Shoulder Involvement in Rheumatic Diseases. Sonographic Findings

GIULIO COARI, FRANCO PAOLETTI, and ANNAMARIA IAGNOCCO

ABSTRACT. ***Objective.*** To distinguish using shoulder sonography the different changes present in rheumatoid arthritis (RA), polymyalgia rheumatica (PMR), and periarticular disorders (PD) of soft tissue of the shoulder.

Methods. Ninety shoulders of patients with RA, 32 with PMR, 122 with PD, and 108 controls were studied sonographically, using a 7.5 MHz linear probe. The following structures were evaluated: long head of biceps tendon, supraspinatus, infraspinatus and subscapularis tendons, subacromial and subscapularis bursae, rotator cuff (thickness), calcifications, and glenohumeral and acromioclavicular joints. Statistical analysis was by Student's t test and chi-squared test.

Results. Involvement of long head of biceps tendon (peritendinous fluid collection, changes of thickness, and/or echotexture) was significantly different between RA and PMR and between PD and PMR. Alterations in thickness and/or fibrillar pattern were evaluated in rotator cuff tendons: supraspinatus tendon was involved with significant differences between PD and both RA and PMR; the changes of subscapularis tendon were present, with significant differences between PD and both the other groups; the alterations of infraspinatus tendon were not statistically different between the 3 groups. Effusion within bursae was present, with significant differences only between RA and PD. The mean thickness of rotator cuff was significantly different between controls (6.2 mm) and both PD (5.3 mm) and RA (5.8 mm), and between PMR (6 mm) and PD. Evaluation of effusion within the glenohumeral joint (capsule–bone distance) showed significant differences between controls (2.4 mm) and both RA (4.2 mm) and PMR (4 mm), between RA and PD (2.6 mm), and between PMR and PD. Calcifications were present only in PD (21.3%) and RA (6.7%), with significant differences. Effusion within the acromioclavicular joint was present in RA (35.5%) and PD (20.5%), with significant differences.

Conclusion. Shoulder sonography showed involvement of all structures in RA, the prevalence of effusion in PMR, and involvement mainly of tendons in PD. (J Rheumatol 1999;26:668–73)

Key Indexing Terms:
SHOULDER RHEUMATIC DISEASES SONOGRAPHY

Shoulder pain may be related to many different disorders[1]. Both articular structures and periarticular soft tissues may be involved with very similar symptoms. For this reason it is often difficult to detect and identify the site of anatomic alterations with clinical examination. Moreover the anatomy of the shoulder girdle is very complex and depends on anatomic integrity of muscles, tendons, and joints[2]. Important structures for articular motility and stability are the rotator cuff tendons (supraspinatus, infraspinatus, subscapularis, teres minor) and the tendon of the long head of biceps. Among the bursae, the most important are the subacromial bursa and subscapularis bursa. The complex anatomy of the shoulder girdle makes it difficult to identify the site of changes when pain and limited motion are present and to assess the type of alterations clinically. In contrast, sonographic studies of soft tissues have successfully evaluated changes of both articular and periarticular structures[3-16]. The noninvasiveness and sensitivity of ultrasonographic analysis and its relatively low cost with respect to other diagnostic techniques (computed tomography, magnetic resonance imaging), make it suitable for patient followup and monitoring of therapy. Moreover the limited availability of the above imaging methods limits their routine use, while sonography is a useful and increasingly available technique for examination of soft tissues.

Our aim was to identify and differentiate the type of changes present in the shoulder in 3 rheumatic diseases with different pathogenesis: rheumatoid arthritis (RA), polymyalgia rheumatica (PMR), and periarticular disorders (PD) of soft tissues of the shoulder, such as rotator cuff or bicipital tendinitis, tendon tears, bursitis, capsulitis.

RA, a chronic inflammatory disease, whose diagnosis is based on internationally accepted criteria, such as those of the American Rheumatism Association[17], is characterized by erosive synovitis with formation of synovial pannus and consequent destruction of both articular and periarticular tissues[18]. Among articular features of RA, the involvement

From the Department of Rheumatology, University of Rome La Sapienza, Rome, Italy.

G. Coari, MD; F. Paoletti, MD; A. Iagnocco, MD.

Address reprint requests to Dr. G. Coari, Istituto di Reumatologia, Università di Roma La Sapienza, v.le del Policlinico 151, 00161 Roma, Italy.

