**LETTERS TO THE EDITOR**

**Does lymphoma ‘cure’ rheumatoid arthritis?**

Prolonged remission in established rheumatoid arthritis (RA) is extremely rare. During a six year follow up of 458 patients Wolfe and Hawley observed two prolonged remissions of more than 48 months, and concluded that ‘once established, RA tends to remain, irrespective of more than 48 months, and concluded that secondary immunodeficiency should be considered during spontaneous cures of well established RA.

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**Insufficiency fracture of the sacrum revealing a pregnancy associated osteoporosis. First case report**

Osteoporosis of pregnancy, usually responsible for spinal or femoral fracture, is rare as it is insufficiency fracture of the sacrum, usually occurring in the elderly. Magnetic resonance imaging (MRI) permitted during pregnancy, led us to diagnose an insufficiency fracture of the sacrum revealing a pregnancy associated osteoporosis, never previously reported to the best of our knowledge. Rheumatologists need to be aware of this new cause of pelvic pain during pregnancy.

A 29 year old pregnant (seventh month) woman presented with a spontaneous acute claudication in conjunction with a left hyperalgesic buttck pain. Her past medical history showed: low back pain, since the second month of her pregnancy, relieved by rest and paracetamol; smoking (10 packet years) stopped at the sixth month of pregnancy; one spontaneous miscarriage at six months responsible for an isoocagual hepatic treatmen

**Table 1 Biological markers**

<table>
<thead>
<tr>
<th>Biological markers</th>
<th>Patient</th>
<th>Normal range for our laboratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum calcium</td>
<td>2.46 mmol/l</td>
<td>2.2-2.6</td>
</tr>
<tr>
<td>Serum phosphorus</td>
<td>1.07 mmol/l</td>
<td>0.98-1.3</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>140 U/l</td>
<td>36-120</td>
</tr>
<tr>
<td>Protdemia</td>
<td>70 g/l</td>
<td>65-75</td>
</tr>
<tr>
<td>Creatinemia</td>
<td>91 µmol/l</td>
<td>45-90</td>
</tr>
<tr>
<td>25 OH vitamin D</td>
<td>4 ng/ml</td>
<td>10-35</td>
</tr>
<tr>
<td>Parathyroid hormone</td>
<td>27 ng/l</td>
<td>10-65</td>
</tr>
<tr>
<td>g1-protein</td>
<td>5.70 mg/ml</td>
<td>4-9</td>
</tr>
<tr>
<td>Antiphospholipid antibody</td>
<td>negative</td>
<td></td>
</tr>
<tr>
<td>Blood count</td>
<td>normal</td>
<td></td>
</tr>
<tr>
<td>CRP</td>
<td>5 mg/l</td>
<td>0-10</td>
</tr>
<tr>
<td>ESR</td>
<td>21 mm/1st h</td>
<td>5-15</td>
</tr>
<tr>
<td>Thyroid hormone</td>
<td>normal</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 1 Magnetic resonance imaging of the pelvis showing on the left part of the sacrum a high signal intensity on T2 weighted sequences.**

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7 Blood count normal
8 CRP=C reactive protein, ESR=erythrocyte sedimentation rate.
No endotoxin detected in plasma of patients with ankylosing spondylitis

Endotoxin has long been known to be an important virulence factor for Gram negative bacteria. It is chemically classified as lipopolysaccharide (LPS) and it is one of the major constituents of the outer membrane of Gram negative bacteria. This molecule is also known to be responsible for many injurious effects of Gram negative bacterial infections and thus is clinically important. The pathogenesis of ankylosing spondylitis (AS) is still unknown. A microbial aetiology has been suggested, as increased faecal carriage of Klebsiella spp, as well as increased antibody values, particularly against the LPS part of K pneumoniae have been reported in AS patients. Furthermore, Wagener et al have shown that endotoxin concentrations were increased in approximately 30% of the AS patients and that a significant correlation was found between the increased endotoxin values and increased C reactive protein (CRP) concentrations. Therefore, it seems that LPS plays an important part in the pathogenesis of AS. In this study we have examined the plasma of 28 hospital AS patients (eight females, 20 males; mean age 43 years (range 26-62)); mean erythrocyte sedimentation rate 22 mm/1st h (range 5-64) and the mean CRP value 12 mg/l (range 0-37) from the Rheumatism Foundation Hospital, Heinita, Finland for the presence of endotoxin. The mean (SD) duration of the disease was 14.5 (9.0) years: less than five years for three patients, 5-10 years for nine patients, and more than 10 years for 16 patients.

