

ULTRASONOGRAPHIC STUDY OF THE PAINFUL SHOULDER IN PATIENTS WITH RHEUMATOID ARTHRITIS AND PATIENTS WITH DEGENERATIVE SHOULDER DISEASE

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Abstract

Objective: to compare ultrasound (US) changes of the painful shoulder between patients with rheumatoid arthritis (RA) and patients with degenerative shoulder disease (DSD).

Patients and Methods: Patients with painful shoulder (n=178) were divided according to clinical diagnosis made by rheumatologist: Group1-77 patients with RA, Group2-101 patients with DSD. US changes were evaluated by linear transducer 7.5 MHz for: long head biceps tendon-LHB, supraspinatus tendon-SSP, infraspinatus tendon-ISP, subscapularis tendon-SSC, subacromial/subdeltoid bursa-SA/SD-B, glenohumeral joint effusion-JE, bone cartilage-BC and humeral erosions-HE. The ultrasound examiner was blinded for clinical findings, diagnosis and patient identification.

Results: Frequent pathological changes were found in: SSP tendon (84.4% RA and 71.7% DSD), LHB tendon (81.8% RA and 69.3% DSD), ISP tendon (58.4% RA and 56.4% DSD) and SSC tendon (49.4% RA and 46.5% DSD) ($p=0.045$ $p=0.058$ $p=0.951$ and $p=0.710$ respectively). Evaluating changes separately, statistical differences were noted in: LHB tenosynovitis, SSP tendon rupture (three times more in RA patients), ISP tendon rupture (five times more in RA patients), as well as in glenohumeral JE, BC reduction and HE ($p=0.019$ $p=0.001$ $p=0.005$ $p=0.000$ $p=0.003$ and $p=0.007$, respectively). LHB tendon pathology (tendinopathy, subluxatio and rupture), SSC tendinopathy, global SSP and ISP tendinopathy as well as bursitis of SA/SD did not show statistical difference between the patient groups. Using logistic regression model, the following set of

items: glenohumeral JE, BC reduction and HE has shown to be distinctive between RA and DSD group.

Conclusion: Ultrasound detected different frequencies of LHB tenosynovitis, SSP and ISP tendon ruptures, glenohumeral JE, BC reduction and HE in RA and DSD patients comparisons. Combination of glenohumeral joint effusion, bone cartilage reduction and humeral erosions was able to identify patients with RA in a population of patients with painful shoulder disease with a moderately high degree of confidence.

Keywords: Musculoskeletal Ultrasound; Painful Shoulder; Rheumatoid Arthritis; Degenerative Shoulder Disease.

Introduction

Painful shoulder is one of the most common conditions in rheumatology. The pain in the shoulder may be caused by various intra and extra-articular pathology mechanisms. Clinical findings of the painful shoulder are similar or even equal for cases with different etiology. Could this issue be more precisely defined if we had a tool in our hands able to distinguish arthritic and non-arthritic pathology of the disease?

Many authors suggest that ultrasound imaging (US) is a good choice for the evaluation of shoulder pathology¹⁻⁵. Rheumatologists have focused on the US ability to detect and monitor joint-related articular and periarticular structural changes^{6,7}. Could inflammatory and non-inflammatory etiology of the painful shoulder syndrome be differentiated by a combination of the US pathology features?

Ultrasound has proved to be an effective, non-invasive, sensitive, reproducible, low-cost and readily available diagnostic tool. Traditionally it has

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been the tool of radiologists. However, there is a widespread increase in interest and use by rheumatologists^{8,9}. Musculoskeletal US is an instrument that potentially extends our clinical examination skills¹⁰. Some authors consider this tool as one of the most useful in the exploration of the shoulder and recommend US together with plain films as the first step examination¹¹. Some limitations of this method are subjectivity, long-term intensive training and different technical characteristics of the US device¹².

AIM of the study

To compare ultrasound changes of the painful shoulder in patients with rheumatoid arthritis and patients with degenerative shoulder diseases. To identify set of items able to make distinction between patients with rheumatoid arthritis and patients with degenerative shoulder diseases.

Patients and Methods

Patients

Outpatients with painful shoulder syndrome were consecutively enrolled from the Rheumatology Institute-Belgrade in a two-year period. A total number of 178 patients was divided into two groups on the basis of clinical diagnosis established by a clinical practice rheumatologist. Group 1 involved patients with rheumatoid arthritis (RA). Their diagnosis was established previously according to the American College of Rheumatology (ARA 1987) criteria. Group 2 involved patients with a first flare of shoulder pain clinically diagnosed as degenerative shoulder disease (DSD). Those patients did not have inflammatory rheumatic disease in their history.

