Case Report

Bacteraemia associated with a vancomycin-resistant *Enterococcus gallinarum* strain harbouring both the *vanA* and *vanC1* genes

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A case of a post-surgical patient who developed a fatal bloodstream infection caused by high-level vancomycin-resistant *Enterococcus gallinarum* is reported. The isolate was found to carry both the *vanC1* and *vanA* genes. This is the first report of an invasive infection associated with a *vanA* *E. gallinarum* isolate in Brazil.

Introduction

Motile enterococcal species, *Enterococcus gallinarum* and *Enterococcus casseliflavus*, usually carry the *vanC1* or *vanC2* gene, respectively, and exhibit low-level intrinsic resistance to vancomycin (Teixeira & Facklam, 2003). Together these species are responsible for about 1–2% of all enterococcal infections in humans. *E. gallinarum* strains have been recovered from different sources, but their association with clinically significant infections, including bacteraemia and endocarditis, has rarely been documented. The presence of *vanC* genes, which confer low-level vancomycin resistance, is not considered an indication for strict patient isolation precautions. However, coexistence of the *vanC1* genotype with the *vanA* genotype may represent an additional threat for the control of enterococcal infections (Ratanasuwan *et al.*, 1999; Reid *et al.*, 2001; Dargere *et al.*, 2002).

We describe a case of bacteraemia caused by a high-level vancomycin-resistant *E. gallinarum* isolate. This is the first report of infection caused by a motile *Enterococcus* isolate harbouring both the *vanC1* and *vanA* genes in Brazil.

Case report

A 70-year-old man was admitted to hospital for surgical repair of an abdominal aortic aneurysm. The patient had a background history of cerebrovascular accident, tabagism and alcoholism. After aneurysmectomy was performed, he was transferred to the intensive care unit (ICU) and subsequently he had post-operative epileptic seizures and became comatose. On the second post-operative day, the patient presented ischaemic complications with lower extremity ischaemia due to embolization. Despite embolectomy, he ultimately came to above-knee amputation. On

the eighth post-operative day, the patient became febrile and an *Acinetobacter* isolate was recovered from a tracheal aspirate specimen. The following antimicrobials were used for treatment while the patient was in the ICU: amikacin, cefepime, ceftazidime and ceftriaxone. On the 20th post-operative day, the patient's clinical condition deteriorated and was complicated by acute renal failure. The antibiotic regimen was then shifted to vancomycin and meropenem. Three consecutive cultures of peripheral blood were drawn within a 24 h period, and revealed *E. gallinarum* and *Acinetobacter* sp. growth. The patient died on the 25th post-operative day due to sepsis, renal failure and surgery complications.

The *E. gallinarum* isolate was identified by conventional physiological tests (Teixeira & Facklam, 2003). Results of disc diffusion tests indicated that the *E. gallinarum* isolate was susceptible to ampicillin, fosfomicin, linezolid, nitrofurantoin and high levels of streptomycin, showed intermediate resistance to ciprofloxacin, norfloxacin and rifampicin, and was resistant to chloramphenicol, erythromycin, teicoplanin, tetracycline and vancomycin, according to the guidelines provided by the Clinical and Laboratory Standards Institute (2005). The isolate was also high-level resistant to gentamicin (HLR-GE) and harboured the *aac(6')-aph(2'')-Ia* gene, as detected by PCR performed according to Swenson *et al.* (1995). Determination of MIC values by the E-test (AB Biodisk) revealed the occurrence of high-level glycopeptide resistance ($>256 \mu\text{g ml}^{-1}$ for vancomycin and $96 \mu\text{g ml}^{-1}$ for teicoplanin). PCR assays for detection of vancomycin-resistance genes were performed with primers pairs previously defined (Satake *et al.*, 1997) and showed that the *E. gallinarum* strain harboured both the *vanC1* and *vanA* genes.

Discussion

First reported in the late 1980s, high-level vancomycin-resistant enterococci (VRE) have rapidly become established as important nosocomial pathogens. The origins of the vancomycin resistance-associated genes in enterococci are unknown, but the selective pressures favouring the emergence of VRE are better understood. Results of different investigations have shown an association between colonization with VRE and previous oral or parenteral administration of vancomycin. In addition, an association between VRE and use of antimicrobial agents other than vancomycin, such as cephalosporins or metronidazole, has been demonstrated (Carmeli *et al.*, 1999).

The present report illustrates the need to increase awareness of invasive diseases due to motile enterococcal species. Additionally, our findings documenting the occurrence of a motile enterococcal isolate carrying the *vanA* gene, and the association of such a characteristic with HLR-GE, suggest that these micro-organisms may also represent additional and important reservoirs of resistance genes in the nosocomial setting. Corso *et al.* (2005) showed that *E. gallinarum* clinical isolates were capable of capturing *vanA* elements and transferring them to an *Enterococcus faecium* reference strain, reinforcing that strict control measures should be taken in hospitals where bacteria act as reservoirs of unusual resistance genotypes. In Brazil, faecal colonization by high-level vancomycin-resistant *E. gallinarum* has already been described (Camargo *et al.*, 2004) but, to the best of our knowledge, this is the first documented case of infection.

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