ISOLATION OF VANCOMYCIN-RESISTANT ENTEROCOCCI IN QUEENSLAND, CASE 1

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Abstract

Vancomycin-resistant enterococci are increasingly being reported from many parts of the world. We describe a case of peritonitis with *Enterococcus faecium* exhibiting the *van A* phenotype. The organism was resistant to vancomycin, teicoplanin, amoxycillin and high levels of streptomycin. Rectal swabs from more than 25 other patients who were in the hospital at the same time were negative. No staff members were found to be colonised. Infection control measures were effective in preventing the spread of the resistant *Enterococcus faecium.* Regular surveillance of enterococcal isolates and faecal specimens or rectal swabs of patients at high risk may be justified to determine the level of vancomycin resistance in Australian hospitals. ***Comm Dis Intell* 1996; 20; 400-40**1.

Introduction

Enterococci are common nosocomial pathogens and are intrinsically resistant to a large number of antibiotics. Amoxycillin is the drug of choice for most infections, with vancomycin being used in cases of amoxycillin resistance or penicillin allergy. If there is amoxycillin and vancomycin resistance, teicoplanin is usually the only readily available alternative. However, enterococci with the *van A* phenotype, resistance to teicoplanin as well as vancomycin exists.

Vancomycin-resistant enterococci (VRE) have been described in Europe and the United States of America since the late 1980s. Three phenotypes (*van A*, *van B* and *van C*) are recognised. The first case in Australia was described at the Australasian Society for Infectious Diseases meeting in Darwin in May 1995[1]. Few cases have been detected in Australia since then.

We describe a case of infection with *van A* phenotype vancomycin-resistant *Enterococcus faecium* in a patient in Queensland. We also report on an investigation into carriage by staff members and other patients.

Case report

A 65 year old man with a history of end-stage renal failure (treated with chronic ambulatory peritoneal dialysis) presented with peritonitis in August 1996. He had a history of hypertension and aortic stenosis requiring prosthetic heart valve replacement. He had not received medical care outside Queensland, nor had he been accommodated near patients from interstate or overseas. Several courses of vancomycin had been required as empiric therapy for suspected peritonitis.

Peritoneal fluid bags grew *Bacteroides fragilis*. A perforated diverticular abscess was suspected and laparotomy performed. The peritoneal fluid obtained during laparotomy grew *Enterococcus faecium*, methicillin-resistant *Staphylococcus aureus, Lactobacillus* species and *Clostridium perfringens.*

Enterococcus faecium was identified according to the following criteria: nonmotile, nonpigmented, catalase negative Gram-positive cocci, Lancefield group D, growth in 6.5% NaCl, pyrrolydonylarylamidase positive, pyruvate negative. Both API Strep and Vitek GPI identified the organism as *Enterococcus faecium*.

The organism was resistant to amoxycillin (no zone with 10 mcg disc using NCCLS methods) and penicillin minimal inhibitory concentration (MIC) >64 mg/L by E test. It was also resistant to vancomycin (MIC >256 mg/L by E test) and teicoplanin (MIC 32 mg/L by E test). Thus the organism meets the description of the *van A* phenotype. High level resistance to streptomycin was demonstrated (MIC >2,000 mg/L). There was no high level resistance to gentamicin. The organism was resistant to ciprofloxacin (no zone with 5 mcg disc) and trimethoprim-sulphamethoxazole (no zone with 1.25/23.75 mcg disc). A 33 mm zone was found with pristinamycin (15 mcg disc).

The patient was transferred to a single room. Disposable gloves and a plastic apron were worn by doctors and nurses entering the room. A stethoscope, sphygmomanometer and thermometer were dedicated to the room.

Rectal swabs were collected from all patients who had been in the same ward as the index patient. These were plated onto blood agar containing vancomycin (3 mg/L), colistin (7.5 mg/L), nystatin (12,500 IU/L) and gentamicin (8 mg/L). None of the six patients tested was positive. Rectal swabs from twenty other renal unit patients were negative for VRE. A rectal swab obtained subsequently from the index patient, plated onto the antibiotic supplemented blood agar grew the resistant *Enterococcus faecium*.

Environmental samples collected from the bed, door handle and drawers around the patient were negative for *Enterococcus faecium*.

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Medical, nursing, paramedical and environmental services staff working in the patient's ward were tested for hand carriage of the resistant enterococcus. A modified glove fluid method was used. Twenty millilitres of trypticase soy broth with 2% Tween 80 was placed in a plastic bag. The hands of the staff member were placed individually in the broth and massaged externally for 30 seconds. The broth was then plated onto blood agar. Thirteen staff members were tested. None had hand carriage of VRE. One registered nurse was found to have hand carriage of a vancomycin-resistant catalase negative Gram-positive coccus which grew on, and blackened, bile aesculin plates. This organism was subsequently identified as *Leuconostoc* species.

Discussion

It is not unexpected that infections with vancomycin-resistant enterococci are being found in Australian patients. It is probable that other patients who have been colonised have not yet been detected. What can be done to identify such patients?

Guidelines produced by the Hospital Infection Control Practices Advisory Committee in the United States of America recommend that even hospitals without known cases should monitor for VRE[2]. This can be done by periodic testing of enterococcal isolates for vancomycin resistance. In addition, periodic screening of rectal swabs (or faecal specimens) from high risk patients can be performed. High risk patients include those in intensive care units, those with end-stage renal failure and those with a history of vancomycin usage. Patients treated with chronic ambulatory peritoneal dialysis are frequently given vancomycin as empiric treatment for peritonitis or catheter exit site infections. They become exposed to low levels of vancomycin for prolonged periods, thus creating an environment for the development of vancomycin resistance.

As *van A* or *van B* phenotypes can be induced by vancomycin use, there is logic in restricting vancomycin use to prevent the development of VRE. Vancomycin use should be discouraged in the following circumstances: *Clostridium difficile* colitis, routine surgical prophylaxis, treatment in response to a single blood culture positive for coagulase negative *Staphylococcus*, initial empiric treatment of febrile neutropaenic patients and treatment of Gram-positive infections in renal failure patients purely for dosing convenience[2].

The patient described here probably developed VRE as a result of exposure of his endogenous enterococcal flora to low levels of vancomycin over a prolonged period. The negative results from many other patients indicate that the prompt use of appropriate infection control measures can prevent nosocomial spread to other patients. No further cases have been identified in the hospital.

The American guidelines appear reasonable to consult if a patient colonised or infected with VRE is detected[2]. Patients with VRE should be housed in a single room or in a multibed room with other patients with VRE. Gloves and plastic aprons should be worn when nursing the patient. Ward contacts of the index patient should be screened, and the patient isolated if VRE is detected. Strict hand washing procedures should be observed. Prolonged intestinal carriage of VRE by patients is well described and efforts should be made to add alerts to the patient's chart so that appropriate isolation can be made on readmission.

Acknowledgments

The clinicians and nursing staff involved in staff and patient surveillance are thanked for their cooperation. Dr Joan Faoagali and Mr Adrian Whitfield (Royal Brisbane Hospital) performed the teicoplanin E test.

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