MAJOR ARTICLE

Preventing the Influx of Vancomycin-Resistant Enterococci into Health Care Institutions, by Use of a Simple Validated Prediction Rule

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Background. The goal of this study was to develop a validated prediction rule for identification of patients harboring vancomycin-resistant enterococci (VRE) at hospital admission.

Methods. A model for the prediction of patients harboring VRE at admission was created and validated by assigning weighted point values to independent risk factors associated with harboring VRE at admission, in 2 different cohorts of patients from 2 tertiary care hospitals in Boston, Massachusetts. Patients with VRE isolated from clinical culture samples collected within 48 h of hospital admission were compared with patients not harboring VRE. To assess the diagnostic accuracy of the prediction rule, the main outcome measures were patient demographic characteristics, comorbid illnesses, hospitalizations, and antibiotic exposure.

Results. A total of 412 patients were enrolled. A risk index score was derived by using the following 6 independent risk factors associated with VRE recovery within 48 h of hospital admission: previous isolation of methicillin-resistant *Staphylococcus aureus* (MRSA), whether the patient was receiving long-term hemodialysis, transfer from a long-term care facility, antibiotic exposure, prior hospitalization, and age >60 years. On the basis of a point score $\geqslant$10, the sensitivity, specificity, and positive and negative predictive values of this prediction rule were 44%, 98%, 81%, and 90%, respectively.

Conclusions. This validated clinical prediction rule provides a novel strategy for the identification of patients at high risk of harboring VRE at hospital admission. Implementation of this rule may reduce the influx of VRE into health care institutions and the overall prevalence of VRE, by targeting VRE-screening measures and contact isolation precautions for these high-risk patients.

Widespread acquisition of vancomycin-resistant enterococci (VRE) is associated with grave implications in health care, including high morbidity and mortality rates and excess health care costs [1]. The recent recovery of strains of vancomycin-resistant *Staphylococcus aureus* (VRSA), which were produced by the transfer of vancomycin-resistant genes from VRE to *S. aureus* isolates [2, 3], further emphasizes the urgent need to prevent de novo acquisition of VRE.

VRE have become endemic in numerous health care institutions [4–7]. This endemic state is achieved by a constant influx of VRE into the health care setting, from newly admitted patients who are colonized or infected with VRE, followed by cross-transmission between hospitalized patients with de novo VRE acquisition and an efflux of VRE from the health care setting, owing to patient discharge or death. Developing effective prevention strategies necessitates an understanding of the various components required to achieve this endemic state.

The majority of prevention strategies focus on the middle component—that is, the prevention of cross-transmission between hospitalized patients. However, the influx of VRE into the health care setting may be an even more important factor in establishing endemicity, because eradication of VRE from a health care institution could be achieved, in theory, by prevention of this influx [8]. A mathematical model describing the transmission dynamics of VRE in an outpatient hemodialysis unit validates this concept. This model

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demonstrates that, although 100% compliance with hand washing or a nurse-to-patient ratio of 1:1 would substantially decrease the overall prevalence of VRE in the outpatient hemodialysis unit, prevention of the influx of VRE into the unit is the only intervention that completely eradicates VRE from this patient population over time [8].

Impeding the influx of VRE into a health care institution would require a program of active surveillance of culture samples obtained to detect gastrointestinal VRE colonization among all patients admitted to a hospital. Contact isolation precautions would be applied to patients with VRE colonization, to prevent cross-transmission of VRE to other hospitalized patients [9]. Given the low prevalence of VRE at admission, the screening of all newly admitted patients would not be feasible. A more efficient strategy would be to perform targeted screening of patients at high risk of harboring VRE at admission. This strategy would greatly reduce the number of negative culture results during screening and, ultimately, would both decrease the burden on hospital personnel and limit health care costs.

To identify the characteristics of patients at high risk for having VRE colonization at hospital admission, a 2–medical center, 6-year epidemiological analysis of patients harboring VRE was performed. From this analysis, a clinical prediction rule was developed that could identify patients at high risk for having VRE colonization at admission and that could indicate which subgroup of newly admitted patients required screening for VRE and contact isolation. The prediction rule then was validated by analysis of data from a separate cohort of high-risk patients, by means of standard methodological criteria [10].

