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Original article

Prevalence and distribution of cartilage damage at the metacarpal head level in rheumatoid arthritis and osteoarthritis: an ultrasound study

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Abstract

Objectives. To determine the prevalence and distribution of US-detected qualitative cartilage damage at metacarpal heads of patients with rheumatoid arthritis (RA) and hand osteoarthritis (OA).

Methods. Fifty-two RA patients and 34 patients with hand OA were enrolled. US examination of the metacarpal head cartilage from the II to V finger of both hands was performed. A total of 414 metacarpophalangeal (MCP) joints in RA and 266 MCP joints in OA patients were scanned with a linear probe up to 22 MHz. Qualitative assessments using a previously described scoring system for cartilage damage were performed. The prevalence and distribution of cartilage damage were analysed. Multivariate regression analysis was used to determine the predictive value of age, gender, body mass index (BMI), disease duration and the presence of rheumatoid factor and anti-CCP antibodies for US-detected cartilage damage.

Results. The metacarpal head cartilage was positive for cartilage damage in 35.7% (148/414) of MCP joints in RA and in 43.6% (116/266) of MCP joints in OA patients. In RA, the hyaline cartilage of the II and III metacarpal heads (bilaterally) was the most frequently affected. In OA, cartilage damage was more homogeneously distributed in all MCP joints. Multivariate regression analysis showed that age and disease duration, but not gender, BMI or autoantibody status, were independent predictors of US-detected cartilage damage in RA.

Conclusion. Cartilage damage was found in more than one third of the MCP joints in both RA and OA patients, and in RA patients, the II and III MCP joints were the most damaged.

Key words: rheumatoid arthritis, osteoarthritis, cartilage damage, ultrasonography

Key messages:

1. Ultrasound detects cartilage damage in more than one-third of the joints in RA and OA.

2. In RA patients, the second and third metacarpophalangeal joints are the most damaged.
3. Using a 22-MHz probe allows highly detailed direct visualization of the metacarpal head hyaline cartilage.

Introduction

Rheumatoid arthritis (RA) is a systemic inflammatory disease characterized by chronic and symmetric synovitis leading to progressive destruction of joint components [1]. Osteoarthritis (OA) is the most common joint disease and is one of the main causes of pain and disability in older adults [2]. Hyaline cartilage damage variably contributes to joint destruction, and the cartilage of the metacarpal head is a frequent target in both RA and hand OA [3-7].

Conventional radiography (CR) is the standard imaging modality for assessing and monitoring joint damage in RA and OA in daily rheumatologic practice. However, only indirect signs of cartilage damage can be depicted with CR. Therefore, CR may not be sensitive enough to detect early lesions. High-resolution ultrasound (US) with linear probes up to 22 MHz allows for direct and detailed visualization of the metacarpal head cartilage with a resolution power of 0.1 mm. To date, only a limited number of studies have been published with the aim of investigating the ability of US to assess cartilage damage in small joints of the hands in patients with RA and OA [4, 5, 8-10]. The main aim of the present study was to determine the prevalence and distribution of US-detected qualitative findings indicative of cartilage damage at metacarpal heads in patients with RA and hand OA. As a secondary aim, we compared US and CR semi-quantitative scores indicative of cartilage damage.

Methods

Patients

A total of 52 consecutive patients fulfilling the American College of Rheumatology (ACR) 1987 [11] and/or ACR/EULAR 2010 [12] classification criteria for RA and 34 patients fulfilling the ACR criteria for hand osteoarthritis [13] were enrolled in this study. Patients were recruited from the Department of Rheumatology of the Università Politecnica delle Marche at Jesi (Ancona/Italy) between the 5th of September, 2015 and the 15th of December, 2015 and from Department of

Pediatric and Adult Rheumatology, Motol University Hospital, Prague, Czech Republic between the 2nd of February, 2016 and the 30th of April, 2016. Patients younger than 18 years old or with a history of other concomitant arthropathies (i.e., crystal-related arthropathy) were excluded. Joints in which relevant deformation or previous trauma or surgery had occurred were not assessed. The study was performed according to the principles of the Declaration of Helsinki and good clinical practise. All patients gave their informed consent before entry into the study.