Submitted May 20, 1998 revision accepted August 7, 1998.

of the shoulder varies from 33 to 75%[19], but it usually appears in those cases with progressive and severe disease[20]. The involvement of periarticular soft tissues causes bursitis, lesions of the long head of biceps tendon, and inflammation and possible destruction of the rotator cuff tendons.

PMR is a clinical syndrome characterized by inflammatory involvement of the shoulder and pelvic girdles, with pain and stiffness of at least 4 weeks' duration[21]. Proposed diagnostic criteria include elevated erythrocyte sedimentation rate (ESR) > 40 mm[22]. The disorder is very closely related to giant cell arteritis[22], and vasculitis is the sole distinguishing feature, found in giant cell arteritis but never in PMR.

The spectrum of PD of the shoulder is commonly characterized by joint pain and restriction of motion. PD are multiform, with possible involvement of many different structures, with the appearance of rotator cuff or bicipital tendinitis, rotator cuff tears, calcific tendinitis, bicipital subluxation or rupture, bursitis, adhesive capsulitis, and frozen shoulder. Because of poor accessibility of the shoulder to clinical examination the precise localization of alterations of those structures is often difficult[2].

Many studies evaluated the involvement either of the shoulder or of single structures in various disorders[2–16,23–26] but differences between the 3 diseases have never been reported.

MATERIALS AND METHODS

Ninety shoulders in 71 patients with RA (19 with bilateral and 52 with monolateral clinical involvement), 32 joints of 16 subjects with PMR, and 122 articulations of the same number of patients with monolateral PD were studied. Diagnosis was confirmed according to recent international diagnostic criteria[17,22]. In all patients the quality of work was light or moderate. Of subjects with RA 50 were female and 21 male; their mean age was 59.9 years (range 21–73) and mean disease duration was 5.5 years (range 0.9–16). Rheumatoid factor was present in 50 cases (70.4%), ESR (mean 48.3 mm; range 33–67) was always elevated, and C-reactive protein (CRP) (mean 52.6 mg/dl; range 36–89) was positive. Radiographs showed juxtaarticular osteoporosis in 17 joints (18.8%), erosions in 28 (31.1%), and calcifications in 7 (7.7%). Sixty-four patients (90.1%) were treated with disease modifying drugs, 43 (60.6%) with corticosteroids, and 68 (95.7%) with nonsteroidal antiinflammatory drugs (NSAID).

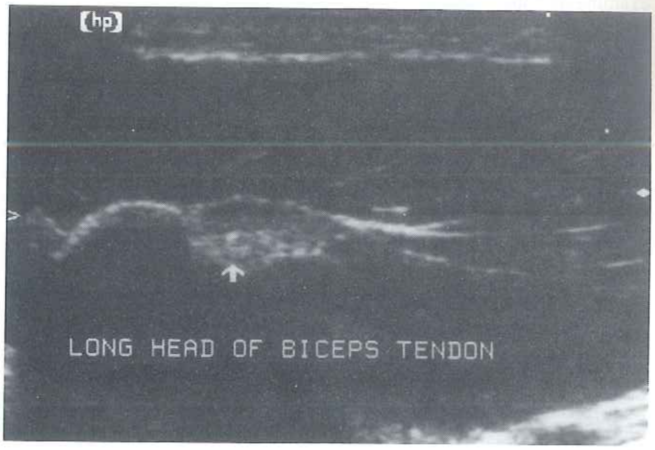
Of patients with PMR 12 were female and 4 male; their mean age was 67.5 years (range 58–76) and mean disease duration was 0.5 years (range 0.2–1.9). ESR was elevated (mean value 56.3 mm; range 51–74); CRP was positive in 12 cases (75%) and the mean value in the entire group was 74.1 mg/dl (range 2–110). Radiographs showed no changes. All patients were treated with oral low dose corticosteroids.

Of subjects with PD 77 were female and 45 male; their mean age was 45.6 years (range 35–61) and the mean disease duration was 0.2 years (range 0.1–0.8). All cases had symptoms of pain and limitation of motion. Shoulder involvement was confirmed by clinical evaluation and no changes of laboratory investigations were found. Radiographs showed the presence of calcifications in 31 shoulders (25.4%). Eighty-one patients (66.4%) were treated with NSAID.

