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Dynamic Risk Prediction for Hospital-Acquired Pressure Injury in Adult Critical Care Patients

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IMPORTANCE: Accurately measuring the risk of pressure injury remains the most important step for effective prevention and intervention. Time-dependent risk factors for pressure injury development in the adult intensive care unit setting are not well understood.

OBJECTIVES: To develop and validate a dynamic risk prediction model to estimate the risk of developing a hospital-acquired pressure injury among adult ICU patients.

DESIGN: ICU admission data were split into training and validation sets. With death as a competing event, both static and dynamic Fine-Gray models were developed to predict hospital-acquired pressure injury development less than 24, 72, and 168 hours postadmission. Model performance was evaluated using Wolbers' concordance index, Brier score, net reclassification improvement, and integrated discrimination improvement.

SETTING AND PARTICIPANTS: We performed a retrospective cohort study of ICU patients in a tertiary care hospital located in San Francisco, CA, from November 2013 to August 2017.

MAIN OUTCOMES AND MEASURES: Data were extracted from electronic medical records of 18,019 ICU patients (age ≥ 18 yr; 21,220 encounters). Record of hospital-acquired pressure injury data was captured in our institution's incident reporting system. The information is periodically reviewed by our wound care team. Presence of hospital-acquired pressure injury during an encounter and hospital-acquired pressure injury diagnosis date were provided.

RESULTS: The dynamic model predicting hospital-acquired pressure injury more than 24 hours postadmission, including predictors age, body mass index, lactate serum, Braden scale score, and use of vasopressor and antifungal medications, had adequate discrimination ability within 6 days from time of prediction ($c = 0.73$). All dynamic models produced more accurate risk estimates than static models within 26 days postadmission. There were no significant differences in Brier scores between dynamic and static models.

CONCLUSIONS AND RELEVANCE: A dynamic risk prediction model predicting hospital-acquired pressure injury development less than 24 hours postadmission in ICU patients for up to 7 days postadmission was developed and validated using a large dataset of clinical variables readily available in the electronic medical record.

KEY WORDS: critical care; electronic health records; forecasting; pressure ulcer; risk assessment; risk factors

Pressure injuries (PIs), also known as pressure ulcers, decubitus ulcers, or bed sores, are a serious form of tissue damage caused by a combination of pressure, friction, shearing forces, and moisture. They most frequently occur over bony prominences such as the back of the head, ischium, sacrum, and heels. Hospital-acquired pressure injuries (HAPIs) are PIs that occur during inpatient stays, often in patients with a variety of risk factors, such as older age,

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decreased perfusion, immobility, poor nutritional status, severe illness, and diabetes (1).

Pressure ulcers have a significant impact on short-term outcomes in the U.S. inpatient populations: patients with at least one pressure ulcer have double the median length of stay and median treatment costs, and more than a five-fold increase in mortality compared with patients without ulcers (2). Altogether, patients who develop the PIs are subject to increased pain, secondary infections, longer hospital stays, and an increased likelihood of mortality due to related complications such as secondary infections and sepsis (2, 3).

Accurate risk assessment plays a critical role in providing preventive measures in a time-sensitive manner. Current prevention measures rely mainly on subjective risk assessment coupled with periodic patient repositioning intended to alleviate or relocate pressure (4). The most well-known PI risk assessment tool is the Braden Scale—a clinical checklist that scores patients' sensory perception, moisture, activity, mobility, nutrition, friction, and shear on a one-to-four scale, with patients scoring below a cutoff (usually 17) considered higher risk (5). Although the Braden Scale has been widely adopted, its clinical utility shows mixed results, with some studies showing it to be a significant predictor for PI (6, 7), whereas other studies failed to show this relationship or found it significant only in an unadjusted analysis (8, 9). A review of clinical studies comparing the efficacy of the Braden Scale and similar risk assessment scales found no positive clinical impact attributable to their application (10). The subjective nature of the Braden Scale combined with its inconsistent clinical utility indicates that a better risk prediction tool for HAPI in the ICU is needed.