US image acquisition

All patients were scanned by a single rheumatologist (JH). US examinations were focused on the assessment of the hyaline cartilage of the metacarpal heads. The metacarpal heads of the second to fifth fingers of both hands were scanned from the radial to ulnar and from the proximal to distal sides to ensure maximal exploration of the hyaline cartilage. Each joint was scanned in both the longitudinal and transverse views. Hands were scanned with the MCP joints in maximal flexion to increase the extent of the cartilage detectable by US. Particular attention was paid to maintaining the probe at an angle of 90 degrees between the direction of the US beam and the cartilage surface. US examinations were performed using a MyLab Twice and MyLab ClassC (Esaote S.p.A. Genoa, Italy) equipped with a linear probe with a frequency ranging from 10 to 22 MHz.

US image interpretation

Healthy hyaline cartilage appears as a homogenously anechoic layer covering the bony cortex of the metacarpal head and is delimited by superficial and deep margins that characteristically appear as thin, sharp, continuous regular and hyperechoic linear interfaces [9]. Cartilage damage was evaluated using the semi-quantitative scoring system proposed by Disler and colleagues: 0=normal hyaline cartilage; 1=loss of the sharpness of the superficial margin of the hyaline cartilage; 2=partial thickness defect of the cartilage layer; 3=full thickness defect of the cartilage layer with a normal subchondral bone profile; 4=complete loss of the cartilage layer and subchondral bone involvement [14]. All US pathological

findings were documented with at least two perpendicular scans. Total scores at both the joint and patient levels were calculated.

Radiographic examination

CR of the hands (performed within the previous 6 months) was performed in the posteroanterior view in a total of 28 patients with RA and 19 with OA, and the films were assessed by an experienced musculoskeletal radiologist (MC) and a rheumatologist (FS) who were blinded to the clinical and US imaging data. Their inter-observer reproducibility was tested in a previous study, yielding a kappa value of 0.81 (data not published yet).

Joint space narrowing was scored at the level of the second to fifth MCP joints (bilaterally) using the van der Heijde scoring system (SvH) [15] and Simple Erosion Narrowing Score (SENS) [16] in RA patients and the Kallman score in OA patients [17].

Statistical analysis

The data are described as the mean and standard deviation (SD), unless stated otherwise. Basic descriptive statistics were computed for all variables, which were subsequently tested for a normal distribution using the Kolmogorov-Smirnov test. RA patients were divided in two subgroups according to disease duration: maximum of two years (early RA) and more than two years (established RA).

The bivariate relationship between the variables was assessed using Spearman's correlation coefficient. The agreement between the US and CR findings was calculated using non-weighted Cohen's kappa coefficients. The US and X-ray grades were matched according to the degree of cartilage damage. In RA patients, US grades of 0-1 were determined as reflective of SvH grade 0, US grade 2 as reflective of SvH grades 1-2, and US grades 3-4 as reflective of SvH 3-4. In OA patients, US grades 0-1 were determined as reflective of Kalmann grade 0, US grade 2 as reflective of Kalmann grade 1, and US grades 3-4 as reflective of Kalmann grade 2-3. Further, US grades 0-1 reflected SENS grade 0 and US grades 2-4 reflected SENS grade 1 (SENS grade 0 reflected Kallmann grade 0 as an expression of normal findings and SENS grade 1 reflected Kalmann grades 1-3 as an expression of pathological findings). The kappa coefficients were assessed

according to the convention suggested by Landis and Koch, who characterized values <0 as an indication of no agreement, 0–0.20 as having poor agreement, 0.21–0.40 as having fair agreement, 0.41–0.60 as having moderate agreement, 0.61–0.80 as having substantial agreement, and 0.81–1.0 as having excellent agreement [18]. The agreement percentages were calculated.

In addition, linear regression analysis was performed with the US sum score of cartilage damage as the dependent variable and with age, sex, body mass index (BMI), and disease duration in OA patients, and with age, sex, BMI, disease duration, rheumatoid factor (RF), and anti-citrullinated protein antibodies (anti-CCP antibodies) positivity in RA patients as the independent variables. Statistical significance was set at p values of less than 0.05. Statistical analysis was performed using SPSS version 23 statistical software (SPSS, Inc., Chicago, IL, USA).

