all cases of CDI with onset during hospitalization or within 72 hours after patient discharge. We subsequently undertook a search of all microbiologically confirmed cases of CDI during the period 2007–2012. Individuals who shared the same surname or same address were identified for additional investigation. All putative case-pairs identified were reviewed to identify potential epidemiological associations; this included ribotyping of available *C. difficile* isolates and, when possible, multiple-locus variable number tandem repeat analysis (MLVA). Six cases of paired CDI were identified.

In pair 1, the index patient, a 74-year-old woman, was admitted for investigation and management of diarrhea. She had had an episode of CDI earlier that year and received a diagnosis of recurrent CDI during this hospitalization. A specimen obtained within 1 day of admission to the hospital was found to be positive for glutamate dehydrogenase (GDH) and *C. difficile* toxin. One week later, the patient's husband (also her main caregiver) developed CDI. The contact patient had multiple comorbidities and his own independent risk factors for CDI. Isolates from both patients were identified as ribotype 027, and they were indistinguishable on MVLA typing.

In pair 2, the index patient, a 76-year-old woman, was admitted to the hospital for investigation of suspected acute colitis after chemotherapy. A stool sample obtained at hospital admission was found to be positive for GDH but negative for *C. difficile* toxin, which suggested *C difficile* colonization rather than CDI. However, because of persistent symptoms, the patient was given metronidazole therapy, to which she responded well. Her husband, a patient with chronic lung disease who required recurrent antibiotic therapy for infective exacerbations in the community, was admitted to the hospital 10 days later with diarrhea. A stool sample obtained the following day was positive for both GDH and *C. difficile* toxin. Both isolates belonged to ribotype 127.

In pair 3, a 39-year-old woman received a diagnosis of CDI in the community after receiving antibiotic therapy for presumed cholecystitis. Her 15-month-old son presented to his primary care physician with diarrhea. At the family's request, a stool sample was tested and was found to be positive for both GDH and *C. difficile* toxin. Ribotyping of the isolates demonstrated that both belonged to ribotype 017.

Review of the paired cases of CDI, taken together with indistinguishable ribotypes and their temporal association, is highly suggestive of an epidemiological link and thus highlights the potential for spread within families. Interestingly, 2 of 3 contact patients had their own independent risk factors for CDI. In addition, the apparent transmission from a GDH-positive but toxin-negative patient to her spouse, who went on to develop active CDI, is also of particular note. Although the clinical significance of isolating *C. difficile* in an infant is not clear, as in the last case-pair, the matching ribotypes suggest a putative link between the 2 cases.

A recent study has suggested that intrafamilial transmission of CDI is infrequent.[1] Our findings corroborate this. We identified 3 case-pairs from a total of 238 confirmed cases of CDI over a 5-year period. However, the database search relied on identification of shared surname and address, and this may have underestimated the frequency of transmission. Nevertheless, we have amended the information on CDI given to patients and their relatives. In particular, we have reinforced the importance of adopting appropriate hand hygiene measures by index case patients and family members (both at home and in the hospital) in an attempt to reduce the risk of intrafamilial spread of CDI.

ACKNOWLEDGMENTS

Potential conflicts of interest. All authors report no conflicts of interest relevant to this article. All authors submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and the conflicts that the editors consider relevant to this article are disclosed here.

Debbie Baishnab, MB, BChir;[1] Kathleen R. Banfield, MSc;[2] Kathleen Jones, BSc;[2] Katharine S. Scott, MB, BS, FRCPath;[3] Nigel C. Weightman, MB, BS, FRCPath;[3] Kevin G. Kerr, MD, FRCPath[3,4]

Affiliations: 1. Department of Microbiology, Leeds Teaching Hospitals Trust, Leeds, United Kingdom; 2. Department of Infection Prevention and Control, Harrogate and District National Health Service Foundation Trust, Harrogate, United Kingdom; 3. Department of Microbiology, Harrogate and District National Health Service Foundation Trust, Harrogate, United Kingdom; 4. Hull York Medical School, York, United Kingdom.

Address correspondence to Kevin G. Kerr, MD, FRCPath, Department of Microbiology, Harrogate District Hospital, Harrogate, North Yorkshire HG2 7SX, UK (kevin.kerr@hdft.nhs.uk).

Presented in part: 8th International Healthcare Infection Society Conference; Liverpool, United Kingdom; November 2012.

