talalgia	4 p (7) 4 h (3.5)	0	0
Vascular signal in plantar fascia insertional tract on PDS	3 p (5.2)	0	0
	3 h (2.6)		
With talalgia	0	0	0

CC: chondrocalcinosis group, OA: osteoarthritis group, p: patients, h: heels, PDS: power Doppler sonography, NE: not evaluable. * $p < 0.05$ vs OA, ** $p < 0.01$ vs OA, *** $p < 0.001$ vs OA, **** $p < 0.0001$ vs OA, $^{\#}$ $p < 0.05$ vs controls, $^{\#\#}$ $p < 0.01$ vs controls, $^{\#\#\#}$ $p < 0.001$ vs controls, $^{\#\#\#\#}$ $p < 0.0001$ vs controls. Statistics are indicated only for the sonographic results.

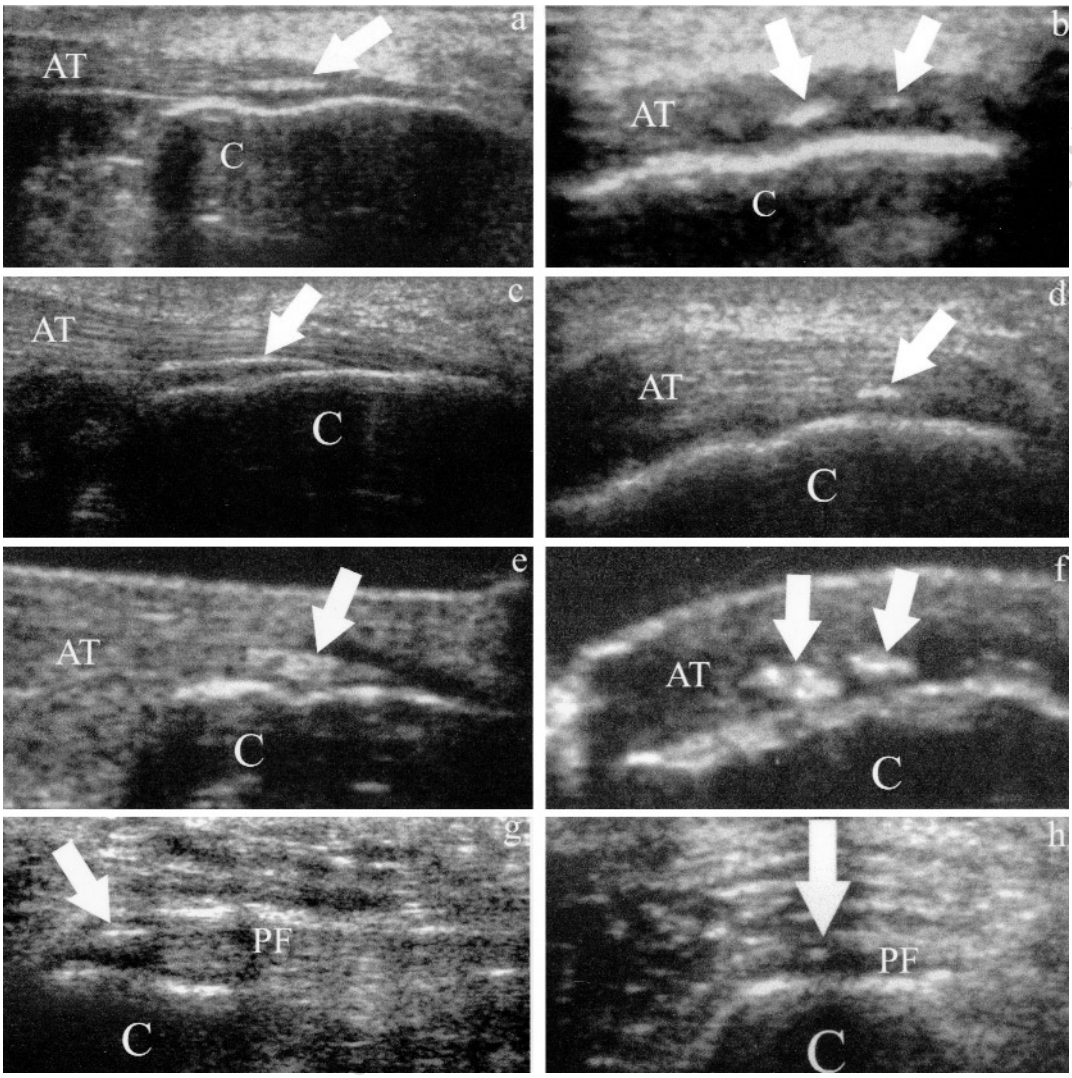

Figure 2. US patterns of tendon calcifications. Posterior (panels a–f) and inferior (g–h) sonograms of the heel of patients with chondrocalcinosis; 10–13 MHz linear probe. Calcific deposits are indicated by arrows. Longitudinal scans (along the major axis of the Achilles tendon and the plantar fascia) are on the left, transverse scans on the right. C: calcaneus, AT: Achilles tendon, PF: plantar fascia. Panels a and b: Multiple thin linear bands; 2 linear calcific deposits are visible in the transverse scan. Note that no definite acoustic shadowing is created by these calcifications, even in longitudinal scan. Panels c and d: Single fine linear band; a single long linear calcification, parallel to the tendon fibrillar structure, is notable particularly in longitudinal scan. Panels e and f: Thick solid bands; 2 thick calcifications, occupying almost all the distal portion of Achilles insertional tract but not in continuity with the cortical bone, create as minimal acoustic shadowing. Panels g and h: Single fine linear band; a linear calcification is present in the superficial portion of the plantar fascia. Mild acoustic shadowing is more evident in transverse scan.

enthesopathy with enthesophytosis and 4 patients with posterior talalgia had retrocalcaneal bursitis; the patterns of calcifications observed in these patients were single fine linear bands in 3 patients and thick solid bands in 2 patients.

PDS showed vascular signals (intratendinous or intrabursal) in all the patients with posterior talalgia (Figures 3 and 4).

All patients with inferior heel pain showed plantar fasciitis with enthesophytosis; however, in 2 cases thin linear calcifications of the plantar fascia were observed. PDS showed no vascular signal in any case of plantar fasciitis with talalgia.

No significant correlation was observed between talalgia and presence of calcifications.

A significant correlation was observed between talalgia and Achilles enthesopathy ($r = 0.78$, $p < 0.0001$), deep retrocalcaneal bursitis ($r = 0.7$, $p < 0.0001$), and plantar fasciitis ($r = 0.31$, $p < 0.001$).