PATIENTS AND METHODS

Derivation set. The derivation study was performed at the Beth Israel Deaconess Medical Center, Boston, and was approved by the hospital's Institutional Review Board. This hospital is a 430-bed tertiary care teaching hospital with 6 intensive care units (ICUs) and an average of 26,000 patient admissions per year.

Patient selection. From July 1997 through December 2001, clinical culture samples yielding enterococci were identified through a review of the computer summary report of the microbiology laboratory. Patients were enrolled only once, at the first recovery of VRE within 48 h of hospital admission during the study period. To avoid bias in identification of patients at high risk of harboring VRE, patients in whom VRE was detected by screening of rectal swab samples were excluded from the analysis.

To identify the risk factors for isolation of VRE from clinical culture samples obtained within 48 h of hospital admission, a case-control study was performed. A case patient was defined as an adult patient (age, $\geq$18 years) from whom VRE was isolated from at least 1 clinical culture sample collected within 48 h of hospital admission. One control subject was selected for each case patient. Control subjects were chosen from among all adult patients admitted to the hospital during the same year and from whom enterococci were not isolated during their hospital stay. If >1 control subject was available per case patient, the control subject who was admitted to the hospital at the date and time closest to those of the case patient was chosen.

Data collection. Electronic medical records for inpatient admissions and outpatient medical visits, including visits to physicians' offices and home visits from nurses, and databases of microbiology results and pharmacy records were reviewed. Data collected at study enrollment included patient demographic characteristics, whether the patient had been transferred from another hospital or was a resident of a long-term care facility or nursing home, previous hospitalization within 1 year of enrollment, ambulatory status, whether the patient was receiving long-term hemodialysis, and surgical procedures undergone during the 30 days prior to study enrollment. A composite score for comorbid illnesses was derived by using the Charlson score [11]. Antibiotics administered for at least 48 h during a 30-day period prior to study enrollment were recorded. Information on inpatient antibiotic exposure during previous hospitalizations was obtained through a computerized pharmacy database. Information on outpatient antibiotic exposure was determined from this database and from records of outpatient and nursing home visits. For the risk-factor analysis, exposure to oral and intravenous antibiotics was analyzed by antibiotic and by class and included penicillins, vancomycin, cephalosporins, antibiotics with predominantly anaerobic activity (metronidazole and clindamycin), aminoglycosides, quinolones, and imipenem. With regard to the prediction rule, antibiotics were analyzed as a group, and the mean number of different antibiotics received in the previous 30 days was used to dichotomize the data. Records of microbiology results also were reviewed for recovery of VRE or methicillin-resistant *S. aureus* (MRSA) in the 12 months prior to enrollment.

Validation set. The validation study was performed at the Brigham and Women's Hospital, Boston, and was approved by the hospital's Institutional Review Board. This hospital is a 750-bed tertiary care center with 8 ICUs, with 10 beds each, and ~40,000 patient admissions per year. Criteria for patient inclusion and definitions for case patients and control subjects that were identical to those used for the derivation set were applied to the validation cohort, with the following 2 exceptions. First, 3 control subjects were chosen for each case patient. Second, to avoid introduction of bias, case patients with a history of harboring VRE were excluded, because all patients with a history of harboring VRE who were admitted to Brigham and Women's Hospital during the study period underwent required screening of rectal swab samples at hospital admission. The

cohort included patients admitted to the hospital during the years of 2001 and 2002.

Microbiological analysis. Enterococci were identified by use of standard methods, including analysis of colony morphology, Gram stain, and standard biochemical tests [12]. API-Rapid Strep Strips (bioMérieux Vitex) were used to identify species. Screening for vancomycin resistance was performed by plating of isolates on brain-heart infusion media agar (Becton Dickinson) with 6 μg/mL vancomycin, and resistance was confirmed by MIC testing using the broth microdilution method [13]. Isolates with an MIC of vancomycin of $\geq$32 μg/mL were classified as resistant.

