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Epidemiology of colonisation of patients and environment with vancomycin-resistant enterococci

Dr Marc JM Bonten MD ^a ... Robert A Weinstein MD ^{a, b}

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Summary

Background

Vancomycin-resistant enterococci (VRE) have emerged as nosocomial pathogens during the past 5 years, but little is known about the epidemiology of VRE. We investigated colonisation of patients and environmental contamination with VRE in an endemic setting to assess the importance of different sources of colonisation.

Methods

Between April 12, and May 29, 1995, cultures from body sites (rectum, groin, arm, oropharynx, trachea, and stomach) and from environmental surfaces (bedrails, drawsheet, blood-pressure cuff, urine containers, and enteral feed) were obtained daily

from all newly admitted ventilated patients in our medical intensive-care unit (MICU). Rectal cultures were obtained from all non-ventilated patients in the MICU. Strain types of VRE were determined by pulsed-field gel electrophoresis.

Findings

There were 97 admissions of 92 patients, of whom 38 required mechanical ventilation. Colonisation with VRE on admission was more common in ventilated than in non-ventilated patients (nine [24%] *vs* three [6%], $p < 0.05$). Of the nine ventilated patients colonised with VRE on admission, one acquired a new strain of VRE in the MICU. Of the 29 ventilated patients who were not colonised with VRE on admission, 12 (41%) acquired VRE in the MICU. The median time to acquisition of VRE was 5 days (interquartile range 3–8). Of the 13 ventilated patients who acquired VRE, 11 (85%) were colonised with VRE by cross-colonisation. VRE were isolated from 157 (12%) of 1294 environmental cultures. The rooms of 13 patients were contaminated with VRE, but only three (23%) of these patients subsequently acquired colonisation with VRE. Pulsed-field gel electrophoresis of 262 isolates showed 20 unique strain types of VRE.

Interpretation

Frequent colonisation with VRE on MICU admission and subsequent cross-colonisation are important factors in the endemic spread of VRE. Persistent VRE colonisation in the gastrointestinal tract and on the skin, the presence of multiple-strain types of VRE, and environmental contamination may all contribute to the spread of VRE.



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