Should clindamycin be used in treatment of patients with infections caused by erythromycin-resistant staphylococci?

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Sir

I read the study by Panagea et al.1 with great interest. The researchers speculate about the relevance of rapid in vitro development of resistance to actual clinical response when patients infected with Staphylococcus aureus isolates exhibiting inducible resistance to clindamycin (ICR) are treated with clindamycin.

In this correspondence I describe three patients in our hospital who were recently treated with clindamycin for infections with methicillin-resistant Staphylococcus aureus (MRSA) exhibiting ICR.

Patient 1, a 76 year-old man, was admitted to hospital for investigation of a possible right iliac fossa mass. The patient was reviewed when he continued to have a spiking temperature for 48 h despite empirical treatment with piperacillin and gentamicin. On examination, the venflon insertion site in the right cubital fossa was discharging pus, with cellulitis extending to the forearm and upper arm. Blood cultures taken earlier became positive at the time of referral and a Gram’s stain showed the presence of Gram-positive cocci, suggestive of staphylococci. Treatment with flucloxacillin was commenced but discontinued within 24 h when blood, and wound cultures from the venflon site, were positive for MRSA, exhibiting ICR. Clindamycin (300 mg administered every 6 h) was given intravenously for 4 days and orally for a further 12 days. The patient did not develop any diarrhoea during treatment. The response to the treatment was excellent with resolution of cellulitis and fever. Wound swabs taken during and at the conclusion of the treatment showed scanty growth of MRSA, which continued to exhibit ICR.

Patient 2, a 60 year-old woman, was admitted with cellulitis of the left leg extending from the ankle to the knee. Wound cultures were taken and empirical treatment with flucloxacillin and benzyl penicillin was commenced. MRSA exhibiting ICR was isolated from the wound swabs. Benzyl penicillin and flucloxacillin were replaced by clindamycin (300 mg administered every 6 h) given intravenously for 2 days and orally for a further 12 days. At the end of treatment, the cellulitis was completely resolved and wound swabs did not yield any MRSA. The patient did not develop any diarrhoea during treatment.

Patient 3, an 85 year-old woman, was admitted with spiking fever and a pelvic abscess probably secondary to carcinoma of the colon. Blood cultures taken during a febrile episode yielded MRSA exhibiting ICR. Further clinical examination revealed that she had extensive cellulitis and a long-standing sinus on the lower part of her back. Swabs from this area also grew MRSA with the same antibiotic sensitivities. Treatment was commenced with clindamycin (300 mg administered every 6 h) given intravenously for 2 days and orally for a further 12 days. After a few days of treatment the fever subsided, the cellulitis resolved completely and the patient’s general condition improved remarkably. Unfortunately, a fortnight later the patient once again developed pyrexia. Blood cultures taken on that occasion yielded MRSA, which were completely resistant to clindamycin. The patient is currently being treated with vancomycin. The patient did not develop any diarrhoea during clindamycin treatment.

The first two patients and possibly the third had a satisfactory clinical outcome to the clindamycin treatment despite a variable bacteriological response.

The reason for this is not clear. It is conceivable that favourable pharmacokinetics, the intra-phagocyte concentrations and the ability of clindamycin to inhibit staphylococcal toxins all play a role in alleviating the clinical manifestations of MRSA infection.2–4

In the light of the restricted range of antibiotics available for the treatment of MRSA and the known limitations of vancomycin, clindamycin should be considered for the management of serious soft tissue infections with MRSA that are either sensitive to clindamycin or exhibit ICR. Provided the patients are monitored closely, fear of development of clindamycin resistance or pseudomembranous colitis should not discourage clinicians from using this antibiotic.5,6

References