Statistical analysis. Quantitative variables were tested for distribution and were compared by using the Kruskal-Wallis test. Differences in group proportions were assessed by using the χ^2 test. Age was made dichotomous at the mean value. Potential risk factors for harboring VRE were analyzed by univariate analysis. Variables with an α value <0.05 were included in the regression analysis, and backward stepwise variable selections were performed, to define the best-fitting model. The discriminant ability of the models with regard to the derivation, validation, and combined cohorts was assessed by means of the area under the receiver operating characteristic (ROC) curve [14]. To develop the prediction rule, a scoring system using point values was developed. The natural logarithm of the OR for each risk factor selected by the logistic regression model was multiplied by 2 and rounded to the nearest integer. Sensitivity and specificity, with 95% CIs, for the prediction rule at different cut-off values, the positive predictive value (PPV), and the negative predictive value (NPV) were obtained by using standard definitions and methods [15, 16]. Statistical analysis was performed by using Intercooled Stata software, version 7.0 (Stata Statistical Software).

RESULTS

Derivation set. The rate of recovery of VRE from clinical culture samples collected within 48 h of hospital admission increased significantly, from 0.56 per 1000 patient admissions in 1998 to 1.52 per 1000 patient admissions in 2001 (P = .001; figure 1). A total of 15, 23, 42, and 43 single-patient VRE isolates (1 isolate was counted per patient per study year) were recovered within 48 h of hospital admission, during the years 1998, 1999, 2000, and 2001, respectively. The number of patient admissions from July 1997 through December 1997 was not available.

Patient characteristics. From July 1997 through December 2001, a total of 3100 clinical culture samples yielded enterococci. Of these culture samples, 1455 (47%) were obtained within 48 h of hospital admission; of these 1455 samples, 165 (11%), collected from 121 patients, yielded VRE. Only the first

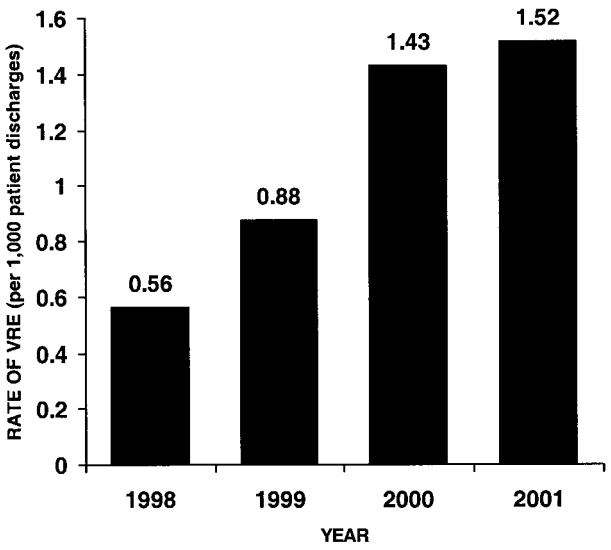

Figure 1. Rates of recovery of vancomycin-resistant enterococci (VRE) from clinical culture samples obtained within 48 h of hospital admission (determined on the basis of 1 isolate per patient, for each year of the study period).

VRE isolate recovered for each patient during the derivation-study period was included. Five patients were excluded because of missing demographic and clinical data. Thus, a total of 116 case patients were enrolled. Culture samples were obtained from urine (53%), wounds (24%), blood (15%), and other sites (8%). Table 1 summarizes the clinical and demographic characteristics of patients in the validation cohort.

Risk factors associated with isolation of VRE from clinical culture samples obtained within 48 h of hospital admission. Table 1 lists the unadjusted ORs, 95% CIs, and P values associated with patient demographic and clinical variables. Six independent risk factors associated with VRE recovery within 48 h of hospital admission were identified (table 2). For all 116 case patients, $\geq$2 independent risk factors were selected by the model, and $\geq$3 independent risk factors were selected for 111 case patients (96%). This model correctly identified 81% of the case patients, with a sensitivity of 77% and a specificity of 84%. The point values assigned to each risk factor are shown in table 3.

Validation set. From 2001 through 2002, enterococci were recovered from 409 clinical culture samples. A total of 45 isolates represented single-patient isolates recovered within 48 h of hospitalization, during the study period. With 3 control subjects enrolled for each case patient, a total of 180 patients were enrolled in the validation cohort. Patient demographic and clinical data are shown in table 1.