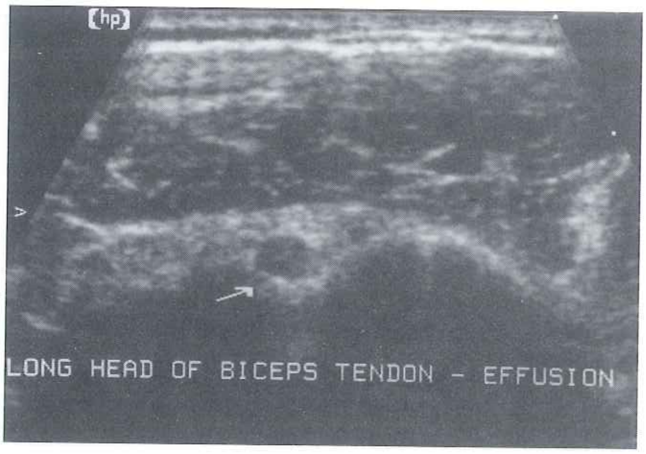
The shoulders of 54 healthy control subjects were examined; 31 were female and 23 male; their mean age was 41.7 years (range 26–57).

The presence of any signs of any other rheumatic diseases with involvement of the shoulder was an exclusion criterion for the study. Sonography was performed by 2 different operators (both rheumatologists) with a 7.5 MHz linear transducer. In all cases investigations included long head of biceps tendon, 3 rotator cuff tendons (supraspinatus, infraspinatus, subscapularis), and subacromial and subscapularis bursae; measurement of total rotator cuff thickness and identification of calcifications and effusions of glenohumeral and acromioclavicular joints. The 4th tendon of rotator cuff (teres minor) was not examined due to frequent difficulties in its sonographic assessment. Evaluation of tendons and bursae and measurement of rotator cuff thickness were by a combination of reported techniques[7,8,23,24]. The mean values of results from measurements of the rotator cuff thickness were calculated in all the 4 groups examined (healthy subjects, RA, PMR, and PD patients). Calcifications were identified by multiple scanning around the shoulder girdle. Effusion of glenohumeral joint was evaluated by transaxillar scan measuring the longest distance between the bony surface of the humerus and the joint capsule, according to Koski's technique[25,26]; then the mean values of the results obtained in the normal shoulder and in the 3 different groups of pathologic joints were calculated. Acromioclavicular joint was examined by longitudinal scanning of the joint, with the shoulder in neutral position; effusion was revealed when the capsule stood convex to the articular space with simultaneous appearance of hypoechoic area within the joint.

Sonographic assessment of long head of biceps tendon was obtained based on the appearance of peritendinous fluid collection (Figure 1), evaluating the thickness of the tendon and studying its fibrillar pattern. The presence of a hypoechoic or sonolucent area around the tendon and within its sheath indicated the presence of biceps tenosynovitis[10,12]. With regard to tendon thickness, thinning was interpreted as a partial rupture of its fibers,



A



B

Figure 1. Transverse scan of long head of biceps tendon: (A) normal tendon; (B) peritendinous fluid collection and tendon thinning.

as well as the presence of a hypoechoic area[4–6,10,11,13] within the tendon fibrillar structure (tear); thickening was considered a sign of degenerative process. The absence of the tendon either in its normal site or in surrounding areas was interpreted as a sign of total rupture[10].

Variations in thickness were evaluated with respect to contralateral tendon. When bilateral involvement of the shoulder was present comparison was made with respect to tendons of healthy subjects. Changes were considered significant when the variation was at least 1/3 of the normal thickness of the tendon[12]. Tendon structure was evaluated studying the homogeneity of its fibrillar pattern and irregularities of its margins[27]; changes were interpreted as a sign of either inflammatory lesion or degenerative process.

Sonographic evaluation of tendon thickness and fibrillar structure was conducted for supraspinatus (Figure 2), infraspinatus (Figure 3), and subscapularis tendons, as well as long head of biceps tendon. Tears of rotator cuff tendons were detected evaluating the appearance of a hypoechoic discontinuity within the tendon fibrillar pattern; a tear was considered full-thickness when the defect extended through the entire tendon, and as partial-thickness when it was limited to a part of the tendon thickness[4–6,10,11,13].