The prevalence of hospitals using electronic medical records (EMRs) presents a significant opportunity to use the granular data collected by EMR, including data from multiple time points, to investigate risk factors related to disease development. The dynamic risk prediction framework allows for time-dependent covariates (11–13). For example, among patients who have not had a HAPI by 24 hours postadmission, risk factors such as laboratory values and medications ordered up until 24 hours postadmission can be used to predict HAPIs. Similarly, among patients who have not had a HAPI by 72 hours postadmission, risk factors up until 72 hours postadmission can be used to predict HAPIs. Dynamic risk prediction has recently been used in several disease areas, including diabetes (11), heart failure (14, 15), and colorectal liver metastases (16). However, many studies predicting PIs have only made static predictions using risk factor data at or up to a single time point (17–22). We sought to develop and validate a dynamic risk score at different time points of a patient's hospital course to accurately predict the risk of developing a HAPI among adult ICU patients.

MATERIALS AND METHODS

Setting

Our tertiary care center located in San Francisco, CA, uses an EMR to integrate medical information, including diagnoses, medications, laboratory results, charting, and procedure codes. The study protocol was reviewed and approved by our Institutional Review Board (IRB) (Human Research Protection Program IRB—Laurel Heights Committee, approval number 13-10753). Since the study was retrospective and met the criteria for minimal risk to participants, the requirement for informed consent was waived. This study followed the Transparent Reporting of a Multivariable Prediction Model for Individual Prognosis or Diagnosis guideline.

Study Design and Population

We conducted a retrospective cohort study among adult (age ≥ 18 yr) University of California San Francisco Health members hospitalized in the ICU from November 13, 2013, to August 31, 2017. Record of HAPI data is captured in our institution's Incident Reporting system, and the information is periodically reviewed by our wound care team to confirm a true HAPI diagnosis. Presence of HAPI during an encounter and date of HAPI diagnosis were provided. Each hospitalization encounter in the ICU was treated independently in the analysis.

Data Collection and Organization

We collected five components from the EMR software EPIC (EPIC Systems Corporation, Verona, WI): 1) demographics, 2) preadmission and inhospital encounter diagnoses, 3) administered inpatient medications, 4) flowsheet variables (measurements of patient taken at bedside by a provider or a machine), and 5) laboratory results (**Supplemental Table 1**, http://links.lww.com/CCX/A850). The time of medication was approximated by the time when ordered.

Development of the Hospital-Acquired Pressure Injury Risk Score

To determine risk factors at different times during a patient's admission, four patient cohorts were created from the eligible patient encounter data: 1) full sample (baseline cohort) to predict any HAPI, 2) subsample excluding encounters with HAPI, death, or discharge from ICU recorded within 24 hours postadmission (24-hr cohort) to predict HAPIs at least 24 hours postadmission, 3) subsample excluding encounters with HAPI, death, or discharge from ICU recorded within 72 hours postadmission (72-hr cohort) to predict HAPIs at least 72 hours postadmission, and 4) subsample excluding encounters with HAPI, death, or discharge from ICU recorded within 168 hours (7 d) postadmission (168-hr cohort) to predict HAPIs at least 168 hours postadmission. A static prediction model, only using baseline predictors, was developed in each of the four cohorts; and a dynamic prediction model, using both baseline predictors and predictors that are updated at specific "landmark times," was developed in each of the three postbaseline cohorts. **Supplemental Figure 1*A–D*** (http://links.lww.com/CCX/A850) includes information on which variables were considered for static and dynamic models.

The following procedures were performed on each of the four cohorts: ICU admissions were randomly split (7:3) into training and internal validation sets (Supplemental Fig. 1*A–D*, http://links.lww.com/CCX/A850). The validation set was reserved for evaluating the resulting risk score developed in the training set. Characteristics of the training and validation samples were described (**Supplemental Table 2*A–D***, http://links.lww.com/CCX/A850). Candidate predictors of HAPI were determined using literature review, clinical judgment, and analyses in the training set. Two senior resident physicians, attending surgeon, and attending critical care physician reviewed the top 110 potential predictors by odds ratio value and discussed their clinical importance and relevance. Only variables with less than 50% missing data were considered as potential predictors. The missing category was analyzed if any variable had greater than 5% missing data.

Fine-Gray subdistribution hazard models were used to account for death during ICU stay as a competing event. Please see **Supplemental Methods** for more discussion of the competing-risks methods (http://links.lww.com/CCX/A850). Unadjusted analysis was performed to assess the association between each candidate predictor and HAPI. If a variable had both continuous and categorical options, the categorical version was used in order to improve utility and interpretation of the resulting risk score. Backward elimination selection was used to develop a multivariable model. Candidate predictors that had associations with p value of less than 0.1 from the unadjusted analyses were included in a multivariable analysis, and covariates not attaining significance at the 0.05 level were sequentially eliminated until all covariates were significantly associated with the outcome.