Accuracy of the prediction rule in identification of patients harboring VRE at hospital admission. A summary of the diagnostic accuracy of the risk index score, stratified by different

Table 1. Demographic and clinical characteristics of the derivation and validation cohorts of patients with and those without vancomycin-resistant enterococci (VRE) recovered from clinical culture samples obtained at hospital admission.

Characteristic	Derivation cohort				Validation cohort			
	VRE negative (n = 116)	VRE positive (n = 116)	OR (95% CI)	P	VRE negative (n = 135)	VRE positive (n = 45)	OR (95% CI)	P
Age, mean years ± SD	63 ± 20	69 ± 13	…	.004	49 ± 19	63 ± 13	…	<.01
Age of >60 years	68 (59)	89 (77)	1.5 (1.1–2.1)	<.01	42 (31)	27 (60)	2.4 (1.4–4)	<.01
Male sex	49 (42)	51 (44)	0.9 (0.7–1.2)	.79	53 (39)	25 (55)	1.9 (0.9–3.8)	.06
Race								
White	80 (70)	90 (78)	1.5 (0.8–2.6)	.17	106 (79)	32 (71)	0.7 (0.4–1.2)	.1
African American	36 (30)	16 (22)	…	…	29 (21)	13 (29)	…	…
Transfer from LTCF or hospital	24 (21)	72 (62)	2.3 (1.7–3)	<.01	5 (4)	16 (35)	4.1 (2.7–6.2)	<.01
Nonambulatory	26 (22)	61 (53)	1.8 (1.4–2.3)	<.01	123 (91)	31 (69)	2.6 (1.6–4.3)	<.01
Reason for admission								
Medical	102 (88)	103 (89)	0.9 (0.6–1.4)	.83	84 (62)	35 (78)	1.7 (0.9–3.3)	.06
Surgical	14 (12)	13 (11)	…	…	51 (38)	10 (22)	…	…
Prior hospitalizations[a]								
0	53 (46)	15 (13)	0.3 (0.2–0.5)	<.01	112 (83)	2 (4)	0.2 (0–0.10)	<.01
1	29 (25)	24 (21)	0.8 (0.6–1.2)	.43	12 (9)	10 (22)	2 (1.1–3.5)	.01
2	12 (10)	21 (18)	1.3 (0.9–1.7)	.09	6 (4)	11 (24)	3.1 (1.9–4.9)	<.01
⩾3	22 (19)	56 (48)	1.8 (1.4–2.3)	<.01	5 (3)	22 (48)	5.4 (3.5–8.2)	<.01
Mean no. ± SD	1.3 ± 1.9	3.1 ± 3.1	…	<.01	0.3 ± 1	2.9 ± 2	…	<.01
Charlson score, mean ± SD	2.4 ± 2.3	3.8 ± 2.2	…	<.01	1.2 ± 1.9	4 ± 2.4	…	<.01
Transplant recipient	1 (1)	6 (5)	1.7 (1.2–2.4)	.06	2 (1)	4 (8)	2.8 (1.5–5.2)	.03
Long-term hemodialysis	5 (4)	25 (21)	1.8 (1.4–2.3)	<.01	1 (1)	9 (20)	4.2 (2.9–6)	<.01
Previous surgery[b]	15 (13)	54 (46)	2 (1.6–2.5)	<.01	4 (3)	13 (29)	3.8 (2.5–5.8)	<.01
Previous recovery of MRSA[a]	3 (2)	35 (30)	2.2 (1.8–2.6)	<.01	1 (1)	9 (20)	4.2 (2.9–6)	<.01
Exposure to ⩾2 antibiotics[b]	22 (19)	70 (60)	2.3 (1.7–3)	<.01	4 (3)	28 (62)	7.64.7–12.1	<.01

NOTE. Data are no. (%) of patients, unless otherwise indicated. LTCF, long-term–care facility; MRSA, methicillin-resistant *Staphylococcus aureus.*

[a] Within 1 year of study enrollment.

[b] Within 30 days of study enrollment.

cut-off values for the derivation, validation, and combined cohorts, is shown in table 4. Figure 2 displays the ROC curves for the derivation, validation, and combined cohorts, for different scores. Extrapolated PPVs and NPVs for different prevalences of VRE recovery at hospital admission are shown in figure 3.