Involvement of subacromial and subscapularis bursae was diagnosed when a hypoechoic or sonolucent image appeared within the bursae, indicating the presence of effusion[6,12,28].

Statistical analysis evaluated differences between healthy subjects and the 3 groups of patients examined (RA, PMR, PD) and between each of the 3 patient groups. Student's t test was used to evaluate the capsule–bone distance and the thickness of rotator cuff. Chi-squared test was used to examine the other variables.

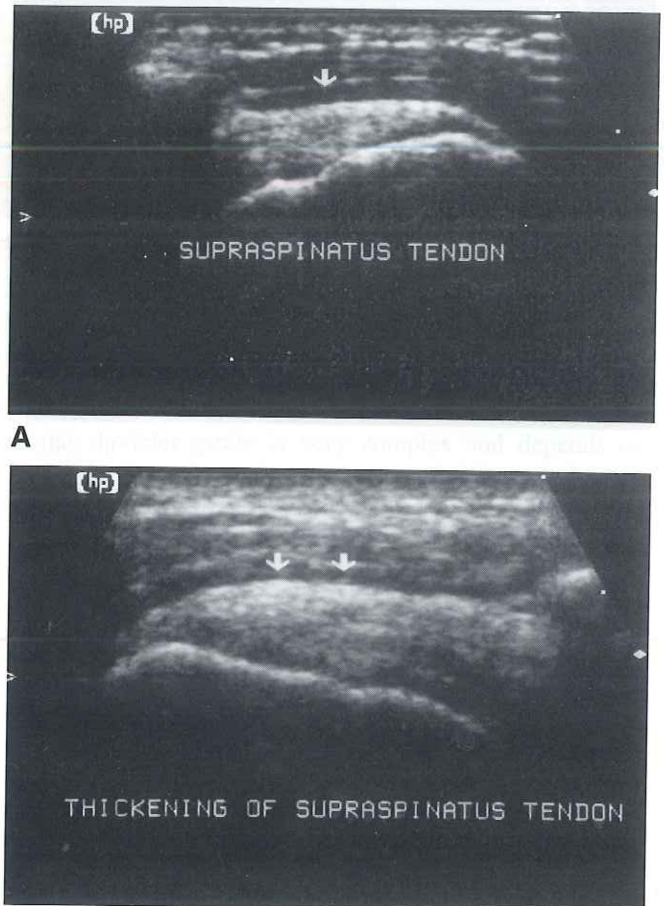


Figure 2. Supraspinatus tendon: (A) normal tendon; (B) thickening.

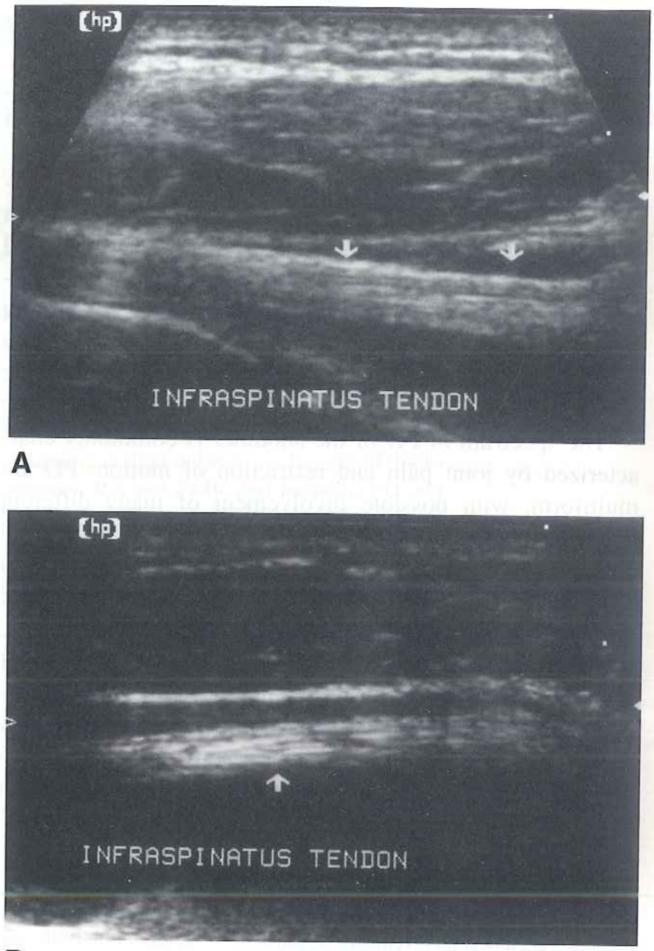


Figure 3. Infraspinatus tendon: (A) normal tendon; (B) thinning.