A regression coefficient-based approach (23) was used to develop a points-based scoring system from the selected predictors in the final models. Plots of the cumulative incidence function (CIF) stratified by quintiles of the risk score were generated, and differences in these risk strata were assessed using Fine-Gray models.

Evaluation of Risk Score Performance

Model performance was evaluated in the validation set using Wolbers' concordance index for prognostic models with competing risks (24), Brier score (25, 26), net reclassification improvement, and integrated discrimination improvement (27–31), and calibration plots. Models including only Braden Scale score categories from the 24-, 72-, and 168-hour cohorts were also performed, translated to risk scores, and validated using Wolbers' concordance index in order to compare their performance to our study's risk score performance.

Statistical Analysis

Hypothesis tests were two-sided, and the significance threshold was set to 0.05. Statistical analyses were performed using SAS (Version 9.4, SAS Institute, Cary, NC) and R (Version 4.0.0, R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Characteristics and Multivariable Fine-Gray Competing Risk Models of Each Cohort

Table 1 and Supplemental Table 2*A–D* (http://links.lww.com/CCX/A850) describe characteristics in each cohort's training and validation sets. **Supplemental Table 3*A–D*** (http://links.lww.com/CCX/A850) contains the final candidate lists of predictors for backward elimination selection, along with descriptive statistics by HAPI/death/censored status, in each cohort's training set. **Supplemental Table 4** (http://links.lww.com/CCX/A850) lists variables with less than 5% missing data by cohort.

TABLE 1.
Characteristics in 24-Hour Cohort Training and Validation Samples

Variable	% (*n*) or Mean ± SD (*n*)	
	Training (*n* = 14,025)	Validation (*n* = 6,012)
Outcome at least 24 hr after admission		
HAPI diagnosis	1.9 (266)	1.8 (111)
Death without HAPI	6.6 (924)	6.6 (394)
Median (IQR) days from admission to HAPI diagnosis	10 (5–19)	12 (6–20)
Median (IQR) days from admission to death	9 (5–17)	9 (5–18)
Characteristics at start of encounter		
Age (yr)	61.3 ± 17.4 (14,025)	61.2 ± 17.1 (6,012)
Male gender	53.1 (7,452)	53.3 (3,207)
Body mass index (kg/m^2)	27.0 ± 7.2 (13,553)	26.9 ± 7.1 (5,797)
Preexisting diagnoses		
Diabetes	21.6 (3,022)	20.9 (1,257)
Chronic pulmonary disease	21.3 (2,988)	22.4 (1,347)
Renal failure	22.0 (3,080)	23.6 (1,414)
Congestive heart failure	14.0 (1,967)	14.7 (885)
Peripheral vascular disease	8.3 (1,164)	8.4 (506)
Laboratory results within 24 hr of admission		
Maximum of WBC values (1,000 cells/μL)	12.9 ± 18.2 (12,662)	12.8 ± 14.0 (5,411)
Minimum of sodium values (mg/dL)	135.4 ± 4.6 (12,555)	135.4 ± 4.6 (5,371)
Minimum of hematocrit values (%)	32.0 ± 6.8 (12,673)	32.0 ± 7.0 (5,423)
Maximum of $Paco_2$ values (mm Hg)	45.2 ± 11.3 (8,003)	45.1 ± 11.1 (3,454)
Minimum of Pao_2 (mm Hg)	98.5 ± 94.6 (7,893)	97.9 ± 93.8 (3,411)
Maximum of creatinine values (mg/dL)	1.4 ± 1.8 (12,645)	1.4 ± 1.8 (5,410)
Minimum of blood pH values	7.33 ± 0.10 (8,049)	7.33 ± 0.10 (3,472)
Minimum of platelet count values (1,000 cells/μL)	191.4 ± 97.9 (12,628)	193.2 ± 98.4 (5,397)
Maximum of lactate serum values (mg/dL)	2.8 ± 2.7 (7,674)	2.9 ± 2.9 (3,312)
Maximum of nonfasting glucose values (mg/dL)	169.9 ± 106.5 (9,742)	171.1 ± 111.8 (4,186)
Medication ordered within 24 hr of admission		
Paralytic	4.7 (627)	4.5 (257)
Vasopressor	25.4 (3,413)	26.7 (1,537)
Antibiotic	63.4 (8,525)	62.5 (3,591)
Antifungal medication	44.1 (5,931)	44.1 (2,532)
Minimum of Braden Scale score within 24 hr of admission	16.3 ± 3.5 (12,780)	16.4 ± 3.5 (5,439)
6–12	14.7 (2,060)	14.2 (851)
13–14	13.0 (1,821)	12.9 (778)
15–16	17.3 (2,423)	17.0 (1,023)
17–23	46.2 (6,476)	46.4 (2,787)
Missing	8.9 (1,245)	9.5 (573)

