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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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References


Desensitisation to allopurinol: A cautionary tale

SIR. Our recent communication describing successful re-institution of allopurinol in a patient who was previously allergic has aroused considerable 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, 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 successfully ‘desensitised’.1 3-6 We report the case of a patient who developed a severe reaction on re-institution 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 azaproprazone 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 umol/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 as recently described.1 Two weeks after reaching the target dose of 0.3 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 x 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 re-institution 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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