Letters to the Editor

Hip bursitis in active polymyalgia rheumatica: Report of a case

Sir,

The cause of musculoskeletal discomfort in the shoulder and pelvic girdles in patients with polymyalgia rheumatica (PMR) is not completely understood. A mild synovitis of the glenohumeral joint has been demonstrated by radionuclide scanning, arthroscopy and synovial biopsy (1, 2). Fluid accumulation in the hip and/or glenohumeral joints was detected in 68% of 19 PMR patients studied by ultrasonography (3). However, joint synovitis can only partially explain the diffuse and broad proximal discomfort in the shoulder and pelvic girdles. In a recent case-control magnetic resonance imaging (MRI) study of the shoulders in patients with active, untreated PMR we found bilateral subacromial and subdeltoid bursitis in 13/13 (100%) of the cases (4). Ten of the 13 patients had also synovitis of the glenohumeral joint. This study suggested that subacromial and subdeltoid bursitis in association with glenohumeral joint synovitis may better explain the diffuse aching in the shoulders and in the proximal portions of the arms than joint synovitis alone.

PMR patients with pelvic girdle involvement usually experience stiffness and diffuse, severe musculoskeletal discomfort in the inguinal region, the buttock and all portions of the thigh down to the knee (5). The anatomical structures involved by the inflammatory process in patients presenting pelvic girdle symptoms have been less thorougly studied.

We describe a 78-year PMR patient with pelvic girdle symptoms in whom MRI demonstrated the prominent involvement of hip bursae. The patient presented with a 2-month history of aching and stiffness in the neck, shoulder and pelvic girdles associated with low grade fever. His previous medical history was negative. Pain was most prominent in the proximal portion of the upper arms and in the thighs and was accentuated during the nocturnal hours and by movement. Physical examination showed tenderness and limited motion in the shoulders. Moreover, localized tenderness was elicited on palpation over the left greater trochanter. Physical findings were otherwise normal. The body temperature was 37.2°C.

Laboratory investigations showed a erythrocyte sedimentation rate of 86 mm/1st hour (Westergren), C-reactive protein 94 mg/L (normal: < 5 mg/L), and normal findings for the complete blood cell count, renal and hepatic function, serum creatine kinase, and protein electrophoresis. Urinary Bence-Jones protein was absent. Rheumatoid factor and serum tumor markers were negative. Radiographs of the chest and pelvis and abdominal ultrasonography detected no pathological findings. Ultrasonography of the shoulders demonstrated bilateral subacromial and subdeltoid bursitis and scanty fluid collection in the glenohumeral joints.

A diagnosis of PMR was made. Before starting corticosteroid therapy, however, an MRI of the hip regions was performed (Philips Gyroscan T5 II, 0.5 Tesla, Eindhoven, The Netherlands). It showed findings consistent with bilateral iliopsoas and trochanteric bursitis associated with mild bilateral hip joint synovitis (Fig. 1). Symptoms rapidly remitted after prednisone 20 mg/day was given.

The MRI findings in this patient suggest that iliopsoas and trochanteric bursitis associated with hip joint synovitis could account for the bilateral diffuse discomfort which he experienced in his inguinal region, buttocks and thighs. The pain in trochanteric bursitis is located along the lateral side of the upper thigh, with radiation from the buttock down toward the knee (6-8). It is deep and aching in quality, frequently worsening at night. Localized tenderness over the greater trochanter can be elicited on palpation, especially in thin subjects (7).

Iliopsoas bursitis is characterized by pain in the groin extending to the medial side of the thigh and is often difficult to detect clinically (9). The procedure of choice in the diagnosis of iliopsoas bursitis is MRI (10).

In conclusion, the MRI findings of trochanteric and iliopsoas bursitis observed in this patient, in association with hip joint synovitis, could explain the diffuse and broad pelvic girdle symptoms seen in PMR. A case-control study is needed to confirm the frequency and severity of hip bursitis in PMR.

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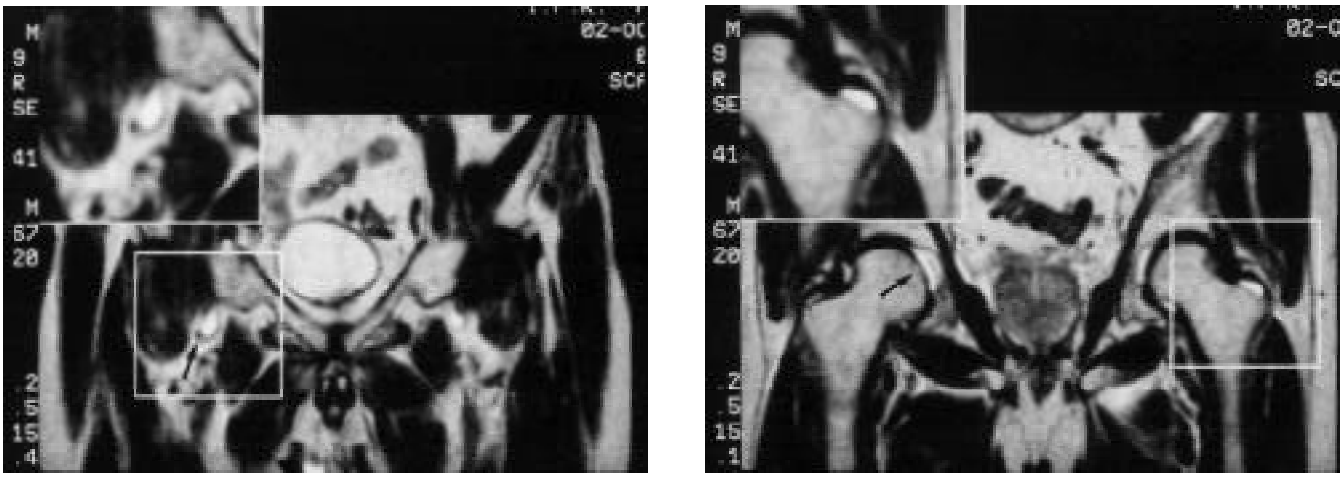

