Community-acquired vancomycin-resistant Enterococcus faecium: a case report from Malaysia

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Vancomycin-resistant enterococci (VRE) are formidable organisms renowned for their ability to cause infections with limited treatment options and their potential for transferring resistance genes to other Gram-positive bacteria. Usually associated with nosocomial infections, VRE are rarely reported as a cause of community-acquired infection. Presented here is a case of community-acquired infection due to vancomycin-resistant Enterococcus faecium. The patient had been applying herbal leaves topically to his cheek to treat a buccal space abscess, resulting in a burn of the overlying skin. From pus aspirated via the skin a pure culture of E. faecium was grown that was resistant to vancomycin with a MIC of 256 µg ml⁻¹ by the E test and resistant to teicoplanin by disc diffusion, consistent with the VanA phenotype. The organism was suspected of contaminating the leaf and infecting the patient via the burnt skin. This case highlights the need for further studies on the community prevalence of VRE among humans and animals to define unrecognized silent reservoirs for VRE, which may pose a threat to public health.

Introduction

The emergence of antimicrobial-resistant organisms, including vancomycin-resistant enterococci (VRE), poses a challenge to clinicians due to the lack of effective antimicrobial agents available against them and associated infection-control implications. VRE were first reported in Europe in 1988, and have since emerged as a global threat to public health. Increasingly recognized as nosocomial pathogens, they tend to colonize or infect severely debilitated hospitalized patients on prolonged courses of antibiotics (Kapil, 2005; D’Agata et al., 2001). Community-acquired infections with VRE, however, have rarely been described (Coque et al., 1996). There are five phenotypes of VRE, i.e. VanA, VanB, VanC, VanD and VanE, recognized by their susceptibility pattern to vancomycin and teicoplanin (Cetinkaya et al., 2000). We report here a case of community-acquired buccal space abscess caused by vancomycin-resistant Enterococcus faecium from the University of Malaya Medical Center, Kuala Lumpur, Malaysia.

Case report

A 38-year-old man presented to the Accident and Emergency Unit of the University of Malaya Medical Center, Kuala Lumpur, with a 3 day history of pain and swelling of the right cheek associated with pain in the right upper and lower teeth. There was no significant past medical history. A firm believer in traditional medicine, he had initially sought the advice of a ‘bomoh’ (the local term for a traditional medicine man) and had been given some herbal leaves to apply over his right cheek, following which he had developed further pain, redness and a burnt area over the affected cheek skin.

On examination, the man was febrile, with a temperature of 38.4 °C. There was a swelling over the right cheek area extending from the right zygomatic arch to the right lower border of the mandible. The overlying skin was inflamed, with a burnt, black and dry central area, but no apparent sinus was visible. The patient was unable to fully open his mouth, and palpation revealed the swelling to be tender and fluctuant. A clinical diagnosis of right buccal abscess of the cheek secondary to infected molar roots with cellulitis of the cheek was made, and an orthopantomogram revealed radiolucency over the right mandibular molar alveolar regions suggestive of retained roots. His haemoglobin level was 14.9 g d⁻¹ and white blood cell count was 12.3 × 10⁹ l⁻¹ (82 % neutrophils, 11 % lymphocytes). The renal function was normal.

The patient was admitted to the Ear, Nose and Throat ward and intravenous cefuroxime (750 mg) and metronidazole (500 mg) were administered every 8 h. Aspiration of the swelling under local anaesthetic was also carried out in the ward. About 5 ml of pus was aspirated externally via the right

Abbreviation: VRE, vancomycin-resistant enterococci.
cheek and another 5 ml was aspirated intraorally via the right buccal mucosa, and the specimens were sent for culture and antimicrobial susceptibility testing.

The patient’s condition improved dramatically and by the next day, the fever had subsided, the size of the swelling had decreased and the patient was able to open his mouth. He was discharged with instructions to complete a weeks’ course of oral cefuroxime and metronidazole.

**Microbiological investigations**

The two pus specimens received at the Microbiology Laboratory of the University of Malaya Medical Centre were processed using standard laboratory procedures, and susceptibility testing was in accordance with the NCCLS disc diffusion method (NCCLS, 2004).

The intraorally aspirated pus grew *Streptococcus milleri* that was sensitive to penicillin, erythromycin, clindamycin, imipenem and vancomycin. The pus aspirated externally via the right cheek grew *E. faecium*, which was identified using standard biochemical tests (Cetinkaya et al., 2000) and the API Strept kit (Biomerieux). This was resistant to ampicillin, erythromycin, clindamycin, teicoplanin and vancomycin, and susceptible to amoxycillin clavulanate, imipenem and linezolid. High-level resistance to gentamicin was tested for, using the 120 μg gentamicin disc; however, resistance was not detected. MIC testing using the E test (AB biodisk) performed according to the manufacturer’s guidelines on Mueller–Hinton agar revealed a vancomycin MIC of >256 μg ml⁻¹. PCR amplification revealed the presence of the *vanA* gene.