Blood samples were taken by the Vactainer blood collection system (Becton & Dickinson Diagnostic Instrument System, Towson, MD, USA) into Vactainer tubes containing sodium citrate and kept in melting ice until separation of plasma. The samples were centrifuged immediately at 400 g for 10 minutes at +4°C and plasma carefully removed and stored at −70°C until assayed. All equipment was endotoxin free.

The concentration of endotoxin in plasma was determined by a chromogenic Limulus assay using Coatest kit (Chromogenix AB, Mölndal, Sweden) with a microtitre modification.1 The detection limit was 5 pg/ml; among normal controls, endotoxin values do not exceed this concentration.

None of the AS patients had plasma endotoxin concentrations exceeding the level of 5 pg/ml. This is surprising, as increased gut permeability has been found in AS patients1 and consequently gut microbes might easily pass through the mucosa to enter the circulation. The low serum antibody concentrations against LPS of certain enterobacteria in AS patients support this scenario. Endotoxaemia may be rapidly transitory, however. Endotoxin could leave the circulation at any stage of the disease; for instance, Wagener et al showed a clear correlation between increased endotoxin and CRP values in AS patients. Endotoxin may also be bound to the specific endotoxin binding protein and is therefore not detected by the Limulus test. However, the same assay was used by Wagener et al, who found increased plasma endotoxin concentrations in one third of the AS patients and in up to half of the patients with sacroiliitis and peripheral arthritis or rheumatoid arthritis. In their study the blood sampling and handling was not reported. The Limulus assay is known to be extremely sensitive to contaminants.

In conclusion, in this study we could not confirm the reported findings on increased plasma endotoxin concentrations in patients with AS. However, this does not exclude the concept that endotoxin or LPS may play an important part in the pathogenesis of AS.
patients showed fractures of vertebrae (one fracture in seven patients and ≥ two in 10 patients) and 11 patients of the appendicular skeleton (one fracture in eight patients and ≥ two in three patients).

Mean T score was −3.2 (0.3) (range −5.4 to −0.5) in the spine and −2.8 (0.2) (range −5.0 to −0.5) in the femoral neck. After two years of treatment with cyclic etidronate, intention to treat analysis indicated an increase in BMD of +7.3 (1.1)% in the spine (p<0.001) and of +2.4 (1.2)% in the hip (p<0.05) (fig 1). The increase of BMD in the spine was similar if patients with spinal fractures (n=8) were excluded (+7.9 (2.0), p<0.01).

The significant increase of BMD in the spine during cyclic etidronate therapy is comparable to the treatment effect in postmenopausal osteoporosis. This is the first study to indicate that cyclic etidronate can significantly increase BMD in the hip of men with IOP and substantial bone loss.

This study suggests that cyclic etidronate may be valuable in men with IOP, although double blind controlled prospective studies, with long term follow up are necessary to adequately assess this. In addition, whether cyclic etidronate will beneficially influence fracture rates in men will probably remain unknown, as large groups of patients are required to study this effect and as osteoporosis is less frequent in men than in women.

Authors’ reply

We thank Dr Ehrlich for his interest in our case report of a patient with a posterior interosseous nerve lesion, secondary to rheumatoid arthritis with synovitis of the elbow joint. We were not aware of his paper describing antecubital cysts in rheumatoid arthritis but, as our case was mainly an illustration of an unusual compressive neuropa-thy rather than a description of synovial cysts of the elbow joint, we would not have included the topic of his paper in our literature search. We agree that there is a similarity between the arthrogram in Dr Ehrlich’s paper and that illustrated in our paper. Our case reinforces the conclusions of Dr Ehrlich’s paper 23 years ago that antecubital cysts are relatively common, are frequently overlooked, and should be actively sought when a patient with rheumatoid arthritis presents with symptoms suggestive of a compressive neuropathy in the forearm. We hope that our case report and Dr Ehrlich’s letter will remind rheumatologists to consider this potentially treatable cause of upper limb disability in patients with rheumatoid arthritis.

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Finger drop in a patient with rheumatoid arthritis

I wish to comment on the case report by McDonald and Smith. Although the authors report that a tomographic arthropgram showed no cystic extension of the joint capsule, the two figures are almost identical with those previously seen in antecubital cysts in rheumatoid arthritis. These ‘cysts’ are corollaries to the popliteal cysts and are generally missed in physical examinations because the bulge they produce often disappears into the fleshy part of the forearm. Clearly, they represent an escape for the effusion in a joint distended by severe synovitis and its products. The proximity of major nerves puts them at risk, as the paper by McDonald and Smith shows.
Finger drop in a patient with rheumatoid arthritis

GEORGE E EHRLICH

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