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 ceftiraxone. 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, fosfomycin, 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 μg ml\(^{-1}\) for vancomycin and 96 μg 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 harbouring both the vanC1 and vanA genes.

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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 microorganisms may also represent additional and important reservoirs of resistance genes in the nosocomial setting. Corso et al. (2005) showed that Enterococcus 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 Enterococcus gallinarum has already been described (Camargo et al., 2004) but, to the best of our knowledge, this is the first documented case of infection.

References