HAPI = hospital-acquired pressure injury, IQR = interquartile range.

TABLE 2.
Covariates in 24-Hour Cohort Multivariable Prediction Models and Associated Point Values for Hospital-Acquired Pressure Injury

Model	Variable	Log-SHR (β)	SHR (95% CI)	*p*	Points
Model with static predictors only	Age ≥ 50 yr	0.444	1.56 (1.13–2.16)	0.007	4
	BMI (kg/m²)				
	Underweight: < 18.5 vs normal (18.5–<25)	0.450	1.57 (1.04–2.38)	0.034	5
	Overweight: 25–<30 vs normal (18.5–<25)	−0.226	0.80 (0.57–1.12)	0.187	−2
	Class 1 obesity: 30–<35 vs normal (18.5–<25)	0.284	1.33 (0.93–1.91)	0.124	3
	Class 2 obesity: 35–<40 vs normal (18.5–<25)	0.371	1.45 (0.89–2.37)	0.138	4
	Class 3 obesity: ≥ 40 vs normal (18.5–<25)	0.705	2.02 (1.28–3.20)	0.003	7
Model with both dynamic and static predictors	Age ≥ 50 yr	0.366	1.44 (1.02–2.04)	0.040	4
	BMI (kg/m²)				
	Underweight: < 18.5 vs normal (18.5-< 25)	0.376	1.46 (0.95–2.24)	0.088	4
	Overweight: 25–<30 vs normal (18.5–<25)	−0.224	0.80 (0.56–1.13)	0.208	−2
	Class 1 obesity: 30–<35 vs normal (18.5–<25)	0.273	1.31 (0.90–1.91)	0.152	3
	Class 2 obesity: 35–<40 vs normal (18.5–<25)	0.397	1.49 (0.90–2.47)	0.124	4
	Class 3 obesity: ≥ 40 vs normal (18.5–<25)	0.728	2.07 (1.32–3.26)	0.002	7
	Maximum of lactate serum values within 24 hr of admission (mg/dL)				
	> 3 vs ≤ 3	0.356	1.43 (1.05–1.95)	0.025	4
	Missing vs ≤ 3	−0.343	0.71 (0.51–0.99)	0.046	−3
	Vasopressor medication ordered within 24 hr of admission	0.307	1.36 (1.01–1.83)	0.045	3
	Antifungal medication ordered within 24 hr of admission	0.345	1.41 (1.07–1.86)	0.014	3
	Minimum of Braden Scale score within 24 hr of admission				
	6–12 vs 17–23	0.487	1.63 (1.13–2.36)	0.010	5
	13–14 vs 17–23	−0.054	0.95 (0.61–1.47)	0.808	−1
	15–16 vs 17–23	−0.008	0.99 (0.65–1.51)	0.970	0
	Missing vs 17–23	0.291	1.34 (0.78–2.29)	0.289	3

BMI = body mass index, SHR = subdistribution hazard ratio.

Table 2 and **Supplemental Table 5*A–C*** (http://links.lww.com/CCX/A850) present the results from the multivariable Fine-Gray models in each of the four study cohorts. Supplemental Figure 1*A–D* (http://links.lww.com/CCX/A850) summarizes the numbers of ICU admissions analyzed and HAPI cases included in each analysis.

Twenty-Four-Hour Cohort Dynamic Multivariable Prediction Model

In the 24-hour dynamic model, six variables were significantly associated with higher risk of HAPI: age greater than or equal to 50 years, BMI greater than or equal to 40, maximum of lactate serum values greater than 3 mg/dL, vasopressor medication use, antifungal medication use, and minimum Braden Scale score 6–12 (all data within 24 hr postadmission). Missing lactate serum was also significantly associated with lower risk of HAPI (Table 2).