Results

A total of 52 patients with RA (18 with early RA and 34 with established disease) and 34 patients with OA were consecutively enrolled in this study. Fourty-three RA patients were treated with conventional synthetic disease-modifying antirheumatic drugs (DMARDs) (36 with methotrexate, 3 with leflunomide, 4 with hydroxychloroquine), 21 RA patients received glucocorticoids and 21 RA patients were treated with biological DMARDs (2 with infliximab, 8 with adalimumab, 6 with golimumab and 5 with certolizumab). Twenty-one OA patients were treated with oral non-steroidal anti-inflammatory drugs (NSAIDs), 4 OA patients were treated with topical NSAIDs, 12 OA patients with paracetamol, 6 OA patients with minor opioids and 6 OA patients were treated with chondroitin sulfate. The patients' demographic and clinical characteristics are reported in Table 1.

The US examination of the metacarpal head cartilage from the second to fifth finger of both hands lasted a mean time of 6 minutes. Altogether, 414 MCP joints in RA patients and 266 MCP joints in OA patients were scanned. Eight joints were not recruited due to trauma, surgery or deformation.

Cartilage damage was found by US in a total of 148 (35.7%) out of the 414 MCP joints in RA and in 116 (43.6%) out of the 266 MCP joints in OA. In patients with RA, the least frequently damaged cartilage detected by US (grade 0) was observed

at the level of the IV and V left MCP joints. Conversely, hyaline cartilage in RA patients (grades 3 and 4) was mostly affected at the level of the right II metacarpal head followed by the left II metacarpal head and right III metacarpal head. The least affected areas were the V metacarpal heads bilaterally. In OA patients, the distribution of mostly damaged cartilage (expressed by grade 3) was more homogenous in all MCP joints (Figure 1). There was a tendency for a slightly higher prevalence of US-detected cartilage damage in the right hands, but the difference was not statistically significant. The detailed prevalence and distribution of different US scores for cartilage damage are reported in Table 2.

Relationship between US and CR findings

A significant positive correlation was found between the US total score and either the Sharp van der Heijde score or the SENS score ($r=0.591$, $p<0.001$; $r=0.544$, $p<0.001$, respectively) in patients with RA, and between the US total score and the Kallman score ($r=0.579$, $p<0.001$) in patients with OA (Table 3). The overall agreement between the US and CR findings in patients with RA was substantial ($\kappa=0.630$, $p<0.001$). Moderate overall agreement ($\kappa=0.468$, $p<0.001$) was found between the two imaging techniques in OA patients.

In patients with RA, total agreement between the US and the SENS scores was obtained in 172/220 (78.2%) RA joints: 122/220 (55.5%) joints were judged as normal by both imaging techniques and 50/220 (22.7%) as pathological by both techniques. A total of 39/220 (17.7%) joints were judged as normal with US but pathological with CR, and 9/220 (4.1%) were defined as normal with CR but pathological with US.

Total agreement between the US and the Sharp van der Heijde scores in patients with RA was obtained in 162/220 (73.6%) joints: 121/220 (55%) joints were assessed as normal and 41/220 (18.6%) joints were assessed as pathological by both imaging methods. Normal US but pathological X-ray findings were observed in 40/220 (18.2%) RA joints. Conversely, normal X-ray, but pathological US findings were found in 9/220 (4.1%) RA joints. Nine joints (4.1%) were assessed as pathological with both methods, but a different grade was assigned.

Figure 2 shows a representative set of US images of the metacarpal head cartilage from the second to fifth finger obtained using a dorsal longitudinal view and a corresponding CR picture acquired in the posteroanterior view in the same patient with RA.

In OA patients, total agreement between the two imaging techniques was obtained in 107/150 (71.3%) OA joints: 50/150 (33.3%) joints were judged as normal and 57/150 (38%) joints were judged as pathological by both imaging techniques. Normal US but pathological CR findings were observed in 35/150 (23.3%) OA joints. Conversely, normal CR but pathological US findings were found in 4/150 (2.7%) OA joints. Four joints (2.7%) were judged as pathological by both techniques, but a different grade was assigned by each.

