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Temporal trends and risk factors for healthcare-associated vancomycin-resistant enterococci in adults

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SUMMARY

Background—Published data regarding temporal trends in vancomycin-resistant enterococci (VRE) prevalence within specific regions or healthcare systems are scarce.

Aim—To characterize temporal trends and risk factors for healthcare-associated infections caused by VRE.

Methods—The study included all adult discharges occurring from 2006 to 2014 with an enterococcal infection from three hospitals in a large academic healthcare system. Bivariate analyses were used to identify statistically significant factors associated with vancomycin-susceptible or -resistant infection. Statistically significant variables were included in a final logistic regression model. Trends assessed whether the proportion of enterococcal infections resistant to vancomycin changed over time.

Findings—The sample included 10,186 adults with first-time healthcare-associated enterococcal infection. Significant risk factors ($P \leq 0.05$) for VRE in the final logistic regression model included: tertiary 1 hospital, intensive care unit length of stay, higher Charlson Comorbidity Index, previous immunosuppressive or chemotherapeutic medications, previous hospitalization, renal failure, malignancy, longer length of stay prior to infection, taking an antibiotic prior to infection, being female, and having an infection in winter or spring. Between 2006 and 2014, the rate of resistance varied from 37.1 to 42.9% but there were no significant differences in the proportion resistant to vancomycin over time ($P = 0.36$).

Conclusion—Research targeted at risk factors is important to decrease the amount of VRE infections.

Keywords

Vancomycin-resistant; enterococci; Risk factors; Temporal trends

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Conflict of interest statement

None declared.

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Introduction

Healthcare-associated infections (HCAIs) are frequent (4% of hospitalized patients), often preventable, and associated with medical devices: central line-associated bloodstream infection (BSI), surgical site infection (SSI), catheter-associated urinary tract infection (UTI), and ventilator-associated pneumonia.[1,2] Enterococci are one causative agent of HCAIs; these bacteria are a component of the faecal flora and may be contracted by direct or indirect by faecal–oral transmission; invasive devices are another important portal of entry.[3] *Enterococcus faecium* accounts for the majority of vancomycin-resistant infections and *Enterococcus faecalis* constitutes 2–20% of vancomycin-resistant enterococci (VRE) isolates.[3,4]

Similar to other infectious agents, populations largely affected by VRE include the elderly, those with compromised immune systems, and critically ill patients in the intensive care unit (ICU).[5] Other risk factors include prolonged length of hospital stay, previous exposure to vancomycin, anti-cancer chemotherapeutic agents, immunosuppressants and anti-inflammatory drugs, renal insufficiency, malignancies, comorbidities, and surgical procedures.[4–6]

According to the Centers for Disease Control and Prevention's National Nosocomial Infections Surveillance (NNIS) system, the proportion of HCAIs caused by VRE rose from 0.3% to 7.9% between 1992 and 2004.[7] In addition, infections caused by VRE have been associated with higher treatment costs, prolonged morbidity, and greater mortality rates.[8] However, information regarding temporal trends in VRE prevalence within specific regions or healthcare systems is scarce. Therefore, the aim of this study was to characterize temporal trends and risk factors for HCAIs caused by VRE during a nine-year period (2006–2014) in a large academic healthcare system in New York City.

Methods

Study population

The study was conducted as part of a federally funded project, 'Health Information Technology to Reduce Healthcare-Associated Infection' (National Institute of Nursing Research, National Institutes of Health; R01NR010822). In this project a clinical research database was created of patients hospitalized within three adult acute tertiary care hospitals of the largest healthcare system in New York City. This analysis included all adult (≥18 years) patient discharges from the three hospitals between 2006 and 2014. The hospitals comprised two-tertiary/quaternary care hospitals, designated as hospital 1 and hospital 2 (about 650 and 910 beds), and an approximately 220-bed community hospital designated as hospital 3.

Data collection

The study database was constructed using electronic data from clinical and administrative systems shared between the study institutions.[9] The database drew information from numerous sources, including patients' electronic health record, laboratory and medication administration records, and included admission and discharge data, International

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Classification of Diseases Ninth Revision Clinical Modification (ICD-9-CM) codes, age, sex, comorbidities, and surgical procedures. A subset of this larger dataset was used, with the primary outcome of interest being the first healthcare-associated (i.e. occurring >2 days after hospital admission) BSI, UTI, or SSI caused by *E. faecalis* or *E. faecium*. Infections were identified using a combination of time-stamped microbiology laboratory records and ICD-9-CM procedure and diagnosis codes, based on modified criteria from the CDC National Healthcare Safety Network (NHSN).[10]

