

## Journal summary

### LEADER

#### Rheumatoid arthritis (RA), malignancy, and paraproteins p 657

We know that mortality is increased in RA, and there seems to be little doubt that there is a firm link with lymphoma. The longer the disease is present, the greater the risk. Paraproteins are associated with both RA and malignant conditions so perhaps it is no surprise to see an increase of monoclonal gammopathy in the former as well as in other connective tissue disorders.

### SCIENTIFIC PAPERS

#### Polymorphonuclear cells, oxidative response, and RA p 661

Polymorphonuclear cells are commonly seen in large numbers in the synovial fluid of patients with RA. Only 20% of synovial fluids in this disease showed an oxidative response by these cells, however: it was not seen at all in other forms of arthritis. This is a little surprising—perhaps other cells are responsible for this reaction or there may be more complex immunological explanations.

#### Tumour necrosis factor in RA p 665

Tumour necrosis factor was found in high concentration in the serum of patients with severe RA and in the synovial fluid of nearly half of those examined. This was particularly seen where there was a high leucocyte count, but it was not found in osteoarthritis. Tumour necrosis factor is an osteoclast activating factor and may partially mediate the characteristic inflammatory reaction of RA and also promote bone erosions.

#### Sulphydryl groups, red cells, and RA p 668

The sulphydryl groups on red cell membranes are found mainly on the transmembrane proteins, particularly on the hexose transport protein. They tend to be low in concentration in RA but rise with second line treatment. Changes in the nutritional state of the cells may be significant in explaining why the rise in concentration occurs after disease improvement.

#### Magnetic resonance imaging and defects of articular cartilage p 672

Magnetic resonance imaging promises to be a useful way of observing cartilage defects of the knee non-invasively. How does it correlate with the actual pathology present? Extremely well it seems. It predicts accurately both cartilage thickness and abnormality, something that conventional radiography signally fails to do.

#### Cytokines in synovial fluid in rheumatic diseases p 676

Various peptide cytokines that are induced in the inflammatory process are potentially pathogenic. A study in various rheumatic diseases showed that interleukin-1 $\beta$  concentrations were higher in RA than in osteoarthritis and were seen in intermediate concentration in seronegative spondylarthritis. Others behaved differently, and various clear patterns emerged which may be of importance. Only time will tell.

#### Vitamin D<sub>3</sub> metabolism in primary Sjögren's syndrome p 682

Vitamin D<sub>3</sub> may be immunoregulatory in primary Sjögren's syndrome, at least in vitro. Does it have this role in the living patient? Investigation does not make this clear as yet, and it is not certain whether the observed change in vitamin D<sub>3</sub> metabolism is due to cause or effect.

#### Mast cells in labial salivary glands in Sjögren's syndrome p 685

Mast cells are known to be associated with hypersensitivity reactions but do they have a role in inflammation: recent studies have suggested that they do. This investigation showed that they are heterogeneous but they clearly participate in the inflammatory process. The significance of this participation requires further study, however, as their role is far from clear.

#### Killer cell activity in Sjögren's syndrome and systemic lupus erythematosus (SLE) p 690

Natural killer cell activity is reduced in Sjögren's syndrome and SLE, among other autoimmune diseases. This does not correlate with immune complex concentrations but it may be partially restored in both diseases by interleukin-2 and interferon- $\gamma$ , and by interferon- $\alpha$  in Sjögren's syndrome. This restoration is not complete, however, implying perhaps that decreased production of these cytokines might contribute to the decrease in natural killer cell activity.

#### HLA antigens in juvenile psoriatic arthritis p 694

Juvenile psoriatic arthritis is rare and may precede or follow the occurrence of the rash. In this study most patients did well and none had uveitis. The prevalence of the HLA antigen B17 was increased, as seen in the adult form, but the patients also showed an increase in A2, unlike adults and those with other forms of juvenile arthritis.

#### Significance of low molecular weight C1q in SLE p 698

The concentrations of low molecular weight C1q in the serum were increased in SLE but not in RA or acute poststreptococcal glomerulonephritis. This increase occurred in parallel with rising anti-dsDNA levels, and low molecular weight C1q behaved differently from the normal form, but this may be an artefact. At all events low molecular weight C1q may have a predictive value when monitoring patients with SLE.

