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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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.

The British Journal of Rheumatology is a very famous medical journal and has earned a great reputation all over the world, including China. I have been engaged in rheumatological fields for no more than 2 yr after my 2 yr intern practice in the Great Wall Hospital.

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Reply

Our colleague, Dr Le Sheng Guang, discusses two points in our paper. The first one is a typographical error, the second one is a true error. In the summary, ‘RAV’ should be read instead of ‘LSRA’.

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Treatment of Gout Following Cardiac Transplantation

SIR—After cardiac transplantation, due to the hyperuricaemic effect of the combination of cyclosporine, azathioprine and diuretics, the prevalence of gout increases 8-fold [1]. Allopurinol potentiates azathioprine by inhibiting its metabolism, which can result in potentially fatal pancytopenia [2, 3]. We describe two cases where post-transplant gout was successfully controlled by combining allopurinol with a reduced dose of azathioprine.

Case 1: Fourteen months after cardiac transplantation, a 54-yr-old male taking cyclosporine 100 mg bd, azathioprine 100 mg and frusemide 80 mg daily developed polyarthritis. Serum urate was 0.78 mmol/l, blood urea 17.3 mmol/l and serum creatinine 211 µmol/l. Gout was diagnosed and after a brief course of colchicine, allopurinol 100 mg/day was started, with a reduction in azathioprine to 25 mg/day. Daily blood counts for 10 days and periodically thereafter revealed no changes. The frusemide was reduced to 40 mg daily and then eventually stopped. Three months later, the blood urea had improved to 11.6 mmol/l, creatinine to 186 µmol/l and urate to 0.3 mmol/l. His cardiac and renal function remained stable, and he remained pain free.

Case 2: A 62-yr-old male had been treated with allopurinol since 1990, but this was stopped at the time of his cardiac transplant. Two years later, he developed an acute inflammatory polyarthritis. He was taking azathioprine 50 mg/day, cyclosporine 100–125 mg/day and frusemide 80 mg/day, his serum urate was 0.64 mmol/l, and gout was diagnosed. After a short course of colchicine, he was commenced on allopurinol 100 mg daily, increasing to 200 mg 2 weeks later. The azathioprine was reduced to 25 mg and daily blood counts for the next 10 days revealed no significant change. The serum urate fell to 0.37 mmol/l and the attacks of gout ceased. He was admitted with a further attack of gout in October 1995, when the serum urate had increased to 0.45 mmol/l, probably secondary to an increase in the frusemide. The allopurinol was increased to 300 mg daily and the frusemide reduced to its previous level. The gout attacks have ceased and he remains well.

Cyclosporine, azathioprine and diuretics are the mainstay of post-transplant regimens, but the combination is nephrotoxic and hyperuricaemic [4]. Although no general rule can be made, both cases illustrate that gout in post-transplantation patients can be successfully treated with allopurinol, providing the dose of azathioprine is reduced and careful monitoring of blood counts is performed. As azathioprine is metabolized to 6-mercaptopurine, which is metabolized by xanthine oxidase, concurrent use of the xanthine oxidase inhibitor allopurinol is potentially dangerous [2, 3]. The logical response is to reduce the dose of azathioprine, conventionally by 75% [5, 6]. Azathioprine is also metabolized by hypoxanthine guanine phosphoribosyltransferase (HGPT) and thiopurine methyl transferase (TPMT). The activity of TPMT is genetically determined and those deficient in this enzyme (~1 in 300) metabolize more via the HGPT pathway and risk immunosuppression. Assays of TPMT are now available and, although not measured here, measurement of TPMT will further improve safety [7].

It is understandable that cardiologists should be concerned primarily with the stability of the transplant, and consider that non-fatal biochemical changes are of secondary importance. The latter, however, affect not only quality of life by preventing attacks of gout, but minimize hospital admissions and additional, occasionally toxic, medication. Since transplants are now almost routine, it is time to pay more attention to these secondary issues.

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