(a) **(b)**

Fig. 1. Hip region coronal T2 weighted images. (**a**) Bilateral fluid accumulation in the iliopsoas bursae suggesting bursitis (**arrow**). The inset shows a detail of the right iliopsoas bursitis. (**b**) Enlarged fluid-filled left trochanteric bursa (**inset**) with minimal involvement of the right trochanteric bursa. Mild bilateral fluid effusion is also visible in the hip joint (**arrow**).

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IL-6 and some natural inhibitors of chronic human inflammation in RA and SLE

Sir,

The role of IL-6 in chronic inflammation is hotly debated (1). It is thought to be pro-inflammatory in some experimental models of arthritis (2) while in others it seems to be protective (3). One of the ways by which IL-6 may become protective is through the stimulation of the synthesis of some cytokine antagonists, among which TNF R type I (tumor necrosis factor soluble receptor type I-TNF RI) and IL-1 receptor antagonist (IL-1Ra) are thought to be acute phase reactants (4, 5). Since the role of IL-6 on APPs is one of the major biological actions of the cytokine, the possible inter-relationship between IL-6 and cytokine antagonists belonging to the acute phase proteins might be of considerable interest *in vivo*. We report here on the inter-relationships between IL-6 and natural inhibitors in two chronic diseases, rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE), derived from data on serum circulating levels.

We followed 15 patients with rheumatoid arthritis (RA) longitudinally for a period of six months, seeing them at entry into the study, and at months 1, 3 and 6 (60 assays) and tested serum IL-6, TNF RI (p55), IL-1 receptor antagonist (IL-1 Ra) and several other parameters. We observed a strong correlation between IL-6 and TNF RI, as well as between IL-6 and CRP (C reactive protein) values. However, no such relationship was seen between IL-6 values and IL-1Ra levels (Fig. 1). Thus, in RA we observed that the higher the initial IL6 (interleukin 6) response, the stronger appeared to be the synthesis and shedding of soluble TNF RI, but not that of IL-1Ra.

On the other hand, the relationship between IL-6 levels and TNF RI was confirmed in another chronic inflammatory disease, SLE (28 samples: $R = 0.42$, $p < 0.04$). No relationship was found between IL-6 and IL-1Ra. In SLE high levels of IL-1Ra were seen (17 patients) while the IL-6 levels were half those usually found in RA (2).

Therefore certain natural inhibitors such as TNF RI seem to be strongly correlated with IL-6 in chronic human inflammatory disorders such as RA and SLE, while others such as IL1 Ra are not. This suggests that IL-6 may act as an inducer of the synthesis and shedding of TNF RI.

The available data *in vivo* suggest that IL-6 deserves a new look by those studying chronic inflammatory diseases, as a potential indirect antagonist of the tissue damage induced by TNFα through the synthesis and shedding of TNF RI.

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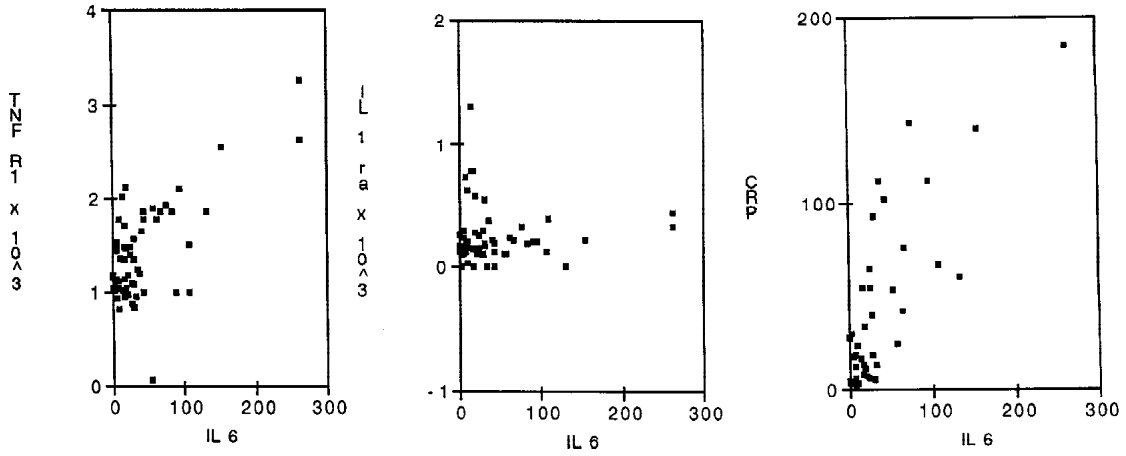

Fig. 1. Correlations over time between IL-6 (pg/ml) and TNF RI (pg/ml), IL-1 Ra (pg/ml), and CRP levels (mg/L) in rheumatoid arthritis.