#### Crithidia luciliae test for anti-double-stranded DNA antibodies and fibronectin p 705

These antibodies are detected in about two thirds of patients with SLE, particularly if it is severe, and are rare in other diseases. Fibronectin may interfere with the *Crithidia luciliae* test because of its specific binding site for DNA. This study showed that this interference is real and its removal enhanced the test. The significance of this and the implications for the diagnosis of SLE are clear.

#### Cell wall streptococcal carbohydrate antigen in rheumatic fever p 708

Some patients exposed to streptococcal group A infection go on to develop rheumatic fever, whereas others do not. There is clearly an immunoregulatory dysfunction operating in

this disease, but its elucidation has escaped us. This study in patients with rheumatic fever, their siblings, and their parents suggested that there is an inherited recessive gene responsible for the high reaction to the streptococcal polysaccharide antigen to the cell wall. This, among other genetic influences, may play a part.

**Leprosy and enthesitis** p 715  
Leprosy still occurs in some parts of the world and occasionally it may be complicated by a chronic peripheral polyarthritis. Ten of 77 patients with leprosy studied had an enthesitis not previously reported, and its clinical and laboratory abnormalities are described. It proved to be unpredictable in some ways, though distinctive laboratory findings emerge.

## CASE REPORTS

**Arthritis in a T lymphotropic virus type 1 (HTLV-1) carrier** p 718  
Viruses may act as a trigger for autoimmune diseases: at least that is an idea being toyed with at the moment. A 57 year old woman is described here with apparent RA who was a clinical carrier of HTLV-1. Perhaps the virus had a triggering role in this case, and this possibility is carefully discussed.

**Lymphoedema of the limbs and RA** p 722  
Lymphoedema is an occasional complication of RA and seven patients with this combination are presented here. The lymphoedema proved to be persistent and resistant to treatment. Reluctantly, only conservative management could be offered, and the authors conclude that the cause is a reduced number of lymphatic vessels.

**Captopril and its antiproteinuric effect** p 725  
The antiproteinuric effect of captopril has been noted in various forms of primary and secondary renal disease. Here its effect is described in a young woman with lupus nephritis and intractable nephrotic syndrome. Captopril treatment was effective, but it is not suggested that we should rely on captopril alone—it should be combined with standard treatment.

**Ankylosing spondylitis, spinal pseudarthrosis, and lung disease** p 728  
Pseudarthrosis is a localised, destructive lesion in ankylosing spondylitis usually seen in the thoracolumbar region of the spine. It was seen here in a man also presenting with fibrobullous lung disease and both lesions closely mimicked tuberculosis. You have been warned.

**Avascular necrosis of the femoral head and trochanteric bursitis** p 730  
The authors suggest that trochanteric bursitis may be the first warning of the presence of avascular necrosis of the hip. Magnetic resonance imaging clearly showed its presence, and in patients at risk from developing this form of hip disease this possibility should be considered. Surgical intervention might be prophylactic against the need for eventual total hip replacement.

## DISPATCH

**From Spain** p 733  
Spain has a population smaller than the United Kingdom yet it has almost as many rheumatologists, though more are needed. To British rheumatologists this is a familiar cry. The connection between the Spanish specialty and ourselves is strong and we trust that it can be maintained. It is good to know that rheumatology is alive and well in the Iberian peninsula.

## NOW AND THEN

**First two decades of British rheumatology** p 734  
George Kersley has been one of the major figures in the development of rheumatology as a specialty in the United Kingdom. He was prominent in the field when it was regarded as an unimportant and minor study of disease and it took great courage to devote his life to it when so-called mainstream colleagues looked down on it. Together with Professor Kellgren, Eric Bywaters, Professor Duthie, Will Copeman, Frank Dudley Hart, and many others he put rheumatology on the medical map. It is fascinating to read his recollections of those heady early days. We owe a great debt to these pioneers.

## REVIEW

**Is there a treatment yet for systemic sclerosis?** p 735  
The short answer is, no, not least because of the heterogeneity of the disease. There are distinct possibilities for the future, however, and there is much that can be done in the way of supportive treatment to help alleviate the real distress of those unfortunate enough to get it. Carol Black discusses these treatments and their place in the management of this fortunately uncommon disease.

EDITOR



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