Because this risk index score would be targeted for patients at high risk of harboring VRE, defined as those patients with a score of ⩾10, the prevalence of this subgroup of patients was estimated by using data from the combined cohort, as follows. We assumed that 2% of patients admitted to an institution will have a score of ⩾10. This percentage was derived from the number of control subjects with a score of ⩾10. Case patients with a score of ⩾10 were excluded, because their contribution to the percentage of patients with a score of ⩾10 was minimal. Thus, of the 66,000 patients admitted per year to both hospitals, 1320 (2%) would have a score ⩾10. During the last 12 months of patient enrollment for the derivation and validation cohorts, 27 and 23 patients, respectively, were found to harbor VRE at hospital admission. Of these 50 patients, we estimated that 22 patients (44% of VRE-positive patients correctly identified by the score) would have a score of ⩾10. If the use of clinical culture samples identifies only 1 of 10 patients with VRE colonization [6], then ~220 patients would have VRE colonization at hospital admission. Thus, the prevalence of VRE among patients with a score of ⩾10 is assumed to be 17% (220 of 1320 patients). At this prevalence, the PPV and NPV of a risk score of ⩾10 are 81% and 89.5%, respectively (figure 3).

Table 2. Logistic regression analysis of risk factors associated with the recovery of vancomycin-resistant enterococci from patients in the derivation cohort, within 48 h of hospital admission.

Risk factor	OR (95% CI)	P
Previous recovery of MRSA[a]	9.36 (2.42–36.24)	.001
Long-term hemodialysis	5.80 (1.66–20.31)	.006
Transfer from LTCF or hospital	4.60 (2.23–9.49)	<.001
Exposure to ⩾2 antibiotics[b]	4.28 (2.02–9.09)	<.001
Previous hospitalization[a]	3.71 (1.65–8.35)	.001
Age of >60 years	2.92 (1.32–6.42)	.008

NOTE. Area under the receiver operating characteristic curve was 0.87. LTCF, long-term care facility; MRSA, methicillin-resistant *Staphylococcus aureus.*

[a] Within 1 year of study enrollment.

[b] Within 30 days of study enrollment.

Table 3. Risk index score for recovery of vancomycin-resistant enterococci at hospital admission, by associated risk factor.

Risk factor	Point value
Previous recovery of MRSA[a]	4
Long-term hemodialysis	3
Transfer from LTCF or hospital	3
Exposure to ⩾2 antibiotics[b]	3
Previous hospitalization[a]	3
Age >60 years	2

NOTE. LTCF, long-term–care facility; MRSA, methicillin-resistant *Staphylococcus aureus*.

[a] Within 1 year of study enrollment.
[b] Within 30 days of study enrollment.

DISCUSSION

Limiting the dissemination of VRE throughout a health care institution requires a comprehensive approach that targets the various components required to sustain VRE endemicity. These components include the influx of VRE into the institution from patients harboring VRE at hospital admission, dissemination of VRE from these patients by de novo acquisition by other hospitalized patients, and an efflux of VRE out of the institution. This 2–medical center study addresses the importance of the influx of VRE into the health care institution, an essential step in the propagation of VRE and the establishment of endemicity. In this study, a significant increase in the number of patients with VRE infection or colonization at hospital admission, from 0.56 patients per 1000 admissions in 1998 to 1.5 patients per 1000 admissions in 2001, was documented. The 3-fold increase over a 4-year period suggests a troubling trend for the future and emphasizes the need for early identification of these patients by means of prompt implementation of measures to limit the at-risk time for the spread of VRE from these patients.

The clinical prediction rule developed and validated in this 2–medical center study provides a very specific test that identifies patients with VRE infection or colonization at hospital admission, among a high-risk group of patients. Six variables were identified, for which data are easily obtained during assessment of patients, and weighted scores were applied: the variables were age of >60 years, hospitalization in the previous year, exposure to ⩾2 antibiotics within the previous 30 days, transfer from another hospital or long-term care facility, requirement for long-term hemodialysis, and history of MRSA infection. When a cut-off value of ⩾10 was used for the point score, the specificity of this prediction rule was 98% for the combined derivation and validation cohorts. When the prevalence of VRE colonization was assumed to be 17% among patients with a score ⩾10 at hospital admission, the PPV and NPV for the prediction rule were 81% and 90%, respectively. The predictive values were similar for both cohorts of study patients, who were from 2 different hospitals, further validating the accuracy of the prediction rule. These results suggest that use of this prediction rule for patients who have a score of ⩾10

Table 4. Accuracy of the prediction rule in identification of patients harboring vancomycin-resistant enterococci (VRE) at hospital admission for the derivation, validation, and combined cohorts, stratified by cut-off values for the risk index score.