Calculation of time for the duration of shoulder pain symptoms was made from the occurrence of the last exacerbation until the evaluation in our hospital. For that period of time selected RA patients didn't change their therapy with disease modified anti rheumatic drugs (DMARD), as well as with corticosteroids (CS). Both groups have been taking standard doses of one nonsteroidal anti-inflammatory drug (NSAID) or analgesics from the beginning of their symptoms.

Disease activity for RA patients was measured by disease activity score with 3 variables-DAS28(3)^{13,14}.

This score was calculated by standard formula, using the number of tender and swollen joints from a 28-joints count list and the Erythrocyte Sedimentation Rate (ESR). The patient's general health, or global disease activity usually measured on a Visual Analogue Scale (VAS) was excluded from the calculation formula, as the current pain could be caused not only by rheumatoid arthritis activity, but also by a painful shoulder syndrome. The DAS28(3) provides a number on a continuous scale indicating the current activity of the rheumatoid arthritis (Table I). Patients with high disease activity indicated with DAS28(3) above 5.1 were excluded, as well as patients with extraarticular manifestations of the rheumatoid arthritis disease.

Exclusion criteria for both groups were fracture or severe trauma of the shoulder. Patients who had comorbidity that might influence shoulder pain (cervical spine degeneration, neuropathy, chest tumor, cardiac pain) were also excluded.

Every patient gave the informed consent to be examined by ultrasound and to participate in the study. This study was approved by local Ethics committee.

Ultrasonography

This examination was performed by a single rheumatologist, experienced in musculoskeletal US examination for seven years. The ultrasound examiner was blinded for the clinical findings, diagnosis and patient identity. All patients were examined with commercially available equipment using a 7.5 MHz linear phased array transducer (Voluson 730 PRO, GE Medical Systems Kretz Ultrasound).

Pathological US findings were detected for long head biceps tendon-LHB, subscapularis tendon-SSC, supraspinatus tendon-SSP, infraspinatus tendon-ISP, subacromial/subdeltoid bursa-SA/SD-B, glenohumeral joint effusion-JE, bone cartilage-BC, and humeral erosions-HE.

Transverse and longitudinal planes were used for all of the following investigated structures. LHB and SSC tendons were examined on the anterior aspect of the shoulder, in the seating position, with the arm held in neutral position, the elbow flexed to 90°, and the forearm in a supinated position on the thigh¹⁵. SSP and ISP tendons were examined with laterally moved transducer, with the patient's shoulder in hyperextension and internal rotation. This position allows the maximal length of tendons to be visualized¹⁶. The SA/SD-B was imaged between the deltoid muscle and the SSP and ISP ten-

Table I. Baseline characteristics of the examined patients

Patient characteristics	Rheumatoid arthritis n=77	Degenerative shoulder disease n=101	p
Age (years), mean (SD)	60.87 (10.24)	61.09 (10.90)	0.651
Female (number), percent (%)	54 (70.1)	63 (62.37)	0.178
Symptom duration (years), mean (SD)	0.84 (0.27)	0.37 (0.16)	0.002**
Erythrocyte sedimentation rate (mm/hr), mean (SD)	24.28 (9.98)	19.7 (7.64)	0.114
Red blood cells (number $\times 10^{12}/L$), mean (SD)	3.48 (0.42)	4.29 (0.57)	0.000**
White blood cells (number $\times 10^9/L$), mean (SD)	5.87 (2.49)	7.84 (2.01)	0.001**
Platelets (number $\times 10^9/L$), mean (SD)	207.4 (66.4)	321.4 (73.7)	0.000**
Hemoglobine (g/L), mean (SD)	112.0 (22.2)	137.8 (15.9)	0.000**
RF positive (number), percent (%) [‡]	63 (81.8)	3 (3.0)	0.000**
ANA positive (number), percent (%) ^{‡*}	9 (11.7)	5 (5)	0.098
Tender joints count (number 0-28), mean (SD)	2.65 (0.98)		
Swollen joints count (number 0-28), mean (SD)	1.41 (0.98)		
VAS (mm), mean (SD) ^{‡‡}	6.30 (1.50)	6.43 (2.10)	0.739
DAS28/3 (number), mean (SD) ^{‡‡*}	3.73 (0.61)		
Diclofenac therapy (number), percent (%)	61 (79.22)	82 (81.19)	0.744
Glicocorticoid therapy (number), percent (%)	51 (66.23)		