Relationship between age, sex, disease duration and presence of auto-antibodies with US cartilage damage

In patients with RA, we found significant associations between US cartilage damage and age along with disease duration ($r=0.457$, $p<0.001$, $r=0.276$, $p<0.05$, respectively). Sex, BMI, RF positivity nor anti-CCP autoantibody positivity were not significantly correlated with US cartilage damage (Table 3). Multivariate regression analysis confirmed that even when controlling for effect of other variables in the model, age ($\beta=0.577$, $p<0.001$) and disease duration ($\beta=0.308$, $p=0.009$) (adjusted $R^2=0.425$, $F_{6,42}=6.9$, $p<0.001$), but not sex, BMI or autoantibody status contributed significantly to explain US cartilage damage as the dependent variable (Table 4).

In patients with OA, none of the variables were associated with US cartilage damage (Table 3). Also multivariate regression analysis confirmed, that this set of predictors is not related to US cartilage damage in our cohort of OA patients – see Table 4 (adjusted $R^2=0.046$, $F_{4,21}=1.3$, $p=0.303$).

Discussion

To our knowledge, this study is the first to provide US data regarding the prevalence and distribution of metacarpal head cartilage damage in patients with

RA and hand OA using a linear probe with a frequency reaching 22 MHz. While there is a consistent body of evidence supporting the value of US in the evaluation of synovitis and bony changes in RA, very little data is currently available in the literature on cartilage and its morphostructural abnormalities.

In the last two decades, several researchers have aimed to evaluate cartilage thickness using US in order to monitor the decrease in articular thickness in RA and OA [8, 19-22]. However, as shown by Torp-Pedersen *et al*. in 2010, in most of the studies the cartilage thickness was underestimated or overestimated due to incorrect measuring (not including the leading interface as part of the cartilage or by using oblique insonation of the cartilage) [23]. In 2012, US experts evaluated cartilage abnormalities in the MCP joints of OA patients (including the superior leading interface as a part of the cartilage), and they showed that US is a reliable tool for the assessment of cartilage damage in the small joints of the hand with very good inter- and intra-observer reliability [9]. More recently, Mandl *et al*. investigated the relationship between metacarpal cartilage thickness measured by US, CR measured as JSW and JSN evaluated by X-rays by the van der Heijde modified Sharp methods and anatomical measurements performed on cadaver specimens, and reported positive agreement between cartilage thickness values evaluated by all methods [5].

Nevertheless, as cartilage thickness may also vary in healthy individuals depending on weight, height and gender, [24-26] thinner cartilage does not necessarily indicate a pathological finding. Therefore, in our study, we have focused on the investigation of the prevalence and distribution of cartilage damage expressed by a US semi-quantitative scoring system defining 5 stages of damage as proposed by Disler [14] and used later by Filippucci et al. [4].

We have found that cartilage damage at different stages was more homogenously distributed in patients with OA rather than in patients with RA (Figure 1). This could possibly be explained by the fact that cartilage damage generally progresses more slowly in OA. However, we can hypothesize that once cartilage damage occurs in patients with RA it progresses more rapidly, probably as a result of aggressive inflammation.

Moreover, focusing our attention on the highest scores of cartilage damage (i.e., grades 3 and 4), we found that in RA the metacarpal heads of the second fingers

were most severely affected, followed by those of the third fingers. In OA patients, the distribution of the highest scores of cartilage damage was more homogenous in all MCP joints. This might potentially be explained by joint inflammation being more frequently present at the level of the second and third MCP joints rather than at the level of IV or V MCP joints in RA patients. Conversely, we suppose that in patients with hand OA, biomechanical stress and metabolic factors that cause cartilage damage are more homogeneously distributed among MCP joints. The cartilage of the right hand was slightly more affected than that of the left hand, again, probably on account of the more excessive mechanical stress of the right hands in right-handed participants in this study; however, this difference was not statistically significant.

