Successful treatment of MRSA endocarditis in a haemodialysis patient

Sir,

Infective endocarditis is a recognized complication of haemodialysis and is often associated with access-line related sepsis [1]. In spite of the increasing dependence on such lines for dialysis access and the increasing prevalence of methicillin resistant Staphylococcus aureus (MRSA) as a pathogen in renal patients, MRSA endocarditis is still rare in this population [2].

Case. A 65-year-old man on haemodialysis developed acute loss of function of his fistula and a vascatheter was inserted for temporary dialysis access. Two weeks later an exit site infection was noted. The area was swabbed, blood cultures taken and the line replaced. Intravenous vancomycin treatment commenced. The following day the patient became pyrexial and admitted to hospital. The previous day’s culture grew MRSA. Regular i.v. vancomycin was continued (500 mg after dialysis three times a week) and oral fuscidic acid was added. In spite of these measures he remained pyrexial and blood cultures continued to grow MRSA (Figure 1). A trans-thoracic echocardiogram showed no vegetations. Fuscidic acid resistance developed on the tenth day. The drug was stopped and gentamicin added. All lines were removed and intermittent femoral access was instituted for each dialysis session. He remained pyrexial and positive blood cultures persisted. He developed a widespread bilateral cavitating pneumonia. Bronchio-alveolar lavage grew MRSA. Regular monitoring of the minimum inhibitory
concentration and minimum bactericidal concentration of antibiotics for the organism suggested insufficient antibacterial activity with vancomycin and gentamicin so i.v. rifampicin was added. He developed acute mitral regurgitation. Transoesophageal echocardiogram (TOE) confirmed vegetations on the mitral valve leaflets. Medical treatment continued and it was not until the 28th day that the blood cultures were finally sterile. The rifampicin was converted to oral therapy, but a small rise in C reactive protein (CRP), increased temperature and a suboptimal serumcidal titre (back titration), necessitated the reversion to the i.v. route. Treatment continued with three i.v. antibiotics until the CRP and temperature were normal. This took a further two full weeks of treatment (Figure 1). Repeat chest X-ray and TOE at 3 months showed clear lung fields and resolution of vegetations. Two years later his mitral incompetence continues though he has minimal cardiac symptoms.

Comment. MRSA endocarditis is well recognized and often occurs in association with i.v. drug abuse. The condition is still a rarity in the haemodialysis population but given the increased use of central venous catheters for dialysis access and the increasing prevalence of MRSA colonization and infection in most renal units, its incidence is likely to increase. MRSA endocarditis can be a more indolent infection than endocarditis due to methicillin sensitive Staphylococcus aureus depending on the phage type of each, but MRSA endocarditis tends to be seen in more vulnerable subpopulations. The clinical outcomes of infection with either organism are similar [3]. The patient reported had MRSA phage type 15, one of the endemic MRSA infections seen in British hospitals.

Our case illustrates some typical features [4]. The response to treatment can be protracted. Successful treatment usually requires the use of antibiotic combinations tailored to the individual’s bacteraemia by tight microbiological monitoring. Glycopeptide tolerance can be a problem [5]. The protracted course does not seem to affect the outcome adversely. Our case illustrates that continued conservative treatment could be successful even in the context of acute valvular incompetence.

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