RESULTS

Results are reported in Table 1.

In normal shoulders only slight changes of tendon echotexture were found and these were present in 5 cases (1.2%). Involvement of the long head of biceps tendon was found totally in 47 RA shoulders (52.2%) (in 16 cases more than one change was present contemporaneously), in 5 PMR joints (15.6%) and in 67 (54.9%) PD joints (18 with contemporaneous alterations). The most common change in RA and PD was the presence of peritendinous fluid collection. In PMR this alteration was not found, but changes of tendon echotexture and thickening were noted. Comparing individual groups, significant differences were found between RA and PMR ($p < 0.001$) and between PD and PMR ($p < 0.005$).

Rotator cuff tendons were involved more often in PD (99 tendons, 27%) versus both RA (20 tendons, 7.4%) and PMR (5 tendons, 5.2%) with statistically significant differences ($p < 0.000001$). In many cases more than one change was found contemporaneously in the same shoulder. Referring to

Table 1. Changes of structures examined with sonography of the shoulder in healthy controls and in the 3 groups of patients studied.

Structure	Change	Controls, n (%)	RA, n (%)	PMR, n (%)	PD, n (%)
Long head of biceps tendon	Effusion		34 (32.2)		61 (50)
	Thickening		8 (8.9)	2 (6.2)	
	Thinning/tears		3 (3.3)		1 (0.8)
	Total rupture		2 (2.2)		6 (4.9)
	Echotexture changes	2 (1.8)	18 (20)	3 (9.4)	21 (17.2)
No. of SH involved		2 (1.8)	47 (52.2)	5 (15.6)	67 (54.9)
Subscapularis tendon	Thickening		1 (1.1)		6 (4.9)
	Thinning/tears		2 (2.2)	1 (3.1)	18 (14.7)
	Total rupture				2 (1.6)
	Echotexture changes	1 (0.9)	5 (5.5)	1 (3.1)	4 (3.3)
No. of SH involved		1 (0.9)	5 (5.5)	1 (3.1)	23 (18.8)
Supraspinatus tendon	Thickening		4 (4.4)		19 (15.6)
	Thinning/tears		4 (4.4)		41 (33.6)
	Total rupture				3 (2.4)
	Echotexture changes	2 (1.8)	9 (10)	2 (6.2)	44 (36.1)
No. of SH involved		2 (1.8)	9 (10)	2 (6.2)	67 (54.9)
Infraspinatus tendon	Thickening		6 (6.7)		6 (4.9)
	Thinning/tears		1 (1.1)	1 (3.1)	9 (7.4)
	Total rupture				
	Echotexture changes		6 (6.7)	2 (6.2)	8 (6.5)
No. of SH involved			6 (6.7)	2 (6.2)	9 (7.4)
Subacromial bursa	Effusion		16 (17.7)	3 (9.4)	12 (9.8)
Subscapularis bursa	Effusion		3 (3.3)		
Capsule–bone distance	Increased (effusion)		47 (52.2)	21 (65.6)	28 (22.9)
	Mean value, mm	2.4	4.2	4	2.6
Rotator cuff thickness	Decrease		19 (21.1)	2 (6.2)	26 (21.3)
	Mean value, mm	6.2	5.8	6	5.3
Calcifications	Presence		6 (6.7)		26 (21.3)
Acromioclavicular joint	Effusion		32 (35.5)		25 (20.5)

SH: shoulders.

single rotator cuff tendons, the same differences were found for the supraspinatus tendon in the 3 diseases; involvement of subscapularis tendon was present more often in PD versus RA and PMR ($p < 0.01$); no significant differences were found for infraspinatus tendon. In total supraspinatus tendon was involved in 9 cases (10%) of RA, 2 (6.2%) PMR, and 67 (54.9%) PD. Involvement of subscapularis tendon was found in 5 RA cases (5.5%), one PMR (3.1%), and 23 PD (18.8%). Changes of infraspinatus tendon were present in 6 RA joints (6.7%), 2 PMR (6.2%), and 9 PD (7.4%).