Finally, we have investigated the relationship between cartilage damage detected by US and CR. We have correlated the CR findings with qualitative cartilage damage evaluated by a US scoring system. We have found a significant positive correlation between the US total score and either the Sharp van der Heijde score or the SENS score in patients with RA, and between the US total score and the Kallman score in patients with OA. Total agreement between the two imaging methods was obtained in 162/220 (73.6%) RA joints and in 107/150 (71.3%) OA joints.

Normal X-ray findings with pathological US findings were found in 9/220 (4.1%) RA joints and in 4/150 (2.7%) OA joints. This could be explained by the fact that CR provides only indirect pathological findings of cartilage damage and that US with a high-frequency probe has a much higher sensitivity for detecting even minimal morpho-structural changes of the cartilage layer than CR.

Conversely, pathologic X-ray findings and normal US findings were found in 40/220 (18.2%) joints in RA patients and in 35/150 (23.3%) joints in OA patients. Possible explanations include the fact that joint dislocation may not be associated with cartilage damage. Moreover, this disagreement might be explained by the main drawback of US, which is related to the fact that the width of the acoustic window may be reduced by the limited range of flexion of the MCP joint and the fact that US exploration of the cartilage surface may be insufficient at the lateral sides of joints if they are not approachable with a US probe. Finally, US cannot assess the hyaline cartilage at the base of the proximal phalanx. Since US is free of

radiation hazards, such a direct evidence of cartilage lesion can be evaluated overtime using a short-term follow-up schedule allowing an earlier detection of its fast progression, especially in RA patients with active disease.

However, there are also some limitations to our study. First, patients with RA and OA were not perfectly age-matched. However, since RA is more likely to affect young, fertile people, while elderly people are more likely to be affected by OA, this age difference was expected. Moreover, quite homogenous distribution of age in our cohort of OA patients probably contributed to its non-significant association with cartilage damage. Second, the disease duration was longer in patients with RA than those with OA in our cohort; however, it is important to note that cartilage alterations in OA usually develop asymptomatically during the first years of the disease. Therefore, we suggest that cartilage alterations probably appeared several years before diagnosis in the OA patients.

In conclusion, the present study provides detailed evidence regarding both the prevalence and distribution of cartilage damage in RA and OA patients at the MCP joint level. Cartilage damage was found in more than one third of the joints in both RA and OA patients, and in RA patients, the II and III MCP joints were the most damaged. Further investigation with a larger cohort of patients is required to confirm our data.

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Disclosure statement

J.H. has received speaking fees from Abbvie, MSD and Roche. E.F. has received speaking fees from Abbvie, Bristol-Myers Squibb, Celgene, Roche and UCB Pharma. F.S. has received speaking fees from AbbVie, Roche, MSD Italia, Eli-Lilly

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Figures

Fig 1. Prevalence and distribution of cartilage damage detected by ultrasonography.

Prevalence and distribution of different scores of US cartilage damage measured semi-quantitatively at the level of second to fifth metacarpophalangeal joints in patients with RA (A) and OA (B) and distribution of the highest scores of the cartilage damage in RA (C) and in OA (D).

Fig 2. US and CR findings of cartilage damage at the MCP level.
This mosaic allows one to perform a comparative pictorial analysis of US and CR findings of cartilage damage at the MCP level (second to fifth from right to left side) in a patient with RA. US images were obtained in longitudinal dorsal view.

Tables

Table 1 Baseline demographic and clinical characteristics of patients with rheumatoid arthritis and hand osteoarthritis

Characteristics	RA	OA

Number of patients	52	34
Gender, F/M	37 / 15	27 / 7
Age, years	58 ± 14.7	64.4 ± 10.8
Disease duration, years	9 ± 9	4.7 ± 3.7
BMI	23.8 ± 3.2	26.4 ± 3.1
CRP, mg/l	15 ± 18.7	
ESR, mm/first hour	24.6 ± 22.3	
DAS28-ESR	3.65 ± 1.3	
RF positivity, n (%)	34 (65.4%)	
Anti-CCP positivity, n (%)	30 (57.7%)	

The values are the mean ± SD, unless stated otherwise.

