Correspondence


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 (McEerath, 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 afebrile. 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 ceftazidime, 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 afebrile 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 teicoplanin 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.

Sir,

A previously healthy 61 year old male presented with cephalaxin induced toxic reaction for an upper resp tract infection. Two days later she developed drowsiness and rash. She was discharged and re-presented on the 3rd day with a 10-day history of rash and fever. She was afebrile and had a well-distributed maculopapular rash. There was no evidence of gastrointestinal upset or renal failure. Hepatic and respiratory systems were normal.

She was admitted to the hospital and treatment was initiated with intravenous hydration, antihistamines and a short course of oral prednisone. The rash resolved and she was discharged on the 5th day.

Cephalaxin induced toxic reation in a healthy individual.

Sir,

A previously healthy 61 year old male presented with cephalaxin induced toxic reaction for an upper resp tract infection. Two days later she developed drowsiness and rash. She was discharged and re-presented on the 3rd day with a 10-day history of rash and fever. She was afebrile and had a well-distributed maculopapular rash. There was no evidence of gastrointestinal upset or renal failure. Hepatic and respiratory systems were normal.

She was admitted to the hospital and treatment was initiated with intravenous hydration, antihistamines and a short course of oral prednisone. The rash resolved and she was discharged on the 5th day.

Cephalaxin induced toxic reation in a healthy individual.
Correspondence

V. GREK
F. ANDRIEN
J. COLLIGNON
G. FILLET
Department of Hematology.
C.H.U. Sart-Tilman, 4000 Liège, Belgium

References

Cephalexin induced toxic epidermal necrolysis

Sir,

A previously healthy 61-year-old woman was prescribed cephalexin by her general practi-
tioner for an upper respiratory tract infection. Two days later she developed a pruritic, ery-
thematosus 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), hypo-
tensive and dehydrated. Over the next 72 h she de-
veloped 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 deterioration liver function and the development of an Enterococcus faecium sepsicaemia, treated with vancomycin and netilmicin. Her skin condition was managed conservatively with saline bathing, silver sulphadiazine (famazine) and paraffin gauze (Jetone) dressing. Four weeks after admission to the Intensive Care Unit the skin had almost recovered. Five weeks after admission she developed an 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 supplicative cholangitis and this was presumed to be the source of her sepsicaemia.

Toxic epidermal necrolysis (TEN) is character-
ized 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 reac-
tions is not clearly recorded. We know of only one other definite case of TEN associated with cephalexin (Hogn & Rooney, 1987) and one possible case in which TEN occurred after cephalaxin and thiouridine 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.

J. DAVE
R. HEATHCOCK
L. FENELON
D. J. BLENAV
N. A. SIMMONS

*Public Health Laboratory, The William Harvey Hospital, Kennington Road, Wellingborough, Northants
\Department of Clinical Bacteriology and Virology and Intensive Care Unit, Guy's Hospital, Guy's Tower, St Thomas' Street, London SE1 9RT, UK

References
Burt, R. A. P. (1983). A review of the drug events reported by 12,917 patients treated with cepha-
lexin. Postgraduate Medical Journal 59, 47-56.