The presence of effusion within bursae was found more frequently in RA versus the other 2 patient groups, but the finding was statistically significant only when compared to PD ($p < 0.05$).

Total rotator cuff thickness was measured and mean values were calculated: in the control group the mean thickness was 6.2 mm, in RA 5.8 mm, in PMR 6 mm, and in PD 5.3 mm. Thickening was not found, but thinning was present in all the 3 patient groups versus healthy subjects: the differences were statistically significant between controls and both PD ($p < 0.0001$) and RA patients ($p < 0.005$), but not with respect to PMR subjects. Comparing the 3 groups of patients to each other, no significant differences were found between RA and PMR and between RA and PD; in contrast, results were significantly different between PMR and PD ($p < 0.01$).

With regard to effusion of glenohumeral joint with axillary scanning, it was present in 47 RA shoulders (52.2%), 21 PMR (65.6%), and 28 PD (22.9%); differences were statistically significant between PD and both RA and PMR ($p < 0.00005$). The mean values of the longest distance between the bone and the capsule were 2.4 mm in healthy subjects, 4.2 mm in RA, 4 mm in PMR, and 2.6 mm in PD. They were significantly different between healthy subjects and both the RA ($p < 0.000001$) and PMR patients ($p < 0.00005$); in contrast differences were not significant with respect to PD. The comparison among the 3 groups of patients to each other indicated that the values in RA and PMR were similar, and this finding was confirmed by statistical analysis (nonsignificant differences). However, significant differences were present between RA and PD ($p < 0.000001$) and between PMR and PD ($p < 0.00001$).

Calcifications of periarticular soft tissues were found more often in PD, while they were never present in PMR. The differences between PD and RA were significant ($p < 0.01$).

Effusion of acromioclavicular joint was present in RA and in PD, with statistically significant differences ($p < 0.05$) between the 2 groups. Effusion was not found in PMR.

DISCUSSION

Sonographic evaluations of the shoulder have been reported in healthy subjects[7,25] and in various diseases: RA[3,26,29], trauma[4–6], ankylosing spondylitis[3], hemodialysis related arthropathy[23], juvenile polyarthritis[3], psoriatic arthritis[3,26,29], PMR[30]. Single structures of the shoulder have been studied: rotator cuff tendons[4,5,7,9,11,14], bursae[6,28], long head of biceps tendon[10,15], glenohumeral joint[25,26], and acromioclavicular joint[29]. However, comparative sonographic research about changes of all the structures of the shoulder girdle in rheumatic disease had never been published.

Our sonographic study showed different kinds of shoulder involvement in the 3 rheumatic diseases examined: in RA, changes of all structures were found, with alterations of both articular and periarticular tissues; in PMR we noted the highest frequency of effusion within the glenohumeral joint; in PD, involvement of tendons was prevalent.

In RA and PMR, results confirm the presence of synovitis of glenohumeral joint, with consequent appearance of effusion. The significantly increased bone–capsule distance in RA and PMR with respect to controls illustrates this finding. Indeed synovitis is the most important pathogenetic factor in both RA and PMR. RA is characterized by synovial pannus, which is responsible for most articular alterations; in PMR the inflammatory involvement of synovial tissue is well known and has been described[30–32]. In contrast, in PD involvement of periarticular soft tissues is prevalent; when effusion of glenohumeral joint was found it was mild and consequently the mean value of increase of the capsule–bone distance was not significantly different with respect to normal shoulders. However it was significantly different with respect to RA and PMR patients. The appearance of glenohumeral joint effusion in PD could be explained by the frequent communication between the joint space and both the sheath of the long head of biceps tendon and subacromial bursa, with consequent possible detection of effusion during fluid collection within those 2 structures.

In PD, presence of effusion within either the sheath of long head of biceps tendon or the subacromial bursa may depend on 2 factors: (1) the passing of fluid from the glenohumeral joint to those 2 structures due to a communication between them (in those cases fluid is also found in glenohumeral joint); (2) rotator cuff pathology or other soft tissue abnormalities that cause secondary production of fluid by the tendon sheath or the bursa.