Bloodstream infection was defined as positive *E. faecalis* or *E. faecium* blood culture with no positive culture with the same organism at another body site within the previous 14 days. In a previous study we verified that the definition of BSI was 100% congruent with a review of 122 medical records.[11] UTI was defined as a positive *E. faecalis* or *E. faecium* urine culture with $\geq 10^5$ colony-forming units (cfu) per mL and no more than one other species of micro-organism, or 10^3–10^5 cfu/mL plus pyuria. SSI was defined as positive *E. faecalis* or *E. faecium* wound culture within 30 days of an ICD-9-CM-documented National Healthcare Safety Network (NHSN) operative procedure.[12] Additionally, individual patient records were linked using their medical record number, and each patient's first positive VRE infection was used to remove patients with multiple cases of VRE infections. The year and season of each infection (winter, December–February; spring, March–May; summer, June–August; autumn, September–November) were also recorded. Vancomycin susceptibility patterns were obtained from the clinical microbiology reports.

Patients' demographic characteristics and medical conditions were collected from electronic sources. The institution's time-stamped electronic medication administration record was used to determine whether patients received antibiotics or other medications that might increase patient risk of infection (chemotherapeutic agents, immunosuppressants, and anti-inflammatory drugs), which we term 'high-risk medications' at least 24 h before infection. Comorbidities (diabetes, renal failure, and malignancies) and the Charlson Comorbidity Index were collected.[13] Other data collected included patients' age, sex, length of hospitalization, hospital location, length of stay prior to infection (calculated as the difference between first date of infection and date of admission), ICU stay prior to admission, prior within-network hospitalization, and admission from a nursing facility.

Identification of organisms was done in the study institution's clinical microbiology laboratory using Clinical and Laboratory Standards Institute standards; additionally, Vitek2 (bioMérieux, Inc., Durham, NC, USA) and/or E-test were used for antibiotic susceptibility testing.[14,15]

Statistical analysis

The frequency of BSI, UTI, and SSI caused by vancomycin-susceptible and -resistant *E. faecalis* and *E. faecium* were recorded by year. Initially, bivariate analyses using χ^2-tests for categorical variables or simple logistic regression for continuous variables were used to identify statistically significant ($P \leq 0.05$) factors associated with a susceptible or resistant enterococcal infection, including age, sex, hospital, ICU stay prior to infection, Charlson Comorbidity Index, diabetes, renal failure, malignancy, length of hospital stay prior to infection, antibiotic and high-risk medication use prior to infection, season and year (2006–

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2014), prior stay in a nursing facility, prior in-network hospitalization, infection site (BSI, UTI, or SSI), and medical invasive device. Those variables that were statistically significant in the bivariate analyses were included in a multivariate logistic regression model in a stepwise forward fashion. All analyses were performed using SAS version 9.4. The Cochran–Armitage test for trends was used to assess whether the proportion of *E. faecalis* and *E. faecium* infections resistant to vancomycin changed over time.

Results

A total of 10,186 adults with first-time healthcare-associated enterococcal infections were identified from 2006 through 2014: 4094 patients (40.2%) with VRE and 6092 (59.8%) with susceptible strains. Differences between patients with antimicrobial-resistant versus -susceptible enterococcus infections are summarized in Table I. In the final multivariable model (Table II), the significant risk factors of resistance were tertiary 1 hospital vs tertiary 2 (59.4% and 34.1%, respectively; odds ratio: 2.57; 95% confidence interval: 2.30–2.88), having stayed in the ICU (52.1%; 1.54; 1.38–1.71), higher Charlson Comorbidity Index score (1.04; 1.02–1.06), prior high-risk medications (43.5%; 8.19; 5.6–11.26), prior hospitalization (41.9%; 1.52; 1.37–1.68), renal failure (48.9%; 1.75; 1.58–1.94), having a malignancy (52.6%; 1.46; 1.29–1.66), longer length of stay prior to infection (1.03; 1.02–1.03), taking an antibiotic prior to infection (48.3%; 23.72; 17.55–32.07), being female (42.3%; 1.29; 1.17–1.43), and having an infection in winter or spring versus summer or autumn (41.7% and 38.4%, respectively; $P = 0.009$).

Of the total number of first-time enterococcus infections over the study period from 2006 to 2014, the rate of resistance varied from 37.1% to 42.9% (Figure 1). However, there were no significant differences in the proportion resistant to vancomycin over time (two-sided Cochran–Armitage trend: $P = 0.36$).