A significant correlation between talalgia and vascular

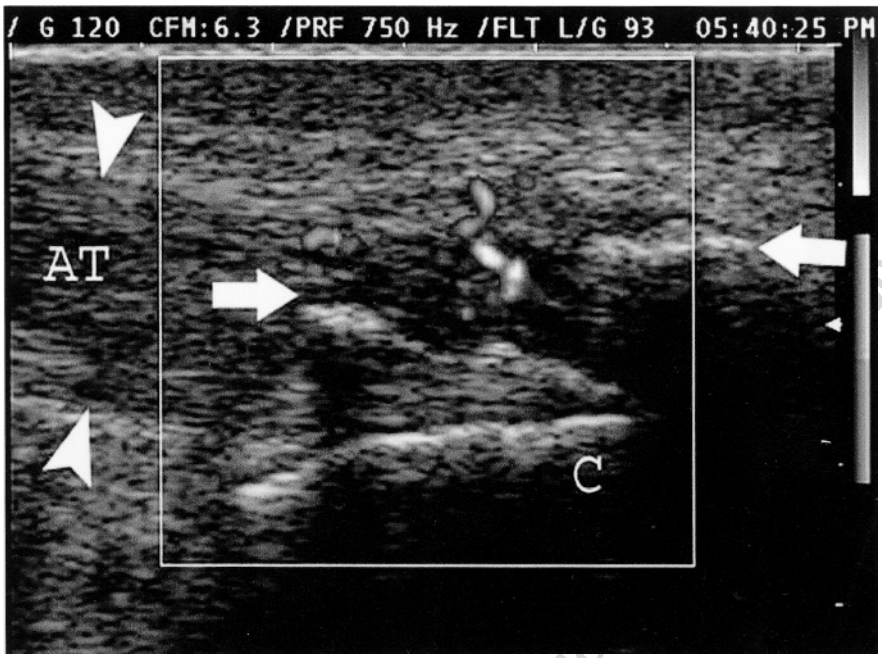

Figure 3. Posterior longitudinal sonogram of the heel of a patient with CC and talalgia; 10–13 MHz linear probe. US reveals thickening and hypoechogenicity of the Achilles insertional tract (between arrowheads), with a gross enthesophyte (arrow on the right) and a linear calcification (arrow on the left) with mild acoustic shadowing. PDS reveals a looping vessel in the midportion of the insertional tract.

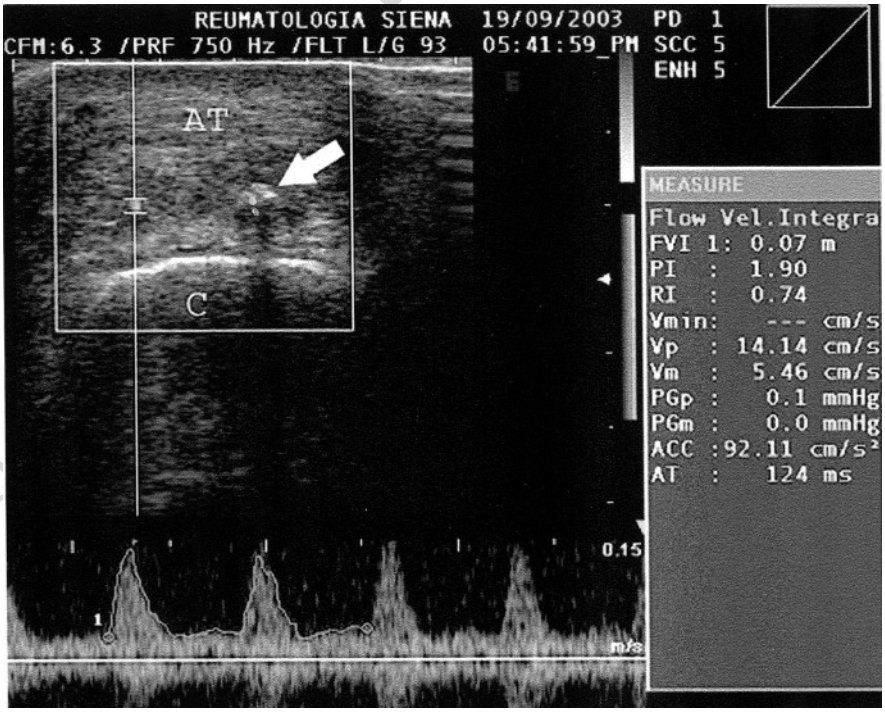

Figure 4. Posterior transverse sonogram of the heel of a patient with CC and talalgia (the same patient as in Figure 2); 10–13 MHz linear probe. Transverse scan shows lack of correlation between the calcification and intratendinous vascularization. Spectral wave analysis of the vessel indicates low values of resistance index (RI) and pulsatility index (PI) as reported in inflammatory vascularizations.

signals on PDS was observed in Achilles enthesopathy ($r = 0.91$, $p < 0.0001$) and deep retrocalcaneal bursitis ($r = 0.65$, $p < 0.0001$).

Achilles tendon calcifications were more frequent in men (16 patients) than in women (17 patients), but this difference was not significant. Plantar fascia calcifications were more frequent in women (5 patients) than in men (4 patients), but this difference was not significant.