In RA the signs of tenosynovitis of the long head of biceps tendon and bursitis of subscapularis and subacromial bursae confirm[3,18–20] the inflammatory involvement also of periarticular soft tissues. Moreover, the typical erosive synovitis and consequent destruction of articular and periarticular structures can account for the significant reduction of total rotator cuff thickness with respect to both controls and the PMR group, for possible lesions of the tendons of rotator cuff.

Our study showed that in PMR articular involvement of shoulder girdle was limited to the glenohumeral joint, as effusion is never present within the acromioclavicular joint; in contrast, the finding was frequent both in RA and PD. This result could be explained by the global involvement of the shoulder in RA; in PD it may indicate the importance of that joint in the mechanical integrity of the shoulder girdle, as it could be consequent to the changes of periarticular structures. In particular in RA, our study showed that sonography reveals more frequently than clinical examination the involvement of acromioclavicular joint, even if it is possible that sonography fails to detect mild effusion of that joint. Nevertheless, if sonographic examination is carried out carefully (comparative study with contralateral joint, the finding of convexity of the capsule with simultaneous appearance of a hypoechoic area within articular space) most cases of effusion should be detected.

The measurements of total rotator cuff thickness showed that significant differences were present between controls and PD, controls and RA, and between PMR and PD. This finding may further indicate the presence only of joint synovitis in PMR, while in RA even periarticular involvement is present, with appearance of inflammatory and erosive processes of tendons and consequent possible lesions of rotator cuff. The involvement of rotator cuff tendons is the most important feature of PD and the significantly different results with respect to both healthy subjects and PMR patients are justified by the main periarticular changes that characterize the disease. Calcifications of periarticular soft tissues were present in PD and RA; but sonography is not the most accurate diagnostic method for their research.

Sonography reveals the different location of changes of the shoulder girdle. The girdle's complex anatomy makes it difficult to identify alterations by clinical examination, and the application of other diagnostic techniques such as magnetic resonance imaging is limited by the high costs and low availability of equipment. In contrast, sonography is a noninvasive and low cost imaging method that has been applied successfully to the study of the musculoskeletal system during recent years and it continues to evolve with the advent of new ultrasound methods[29]. As well, sonographic study of the shoulder appears to be valuable and easily repeatable for monitoring the therapeutic response. The possibility of ultrasound guided interventions[33] improves the usefulness of sonography. Although the practitioner will require a long trial period to carefully evaluate the complex anatomy of the shoulder, our findings confirm the validity of this method. Thus a widespread application of ultrasonographic analysis is hoped for.