[‡] RF – rheumatoid factor^{‡*} ANA – antinuclear antibodies^{‡‡} VAS – global disease activity measured on a Visual Analogue Scale of 100 mm^{‡‡*} DAS28/3 – disease activity score incorporating Erythrocyte sedimentation rate, Tender joints count and Swollen joints count

dons. For the visualization of the joint effusion, distance between joint capsule and inferior margin of ISP tendon above 2 mm was considered as positive sign. The humeral articular cartilage was seen between the SSP and ISP tendons and the humeral head.

Ultrasonography finding for every of the investigated feature was graded as 0 or 1 (absence or presence of the US pathological findings). Subdivision of pathological findings was conducted as recommended by Iagnocco et al¹⁷: for LHB tendon as tendinopathy (transversal diameter above 3 mm in women and 3,3 mm in men³), tenosynovitis (presence of fluid in tendon's sheath), subluxation from groove and tendon rupture; for SSP and ISP tendons as focal tendinopathy (focal hypertrophy above 5 mm-longitudinal scan, focal hypoechogenicity), global tendinopathy (global hypertrophy above 5 mm- longitudinal scan, uniform hypoechogenicity with/without small anechogenic areas as a sign of threatening partial rupture), complete tendon rupture (nonvisualisation of tendon) and presence of calcifications. Pathological findings of SSC tendon were characterized as tendinopathy (hypertrophy and hypoechogenicity), nonvisuali-

zation of tendon (established as complete tendon rupture) and presence of calcifications. Bursitis with or without synovial proliferation was established as distension of SA/SD-B above 2 mm with anechogenic or hypoechogenic fluid. Normal cartilage thickness was considered to be 2 mm. The visualized humeral head and great tuberosity were evaluated for bone erosions. Since only a part of humeral head cartilage is seen with sonography, some of the authors suggest the term tuberosity erosions to be used instead. Nevertheless, Bruyn and Naredo have described humeral head erosions in detecting destructive and inflammatory shoulder abnormalities⁷. Therefore, we have adopted the term humeral erosions (HE) for both humeral head and tuberosity erosions.

Statistical analysis

Differences between groups were evaluated by nonparametric Yates' corrected chi-square test considering pathological ultrasound findings. Chi square and Fisher's exact test were used for the calculation of the differences in US pathology subdivided for every of the investigated features. Independent samples T test was used in calculation of

Table II. Ultrasonography findings

Pathological Ultrasound (US) changes	Rheumatoid arthritis n=77		Degenerative shoulder disease n=101		Yates' corrected chi-square (2-tailed)
	No	%	No	%	p
Long head biceps tendon-LHB	63	81.8	70	69.3	0.058
Supraspinatus tendon-SSP	65	84.4	72	71.7	0.045*
Infraspinatus tendon-ISP	45	58.4	57	56.4	0.951
Subscapularis tendon-SSC	38	49.4	47	46.5	0.710
Subacromial/subdeltoid bursa-SA/SD-B	35	45.5	45	44.6	0.960
Glenohumeral joint effusion-JE	22	28.6	5	5.0	0.000**
Bone cartilage reduction-BC	23	29.9	12	11.9	0.003**
Humeral erosions-HE	12	15.6	4	4.0	0.007**

*p<0,05

**p<0,01

between-groups differences for continue variables. A probability (2-tailed) value less than 0.05 was considered to be statistically significant and less than 0.01 was considered to be highly statistically significant.

Logistic regression model was used to identify set of items that allows discrimination between RA and DSD groups. Logistic regression was used to test hypothesis about relationship between a categorical dichotomous outcome variable (inflammatory or non-inflammatory nature of a painful shoulder syndrome) and several categorical predictor variables (pathological US findings for LHB tendon, SSP tendon, ISP tendon, SSC tendon, SA/SD bursa, glenohumeral JE, BC reduction and HE). Goodness-of-fit test was used to assess the fit of a logistic model against actual outcomes.

All analyses were carried out using commercial statistical program SPSS 16.0.

