ISOLATION OF VANCOMYCIN-RESISTANT ENTEROCOCCI IN QEENSLAND, CASE 2

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Abstract

A case of vancomycin-resistant enterococcus (VRE) colonisation is reported. The organism was not isolated from other patients sharing a room with the index case or from the environment. The microbiology laboratory plays an important role in the detection of VRE and in alerting the infection control, medical and nursing staff. Nosocomial transmission of VRE can be prevented by adherence to appropriate infection control procedures. The occurrence of VRE can be prevented by the appropriate use of vancomycin. ***Comm Dis Intell* 1996;20:402-403.**

Introduction

A reduction in the morbidity and mortality due to many bacterial diseases has been documented since antimicrobial agents were introduced for general use in the 1940s[1,2,3]. However, due to the widespread use of antimicrobials, drug resistance has emerged as a major public health problem in both community and institutional settings. Increased microbial resistance has resulted in prolonged hospitalisations and higher death rates from infections. In addition it has necessitated the use of more expensive, and often more toxic, drugs or drug combinations resulting in higher health care costs[4].

Case report

In June 1996, vancomycin-resistant *Enterococcus faecalis* (VRE) and methicillin-resistant *Staphylococcus aureus* (MRSA) were isolated from a groin swab. The swab was collected because the patient had a rash. The patient had been hospitalised in Queensland for four months with a chronic illness. During this time the patient had received several courses of vancomycin. The VRE was a possible *van B* phenotype, with minimal inhibitory concentration (MIC) to vancomycin 16 mg/L (intermediate), MIC to teicoplanin <4 mg/L (sensitive), ampicillin MIC <2 mg/L (sensitive), and no high level resistance to gentamicin.

An investigation was set up to determine whether patients who had shared a room with the index case were colonised with VRE and whether the resistant organism was present in the environment. A rectal swab was collected from the patient with VRE (the index case) for screening. This yielded VRE on culture. Rectal swabs were also collected from four patients who had shared a room with the index case and from another patient who had previously been in the same room for a month. These were all screened for VRE and found to be negative. Environmental samples were collected from 20 sites in the patient's room and cultured. No VRE were isolated, although non-VRE *Enterococcus faecalis* was found in some areas including curtain rails.

The index patient was isolated in a single room. The room in which the patient had been nursed was subjected to 'terminal' cleaning and the room closed over the weekend. A second groin swab collected two weeks later yielded VRE and MRSA on culture.

The patients who had shared a room with the index case were nursed together in one area.

Discussion

This appears to have been an isolated case. The laboratory has been screening for VRE routinely since October 1995. Rectal swabs collected weekly from haematology/oncology and intensive care patients were screened for VRE using blood agar with amikacin 8 mg/L and vancomycin 6 mg/L. About 400 rectal swabs have been screened and no VRE detected. Our patient did not fulfil the criteria for routine screening and hence was not screened by this method. However this laboratory method would not have detected VRE in this patient as the isolate did not have high level resistance to aminoglycosides and would therefore not have grown on our selective medium. As a result of this an alternative VRE screening medium is currently being investigated.

The microbiology laboratory plays a fundamental role in the surveillance and control of VRE. This is achieved through the use of good technical procedures and prompt reporting of VRE to the medical, nursing and infection control staff.

Enterococci should be identified to species level. Antimicrobial susceptibility testing on enterococci isolated from blood, sterile body sites and other sites (as clinically indicated) should include determination of vancomycin resistance as well as high level resistance to penicillin and aminoglycosides. The laboratory's method of susceptibility testing should include use of the control organism *Enterococcus faecalis* ATCC 51299. This strain has a moderate level of vancomycin resistance mediated by the *van B*

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gene which, unlike high level resistance mediated by the *van A* gene, is difficult to detect.

Efforts to contain VRE and prevent its spread to others is necessary for the management of patients colonised with this organism. Affected patients should be isolated and standard infection control principles adhered to. Particular attention should be paid to the decontamination and disinfection of the environment around the patient. The patient should remain in isolation while colonised with VRE or if readmitted without VRE 'clearance'. The patient may be 'delisted' if rectal and lesion swabs for VRE are persistently negative (three cultures on consecutive weeks) in hospital.

A record of VRE cases should be kept for the epidemiological tracking of cases including their location, antibiotic history and risk factors. Vancomycin use should be reserved for specific conditions and hospitals should develop guidelines for the proper use of vancomycin[5].

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ACUTE FLACCID PARALYSIS SURVEILLANCE IN AUSTRALIA: THE FIRST YEAR

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Abstract

Surveillance for acute flaccid paralysis commenced through the Australian Paediatric Surveillance Unit in March 1995. Thirty-five cases were reported in the first year, giving an estimated incidence of 0.90 cases per 100,000 children under the age of 15 years. Nearly half the cases were Guillain-Barre syndrome. No cases of poliomyelitis were identified. This surveillance scheme will assist in the process of certification of the eradication of poliomyelitis in Australia and the World Health Organization Western Pacific Region. ***Comm Dis Intell* 1996;20:403-405.**

Introduction

The World Health Organization (WHO) aims to eradicate poliomyelitis from the world by the year 2000[1]. Poliomyelitis has already been eradicated from the Americas[2]. For a country to be declared polio free it needs to meet a number of requirements, including polio vaccination coverage of more than 80%, no confirmed poliomyelitis cases for three years and adequate surveillance and investigation of suspected poliomyelitis cases.

Australia has not had any poliomyelitis cases reported through the National Notifiable Diseases Surveillance System since one case was reported in 1986, one case in 1978 and two cases in 1977[3]. The WHO however considers the detection and investigation of all cases of acute flaccid paralysis (AFP) as an essential and sensitive method of detecting wild poliovirus.

The differential diagnosis of acute flaccid paralysis includes Guillain-Barre syndrome, transverse myelitis and traumatic paralysis[4]. Other viruses (for example enterovirus types 70 and 71) may mimic polio. All these events are rare and little is known about the incidence, clinical course and outcomes of AFP in Australia.

In March 1995, surveillance of acute flaccid paralysis commenced through the Australian Paediatric Surveillance Unit (APSU). The aims of the study were to describe the incidence, causes and clinical picture of AFP cases in Australia and to determine whether any cases of AFP are caused by paralytic 'wild' poliovirus.

Methods

A case of acute flaccid paralysis was defined as a child aged less than 16 years with:

acute onset of flaccid paralysis in one or more limbs

or

acute onset of bulbar paralysis.

The Australian Paediatric Surveillance Unit (a unit of the Australian College of Paediatrics) conducts active, prospective national surveillance of selected rare paediatric

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