The left panel of **Figure 1** presents plots of the CIF for HAPI at least 24 hours postadmission stratified by quintiles and their associated point values of the dynamic model risk score. The corresponding estimates of the CIFs for death without HAPI at least 24 hours postadmission are presented in the right panel of Figure 1.

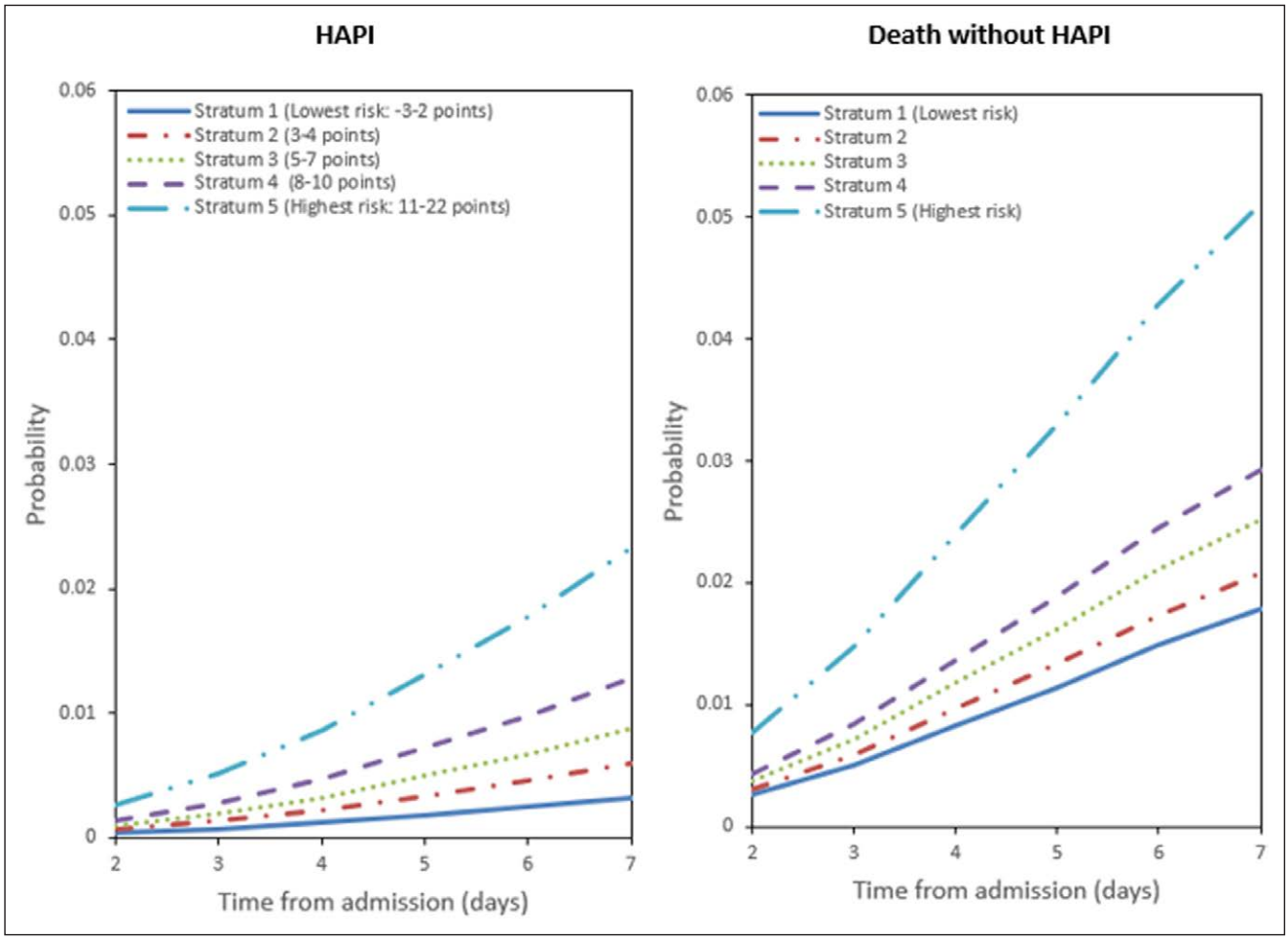

Figure 1. Cumulative incidence function for hospital-acquired pressure injury (HAPI) and competing risk death at least 24 hr from admission by quintiles of dynamic model risk score.