Results

The two groups were homogeneous considering gender and age ($p=0.178$; $p=0.651$) (Table I). Average duration of painful shoulder symptoms was 9 months in RA and 4,5 months in DSD group ($p=0.002$). Average duration of RA disease was 7.29 ± 1.32 years.

In RA group mild anaemia was registered, which was of high significance comparing to DSD group ($p=0.000$) (Table I). The number of white blood cells and platelets has shown to be in normal stan-

dard values, although it was significantly different between the observed groups ($p=0.001$ and $p=0.000$ respectively).

Statistical difference between groups was observed with regard to rheumatoid factor test ($p=0.001$). There was no statistical difference considering antinuclear antibodies, erythrocyte sedimentation rate and Visual Analogue Scale (VAS) ($p=0.098$ $p=0.114$ and $p=0.739$ respectively) (Table I). Mean disease activity for RA patients measured by disease activity score with 3 variables-DAS28(3) was moderate (Table I).

All RA patients were treated with DMARD at the time of examination, doses unchanged from the symptom's onset. They were treated with: Methotrexate 65 patients (87.01%), Sulfasalazine 7 patients (9.09%) and Resochine 3 patients (3.90 %). Doses for Methotrexate were of 10.0 mg weekly (53 patients-81.54%), 12.5 mg weekly (7 patients-10.77%) and 15 mg weekly (5 patients-7.69%).

All patients were treated with NSAIDs, majority of them with Diclofenac in both groups (Table I). Other were treated with Naproxen (6 (7.79%) in RA group and 7 (6.93%) in DSD group), and with Paracetamol or other NSAID (10 (12.98%) from RA group and 12 (11.88%) from DSD group).

Fifty-one (66.23%) rheumatoid patients and none from the DSD group were treated with glucocorticoids per os (prednisone from 7,5 mg to 10 mg daily). None of the patients were treated with local glucocorticoid infiltrations.

The most frequent pathological US changes in the RA painful shoulder group, as well as in the

DSD painful shoulder group, were found in SSP tendon, LHB tendon and ISP tendon, followed by SSC tendon and SA/SD bursitis (Table II).

Comparing separately between groups every of the investigated feature, statistically significant borderline differentiation in RA and DSD groups was found in LHB tendon (Yates' corrected chi-square test 0.058) (Table II). Subdivision of the pathological findings showed that tenosynovitis of LHB was more frequently found in RA than in DSD group ($p=0.019$) while tendinopathy, subluxation and rupture were found almost equally in both groups of patients ($p=0.252$, $p=0.177$ and $p=0.685$ respectively) (Figure 1).

Furthermore, pathological changes of SSP tendon were of borderline significance (Yates' corrected chi-square test 0.045) (Table II). Differences were observed in SSP tendon rupture, which was

presented in RA patients 3 times more often than in DSD group ($p=0.001$) (Figure 2). Global tendinopathy was more frequently found in DSD than in RA patients, but with no statistical difference ($p=0.092$). Focal tendinopathy and calcifications in RA group did not differ from DSD group ($p=0.560$ and $p=0.256$ respectively) (Figure 2).

Pathological ultrasound findings of the ISP tendon were presented almost equally in the investigated groups (Table II). After stratifying of pathological changes, significant statistical difference was found between groups in rupture ($p=0.005$), but focal tendinopathy, global tendinopathy and calcification did not prove to be significant different ($p=0.149$, $p=0.572$ and $p=0.387$ respectively) (Figure 3).

Pathological ultrasound findings of SSC tendon were almost equally found in both groups of patients ($p=0.710$) (Table II), as well as SSC tendinopathy and calcification ($p=0.671$ and $p=0.585$ respectively) (Figure 4). Considering SA/SD-B there was no statistical significance between groups.

In addition, we have recorded glenohumeral joint effusion significantly more often in RA compared to DSD patients (Yates' corrected chi-square test $p=0.000$) (Table II). Further findings with statistical difference between groups were bone cartilage thickness reduction (Yates' corrected chi-square test 0.003). Humeral erosions were more frequently found in RA patients which was statistically proved with high significance, also ($p=0.007$) (Table II).

