

Fat Suppression Magnetic Resonance Imaging in Shoulders of Patients with Polymyalgia Rheumatica

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ABSTRACT. Objective. To evaluate the sites of inflammatory process in the shoulders of patients with polymyalgia rheumatica (PMR) using fat suppressed magnetic resonance imaging (MRI).

Methods. Six consecutive, untreated new patients with PMR were investigated. Five patients with early rheumatoid arthritis (RA) and 4 patients with early psoriatic arthritis (PsA) with bilateral shoulder symptoms served as a control group. Bilateral shoulder fat-suppressed MRI sequences were performed in all patients and controls. We evaluated the presence of joint synovitis, bursitis, tenosynovitis, and bone and soft tissue edema.

Results. Bilateral subacromial/subdeltoid bursitis was found in all patients with PMR, in 1/5 (20%) patients with RA ($p < 0.05$), and in none with PsA ($p < 0.02$). Glenohumeral synovitis was present in all case and controls. Biceps tenosynovitis was observed in 4/6 (67%) patients with PMR, in 4/5 (80%) with RA (not significant, NS), and in all 4 patients with PsA (NS). No evidence of bone edema adjacent to the joint capsule and enthesal insertions or in the soft tissues was present in either cases or controls.

Conclusion. The absence of extracapsular abnormalities in the early shoulder disease of PMR does not confirm the hypothesis of a capsular-based disorder. (J Rheumatol 2004;31:120–4)

Key Indexing Terms:

POLYMYALGIA RHEUMATICA

BONE EDEMA

FAT SUPPRESSED MAGNETIC RESONANCE IMAGING

Shoulder pain is the presenting feature in the majority of patients with polymyalgia rheumatica (PMR). The prominent and diffuse shoulder discomfort radiating distally toward the elbows may be only partially explained by the observed mild joint synovitis¹. In 2 recent imaging studies we found that bilateral subacromial and subdeltoid bursitis was the most frequent lesion present in almost all patients. Biceps tenosynovitis and glenohumeral synovitis were present in around 2/3 of the patients^{2,3}. The inflammatory involvement of these 3 synovial structures may contribute to the diffuse discomfort in the shoulder girdle observed in patients with PMR.

Recently, McGonagle, *et al*, using fat suppressed magnetic resonance imaging (MRI), suggested that bone

and soft tissue edema adjacent to the joint capsule could represent the primary lesion of PMR⁴.

We planned a case-control study to evaluate the type of lesions present in a consecutive series of patients with PMR, rheumatoid arthritis (RA), and psoriatic arthritis (PsA) with symptomatic shoulder involvement using fat-suppressed MRI.

MATERIALS AND METHODS

Six consecutive untreated new patients, 4 men and 2 women, with a diagnosis of PMR (Healey criteria)⁵ who attended the outpatient clinic of Prato and Reggio Emilia rheumatology centers were included in the study. All these patients had shoulder girdle involvement and none had clinical evidence of giant cell arteritis (GCA). The median age at onset of disease was 72 years (range 66–81) and the median disease duration 3 months (range 2–5).

Two control groups were considered. The first consisted of 5 consecutive patients, 3 women and 2 men, with early RA, who had symptomatic bilateral shoulder involvement. The median age at onset of disease was 48 years (range 42–59). The second consisted of 4 consecutive patients, 2 women and 2 men, with PsA and clinical evidence of bilateral shoulder involvement. The median age at onset of disease was 51 years (range 38–70). Three patients had only peripheral involvement and one had both axial and peripheral involvement. Both control groups had a disease duration < one year (median 7 months, range 4–10). All control patients were treated with nonsteroidal antiinflammatory drugs and only one patient with RA was treated with second-line therapy (cyclosporine).

MRI scanning utilizing a 1.5-T superconductive magnet system (Signa-MR/i ES, GE Medical Systems, Milwaukee, WI, USA) was performed using a 17-cm shoulder coil with the patients in supine position. Pulse sequences included axial and sagittal fast spin echo (FSE) T2-weighted sequences with fat saturation (3500 ms repetition time, 60 to 70 ms echo time, and 3 to 4 excitations) and sagittal gradient echo T2-weighted 3

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dimensional sequences (24 ms repetition time, 6.6 ms echo time, and 2 excitations). The axial section was 4–5 mm thick, and the sagittal section was 3–4 mm thick; both had an intersection gap of 0.6–1 mm. The field of view was 16 × 12 cm; the matrix size was 256 × 224 cm or 512 × 224 cm.

