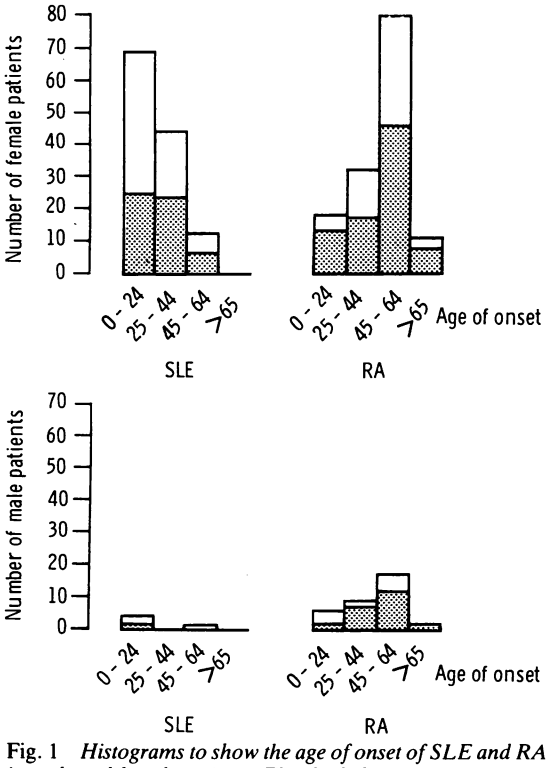

Fig. 1 *Histograms to show the age of onset of SLE and RA in male and female patients. The shaded areas represent new referrals during the two year period under study.*

HLA, may be useful in explaining these ethnic differences. In this regard the observation that insulin dependent diabetes is associated with DR3/4 in Caucasians but with DR3/9 in Orientals is of interest.

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Lymphadenopathy in rheumatic patients

SIR, The paper in the *Annals* by Drs Kelly, Malcolm, and Griffiths makes the interesting point that lymphadenopathy may be an early feature of inflammatory polyarthritis, and in their series two cases were misdiagnosed and treated by radiotherapy as cases of malignant lymphomata.[1] In the 1960s at Westminster Hospital we saw two similar cases, a 15 year follow up showing only rheumatoid disease and no malignancy. The fact that lymph node enlargement was common in relation to inflamed arthritic joints did not seem to be appreciated by my fellow examiners in the Membership examinations of the late 1960s, hence the article quoted by Kelly *et al* (Robertson *et al* 1968[2]).

A review of the case records of 35 patients treated between 1950 and 1966 at Westminster Hospital for giant follicular lymphoma by radiotherapy showed at that time no evidence of inflammatory arthritis of any sort. Of the 100 cases of rheumatoid arthritis we reported in 1968,[2] 82% had lymph node enlargement compared with 52% in the control series matched for age and sex, figures much higher than those of Short *et al* who found 29·4% in those with rheumatoid arthritis and 8·9% in controls.[3] As in our study all palpable glands were reported and an attempt was made to grade their enlargement. In our study we found glands most commonly and most enlarged in axillae, inguinal, and epitrochlear areas in relation to actively inflamed joints, but cervical nodes were present in arthritic and control subjects in equal number (26% and 23%) and were of similar size. Lymphograms done in 15 patients with rheumatoid arthritis by W F White showed non-specific inflammatory changes which, though different from malignant lymphoma, were not sufficiently distinctive to be diagnostic. We therefore considered lymph node enlargement a common finding in adult patients, in relation to inflamed joints, but only rarely part of a generalised lymphadenopathy (as in Felty's syndrome), though this is seen in young patients with chronic juvenile arthritis more often. Kelly *et al* point out that in inflammatory arthritis, lymphadenopathy may occur early on in

the disease course and may cause very real diagnostic confusion and they wisely confined their biopsies to supraclavicular and cervical areas.

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Desensitisation to allopurinol: A cautionary tale

SIR, Our recent communication describing successful reinstitution of allopurinol in a patient who was previously allergic has aroused a considerable amount of interest.[1] We feel it would be appropriate to describe a similar case where the desensitisation regimen failed and the patient suffered potentially life threatening adverse events. Allopurinol is the drug of first choice for the maintenance of patients suffering from gout. Its unique mode of action and high therapeutic index have revolutionised the long term treatment of this condition since the introduction of the drug in 1962. Adverse effects are relatively common, though seldom life threatening,[2] and skin rashes are the most frequently reported side effect. Uricosuric therapies with, if necessary, the addition of colchicine are generally reserved for those patients allergic to allopurinol. Patients with allopurinol hypersensitivity in whom these therapies have failed have been sucessfully 'desensitised'.[1 3–6] We report the case of a patient who developed a severe reaction on reinstitution of allopurinol therapy after uneventfully completing a previously successful desensitisation programme.

Case report

A 54 year old woman with a 12 year history of polyarticular gout developed an urticarial rash, swelling of the head and neck, and stridor after treatment with allopurinol four years earlier. Colchicine produced diarrhoea and she was taking azapropazone 1·2 g, indomethacin 100 mg p.r., and probenecid 1 g daily. She claimed good compliance but suffered two acute attacks of gout yearly. Her serum urate was inconsistently raised (0·27–0·61 mmol/l) during outpatient follow up and she was admitted with a gouty flare.

On admission her right forefoot and ankle were swollen, there was desquamation and erythema of the overlying skin. Aspiration of the affected joints was not attempted. Her renal function and serum lipids were normal (creatinine 90 μmol/l, urea 4·6 mmol/l, total lipids 8·1 g/l), fasting uric acid was raised (0·32 mmol/l), and *x* rays of her wrists and feet showed progression of the erosive joint damage.

In view of the failure of uricosuric therapy we instituted allopurinol desensitisation using the regimen of Fam *et al*[3] as recently described.[1] Two weeks after reaching the target dose of 300 mg/day she suffered the sudden onset of stridor, neck swelling, and a generalised itchy, lumpy rash. These symptoms were similar to those after her first exposure to allopurinol. She consulted her general practitioner who promptly stopped the drug. Within one week she was back to normal. Her eosinophil count was normal before, during, and after the desensitising doses of allopurinol (0·07, 0·08, 0·05×10/l respectively).

This regimen has been compared with the desensitisation of patients allergic to sulphasalazine; this assumes that the unknown mechanisms of hypersensitivity to the two drugs are comparable, though there is no evidence that this is so. Sulphasalazine desensitisation fails in approximately 20% of patients (A D Turner, personal communication). Although allopurinol desensitisation has not been widely used, all previous reports have been of successfully treated patients.[1 3–6] We suggest that if, as this case illustrates, serious side effects can occur without warning weeks after reinstitution of therapy, then the nature and severity of the original adverse event must be taken into consideration before embarking on such a course.

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