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Letters

Anaemia in rheumatoid arthritis

SIR, The unreferenced assertion by Farah, Sturrock, and Russell that iron deficiency anaemia is a common feature of rheumatoid arthritis and is often caused by an asymptomatic peptic ulcer[1] should not go unchallenged. The diagnosis of iron deficiency in patients with inflammatory disease is difficult, unless bone marrow estimations of iron stores are performed. Perhaps the authors would care to support the statement regarding the prevalence of iron deficiency in rheumatoid arthritis with a suitable reference.

In our experience upper gastrointestinal lesions, including asymptomatic peptic ulceration, are common in patients taking non-steroidal anti-inflammatory drugs and are found no more frequently in patients whose anaemia is microcytic, or those with positive faecal occult bloods, than in those with a normocytic anaemia or negative faecal occult bloods.[2] This observation together with other evidence[3] strongly suggests that upper gastrointestinal lesions are not the cause of iron deficiency when it does occur in rheumatoid arthritis. The mere coincidence of two abnormalities does not imply any causal association.

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References

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2 Doube A, Collins A J. Anaemia in patients with arthritis: Are simple investigations helpful? *Br J Rheumatol* (in press).
3 Doube A. Anaemia and non-steroidal anti-inflammatory drugs. *Br J Rheumatol* 1988;**27**:247–8.

SIR, We were interested to read Dr Doube's comments on our paper with respect to asymptomatic peptic ulcers causing iron deficiency anaemia. We would certainly agree with him that asymptomatic peptic ulceration is common in patients taking non-steroidal anti-inflammatory drugs but we would emphasise that the gold standard for diagnosing iron deficiency in a rheumatoid patient is a bone marrow examination.[1] In our experience the measurement of faecal occult bloods is a very unreliable test of blood loss from gastrointestinal lesions. In a recent study of 58 patients with rheumatoid arthritis who underwent endoscopy for iron deficiency anaemia we found that 55% had upper gastrointestinal lesions, and after treatment for these the anaemia resolved in 20 patients. Twelve patients with normal upper gastrointestinal endoscopy results had a positive faecal occult blood test.

As far as our practice is concerned 40% of patients with rheumatoid arthritis with anaemia were iron deficient.

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References

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Absence of an association between ankylosing spondylitis and IgA nephropathy

SIR, In their recent article Peeters *et al* reported on IgA containing immune complexes in ankylosing spondylitis.[1] They stated that ankylosing spondylitis is associated with IgA nephropathy. This statement is not based on epidemiological studies, however, but on reports of the co-occurrence of ankylosing spondylitis and IgA nephropathy. Wall *et al* described an increased incidence of recurrent haematuria in 32 patients with ankylosing spondylitis, but in only one patient was the diagnosis of IgA nephropathy confirmed.[2] The report by Jones *et al* also showed an increased incidence of recurrent haematuria in patients with ankylosing spondylitis[3]: in only one of 51 patients was the diagnosis IgA nephropathy proved. Swaak *et al* found no evidence of IgA nephropathy in any of 40 patients with ankylosing spondylitis.[4] Likewise, Calin reported the absence of any evidence of IgA nephropathy in 68 patients suffering from ankylosing spondylitis.[5] From these studies it can be concluded that recurrent haematuria occurs in up to 20% of patients suffering from ankylosing spondylitis, whereas IgA nephropathy proved by biopsy is present in about 2%.

The prevalence of IgA nephropathy in general populations is estimated to be between 0·2 and 4%.[6] Thus studies of larger populations of patients with ankylosing spondylitis are needed to judge the real prevalence of IgA nephropathy in this disease.

Recently, we reviewed the data from 276 consecutive patients with ankylosing spondylitis (New York criteria) seen at our outpatient clinic between 1984 and 1987. Twenty five patients had recurrent haematuria (more than one observation of ≥10 red blood cells per high power field). In seven patients the haematuria did not reappear during follow up. In eight patients another explanation for haematuria was found (renal stones twice; urinary tract infections five times; malignant tumour of the kidney once). In five patients recurrent haematuria persisted over