Anti-CCP, anticyclic citrullinated peptide antibody; BMI, body mass index; CRP, C-reactive protein; DAS28 score, Disease Activity Score for 28 joints with erythrocyte sedimentation rate; ESR, erythrocyte sedimentation rate; F, female; M, male; OA, Osteoarthritis; RA, Rheumatoid arthritis; RF, rheumatoid factor (latex fixation test)

Table 2 Prevalence and distribution of different US scores of cartilage damage measured semi-quantitatively at the level of second to fifth MCP joints in patients with RA and OA

RA (all patients)			grade 0		grade 1		grade 2		grade 3		grade 4	
MCP	II	R	34	65.40%	2	3.80%	4	7.60%	4	7.60%	8	15.10%
MCP	III	R	27	51.90%	1	1.90%	14	26.60%	4	7.60%	6	11.40%

MCP	IV	R	32	61.50%	0	0.00%	13	24.70%	2	3.80%	5	9.50%
MCP	V	R	33	66.00%	1	2.00%	10	19.70%	2	3.90%	4	7.90%
MCP	II	L	35	67.30%	0	0.00%	7	13.30%	2	3.80%	8	15.10%
MCP	III	L	31	59.60%	2	3.80%	12	22.80%	1	1.90%	6	11.40%
MCP	IV	L	37	71.20%	0	0.00%	8	15.20%	4	7.60%	3	5.70%
MCP	V	L	37	71.20%	0	0.00%	9	17.10%	1	1.90%	5	9.50%
total			266	64.30%	6	1.40%	77	18.60%	20	4.80%	45	10.90%
RA (early disease)			grade 0		grade 1		grade 2		grade 3		grade 4	
MCP	II	R	14	87.50%	0	0.00%	2	12.50%	0	0.00%	0	0.00%
MCP	III	R	10	62.50%	0	0.00%	6	37.50%	0	0.00%	0	0.00%
MCP	IV	R	10	62.50%	0	0.00%	6	37.50%	0	0.00%	0	0.00%
MCP	V	R	9	60.00%	1	6.70%	5	33.30%	0	0.00%	0	0.00%
MCP	II	L	14	87.50%	0	0.00%	2	12.50%	0	0.00%	0	0.00%
MCP	III	L	11	68.80%	1	6.30%	4	25.00%	0	0.00%	0	0.00%
MCP	IV	L	13	81.30%	0	0.00%	3	18.80%	0	000%	0	0.00%
MCP	V	L	13	81.30%	0	0.00%	3	18.80%	0	0.00%	0	0.00%
total			94	74.00%	2	1.60%	31	24.40%	0	0.00%	0	0.00%
RA (established disease)			grade 0		grade 1		grade 2		grade 3		grade 4	
MCP	II	R	20	55.60%	2	5.60%	2	5.60%	4	11.10%	8	22.20%
MCP	III	R	17	47.20%	1	2.80%	8	22.20%	4	11.10%	6	16.70%
MCP	IV	R	22	61.10%	0	0.00%	7	19,40%	2	5.60%	5	13.90%
MCP	V	R	24	68.60%	0	0.00%	5	14.30%	2	5.70%	4	11.40%
MCP	II	L	21	58.30%	0	0.00%	5	13.90%	2	5.60%	8	22.20%
MCP	III	L	20	55.60%	1	2.80%	8	22.20%	1	2.80%	6	16.70%
MCP	IV	L	24	66.70%	0	0.00%	5	13.90%	4	11.10%	3	8.30%
MCP	V	L	24	66.70%	0	0.00%	6	16.70%	1	2.80%	5	13.90%
total			172	59.90%	4	1.40%	46	16.00%	20	7.00%	45	15.70%
OA patients			grade 0		grade 1		grade 2		grade 3		grade 4	
MCP	II	R	19	57.60%	2	6.00%	10	29.70%	2	5.90%	0	0.00%
MCP	III	R	14	42.40%	0	0.00%	17	50.50%	2	5.90%	0	0.00%
MCP	IV	R	17	51.50%	3	8.90%	11	32.70%	2	5.90%	0	0.00%
MCP	V	R	22	66.70%	2	6.00%	8	23.80%	1	2.90%	0	0.00%
MCP	II	L	26	78.80%	0	0.00%	6	17.80%	1	2.90%	0	0.00%
MCP	III	L	12	36.40%	1	3.00%	19	56.50%	1	2.90%	0	0.00%
MCP	IV	L	15	44.10%	2	5.80%	16	46.40%	1	2.90%	0	0.00%
MCP	V	L	25	75.80%	1	3.00%	6	17.80%	1	2.90%	0	0.00%
total			150	56.60%	11	4.20%	93	35.10%	11	4.20%	0	0.00%

