Outbreaks of vancomycin-resistant *Enterococcus faecium* in acute care pediatric hospitals

To the Editor:

We read with interest the recent article entitled “An outbreak of vancomycin-resistant *Enterococcus faecium* in an acute care pediatric hospital: Lessons from environmental screening and a case-control study” (1). We had a similar experience in our facility (the Stollery Children’s Hospital [SCH]; Edmonton, Alberta), and would like to share our findings because there are very little data on this subject in Canadian pediatric centres.

**Setting and methods**

The SCH is a 140-bed, tertiary care pediatric facility that serves as a referral centre for over 1.7 million children from western and northern Canada. Before December 2007, routine surveillance for patients with vancomycin-resistant *Enterococcus faecium* (VRE) colonization was not performed within the SCH. However, the infection control unit conducted prospective surveillance for VRE infection from pediatric clinical specimens such as blood, wound swabs or urine. From January 2001 to November 2007, there were no cases of VRE from clinical isolates. In December 2007, a standardized antibiotic-resistant organism screening tool was implemented in all hospital facilities in our health region. Subsequently, all patients admitted for a minimum of 24 h in any health care facility within the previous six months were screened for antibiotic-resistant organisms, including VRE. Rectal swabs were sent for VRE culture, and results were usually obtained within a few days (range two to seven days); further nonroutine testing on the isolates included pulsed-field gel electrophoresis to determine strain identity for the outbreak (2,3). When patients with VRE were identified, the following were performed – isolation and identification of other positive children (contact and prevalence screens), source control (both carriers and environment) and education (4).

**The outbreak**

The first child with VRE was identified on December 18, 2007, on a general pediatrics ward; 15 children were further identified on two additional wards between December 19, 2007, and January 28, 2008 (Figure 1). None of these children had clinical infection associated with VRE; all had positive cultures from rectal swabs (therefore, they were considered to be carriers). The organisms were of the vanA genotype – *E. faecium*; strain typing showed a single clone associated with all but one of the patients (one other patient identified with VRE during this time, but was not included in the outbreak investigation, had a vanB phenotype). Fifteen patients had at least one admission in the previous year, and seven had more than two admissions. Ten (63%) patients had previous negative swabs before their positive swab. The median age was 10.6 years (range seven weeks to 17 years; seven were teenagers). Underlying diagnoses included non-VRE-related infections (n=5; Lemierre’s syndrome, empyema, cellulitis, peritonitis and pyleonephritis), respiratory (n=5; cystic fibrosis), gastrointestinal (n=2; ulcerative colitis and irritable bowel syndrome), oncological (n=1) and other syndromes (n=3; cerebral palsy, congenital myopathy and trisomy 21). Six children had gastrostomy tubes, and one child was on nasogastric feeds. None of the environmental isolates were positive for VRE.

**Interventions**

Any child with VRE was placed on contact precautions. Roommates of children with VRE were also placed on contact precautions until their VRE status was determined. Once the outbreak was identified, known contacts were screened, a prevalence screening of the entire hospital was performed and weekly prevalence screening on any units with a known VRE case were performed. Any child who was positive for VRE was instructed to use 2% chlorhexidine washcloths daily. Environmental swabs for VRE from randomly chosen objects on the affected units and play areas were performed. In addition to routine terminal cleanings of rooms, the ‘teen room’ underwent a special cleaning. Infection control practitioners re-educated the staff about VRE, and information sheets were provided for staff, patients and families.

**Discussion**

The combined experiences at the SCH and the Hospital for Sick Children (HSC) in Toronto, Ontario, suggest a need for increased screening of VRE in Canadian pediatric institutions. Clinical isolates of VRE do not seem to
indicate the carriage rate of this organism. The outbreaks described at SCH and HSC were both detected because of active screening for VRE. Had we not implemented our screening tool, it is likely that we would have ongoing transmission on the pediatric units because of the lack of positive clinical isolates.

The HSC outbreak study found cephalosporin use as an independent risk factor for VRE colonization. Given that many of the patients in our outbreak had underlying medical issues for which they received antibiotics, it may have been a predisposing factor. Given the frequency of use of this antimicrobial class in other pediatric institutions, this would be another good reason for other pediatric facilities to consider screening children for VRE.

We would favour a broad screening approach instead of targeting certain populations (such as those with oncological diagnoses). In contrast to the HSC, our outbreak extended into the general pediatrics population – only one patient had an underlying oncological diagnosis. Therefore, unlike the HSC that screens children admitted to the hematology/oncology ward, we continue to screen all patients admitted to our facility with a recent history of admission (minimum of 24 h in any health care facility within the past six months).

The HSC reported using a polymerase chain reaction (PCR)-based assay for their outbreak. PCR is advantageous over culture techniques because of the quick turnaround time. We found that VRE culture has a turnaround time of at least two days; the children were isolated during that time. PCR, with a faster turnaround time, is more desirable to avoid unnecessary isolation, and if isolation of contacts was not possible, potentially preventable transmission.

In addition to the usual practices for limiting VRE, we elected to use 2% chlorhexidine washcloths to reduce the bioburden of VRE on the patients, on health care workers’ hands and on environmental surfaces. We have had success in reducing the transmission of VRE on adult units in our facility – and used a similar protocol in pediatric patients – using the wipes for daily body cleansing for one month or until discharge (whichever was shorter) (5-7).

Since this outbreak, we have had no further evidence of VRE outbreaks in our facility. Although not formally assessed, control measures including isolation and identification of other VRE-positive children (contact and prevalence screens), source control (both carriers and environment) and education were successful in containing this outbreak (8).

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REFERENCES