Cohort, cut-off value	Sensitivity, % (95% CI)	Specificity, % (95% CI)	False-positive result, % (95% CI)	False-negative result, % (95% CI)
Combined (n = 412)[a]				
⩾10	44 (36–52)	98 (95–99)	2 (1–5)	56 (48–64)
⩾9	56 (48–64)	97 (94–99)	3 (1–6)	44 (36–52)
⩾8	73 (66–80)	92 (88–95)	8 (5–12)	27 (20–34)
⩾7	75 (68–82)	92 (88–95)	8 (5–12)	25 (18–32)
Derivation (n = 232)[b]				
⩾10	49 (40–59)	96 (90–99)	4 (1–10)	51 (41–60)
⩾9	59 (49–68)	93 (87–97)	7 (3–13)	41 (32–51)
⩾8	75 (66–82)	84 (77–90)	15 (9–23)	25 (17–34)
⩾7	77 (68–84)	84 (77–90)	15 (9–23)	23 (16–32)
Validation (n = 180)[c]				
⩾10	31 (18–47)	100 (97–100)	0 (0–3)	69 (53–81)
⩾9	49 (34–64)	100 (97–100)	0 (0–3)	51 (36–66)
⩾8	69 (53–81)	99 (96–100)	1 (0–4)	31 (18–47)
⩾7	71 (56–83)	99 (96–100)	1 (0–4)	29 (16–44)

[a] No. of patients VRE positive, 161; no. of patients VRE negative, 251.
[b] No. of patients VRE positive, 116; no. of patients VRE negative, 116.
[c] No. of patients VRE positive, 45; no. of patients VRE negative, 135.

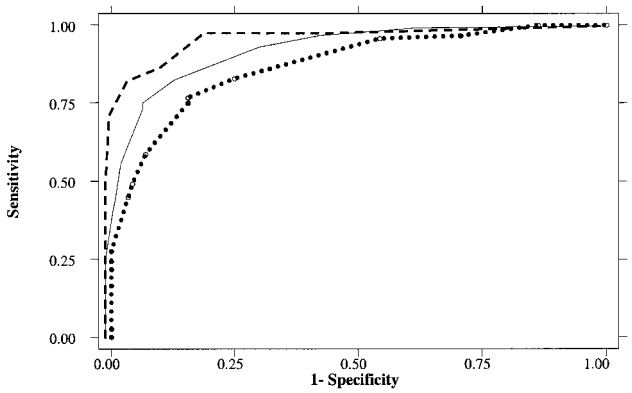

Figure 2. Receiver operating characteristic (ROC) curves displaying sensitivity versus specificity for scoring system comparing patients with and those without vancomycin-resistant enterococci colonization at hospital admission, for the derivation cohort *(dotted line),* the validation cohort *(dashed line),* and the combined cohort *(solid line).* The area under the ROC curve is 87% for the derivation cohort, 96% for the validation cohort, and 92% for the combined cohort.

at hospital admission would correctly identify a large proportion of patients harboring VRE at hospital admission.

Screening programs for identification of VRE colonization among patients already hospitalized have been studied extensively [17–22]. These programs are based on the important premise that, for every patient from whom VRE is recovered from clinical culture samples, many other patients with VRE colonization have not been identified [6, 23, 24]. Tremendous benefits from these programs have been achieved by decreasing over time the overall prevalence of VRE and the number of VRE infections [18, 20]. The cost-effectiveness of this approach also has been demonstrated [18, 25, 26]. These strategies target the middle component of the endemic state—that is, cross-transmission among hospitalized patients. The clinical prediction rule in this study provides an additional strategy by targeting the influx of VRE into the hospital setting and identifying patients harboring VRE at hospital admission. This strategy would limit the potential for VRE dissemination from these patients with unrecognized reservoirs at the start of their hospitalization, as opposed to the strategies mentioned above, which use screening programs to target patients already hospitalized. Although the influx of VRE into the hospital would not change, the benefit of early detection is obvious, because the time period in which VRE dissemination could occur would be greatly reduced. By limiting the implementation of this clinical prediction rule to only those patients with a point score of $\geqslant$10 at hospital admission, the number of patients requiring screening would be greatly reduced, increasing the feasibility of this strategy. The minimal cost and inconvenience associated with obtaining and processing a rectal swab sample also facilitates the successful implementation of this screening program [18].