In the Logistic Regression Analysis, statistical significance of individual regression coefficients (i.e., β s) was tested using the Wald chi-square statistic (Table III). A combination of glenohumeral joint effusion-JE, bone cartilage reduction-BC and humeral erosions -HE has shown to be distinctive between RA and DSD patients. Those were significant predictors of inflammatory nature of the painful shoulder syndrome ($p<0.05$). The inferential goodness-of-fit test was the Hosmer-Lemeshow test (Table III), that yielded a χ^2 (8) of 2.380

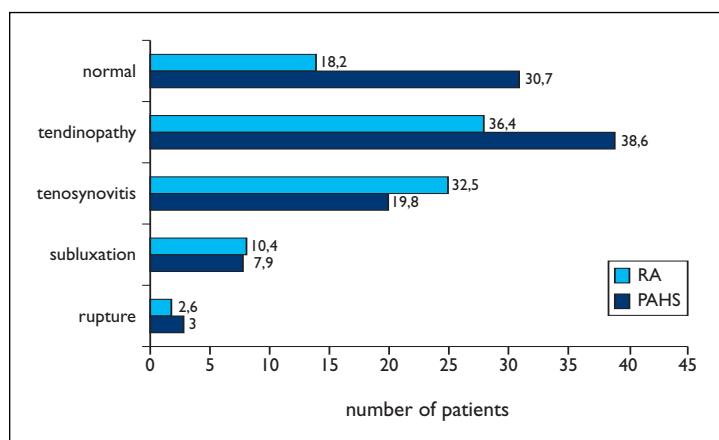


Figure 1. Ultrasound findings of LHB tendon (Number behind the bar represents percentage of patients)

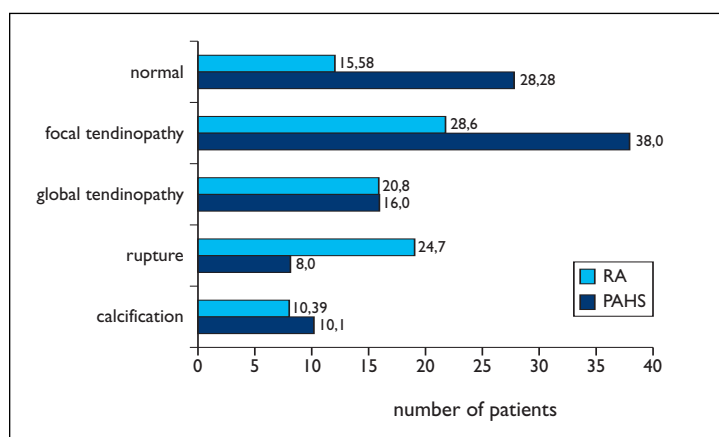


Figure 2. Ultrasound findings of SSP tendon (Number behind the bar represents percentage of patients)

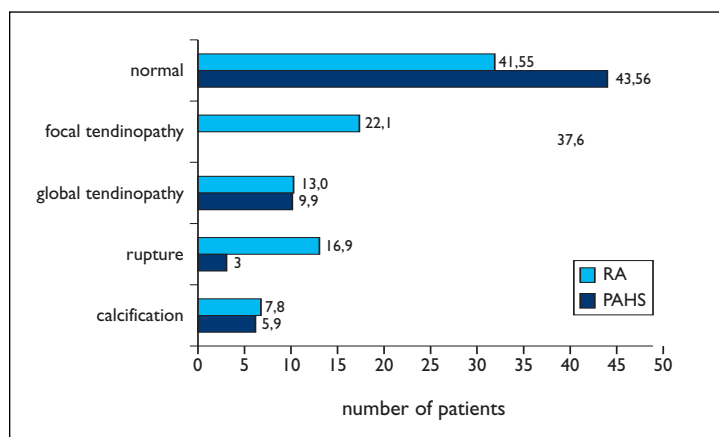


Figure 3. Ultrasound findings of ISP tendon
(Number behind the bar represents percentage of patients)

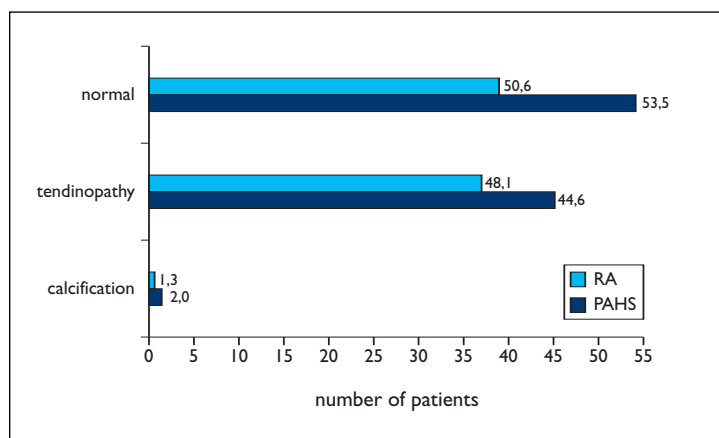


Figure 4. Ultrasound findings of SSC tendon
(Number behind the bar represents percentage of patients)

