Drug points

Eczema after intravenous infusion of immunoglobulin

Drs C Baruch and J C McMillan (Belfast City Hospital, Belfast BT4 7DA) write: The use of intravenous immunoglobulin in high doses has become an accepted treatment for patients with autoimmune thrombocytopenia. The intravenous globulin concentrate prepared by the Swiss Red Cross (Sandoglobulin) has been evaluated in the laboratory,1 and only a few minor adverse reactions have been reported in clinical trials2; the Committee on Safety of Medicines has reported no records of adverse reactions. We report the occurrence of severe eczema after infusion of Sandoglobulin.

The patient was a 75 year old woman who fulfilled the criteria for idiopathic thrombocytopenic purpura when thrombocytopenia was first noted in 1982. After initially responding to steroids she became refractory, and Sandoglobulin infusions (1 g/kg daily for two days) were started in April 1986. No adverse reactions were noted, and her platelet count rose from 6 × 10^10/L to 15 × 10^10/L. She received a second course of Sandoglobulin seven weeks later, but at review one month after the infusion she complained of a rash which had appeared one week previously and had been diagnosed by her general practitioner as "allergic." She was successfully treated with topical steroids. Calamine provided no relief, but she was no longer short of breath. She was given a third course of Sandoglobulin after another interval of six weeks, and at review two weeks after the infusion she had a florid generalised eczematous eruption. Calamine had no effect. The clinical appearance was consistent with acute excoriated eczema, and a skin biopsy specimen suggested a drug reaction.

Cutaneous reactions to food, drug, and other exogenous antigens are associated with blood products, while eczematous reactions are rare. Cutaneous reactions after infusion of hydroxyethyl starch have been reported.3 The manufacturers of Sandoglobulin have received one report of a case from the United States, in which sensitisation to porcine pepino (1:1000) to prevent thrombosis was suspected to be due to previous exposure to insulin (EM Thompson, personal communication). Despite extensive studies we found no proof of a humoral or cellular mechanism. Our patient has required several additional infusions of Sandoglobulin, which were given with concomitant administration of hydrocortisone and chlorphenamine together with topical steroid applications. The patient has subsequently been successfully treated with excoriation and moisturiser, with each infusion, although she continues to suffer from widespread eczema.


Calcinus antigoni and psoriasis

Drs C C Harland, C M E Rowland Payne, S MNeill, and C W M Cooper (Westminster Hospital, London SW2) write: Exacerbations of psoriasis are an important adverse effect of β blocker treatment, as Dr J Savola and colleagues (12 September, p 673) recently reported. We have observed similar exacerbations after withdrawal of β blockers in an unspecified number, however, β blockers were replaced by calcium antagonists. As calcium is implicated in the pathogenesis of psoriasis this may be pertinent. The interest in calcium and psoriasis stems from anecdotal reports. Hypocalcaemia, independent of serum albumin, has been associated with exacerbations of generalised purpuritic psoriasis.1 Generalised purpuritic psoriasis has also been precipitated by parathyroidectomy.2 In both instances the condition improved with calcium replacement.