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Elimination of vancomycin-resistant enterococci from a neonatal intensive care unit following an outbreak

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Summary A policy of weekly faecal cultures for vancomycin-resistant enterococci (VRE) was instituted following the investigation of an outbreak of VRE in our neonatal intensive care unit in 2005. We found that 11 of 18 patients were infected or colonised during the outbreak, including three cases of bloodstream infection and one case of meningitis. This report describes the utility of the surveillance policy in maintaining a VRE-free environment. The outbreak investigation showed that all VRE isolated were *Enterococcus faecium* of the vanA type. Pulsed-field gel electrophoresis suggested that the outbreak was caused by a single strain. Control of the outbreak was achieved by enhanced contact isolation precautions, cohorting of patients and staff, improved environmental decontamination and closure of the unit to new admissions. The patients with bloodstream infections and meningitis were treated successfully with linezolid. Approximately one year after the outbreak, weekly surveillance detected two patients with faecal carriage of VRE whose periods of admission overlapped. Early intensive intervention was associated with disappearance of the organism from the neonatal intensive care unit. No further cases of colonisation or disease have occurred in the unit in the two and a half years since then.

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Introduction

Vancomycin-resistant enterococci (VRE) have become a frequent problem in healthcare settings. VRE infection or colonisation results from transmission from an infected source. Transfer of the genes conferring vancomycin resistance from VRE to other enterococci or other organism has also been observed.[1] Data from epidemiological studies have shown that the presence of VRE in stool cultures obtained in neonatal intensive care units appears to be quite rare, except when infants are screened in the context of an outbreak.[2–5] Colonised neonates have factors known to be associated with VRE carriage, such as low birth weight or long-term antibiotic therapy.[4] Most outbreaks among paediatric and neonatal intensive care units (NICUs) have been due to vancomycin-resistant *E. faecium* of the VanA type.[5,6]

In April 2005, we identified our first three hospitalised neonates with VRE infections, all of whom were identified within a period of 24 h. We describe our investigation and control of the outbreak, the evolving epidemiology of VRE colonisation and infection in our high risk NICU infants, the interventions used to control VRE transmission and the use of weekly faecal surveillance cultures for early detection of VRE carriage in order to prevent silent spread in the unit and additional outbreaks.

The outbreak

The event began with the appearance of systemic VRE infection in three premature neonates. It soon became apparent that the organism had previously become well established in the unit as shown by the discovery of intestinal colonisation in several other patients along with environmental contamination.

Case 1

A 520 g baby boy was born to a G_1P_0 23-year-old mother at 30 weeks of gestation, by caesarean section for fetal distress. Apgar scores were 7 and 9 at 1 and 5 min respectively. Congenital cytomegalovirus infection was diagnosed with ensuing intrauterine growth restriction, cerebral ventriculomegaly, thrombocytopenia and abnormal liver functions. During his extended hospital stay, he developed multiple complications of prematurity including respiratory distress requiring prolonged mechanical ventilation and oxygen therapy. After birth he received a three-day course of ampicillin and gentamicin until primary cultures were proven negative. He also received a three-day course of vancomycin and cefotaxime followed by another seven days of vancomycin treatment for coagulase-negative staphylococcal sepsis diagnosed at seven days of age, and a three-day course of vancomycin and ceftazidime at 19 days of age due to abdominal distension, until cultures were proven negative. At 26 days, four days after completion of the last vancomycin treatment course, his respiratory condition worsened, and he developed abdominal distension, hyperglycaemia and severe thrombocytopenia. Peripheral blood cultures grew *E. faecium* resistant to vancomycin [minimum inhibitory concentration (MIC) >256 mg/L, Etest, AB Biodisk, Sweden] but susceptible to linezolid (MIC 2 mg/L), and *Enterobacter cloacae*. The same vancomycin-resistant *E. faecium* grew subsequently from rectal swabs. The treatment with vancomycin and amikacin was replaced by linezolid and ceftazidime according to drug susceptibilities. Blood cultures obtained on day 3 of treatment (following one day with vancomycin and amikacin and one day with linezolid and ceftazidime) were negative and remained so following cessation of treatment. The patient's clinical condition improved and no complications were observed during linezolid treatment. Complete blood counts were monitored during therapy, and no abnormalities were noted. Liver functions were stable without further deterioration. Stool cultures, however, remained positive for VRE throughout his hospitalisation.

Case 2

An 810 g baby boy, the third of quadruplets, was born by caesarean section to a G_3P_1 AB_2 31-year-old pre-eclamptic mother at 31 weeks of gestation induced by in-vitro fertilisation. Apgar scores were 9 and 10 at 1 and 5 min respectively. Physical examination was normal. His clinical course was uneventful except for oxygen requirement during the first 10 h. After birth he received a three-day course of ampicillin and gentamicin until primary cultures were proven negative. At six days he suffered from recurrent apnoea and hyperglycaemia. Peripheral blood culture and central venous umbilical catheter tip grew *E. faecium* resistant to vancomycin by Etest (MIC >256 mg/L) but susceptible to linezolid (MIC 1.5 mg/L). Cerebrospinal fluid (CSF) culture was negative. A rectal swab taken subsequently was also positive for this organism. Initial treatment with vancomycin and cefotaxime was changed to linezolid according to drug susceptibility. Blood culture obtained on