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Allergic cross-reaction of teicoplanin and vancomycin

Sir,

Teicoplanin is a glycopeptide antibiotic similar to vancomycin, but differs in its possibly lower toxicity and its availability by intramuscular administration. The use of teicoplanin as an alternative therapy for patients allergic to vancomycin is not yet clearly defined. Thirteen cases in which teicoplanin was safely given to patients who were allergic to vancomycin (Van Laethem *et al.*, 1984; Schlemmer *et al.*, 1988; Smith *et al.*, 1989; Wood & Whitby, 1989) and one case of allergic cross-reactivity of teicoplanin and vancomycin (McEralth, Goldberg & Neu, 1986) have been previously reported.

A 52-year-old man who had a refractory low grade non-Hodgkin's lymphoma with 85% bone marrow infiltration was treated with aggressive chemotherapy. He developed a pyrexia without evidence of infection and was empirically treated with piperacillin and vancomycin. After 48 hours, the piperacillin was changed to ceftazidime. 24 h later, he was apyrexial. Ten days later, his temperature rose again and diffuse erythematous cutaneous lesions appeared, increasing after each vancomycin perfusion. Vancomycin was discontinued and over the next 24 h, the fever and the rash resolved. Nine days later, the patient developed a *Staphylococcus epidermidis* septicaemia. He was initially treated empirically with amikacin and cefotetan, but after bacteriological documentation of his infection with imipenem, rifampicin and doxycycline. His fever persisted and teicoplanin was given by slow intravenous administration (400 mg in 2 h). The patient immediately had a rash followed by dyspnoea and severe bronchospasm, which resolved with the discontinuation of teicoplanin and the intravenous administration of methylprednisolone. He became apyrexial after granulocyte transfusions.

Our case confirms the existence of allergic cross-reactions between of teicoplanin and vancomycin. From the literature, the incidence of such cross-reactions seems to be low, which could be explained by the relatively low incidence of allergic reaction to vancomycin coupled with limited use of teicoplanin as an alternative to vancomycin. Our report also shows that reactions to teicoplanin can be severe. In conclusion, caution should be exercised before using teicoplanin in a patient allergic to vancomycin.

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Cephalexin induced toxi

Sir,

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Cephalexin induced toxic epidermal necrolysis

Sir,

A previously healthy 61-year-old woman was prescribed cephalexin by her general practitioner for an upper respiratory tract infection. Two days later she developed a pruritic, erythematous rash over her trunk and limbs and the cephalexin was stopped. After a further three days she was admitted to hospital confused, pyrexial (temperature 40°C), hypotensive and dehydrated. Over the next 72 h she developed ulceration involving 75% of her skin surface area including lips and genitalia, and despite intravenous fluid replacement and vasoactive drugs, developed polyuric acute renal failure, hepatic dysfunction, fits, acute respiratory failure requiring ventilation and disseminated intravascular coagulation. A skin biopsy confirmed the clinical diagnosis of toxic epidermal necrolysis.

Her overall condition improved with general intensive care (including haemofiltration and parenteral nutrition) but her course was complicated by deteriorating liver function and the development of an *Enterococcus faecium* septicaemia, treated with vancomycin and netilmicin. Her skin condition was managed conservatively with saline bathing, silver sulphadiazine (flamazine) and paraffin gauze (Jelonet) dressings. Four weeks after admission to the Intensive Care Unit the skin had almost recovered. Five weeks after admission she developed acute, irreversible bronchospasm of unknown aetiology, such that it was impossible to ventilate her. She had a cardiac arrest related to hypercapnoea (arterial carbon dioxide tension 30 kPa) and died. At autopsy she was found to have suppurative cholangitis and this was presumed to be the source of her septicaemia.

Toxic epidermal necrolysis (TEN) is characterized by epidermal necrosis and skin peeling due to sub-epidermal or intra-epidermal splitting (Swartz, 1990). It can masquerade as the staphylococcal scalded skin syndrome and has been associated with a reaction to a number of drugs, in particular sulphonamides and barbiturates (Swartz, 1990). The incidence of skin rash following cephalexin therapy is reported to be from 0·6–1·1% (Ueda, 1969; Anonymous, 1970; Dash *et al.*, 1972; Burt, 1983), but the exact type of these skin reactions is not clearly recorded. We know of only one other definite case of TEN associated with cephalexin (Hogan & Rooney, 1987) and one possible case in which TEN occurred after cephalexin and thioridazine therapy (Harnar, Dobke & Simoni, 1987). Both of these cases occurred in the United States. Our patient received only cephalexin before developing TEN and we believe it to be the first reported case of cephalexin-induced TEN in the United Kingdom.

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