and was not significant ($p=0.967$), suggesting that the model was well fit to the data. Two additional descriptive measures of goodness-of-fit presented in Table 3 are R^2 indices, defined by Cox and Snell and Nagelkerke. The percent concordant was found to be 71.2%.

Discussion

Painful shoulder complains are often seen in daily rheumatology practice. Several pathological entities have similar clinical picture. Clinical assessment and conventional radiography, currently the most widely used approaches, showed low accuracy as lesion diagnostic tools¹⁸. They usually detect changes after the destructive structural pro-

cess of the shoulder has been occurred. Magnetic resonance imaging (MRI) is an effective technique for assessing shoulder joint, but MRI is an expensive method, with limited availability. Besides that, most of the patients with shoulder pain prefer sonography to MRI¹⁹.

Ultrasound has a number of advantages, such as lack of irradiation, wide availability, dynamic approach, speed, reproducibility and low cost^{11,20}.

US enables quite accurate evaluation of changes in patients with painful shoulder. The question is if the US findings can differentiate different primary pathologies (inflammatory arthropathies vs non-inflammatory).

Mean duration of the painful shoulder syndrome was found to be highly significant different between groups. Some of the main reasons why our patients with RA were not examined earlier were: high level of pain bearing, belief that pain could pass without any treatment, belief that they could be self-cured with NSAID and the expected long waiting list for rheumatologic examination.

Patients with RA were selected on the basis of their disease activity score (DAS28/3), which enrolled them into either a low-activity or a mild-activity disease group. This enabled us

to presume that shoulder pain was not caused by the actual flare of rheumatoid arthritis itself. As we stated before this presumption could not be assumed for a high disease activity group and due to that, those patients were excluded from evaluation.

The most frequent US pathological changes for the RA painful shoulder group were found in SSP tendon (65%), LHB tendon (63%) and ISP tendon (45%). Investigating painful rheumatoid shoulder, Alasaarela and Alasaarela found biceps tendinitis (57%) and changes in the supraspinatus tendon (33%) to be the most frequent tendinopathy changes²². LHB and SSP tendon lesions were also the most common findings of the rheumatic shoulder in the work of Keysser and Osthus²⁵. In our DSD painful shoulder group, frequent pathological

Table III. Logistic Regression Analysis of ultrasound examination of 178 patients with shoulder pain in regard to clinical diagnosis

Statistical tests of individual predictors	β	SE β	Wald χ^2	df	p	e β (odds ratio)
Constant	1.184	0.448	6.990	1	0.008	3.266
US LHB	-0.499	0.418	1.424	1	0.233	0.607
US SSP	-0.786	0.427	3.397	1	0.065	0.456
US ISP	0.753	0.394	3.645	1	0.056	2.123
US SSC	0.104	0.375	0.078	1	0.781	1.110
US SA/SD-B	0.311	0.327	0.907	1	0.341	1.365
US JE	-1.278	0.509	6.306	1	0.012*	0.279
US BC	-0.980	0.442	4.916	1	0.027*	0.375
US HE	-1.627	0.706	5.305	1	0.021*	0.197
Overall model evaluation			χ^2	df	p	
Hosmer&Lemeshow test			2.380	8	0.967	

*p<0,05

Cox and Snell $R^2 = 0.167$ Nagelkerke R^2 (Max rescaled R^2) = 0.223

changes were found in SSP tendon (71.7%), LHB tendon (69,3%) and ISP tendon (56.4%). SSP and LHB tendon lesions were also the most common pathological findings of non-inflammatory painful shoulder syndrome in other studies^{15, 23, 24}.

Tenosynovitis of LHB, as well as SSP and ISP tendon ruptures in the present study were significantly more often observed in RA than in DSD patients. The long-standing inflammatory process has probably played the leading role.