Discussion

The overall proportion of VRE HCAI in this study was slightly higher than that reported by CDC for US hospitals during the same time-period (40.2% versus 30%).[16] Many of the risk factors identified in this study were consistent with those previously reported, including previous exposure to antibiotics, greater acuity, more comorbidities, prior hospitalizations and longer length of hospital stay.[17–24] One study found that prior exposure to vancomycin increased the odds of VRE infection between 2.3- and 11.0-fold.[20] Other studies have also reported that antibiotic exposure prior to infection, renal failure, and presence of malignancy were significant risk factors for VRE infection.[18–21] Furthermore, prior ICU stay, longer length of stay prior to infection, and prior hospitalization have all been found to be significant risk factors for a VRE infection.[23,24] The current literature also supports a previous stay in a nursing facility as a risk factor for VRE infection, but our study found no such association ($P = 0.93$).[25–27] One possible reason for this is that patients acquired the infection at the nursing home, and thus it would have been identified on admission and not considered a hospital-associated infection.[28,29]

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In addition to risk factors previously identified, we also found that rates of resistance were significantly higher in one tertiary care study hospital, even when confounding by other known risks factors was controlled. This was unexpected since both hospitals are part of the same network and share the same infection prevention staff, procedures, and policies. This association might be due to environmental or host factors unaccounted for in these analyses; or, despite similar policies, implementation of infection control practices such as antimicrobial prescribing or barrier precautions may have varied between the two hospitals.[30–32] This finding highlights the importance of local surveillance to identify institution- or unit-specific problems and priorities for infection prevention.

Finally, patients hospitalized during winter and spring seasons were significantly more likely to have a resistant infection when compared to those admitted during summer and autumn. This could be the result of the increase in patient volume in the colder months or, as noted by Nelson, there may be changes in the immune system associated with seasonality.[33]

The results highlight that there was no significant reduction in the proportion of enterococcal infections that were vancomycin resistant. One contributing factor could be the ability of VRE to persist on surfaces for long periods of time.[34–36] For example, Huang and colleagues found the odds of acquiring VRE when staying in a room where a previous VRE-positive patient stayed were 1.4-fold greater than the odds of acquiring VRE when a VRE-negative patient had stayed in that room.[37] A second contributing factor may be related to antibiotic prescribing patterns of clinicians and the excessive or unnecessary use of broad-spectrum agents such as vancomycin, emphasizing the importance of antimicrobial stewardship programmes. Results of such programmes would not be expected to be immediate, and would likely take some years to have an impact.

In addition to confirming previously identified risk factors for VRE, the results of this study highlight the importance of identifying local infection prevention practices and patterns of resistance, since, even within a single healthcare system, our findings varied significantly across hospitals. Furthermore, this study emphasizes the need for high-level surveillance in which microbiology results, clinical information, and admission details are gathered into a single patient record across hospitals for ongoing accurate surveillance of risk factors for resistance. This is supported by the problem of emerging antibiotic resistance that has been difficult to combat, which supports the importance of moving into patient information systems that collect these data items in an integrated manner. Finally, it is clear that the proportion of infections resistant to vancomycin is not significantly different, emphasizing the importance of antimicrobial stewardship programmes.

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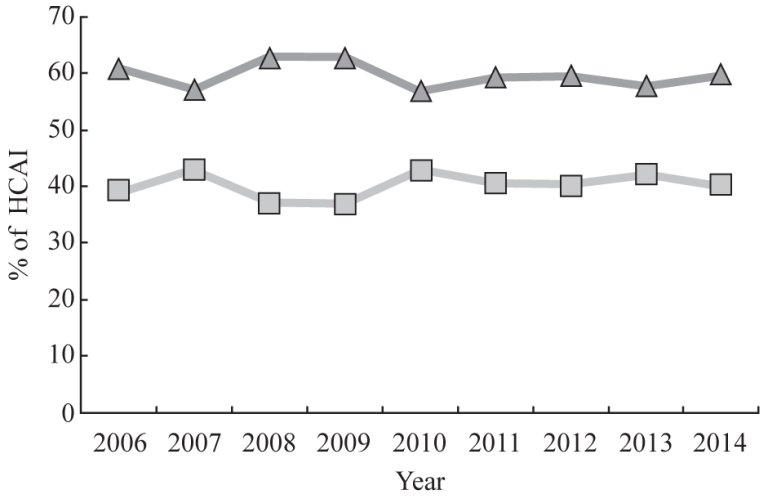

Figure 1.

Percentages of *Enterococcus faecalis* and *Enterococcus faecium* healthcare-associated infection (HCAI) that were susceptible (triangles) and resistant (squares) to vancomycin per year in three New York City Hospitals, 2006–2014. Pearson χ^2-test for independence across years: $P = 0.009$. Two-sided Cochran–Armitage test for trend: $P = 0.36$.