Infect Control Hosp Epidemiol 2013;34(4):442-443

 0899-823X/2013/3404-0020$15.00. DOI: 10.1086/669873

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East North Central Region Has the Highest Prevalence of Vancomycin-Resistant *Enterococcus faecalis* in the United States

To the Editor—We read the article of Hayakawa et al[1] with great interest. The report describes the growing prevalence of vancomycin-resistant *Enterococcus faecalis* in Michigan, a state that also has the most reports of vancomycin-resistant *Staphylococcus aureus*. Similar findings were reported in the tigecycline evaluation and surveillance trial (TEST).[2] During the 2004–2009 period, 4.6% of 3,753 *E. faecalis* isolates were

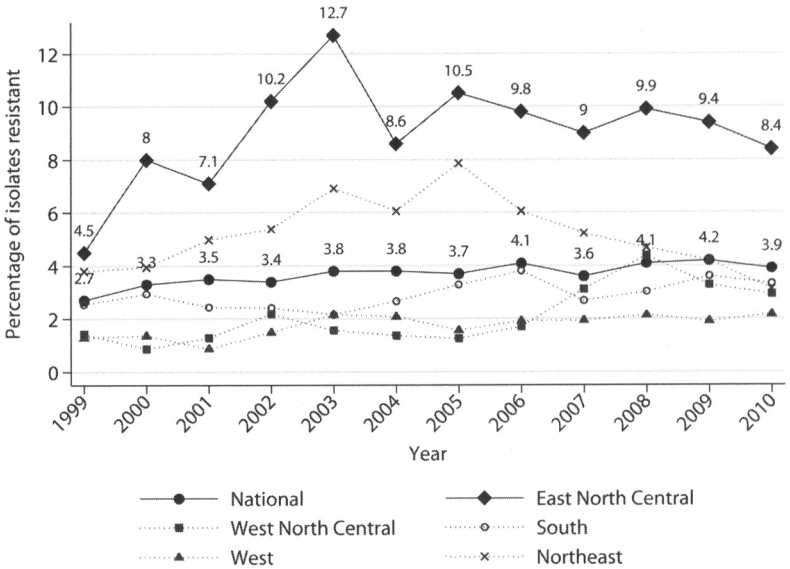

FIGURE 1. Proportion of vancomycin-resistant *Enterococcus faecalis* in the United States by region. The Midwest census region was split into the West North Central and East North Central divisions to highlight the higher prevalence of resistance among isolates from the latter.

vancomycin resistant, with the highest rates of 7.6% in the East North Central region of United States. Here we report rates and trends of vancomycin-resistant *E. faecalis* in the East North Central region compared with national rates from 1999 to 2010.

The antimicrobial susceptibility data were obtained from the Surveillance Network Database (Eurofins-Medinet). The data are described in detail elsewhere[3] and have been widely used to characterize regional and national trends in antibiotic susceptibility.[4-7] The 287 microbiological laboratories in the network were selected on the basis of geographic and demographic criteria to be representative of hospitals at the level of US Census Bureau regional divisions. Participating sites are required to submit reports for all bacterial isolates for which species identification and antibiotic sensitivities are recorded. Categorical susceptibility results are based on the Clinical Laboratory Standards Institute criteria adopted by the facility at the time of testing and reflect results as they were reported to the treating physician.

The analysis considered all outpatient and inpatient (including inpatient, outpatient, intensive care unit, and long-term care) isolates reported between January 1999 and July 2010 that were identified as *E. faecalis*. Perirectal surveillance cultures were not included in the database, and the data were filtered to retain only isolates that were tested to vancomycin. We then looked at the national and regional proportion of isolates that were reported as resistant to vancomycin throughout the study period.

Overall, in the East North Central region, a total of 44,108 isolates of *E. faecalis* between 1999 and 2010 were tested to vancomycin, of which 8.8% (n = 3,858) were resistant to vancomycin. At the national level, the pooled proportion of vancomycin-resistant *E. faecalis* for 1999–2010 was 3.6% (13,022 of 354,462 *E. faecalis* isolates). The Northeast region was behind the East North Central region, with 5.2% of the *E. faecalis* isolates resistant to vancomycin (for yearly national and regional trends, see Figure 1). Figure 1 also indicates a steady rise in the proportion of vancomycin-resistant *E. faecalis* isolates in the United States, from 2.7% in 1999 to 3.9% in 2010.

