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Ultrasonographic study of painful shoulder

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Painful shoulder is a very common condition in clinical rheumatology. However, knowledge of the lesions responsible for shoulder pain in most patients has been limited to clinical examination and plain radiography in clinical practice. High frequency ultrasonography is an accurate,^{1–4} non-invasive, and cheap imaging technique available in clinical rheumatology for evaluating patients with painful shoulder. However, dependence on the skill of the operator has been considered to be the main disadvantage of ultrasound. Diagnostic results are affected by the quality of the equipment, examination technique, sonographer experience, and sonographic diagnostic criteria.

We compared the ultrasonographic findings in two groups of patients with clinically diagnosed periarticular disorders, with a first flare of shoulder pain—group I: 228 patients (228 shoulders); group II: 110 patients (122 shoulders). Patients with previous trauma or chronic inflammatory arthritis were excluded.

Each group was examined in Italy or in Spain by a different rheumatologist (AI, Rome, Italy and EN, Madrid, Spain) using a different commercially available real time machine (Image Point Hx, Agilent Technologies/HP and Sonoline, Versa, Siemens, Seattle, USA, respectively) with a 7.5 MHz linear phased array transducer. Both rheumatologists used the same scanning technique and the same sonographic diagnostic cri-



Figure 1 Sonographic imaging of a supraspinatus tear. Transverse sonogram. Note the presence of fluid (F) filling the defect of the supraspinatus tendon (SS). DM, deltoid muscle; HH, humeral head.

Table 1 Ultrasonographic findings in symptomatic shoulders

Shoulder lesions	Group 1 (228 shoulders) % of shoulders	Group 2 (122 shoulders) % of shoulders
Supraspinatus lesions	67	66
Infraspinatus lesions	25	20
Subscapularis lesions	16	11
Biceps tendon lesions	28	32
Biceps sheath effusion	30	26
SA-SD bursitis	16	22
ACRCL involvement	63	61
RC calcification	15	19
GH effusion	12	7

SA-SD, subacromial-subdeltoid; ACRCL, acromioclavicular; RC, rotator cuff; GH, glenohumeral.
p>0.05 for all results.

teria for shoulder lesions.^{5,6} A χ^2 test was used to compare quantitative variables. A value of $p < 0.05$ was considered significant.

Group I comprised 132 women and 96 men with a mean age of 45.6 years (range 18–64). The mean duration of symptoms was 3.3 months (range 1–8). Group II comprised 81 women and 29 men with a mean age of 54.5 years (range 25–75). The mean duration of symptoms was 8.6 months (range 0.5–36).

The sonographic pathologic findings in the painful shoulders were similar for both groups ($p > 0.05$) (table 1). In most patients various different periarticular structures were affected. Supraspinatus tendon lesions were the most common

pathological finding (fig 1). Infraspinatus and subscapularis abnormalities were seen less often. Increased fluid within the subacromial-subdeltoid bursa and biceps tendon sheath were also very common, as were degenerative changes in the acromioclavicular joint.

Our results are consistent with those previously reported.⁷⁻¹⁰ Ultrasound provides a valuable method for studying painful shoulders in daily practice and clinical research. The scanning technique and pathological criteria should be standardised to achieve optimum widespread use of ultrasonography in rheumatology.

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Synovial lymphocyte responses if tested fresh not frozen can incriminate microbial intrasynovial DNA and RNA

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In a previous issue of the *Annals*, Sibia and Limbach reviewed the microbiology of "infectious arthritis" and described various ways in which the agents might be related to the arthritis.¹ One approach, for which there is considerable support, was, however, not discussed.

In 1980 synovial lymphocytes were found to respond maximally to stimulation by either chlamydia or ureaplasma antigens in cases of sexually transmitted reactive arthritis.² In 1985 synovial responses were reported in eight cases of enteric and 12 cases of sexually transmitted reactive arthritis.³ Responses to the relevant antigens of each category differentiated the enteric from the sexually transmitted cases. Additionally, peripheral blood lymphocytes in all eight enteric cases and in eight of the 12 sexually transmitted cases responded negligibly or only minimally to the antigens that gave significant synovial responses. In 1991 a review of 12 cases of enteric reactive arthritis showed that the maximal synovial responses to the relevant enteric antigen in 10 cases of salmonella, shigella, or yersinia reactive arthritis would unequivocally differentiate them from the responses in two cases of campylobacter reactive arthritis; the results also indicated that some cross reactivity occurred within the salmonella, shigella, and yersinia group.⁴

These observations on the responses of synovial lymphocytes to the causative antigen in reactive arthritis have been confirmed in several countries between 1989 and 1994.⁵⁻⁷ However, the data from some studies have shown that the stimulation indices from the responses in the Vancouver experience are higher and more specific than those of other

laboratories and one laboratory has downgraded the importance of this approach.⁸ Technical differences between laboratory procedures are hard to define, but the use of fresh as opposed to stored frozen lymphocytes for the tests differentiates the Vancouver laboratory from several others. An early investigation of lymphocyte responses performed in the Vancouver laboratory in the late 1970s showed that the use of stored liquid nitrogen frozen lymphocytes negated or greatly reduced the response to antigenic stimulation, although the lymphocytes still responded to phytohaemagglutinin (PHA). In consequence, fresh lymphocytes were always employed subsequently. The assumption was made that freezing caused loss of associated antigen-processing macrophages, which are required for antigen responses, but are not needed for PHA and other mitogen responses. It is noteworthy that the study referred to above, in which synovial responses were considered unhelpful, did use stored frozen synovial mononuclear cells. Another study found a lack of correlation between the detection of *Chlamydia trachomatis* DNA in synovial fluid and the presence of an antichlamydial immune response,⁹ but again, frozen synovial mononuclear cell samples were employed.

It is now clear that DNA or RNA from a variety of micro-organisms can be found within the synovium of arthritic, but also of normal and degenerative, joints. To incriminate intrasynovial organisms as a cause of a patient's arthritis is difficult. The Vancouver experience of a 12 year study of 360 patients with many types of arthritis has indicated that the response of synovial lymphocytes to microbiological antigen stimulation can provide such incriminating