Bilateral MRI was done on all cases and controls. Scans from each shoulder were examined by 2 radiologists who were blinded to clinical diagnosis. The joint space, subacromial and subdeltoid bursae, and synovial sheaths of the long head of the biceps were evaluated for fluid collection. As previously reported measurement of fluid accumulation was graded using a semiquantitative scale (0 = no accumulation; 1 = sufficient accumulation to allow visualization of the articular shoulder structure, periarticular shoulder structure, or both; 2 = moderate accumulation; 3 = sufficient quantity to stretch the walls of the structures).

The location of bone edema was described. The extension of bone edema was graded as follows: 0 = absence; 1 = mild; 2 = moderate; 3 = diffuse.

Statistical analysis was done using the SPSS statistical package (SPSS Inc., Chicago, IL, USA). Chi-square test was used to compare the results.

RESULTS

As shown in Table 1, bilateral subacromial/subdeltoid bursitis was found in all patients with PMR, in 1/5 (20%) patients with RA ($p < 0.05$), and in none with PsA ($p < 0.02$). Unilateral subacromial/subdeltoid bursitis was present in 3 patients with both RA and PsA. Glenohumeral synovitis was present in all cases and controls. Biceps tenosynovitis was observed in 4/6 (67%) patients with PMR, in 4/5 (80%) with RA (not significant, NS), and in all 4 patients with PsA (NS). No evidence of bone edema adjacent to the joint capsule and enthesal insertions or in the soft tissues was present in cases or controls (Figures 1, 2, and 3).

Rapid improvement of symptoms was observed in all patients with PMR after corticosteroid therapy, and none met the American College of Rheumatology 1987 criteria for RA⁶ or developed articular erosions or clinical manifestations related to spondyloarthropathies (enthesitis, dactylitis, inflammatory spinal pain) during the followup period (median duration 26 mo).

DISCUSSION

In keeping with our previous reports^{2,3}, our results confirm that bilateral subacromial/subdeltoid bursitis is the prominent lesion in PMR. However, we did not observe bone and soft tissue edema adjacent to the joint capsule or enthesal insertion. In contrast, McGonagle, *et al*⁴ detected the presence of edema at extracapsular sites adjacent to the joint capsule and in the soft tissues in 50% of the examined shoulders. Their findings led them to suggest that the primary site of disease localization in PMR may be the joint capsule and that joint synovitis and bursitis could be secondary to this inflammatory process⁷.

The reason for the differences we found compared to the McGonagle, *et al* study may be related to patient selection. Late onset undifferentiated spondyloarthropathy (SpA) and elderly patients with late onset RA may present with clinical features similar to PMR^{8–11}. In particular, proximal symptoms, systemic manifestations, oligoarthritis of lower limbs, distal pitting edema, and high erythrocyte sedimentation rate may mimic PMR, and only a sufficiently long followup would confirm the correct diagnosis.

An adequate followup of 2 years allowed us to exclude patients with conditions other than PMR. However, this is not sufficient to explain the differences observed in our study. Patients had similar clinical and demographic features with bilateral shoulder involvement and none received corticosteroids before examination.

McGonagle, *et al* employed fat suppression MRI to investigate the inflammatory shoulder changes of patients with PMR^{4,7}. In our previous MRI studies we investigated the shoulder lesions in patients with PMR with T1 and T2 weighted sequences with gadolinium-DPTA enhancement. It is well known that fat suppression MRI is a more precise radiological procedure to detect the presence of fluid in any

Table 1. MRI shoulder findings in patients and controls.

Patient	Diagnosis	Subacromial/Subdeltoid Bursitis*, L/R	Glenohumeral Joint Synovitis, L/R*	Tenosynovitis of Long Biceps Head, L/R	Extracapsular Edema*† L/R
1	PMR	3/2	3/2	3/3	0/0
2	PMR	3/2	3/2	1/3	0/0
3	PMR	1/1	1/1	0/0	0/0
4	PMR	2/2	3/3	3/0	0/0
5	PMR	1/3	0/1	0/0	0/0
6	PMR	2/3	1/0	3/2	0/0
7	RA	0/1	1/2	0/0	0/0
8	RA	1/1	1/1	2/0	0/0
9	RA	0/3	1/1	2/1	0/0
10	RA	0/1	0/1	2/2	0/0
11	RA	0/0	1/2	0/3	0/0
12	PsA	2/0	3/2	3/2	0/0
13	PsA	0/1	1/0	0/2	0/0
14	PsA	1/0	1/2	0/1	0/0
15	PsA	0/0	1/1	1/0	0/0

* Fluid collection was graded with a semiquantitative scale ranging from 0 to 3. † The presence of extracapsular edema was evaluated adjacent to the joint capsule and enthesal insertions and also in the soft tissues.