Even though our results do not indicate a continuous rise in the proportion of vancomycin-resistant *E. faecalis* isolates from 1999 to 2010 in the East North Central region, the regional proportions have always been high compared with the national average. The proportion of vancomycin-resistant *E. faecalis* isolates in this region remained twice the national average during the 1999–2010 period. Eight of the 13 cases of vancomycin-resistant *S. aureus* were reported from southeast Michigan, an area approximately at the center of this region.[8] Coupled with evidence of the horizontal transfer of the *vanA* gene complex from vancomycin-resistant *E. faecalis* to *S. aureus*,[9,10] our findings underscore the importance of monitoring the trends in vancomycin-resistant *E. faecalis* through continuous and timely surveillance.

ACKNOWLEDGMENTS

Financial support. This work was supported by the Extending the Cure Project funded by a grant from the Pioneer Portfolio of the Robert Wood Johnson Foundation.

Potential conflicts of interest. All authors report no conflicts of interest

relevant to this article. All authors submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and the conflicts that the editors consider relevant to this article are disclosed here.

Sumanth Gandra, MD, MPH;[1] Nikolay Braykov, BSE;[2] Ramanan Laxminarayan, PhD, MPH[2,3,4]

Affiliations: 1. Division of Infectious Diseases and Immunology, University of Massachusetts Medical School, Worcester, Massachusetts; 2. Center for Disease Dynamics, Economics, and Policy, Washington, DC; 3. Public Health Foundation of India, New Delhi, India; 4. Princeton Environmental Institute, Princeton University, Princeton, New Jersey.

Address correspondence to Sumanth Gandra, MD, MPH, 55 Lake Avenue, Worcester, MA 01545 (sumanth.gandra@umassmemorial.org).

Infect Control Hosp Epidemiol 2013;34(4):443-445

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Intensified Infection Control Measures to Minimize the Spread of Colistin-Resistant *Acinetobacter baumannii*

To the Editor—The emergence of carbapenem-resistant gram-negative bacteria (GNB) is an increasing source of healthcare-associated infection worldwide and has been associated with adverse clinical outcomes and resource consumption.[1,2] The use of colistin and polymixin B has been resurrected during the past decade, especially in combination drug regimens targeting carbapenem-resistant GNB.[3] To date, the emergence of colistin-resistant GNB has been uncommon, yet it is of global concern.[3] We report a case of pneumonia due to colistin-resistant *Acinetobacter baumannii* infection in a patient who presented to an intensive care unit (ICU), implementation of intensified infection prevention control (IPC) measures, and the ICU monitoring efforts associated with ensuring that there was no subsequent detection of this pathogen in other patients. On September 15, 2012, a 74-year-old man with chronic obstructive pulmonary disease, diabetes, renal failure, and recurrent carbapenem-resistant *A. baumanniii* pneumonia (3 episodes in the previous 12 months) was readmitted to the medical ICU with fever, shortness of breath, and pneumonia. Because of a history of carbapenem-resistant *A. baumannii* infection treated with colistin and cefoperazone-sulbactam, the patient was placed under isolation precautions at hospital admission. Sputum cultures obtained at admission grew colistin-resistant *A. baumannii* (colistin minimum inhibitory concentration, 128 μg/mL), the infection control team was notified, and IPC measures in the 8-bed medical ICU were initiated. The IPC measures included (i) implementation of enhanced contact isolation precautions (ie, strict adherence to hand hygiene protocols before and after patient care and use of gowns and gloves); (ii) active surveillance cultures, defined as culture of rectal swab samples and tracheal aspirates (if the patient received mechanical ventilation), for all patients in the unit (on day 0, day 7, and every week until hospital discharge); (iii) daily environmental cleaning with detergents and with phenolic agents of high-touch surfaces and sites contaminated with body fluids or with blood; (iv) an up-to-date education program for all healthcare workers (HCWs) within the first week of the case detection[4,5]; and (v) delivery of real-time feedback to HCWs regarding IPC compliance. A case patient was defined as a patient with nosocomial colonization or infection with colistin-resistant *A. baumannii* identified by clinical culture more than 48 hours after ICU admission; nosocomial acquisition of this microorganism was defined as a positive active surveillance culture more than 48 hours after admission if the initial surveillance cultures were negative. Active surveillance cultures were performed on tracheal aspirate specimens and rectal swab specimens (if the initial