Achilles tendon calcifications were found to be 39% sensitive, 100% specific, and 77% accurate for the presence of CPPD crystal deposition disease. Plantar fascia calcifications were 15% sensitive, 98% specific, and 54% accurate for the presence of CPPD crystal deposition disease (these values are valid for the comparison between CC and OA groups).

The kappa statistic results showed excellent agreement between US and radiography in detecting Achilles tendon calcifications ($k = 0.86$), plantar fascia calcifications ($k = 0.77$), postero-inferior enthesophytosis ($k = 0.90$), and inferior enthesophytosis ($k = 0.83$). Radiography failed to show 8 calcifications (over a total of 58 revealed with US); 2 of these were single fine linear calcifications and 6 were multiple thin linear calcifications. Radiography failed to show 4 plantar fascia calcifications (over a total of 18 revealed with US).

DISCUSSION

Tendon calcifications in CPPD crystal deposition disease have been described in previous radiological works. Gerster, *et al*[3] described fine linear calcifications in 13% of the Achilles tendons and in 0.5% of the plantar fascia of patients with articular CC, concluding that this radiographic pattern was a useful indirect sign of CPPD crystal deposition disease. Pereira, *et al*[9] described thin linear bands of calcification in 25% of Achilles tendons of patients with CC. Other investigators have described linear calcifications in tendons of patients with articular CC, specifically in gastrocnemius tendon (21–40%)[7-9], in hip adductor tendon (23.5%)[6], in quadriceps tendon (8–21%)[7,9], and in triceps tendon (14%)[9]. Gerster, *et al*[5] described 3 cases of Achilles tendinitis associated with linear calcification of CPPD as a rare presentation of extraarticular CC.

Some recent studies evaluated the role of ultrasonography in the detection of calcifications on hyaline cartilages of the knee[10-13] and on carpal fibrocartilages[11] with encouraging results. One recent work showed the usefulness of US to reveal signs of CC in elbow entheses with no radiographic evidence of CPPD crystal deposition disease[14]. To date our investigation is the first application of high frequency greyscale US and PDS to the study of Achilles tendon and plantar fascia in patients with CPPD crystal deposition disease.

Our results show Achilles tendon calcifications in 57.9% of patients with CC ($p < 0.0001$ vs OA and controls) and plantar fascia calcifications in 15.8% of CC (p not significant vs OA; $p < 0.05$ vs controls). Moreover, no significant difference in postero-inferior and inferior calcaneal enthesophytosis was observed between CC and OA. Such alterations were also present in control subjects (postero-inferior enthesophytosis in 24%, inferior enthesophytosis in 18%), even if these percentages were significantly lower.

These data confirm that calcaneal tendon calcifications are frequent findings in patients with CPPD crystal deposition disease; moreover, these calcifications have a high specificity for CPPD crystal deposition disease. The low sensitivity (39% for Achilles tendon calcifications; 15% for plantar fascia calcifications) suggests a moderate accuracy (77% and 54%, respectively).

The sonographic appearance of tendon calcifications in patients with CC was analogous to previous radiologic descriptions[5-9]; although we could not define the precise crystalline composition of the calcific foci within the insertional tract of the tendon, all the patients had definite diagnosis of CPPD crystal deposition disease, so we can assert that the calcification patterns observed in our study are closely related to this disease. Moreover, no patient with CC showed the calcific patterns described in other pathologic conditions. In particular, homogeneous rounded dense calcific deposits have often been described in hydroxyapatite crystal deposition disease[3,9,26], and small or gross calcific foci, not oriented along the fibrillar entheseal structure, were described in early and late heel enthesitis in the course of spondyloarthritis[15,21,26].