L, left; MCP, metacarpophalangeal joint; OA, osteoarthritis; R, right; RA, rheumatoid arthritis

Table 3 Correlations between US and CR findings of cartilage damage involvement

	US score	SvdH score	SENS score	Age	Disease duration	Height	Weight	BMI	RF+	anti-CCP+
RA patients										
All RA (N=52)										
US score	-----	0.562**	0.547**	0.457**	0.276*	0.245	0.326	0.175	0.098	0.086
SvdH score	0.562**	-----	0.964**	0.173	0.102	0.420*	0.420*	0.058	0.444*	0.153
SENS score	0.547**	0.964**	-----	0.100	0.012	0.411*	0.432*	0.067	0.446*	0.200
Early RA (N=18)										
US score	-----	0.876**	0.824**	0.081	0.311	0.311	0.534*	0.280	0.225	0.612
SvdH score	0.876**	-----	0.984**	0.155	0.435	0.491	0.636**	0.259	0.109	0.253
SENS score	0.824**	0.984**	-----	0.060	0.452	0.602	0.636**	0.137	0.146	0.219
Established RA (N=34)										
US score	-----	0.374	0.411	0.596**	0.216	0.191	0.214	0.099	0.207	0.114
SvdH score	0.374	-----	0.952**	0.191	0.130	0.254	0.325	0.150	0.620**	0.332
SENS score	0.411	0.952**	-----	0.166	0.045	0.212	0.374	0.200	0.623**	0.401

	US score	Kallman score	Age	Disease duration	Height	Weight	BMI
OA patients (N=34)							
US score	-----	0.576**	0.278	0.282	0.295	0.039	0.024
Kalmann score	0.576**	-----	0.063	0.097	0.160	0.159	0.212

**Correlation significant at the 0.01 level; *correlation significant at the 0.05 level.

anti-CCP, anti-citrullinated antibodies positivity; BMI, body mass index; OA, osteoarthritis; RA, rheumatoid arthritis; SENS, Simple Erosion Narrowing Score; SvdH score, Sharp van der Heijde score; RF, rheumatoid factor positivity; US, ultrasound score;

Table 4 Linear regression analysis predicting US cartilage damage in RA and OA

Model-RA	adjusted R2=0.425 $F_{6,42}$ = 6.9, p<0.001	Unstandardized Coefficients		Standardized Coefficients	95.0% Confidence Interval		Significance
		β	Std. Error	β	Lower Bound	Upper Bound	
	(Constant)	-4.005	8.963		-22.095	14.084	0.657
	Age	0.390	0.078	0.577	0.232	0.548	0.000*
	Gender	-3.260	2.540	-0.155	-8.385	1.865	0.206
	Disease duration	0.028	0.001	0.308	0.007	0.048	0.009*
	BMI	-0.569	0.362	-0.186	-1.299	0.161	0.123
	RF	3.871	3.122	0.190	-2.430	10.172	0.222
	anti-CCP	-2.584	2.989	-0.132	-8.616	3.447	0.392
Model-OA	adjusted R2=0.046 $F_{4,21}$ = 1.3, p=0.303	Unstandardized Coefficients		Standardized Coefficients	95.0% Confidence Interval		Significance
		β	Std. Error	β	Lower Bound	Upper Bound	
	(Constant)	-9.723	9.429		-29.331	9.885	0.314
	Age	0.147	0.099	0.330	-0.059	0.353	0.152
	Gender	-0.732	2.309	-0.071	-5.533	4.069	0.754
	Disease duration	0.230	0.309	0.150	-0.413	0.872	0.465
	BMI	0.021	0.022	0.190	-0.024	0.066	0.343

Dependent Variable: US cartilage damage

anti-CCP, anticyclic citrullinated peptide antibody; BMI, body mass index; RF, rheumatoid factor.