Implementation of this prediction rule in the clinical setting would require 2 steps. First, patients with a point score of $\geqslant$10 at hospital admission would be identified as at high risk of harboring VRE. Second, VRE infection–control precautions would be applied, on an empirical basis, to this group of patients until results of the screening rectal culture were available. Alternatively, screening could be done and precautions applied only after VRE were detected in the rectal swab sample. The latter approach would be favored only in those circumstances in which resources are limited. Moreover, many hospitals have mechanisms that track patients harboring VRE and automatically apply precautions at readmission. For these hospitals, the application of this clinical prediction rule will provide an additional benefit by identifying patients not known to have harbored VRE.

There were several limitations in this study that require discussion. First, although a cohort study design in which surveillance rectal culture samples for VRE detection were obtained from every patient at admission to the hospital would have been preferable, the number of patients harboring VRE at hospital admission necessitated a case-control study design, which is optimal for infrequent positive outcomes. Second, although a cost-effective analysis demonstrating the beneficial impact of implementing this clinical prediction rule for the screening of high-risk patients at hospital admission was not performed, numerous cost-effective analyses of VRE-screening programs for hospitalized patients have been performed and have shown substantial cost reductions [18, 25, 26]. For MRSA detection, the screening of high-risk patients at hospital admission, with implementation of contact precautions, is the most cost-effective strategy [27]. Third, although the overall number of patients harboring VRE at hospital admission was low, only clinical culture samples were used to detect potential case patients. If patients had been screened for VRE colonization by culture

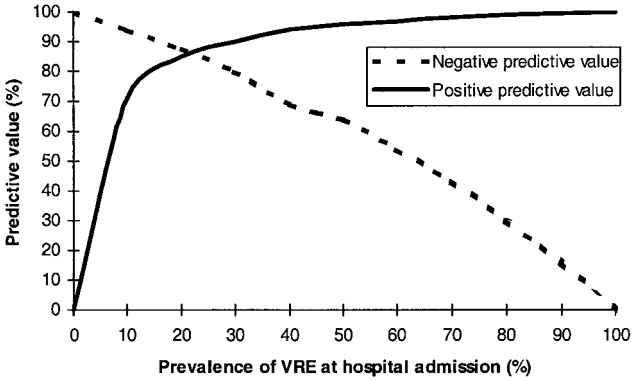

Figure 3. Negative and positive predictive values of the scoring system, for the combined derivation and validation cohorts, by increasing prevalence of patients harboring vancomycin-resistant enterococci (VRE) at hospital admission.

of rectal swab samples, the prevalence would have been substantially higher [6, 14, 23, 24]. Furthermore, the marked increase in the number of patients harboring VRE at hospital admission during the years of the study emphasizes the need to focus on this reservoir of VRE. Last, the PPV of this clinical prediction rule decreases substantially at rates of VRE prevalence of <10% among patients with a score of ⩾10 at hospital admission. Thus, implementation of this clinical prediction rule may be more effective in hospitals with >10% of patients with a point score of ⩾10 and with VRE colonization at hospital admission.

Despite ongoing efforts to limit the increase in the number of patients who harbor VRE, rates of VRE colonization continue to increase throughout the world. The clinical prediction rule developed and validated in this 2-center study provides a novel and diagnostically accurate strategy for the potential prevention of VRE dissemination from the reservoir of VRE entering the hospital and, ultimately, for a decrease in cross-transmission and de novo acquisition of VRE. The minimal cost, inconvenience, and discomfort and the proven reduction in cross-transmission of VRE with the use of screening programs lend further evidence that this strategy could have a substantial impact on the continual increase of VRE colonization and infection.

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