By the time the laboratory had confirmed isolation of VRE, the patient had already been discharged from hospital. He remains well to today.

**Discussion**

The UK, France, Turkey and Malaysia are among the many countries that have reported infection or colonization with VRE, and the spectrum of documented infections includes endocarditis, thrombophlebitis and bacteraemia (Riley et al., 1996; Babcock et al., 2001; Basustaoglu et al., 2001). Community-acquired VRE infections are scarce though, and the only published reports to date have been of urinary-tract infections (Aznar et al., 2004; Taneja et al., 2004). To our knowledge, this is the first community-acquired soft-tissue infection caused by VRE in the literature.

Scientists and researchers have considered the mechanism for the development of vancomycin resistance in *E. faecium*. Possible contributing factors include the frequent parenteral use of vancomycin to treat methicillin-resistant *Staphylococcus aureus* and penicillin-resistant enterococcal infections, as well as the oral use of vancomycin for the treatment of *Clostridium difficile* enterocolitis. Coque et al. (1996) have established that large numbers of VRE may be isolated from hospitalized patients after glycopeptide administration and that this may result in an increased risk of nosocomial transmission as well as spread of vancomycin resistance to other species of bacteria in the gut. The use of avoparcin in animal husbandry in Europe has also been linked to the emergence of VRE, which can reach humans via the food chain (WHO, 1997). Other glycopeptides like teicoplanin and ristocetin, and non-glycopeptide agents such as bacitracin, polymixin B and robenidine (used in the treatment of coccidial infections in poultry) can also induce vancomycin resistance (Lai & Kirsch, 1996). Other farm animals or pets, including horses, dogs, chickens and pigs, may also be potential sources of VRE (Cetinkaya et al., 2000).

The source of the VRE in our patient was not identified, but this patient had no direct exposure to livestock or pet faeces. However, we suspect that the source of the VRE might have been the medicinal leaf that the ‘bomoh’ applied to his outer cheek, which possibly was contaminated with VRE from a community source and infected the cheek through skin burns caused by the leaf. Traditional medicine from a ‘bomoh’ is a form of alternative medicine available in Malaysia that is especially popular among the rural community. In this case, the leaf itself caused a burn on the patient’s skin, which probably led to the entry of the VRE. Also evident is the need for further studies on the community prevalence of VRE among both the animal and human population in Malaysia to define the extent of VRE in the community.

Typically resistant to multiple antimicrobial agents, VRE infections present a therapeutic challenge due to the limited treatment options available. Penicillin or ampicillin with or without a synergistic option (aminoglycosides) is a favourable choice in cases of penicillin-susceptible VRE. Published reports have also described successful treatment of VRE infections with teicoplanin and linezolid (Cetinkaya et al., 2000; Babcock et al., 2001). Our patient responded to drainage of the pus and treatment with cefuroxime and metronidazole. As these antimicrobials are not effective against enterococci, it is likely that the VRE infection of the cheek resolved with surgical drainage without the need for specific anti-VRE antimicrobial therapy.

It is worrying that we have detected VRE with the *vanA* gene, which, along with the *vanB* gene, is plasmid-borne and hence readily transferable from organism to organism (CDC, 1999). This would be especially menacing if spread were to occur to methicillin-resistant *Staphylococcus aureus*, as it would result in highly limited treatment options. Conjuga-tive transfer of the *vanA* gene from enterococci to *S. aureus* has been demonstrated in a laboratory setting (Noble et al., 1992), and since 2002, three cases of vancomycin-resistant *S. aureus* possessing the *vanA* gene have been reported from clinical settings (Chang et al., 2003; Tenover et al., 2004; CDC, 2004). In the first case, the *vanA* gene from the vancomycin-resistant *S. aureus* was postulated to have originated from VRE that were also isolated from the patient...
(Chang et al., 2003). The implications of this finding have been the cause for much concern among scientists and clinicians.

Infection-control measures are always a priority when VRE is isolated from a hospitalized patient, but in this case, by the time we had confirmed VRE in the pus sample, the patient had already been discharged. It is worrying that the patient had been in the open ward for 2 days, providing a silent reservoir for the dissemination of VRE. No screening of patients was done, and there is a possibility that transmission to asymptomatic carriers may have occurred.

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References