Minimal fluid accumulation in the subacromial/subdeltoid bursa is able to be detected by US examination. Subacromial/subdeltoid bursitis was registered in 45.5% of our RA patients, and in 44.6% of DSD patients, with no statistical difference. In the previously published reports, for RA patients bursitis was found either in lower percentage versus ours-from 14.3% to 30%^{23, 26, 27}, or in higher percentage versus ours-from 58% to even 69%^{22, 28}. Other studies of DSD patients found bursitis in 29%-45%^{18, 29}. Our data noted glenohumeral joint effusion in 28.6% of RA patients and only in 5.0% of DSD patients. Other reported studies described this finding in 15.6% to 58% of RA patients^{22, 23, 26, 27, 30} and in 8% to 29% of DSD patients^{24, 31}.

In spite of some dilemmas, several investigators have emphasized the ability of ultrasound in detecting erosions of the humeroscapular joint⁷ with higher sensitivity than radiography^{32, 33}. As we have discussed earlier, the problem is the inaccessibility

and poor visualization of the whole humeral head by ultrasound. Because of that fact, the absence of humeral head erosions should be considered with some reserve.

Williams et al. found that humeral cysts are not related to rotator cuff tear or aging²⁹. In the present study, humeral erosions were found four times more in RA (12%) patients. In other studies, humeral erosions in RA patients were found more frequently, from 20% upon 70%^{34, 35}. The possible reason why humeral erosions appear less in our RA group could lie in good RA disease control, which was due to the inclusion criteria.

According to our knowledge, the set of US painful shoulder pathological features able to distinguish RA and DSD patients was not tested before. In our Logistic Regression Analysis, combination of glenohumeral joint effusion, bone cartilage reduction and humeral erosions was significant predictor of inflammatory nature of the painful shoulder syndrome.

As for the logistic regression, binomial distribution of the dichotomous outcome was robust as long as the sample is random; thus, observations were considered to be independent from each other^{34, 35}.

In terms of the adequacy of sample size, the literature has not offered specific rules applicable to logistic regression³⁵. Sample size calculation for our logistic regression model was based on the work of

Peduzzi et al.³⁶. Using the calculation formula $N = 10k / p$, where k is the number of covariates (independent variables) and p is the smallest number of the proportion of positive cases, the results reported in this study have been considered stable.

Goodness-of-fit statistics showed a good fit of a presented logistic model to the data (Table III). According to classification table, our model correctly predicted 71,2% of the cases at a cut-off value of $p=0.50$. Considering the general fact that percentage of concordant pairs above 75% was more impressive^{34,35}, our predictive accuracy of the logistic regression model could be regarded as moderate.

Limitations of the study

The first limitation of the study was the sample selection. Firstly, the nature of the disease was well-established (disease duration for RA patients was 7 years). This was the case because all patients were recruited from the tertiary health care center. That's why it is not possible to apply the results of the present study to recent-onset RA cases. Further studies are needed to test ultrasound differences for early RA cases, when the achievement of the accurate diagnosis is the most important. Secondly, investigated groups have considerably different duration in the symptom's onset.

The second limitation of this study was ethics, as we were not able to collect the informed consent for all US-identified joint effusion cases in order to perform a joint fluid aspiration. This was the case for the glenohumeral joint effusion as well as for the subacromial/subdeltoid bursa. Thus, the achieved US changes of the synovial fluid which had indicated inflammation were not confirmed by arthroscopy, evacuation and laboratory assessment. Furthermore, the third limitation of the study was economic, thus US findings were not confirmed by MRI or computed tomography (CT). On the other hand, more appropriate imaging tool, the power Doppler ultrasound (PD-US), was not applied because of an absence of standardization with this method⁷. The fourth limitation lies on the ultrasound examination by itself. Since it was performed by a single ultrasound examiner-rheumatologist, there was dependence on personal interpretation of the image, although he was blinded to clinical findings, diagnosis and patient identity.

General limitation of the study was the dependence on the technical characteristics of the US device³⁷.

Conclusions

In the investigated population of patients with painful shoulder and comparing RA and DSD patients, differences were found in tenosynovitis of long head biceps tendon, rupture of supraspinatus and infraspinatus tendons, glenohumeral joint effusion, bone cartilage reduction and humeral erosions.

We want to highlight that the following combination of pathology features: glenohumeral joint effusion, bone cartilage reduction and humeral erosions imaged by ultrasound is able, with a moderately high degree of confidence, to identify patients with established RA in a group of patients with painful shoulder syndrome.

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