Another interesting observation is the lack of definite acoustic shadowing behind almost all the calcifications observed in CC patients. This phenomenon was also evident in the illustrations shown in other US investigations[10-13], but the matter was not discussed. We speculate that the scarce US attenuation is due to the particular (macroscopic and microscopic) structure of these CPPD calcifications. The histologic and crystallographic study by Gerster, *et al*[3] demonstrated that the microscopic crystals of CPPD are accumulated between tendon bundles without a preferential orientation or ordinate structure. By contrast, dystrophic tendinous calcifications and enthesophytes show a bony structure[27-29] — these lesions are clearly identified by US as a marked acoustic shadowing[15]. It is possible that the unstructured deposits of CPPD crystals allow ultrasound penetration. Moreover, the thin linear CPPD calcifications have small transverse diameter, and when it is less than 2 mm, the acoustic shadowing is often not apparent.

Thus, the calcific structure of the deposits observed in CC patients is confirmed by the coincidence with radiographic images. Our study shows excellent agreement between US and radiography in detecting calcifications and enthesophytosis. Radiography failed to show only 8 Achilles tendon calcifications (over a total of 58 revealed with US) and 4 plantar fascia calcifications (over a total of 18 revealed with US). This could be due to uncorrected radiographic imaging, but also to low density of the thin lin-

ear calcific deposits. It is unlikely that these linear deposits were really tendon scars, as they were observed in tendons with absent or minimal ultrasonographic signs of degeneration. Moreover, on US observation, these deposits showed echostructures analogous to those confirmed by radiography.

Inflammatory changes of the calcaneal soft tissues have been observed in CC, with significant differences with respect to OA. Achilles enthesopathy was found in 22.8% of cases of CC ($p < 0.001$), deep retrocalcaneal bursitis was found in 10.5% ($p < 0.05$), and plantar fasciitis was found in 40.3% of cases of CC and in 14% of those with OA ($p < 0.01$), but it was not found in any control subject ($p < 0.0001$ CC vs controls).

These data confirm that CPPD crystal deposition disease may also be associated with inflammatory changes in extraarticular soft tissues.

The presence of vascular signals on PDS was significantly associated with the presence of grey-scale US alterations of the Achilles insertional tract, plantar fascia insertional tract, and deep retrocalcaneal bursae.

However, PDS adds other information to grey-scale US observations. The intratendinous and peritendinous vascular signals observed in 11.4% of the Achilles tendons and 2.6% of the plantar fascia were not strictly related to the calcification (as shown in Figures 3 and 4), but they appear at tendinous sites where there are prevalent degenerative-reactive aspects (at the posterior-superior angle of calcaneus, where a deep retrocalcaneal bursa lies, at the distal portion of the insertional tract where traction forces contribute to the development of enthesophytes, or at the preinsertional region of the Achilles tendon). The absence of strict correlation between talalgia and presence of CPPD calcifications has also been observed in previous studies[3,5].

Moreover, the presence of vascular signal on PDS correlates with talalgia in the course of Achilles enthesopathy (Figures 3 and 4) and deep retrocalcaneal bursitis, but not in the course of plantar fasciitis. This could be due to the prevalence of degenerative changes in many cases of plantar fasciitis (demonstrated by hypoechogenicity and thickening of the plantar fascia insertional tract on US observation), and this could explain the large discrepancy between the US-diagnosed plantar fasciitis and the low prevalence of subcalcaneal talalgia and vascular signal on PDS in these cases.

A recent report[19] describes that vascularization of painful Achilles tendon was not always present, whereas in our study all the cases of painful Achilles enthesopathy (and 2 Achilles enthesopathies without talalgia) showed a vascular signal on PDS. Further studies are required to evaluate the sensitivity and the specificity of PDS in enthesopathy.

Our results show excellent agreement between radiography and ultrasonography and support the latter as a diagnostic tool for suspected cases of chondrocalcinosis, in the assessment of intraarticular and extraarticular calcifications. Moreover, US and PDS may yield immediate correlations between clinical symptoms and pathologic changes. We recommend the use of US and PDS in the initial evaluation of patients with heel pain and suspected chondrocalcinosis.

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