Antineutrophil Cytoplasmic Antibody-Positive Polyarthritis Associated with Minocycline Therapy

Sir—Long-term minocycline therapy has been implicated in the induction of antinuclear antibody (ANA)-positive autoimmune disease: systemic lupus erythematosus-like syndrome and chronic active hepatitis [1–5]. Drug-induced lupus was first described in patients receiving the anti-hypertensive agent hydralazine [6]. Hydralazine can also provoke a systemic vasculitis with a rapidly progressive glomerulonephritis associated with autoantibodies directed against components of the neutrophil cytoplasm with specificity for lactoferrin [7]. Antibodies against lactoferrin have rarely been described in other systemic vasculitic syndromes.

A 32-yr-old male retail manager who had taken minocycline 100 mg daily for 1 yr presented with a 3 month history of sudden onset and progressive fever, malaise, early morning stiffness, symmetrical polyarthritis and weight loss (6 kg). His symptoms were so severe that he was unable to work. Examination revealed low-grade fever (37.8°C) and synovial proliferation of wrists, knees and ankles. There was no evidence of skin, respiratory, renal or neurological disease. Investigations showed blood haemoglobin 11.7 g/dl, ESR 101 mm/h, CRP 129 mg/l (<30 mg/l) and serum IgG 20.1 g/l (<16.1 g/l). Antineutrophil cytoplasmic antibodies (ANCA) with specificity for lactoferrin were detected by ELISA and indirect immunofluorescence. Rheumatoid factor and ANA were negative. Non-steroidal anti-inflammatory drugs were ineffective in controlling his symptoms. Withdrawal of minocycline therapy resulted in a dramatic and complete resolution of symptoms. He returned to work 6 weeks later. Blood haemoglobin, ESR, CRP and IgG were normal, and ANCA was negative.

To our knowledge, this is the first description of minocycline-related ANCA-positive polyarthritis. The dramatic reversal of clinical and laboratory features implicates minocycline as the causative agent. Our findings support the view that minocycline has a propensity for causing a variety of immunologically mediated events.

K. Gaffney, P. Merry
Department of Rheumatology, Norfolk and Norwich Hospital, Brunswick Road, Norwich, Norfolk NR1 3SR.

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responded poorly or not at all. In addition, when antibody levels for all 31 patients were studied, there was found to be a significant inverse correlation between antibody levels and response to drug treatment. Recently, we have enlarged this group of SASP-treated patients to 45. Analysis of response and antibody levels to this antigen preparation has confirmed the significance of this relationship [Spearman's rank correlation Rho = -0.404, P > 0.01 (Fig. 1)]. The reason for this is unclear, but immune mechanisms may be involved.

Further investigation is required to establish whether a relationship exists between antibody levels and response to other DMARDs, and whether it is specific to this antigen preparation.

Clearly, the possibility that levels of serum antibodies to bacterial antigens may be used as markers for the likelihood of response to SASP could have implications regarding the tailoring of SASP therapy.

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S. M. Bradley, H. A. Bird, H. C. Gooi*
Clinical Pharmacology Unit (Rheumatism Research), Chapel Allerton Hospital, Chapeltown Road, Leeds LS7 4SA and *St James's University Hospital, Beckett Street, Leeds LS7 9JT.
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ANCA in RA patients
Sir—I read with interest the article ‘ANCA in RA patients’ [1], and found something conflicting between the summary and the main part of the article.

First, in the third line of the summary, it said that 32% of RA patients had positive indirect immunofluorescence (IIF) stains (P or atypical ANCA), but in the last sentence of this summary, it stated that the overall incidence of ANCA in RA patients was 33% by IIF. When I read the results of the article carefully, I found no data to support the statement ‘thirty-two per cent of RA patients had positive IIF stains’.

Secondly, it said in the summary that LSRA patients were more likely to have anti-HLE antibody. In fact, the results showed that a positive reaction for circulating IgG anti-HLE antibodies was detected in 12 RA patients, including nine of the 28 RAV patients, one of the 31 LSRA patients and two of the 25 Ely RA patients. So it was RAV patients who were more likely to have anti-HLE antibody.