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Table I

Factors associated with HCAI caused by vancomycin-susceptible and -resistant *Enterococcus faecalis* and *Enterococcus faecium* in three New York City hospitals, 2006–2014 (univariate analysis)

Factor	Resistant to vancomycin		*P*-value[a]
	Yes	No	
Total	4094 (40.2%)	6092 (59.8%)	
Mean (range) age (years)	64.7 (0–107)	65.7 (0–104)	0.003[a]
Sex			<0.0001[a]
Male	1714 (41.9%)	2845 (46.7%)	
Female	2380 (58.1%)	3247 (53.3%)	
Hospital			<0.001[a]
Community	286 (7%)	996 (16.4%)	
Tertiary 1	1814 (44.3%)	1238 (20.3%)	
Tertiary 2	1994 (48.7%)	3858 (63.3%)	
ICU prior to infection			<0.0001[a]
Yes	2029 (49.6%)	1868 (30.7%)	
No	2065 (50.4%)	4224 (69.3%)	
Mean (range) CCI	3.37 (0–19)	2.75 (0–16)	<0.0001[a]
Mean (range) days of stay prior to infection	15.83 (0–889)	5.46 (0–348)	<0.0001[a]
High risk medication[b] prior to infection			<0.0001[a]
Yes	4043 (98.8%)	5420 (89%)	
No	51 (1.2%)	672 (11%)	
Prior stay in nursing facility			0.93
Yes	262 (6.4%)	387 (6.4%)	
No	3832 (93.6%)	5705 (96.6%)	
Prior in-network hospitalization			<0.0001[a]
Yes	2697 (65.9%)	3735 (61.3%)	
No	1397 (34.1%)	2357 (38.7%)	
Antibiotic use prior to infection			<0.0001[a]
Yes	4048 (98.9%)	4328 (71%)	
No	46 (1.1%)	1794 (29%)	
Site of infection			<0.0001[a]
Bloodstream	928 (22.7%)	1078 (17.7%)	
Urinary tract	2926 (71.5%)	4356 (71.5%)	
SSI	240 (5.9%)	658 (10.8%)	
Season of infection onset			0.009[a]
Winter	1084 (26.5%)	1523 (25%)	
Spring	1186 (29%)	1647 (27%)	

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Factor	Resistant to vancomycin		*P*-value[a]
	Yes	No	
Summer	944 (23%)	1497 (24.6%)	
Autumn	880 (21.5%)	1425 (23.4%)	
Diabetes			0.30
Yes	1220 (29.8%)	1874 (30.8%)	
No	2868 (70.2%)	4208 (69.2%)	
Renal failure			<0.0001
Yes	2185 (53.5%)	2286 (37.6%)	
No	1903 (46.5%)	3796 (62.4%)	
Malignancy			<0.0001
Yes	1302 (31.9%)	1171 (19.3%)	
No	2786 (68.2%)	4911 (80.8%)	
Year of infection onset			0.009
2006	545 (13.3%)	848 (13.9%)	
2007	615 (15%)	817 (13.4%)	
2008	448 (10.9%)	761 (12.5%)	
2009	485 (11.9%)	823 (13.5%)	
2010	500 (12.2%)	666 (10.9%)	
2011	423 (10.3%)	618 (10.1%)	
2012	362 (8.8%)	537 (8.8%)	
2013	383 (9.4%)	525 (8.6%)	
2014	333 (8.1%)	497 (8.2%)	

HCAI, healthcare-associated infection; CCI, Charlson Comorbidity Index; SSI, surgical site infection.

Numbers in strata may not equal total due to missing values.

[a]Continuous variables assessed using simple logistic regression (Wald χ^2). Categorical variables assessed using Pearson χ^2.

[b]High-risk medications include chemotherapeutic agents, immunosuppressants, and anti-inflammatory drugs.

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Table II

Significant risk factors for vancomycin-resistant enterococcus infection (logistic regression analysis)

Factor	OR	95% CI
Hospital		
Tertiary 2	Reference	Reference
Community	0.63	0.54–0.74
Tertiary 1	2.58	2.30–2.88
ICU stay	1.54	1.38–1.71
Charlson Comorbidity Index	1.04	1.02–1.06
Prior high-risk medication	8.19	5.96–11.26
Prior hospitalization	1.52	1.37–1.68
Renal failure	1.75	1.58–1.94
Maglignancy	1.46	1.29–1.66
Antibiotic use prior to infection	23.72	17.55–32.07
Sex		
Male	Reference	Reference
Female	1.29	1.17–1.43
Season		
Summer	Reference	Reference
Spring	1.16	1.02–1.33
Autumn	1.04	0.90–1.19
Winter	1.17	1.02–1.34
Length of stay prior to infection	1.03	1.02–1.03

OR, odds ratio; CI, confidence interval; ICU, intensive care unit.