REFERENCES

1. Sola ES. Upper extremity pain. In: Wall PD, Melzack R, editors. Textbook of pain. London: Churchill Livingstone; 1994:457–74.
2. Dalton SE. Axial and peripheral joints. The shoulder. In: Klippel JH, Dieppe PA, editors. Rheumatology. London: Mosby; 1998:4,7.1–7.14.
3. Alasaarela E, Takalo R, Tervonen O, Hakala M, Suramo I. Sonography and MRI in the evaluation of painful arthritic shoulder. Br J Rheumatol 1997;36:996–1000.
4. Farin PU, Jaroma HJ. Acute traumatic tears of the rotator cuff: value of sonography. Radiology 1995;197:269–73.
5. Van Holsbeeck MT, Kolowich PA, Eyler WR, et al. US depiction of partial-thickness tear of the rotator cuff. Radiology 1995;197:443–6.
6. Hollister MS, Mack LA, Patten RM, Winter TC III, Matsen FA III, Veith RR. Association of sonographically detected subacromial subdeltoid bursal effusion and intraarticular fluid with rotator cuff tear. Am J Rheumatol 1995;165:605–8.
7. Mack LA, Nyberg DA, Matsen FA. Sonographic evaluation of the rotator cuff. Radiol Clin North Am 1988;26:161–77.
8. Van Moppes FI, Veldkamp O, Roorda J. Role of shoulder ultrasonography in the evaluation of the painful shoulder. Eur J Radiol 1995;19:142–6.
9. Farin PU, Jaroma H. Sonographic findings of rotator cuff calcifications. J Ultrasound Med 1995;14:7–14.
10. Ptasznik R, Hennessy O. Abnormalities of the biceps tendon of the shoulder: sonographic finding. Am J Rheumatol 1995;164:409–14.
11. Farin P, Jaroma H. Sonographic detection of tears of the anterior portion of the rotator cuff (subcapularis tendon tears). J Ultrasound Med 1996;16:221–5.
12. Alasaarela EM, Alasaarela ELI. Ultrasound evaluation of painful rheumatoid shoulders. J Rheumatol 1994;21:1642–8.
13. Hodler J, Terrier B, von Schulthess GK, Fuchs WA. MRI and sonography of the shoulder. Clin Radiol 1991;43:323–7.
14. Middleton WD, Reinus WR, Melson GL, Totty WG, Murphy WA. Pitfalls of rotator cuff sonography. Am J Rheumatol 1986;146:555–60.
15. Farin PU, Jaroma H, Harju A, Soimakallio S. Medial displacement of the biceps brachii tendon: evaluation with dynamic sonography during maximal external shoulder rotation. Radiology 1995;195:845–8.
16. Backhaus M, Manger B. The use of ultrasonography in the rheumatological diagnosis of the shoulder. Rheumatol Eur 1997;26:89–92.
17. Arnett FC, Edworthy SM, Bloch DA, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum 1988;31:315–24.
18. Albani S, Carson DA. Etiology and pathogenesis of rheumatoid arthritis. In: Koopman WJ, editor. Arthritis and allied conditions. Baltimore: Williams & Wilkins; 1997:979–92.
19. Harris ED. Clinical features of rheumatoid arthritis. In: Kelley WN, Harris ED, Ruddy S, Sledge CB, editors. Textbook of rheumatology. Philadelphia: WB Saunders; 1997:898–932.
20. Gordon DA, Hastings D. Rheumatoid arthritis. Clinical features of early, progressive and late disease. In: Klippel JH, Dieppe PA, editors. Rheumatology. London: Mosby; 1998;5,3.1–3.14.
21. Hazleman BL. The vasculitides. Polymyalgia rheumatica and giant cell arteritis. In: Klippel JH, Dieppe PA, editors. Rheumatology. London: Mosby; 1998;7,21.1–21.8.
22. Weyand CM, Goronzy JJ. Polymyalgia rheumatica and giant cell arteritis. In: Koopman WJ, editor. Arthritis and allied conditions. Baltimore: Williams & Wilkins; 1997:1605–16.
23. Coari G, Iagnocco A, Maggi S, et al. Sonographic findings in haemodialysis-related chronic arthropathy. Eur Radiol 1996;6:890–4.
24. Coari G, Iagnocco A. La spalla. The shoulder. In: Coari G, Iagnocco A, editors. Ecografia articolare. Articular sonography. Milano: Masson; 1993:13–21.
25. Koski JM, Axillar ultrasound of the glenohumeral joint. J Rheumatol 1989;16:664–7.
26. Koski JM. Validity of axillar ultrasound scanning in detecting effusion of glenohumeral joint. Scand J Rheumatol 1991;20:49–51.
27. Grassi W, Tittarelli E, Blasetti P, Pirani O, Cervini C. Finger tendon involvement in rheumatoid arthritis. Evaluation with high-frequency sonography. Arthritis Rheum 1995;38:786–94.
28. Van Holsbeeck M, Strouse PJ. Sonography of the shoulder: evaluation of the subacromial-subdeltoid bursa. Am J Roentgenol 1993;160:561–4.
29. Alasaarela EA, Tervonen O, Takalo R, Lahde S, Suramo I. Ultrasound evaluation of the acromioclavicular joint. J Rheumatol 1997;24:1959–63.
30. Koski JM. Ultrasonographic evidence of synovitis in axial joints in patients with polymyalgia rheumatica. Br J Rheumatol 1992;31:201–3.
31. Chou CT, Schumacher HR Jr. Clinical and pathologic studies of synovitis in polymyalgia rheumatica. Arthritis Rheum 1984;27:1107–17.
32. Gordon I, Rennie AM, Branwood AW. Polymyalgia rheumatica: biopsy studies. Ann Rheum Dis 1964;23:447–55.
33. Bradley M, Bhamra MS, Robson MJ. Ultrasound guided aspiration of symptomatic supraspinatus calcific deposits. Br J Radiol 1995;68:716–9.