Figure 1. Axial FSE T2 weighted image with fat saturation of the shoulder in PMR. Presence of moderate (grade 2) subacromial bursitis (black arrows) and moderate (grade 2) glenohumeral joint fluid collection (white arrowheads). Extracapsular soft tissue and bone marrow edema is not detectable.



Figure 2. Sagittal FSE T2-weighted scan with fat saturation of the shoulder in PMR. Grade 2 subdeltoid bursitis (arrowheads) and grade 3 long-head biceps tenosynovitis (arrows). Edema of soft tissues, of bone, and of insertional tract of rotator cuff (supraspinatus and subscapularis muscles) is not visible.

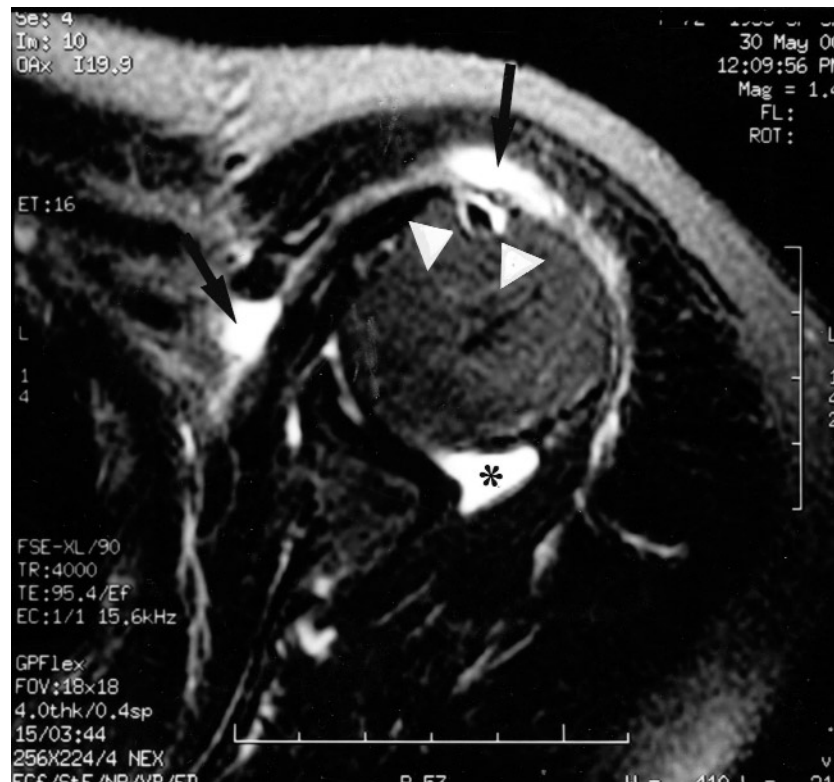


Figure 3. Axial FSE T2-weighted image with fat saturation of the shoulder in PMR. Grade 1 subacromial and subdeltoid bursitis (black arrows), grade 2 glenohumeral joint synovitis (asterisk), and grade 2 long-head biceps tenosynovitis (white arrowheads) with absence of soft tissue and bone edema.

anatomical sites. Indeed, by eliminating the high signal originating from the presence of fat, this technique remarkably enhances the fluid signal from joints, bursae, entheses, bones, and soft tissues.

We used fat suppression MRI to detect the shoulder inflammatory lesions of patients with PMR, as did McGonagle, *et al*⁴. Therefore, the MRI imaging techniques used in the 2 studies were comparable and do not explain the different results.

Based on their results, McGonagle, *et al* suggest the concept of a primary non-synovial pathology in PMR with prominent changes adjacent to the joint capsule⁷. The entheses and joint capsule were also considered to be the primary site of inflammation in SpA. Therefore, they suggested a close relationship between these 2 entities and proposed to include these inflammatory arthropathies in the same group.

However, the demographic, clinical, and immunogenetic characteristics of PMR are completely different from those of SpA. In particular, in the largest series of PMR there is no evidence of the typical manifestations of SpA such as inflammatory spinal pain, enthesitis, dactylitis, and anterior uveitis^{13,14}.

In conclusion, our results confirm that involvement of extraarticular synovial structures is a predominant part of

the inflammatory process of PMR¹². The absence of inflammatory changes in the extracapsular soft tissues adjacent to the joint capsule does not support the hypothesis of a primarily capsular based pathology.

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