There were also statistically significant differences in CIFs for strata 3, 4, and 5 compared with stratum 1. **Supplemental Table 6** (http://links.lww.com/CCX/A850) summarizes these results from all models from the three cohorts. **Supplemental Table 7** (http://links.lww.com/CCX/A850) shows how a clinician can use our dynamic prediction model with a patient case example.

Performance of the Hospital-Acquired Pressure Injury Risk Score

Table 3 and **Supplemental Table 8** (http://links.lww.com/CCX/A850) summarize the performances of all model risk scores in the internal validation set. Supplemental Figure 1*A–D* (http://links.lww.com/CCX/A850) summarizes the numbers of encounters analyzed and HAPI cases included in each analysis. Overall, the 24-hour dynamic model demonstrated the highest discrimination ability for HAPI within 7 days or less postadmission (Table 3). The static model risk score differentiated adequately between patients who did and did not have a HAPI within 2 days postadmission. Discrimination ability deteriorated drastically for greater than or equal to 3 days from admission. The 24-hour dynamic model risk score had concordance indices greater than or equal to 0.7 within 3, 5, and 7 days postadmission, respectively, and discrimination ability was poor for greater than or equal to 8 days postadmission. In contrast, a 24-hour risk score based on Braden Scale score categories alone could only discriminate adequately within 5 days postadmission. The dynamic model produced more accurate risk estimates than the static model between 2 and 26 days postadmission for the 24-hour models.

Calibration plots for each model risk score are presented in **Figure 2** and **Supplemental Figure 2*A–C*** (http://links.lww.com/CCX/A850). The 45° line indicates perfect calibration. For the baseline model risk score, observed and predicted probability of HAPI agreed well for predicted risk values less than or equal

TABLE 3.
Concordance Indices for Models in Internal Validation Set

Day	Concordance Indices						
	Baseline Risk Score	24-hr Risk Scores		72-hr Risk Scores		168-hr Risk Scores	
		Dynamic Model[a]	Static Model	Dynamic Model[b]	Static Model	Dynamic Model	Static Model
1	**0.92**	NA	NA	NA	NA	NA	NA
2	**0.92**	**0.74**	0.60	NA	NA	NA	NA
3	0.66	**0.78**	0.62	NA	NA	NA	NA
4	0.59	**0.72**	0.57	0.69	0.38	NA	NA
5	0.59	**0.75**	0.64	0.65	0.50	NA	NA
6	0.66	**0.72**	0.62	0.67	0.51	NA	NA
7	0.64	**0.73**	0.60	0.69	0.47	NA	NA
8	0.64	0.66	0.59	0.66	0.44	0.62	0.23
9	0.59	0.63	0.57	0.64	0.50	0.54	0.34
10	0.60	0.64	0.58	0.62	0.51	0.59	0.42
20	0.56	0.59	0.58	0.59	0.56	0.56	0.57
30	0.57	0.59	0.59	0.62	0.58	0.53	0.54

NA = not applicable.
[a]The *c*-indices for the 24-hr dynamic model predicting hospital-acquired pressure injury (HAPI) events over the entire prediction interval (> 24 hr) vs only within the 24- to 72-hr interval were 0.61 and 0.74, respectively.
[b]The *c*-indices for the 72-hr dynamic model predicting HAPI events over the entire prediction interval (> 72 hr) vs only within the 72- to 168-hour interval were 0.58 and 0.67, respectively.
Bolded values are ≥ 0.7.

to 9%, with lack of calibration in the upper range of predicted risk; but only less than 3% of encounters had predicted risk greater than 9% (Supplemental Fig. 2*A*, http://links.lww.com/CCX/A850). For the 24-hour model risk score, observed and predicted probabilities were fairly well-calibrated for predicted risk values less than or equal to 10%, and a small percentage (< 2%) of encounters had predicted risk greater than 10% (Fig. 2). For the 72- and 168-hour model risk scores, observed and predicted probability were fairly well-calibrated overall (Supplemental Fig. 2, *B* and *C*, http://links.lww.com/CCX/A850).

DISCUSSION

In this large, single-institution retrospective cohort study, we developed and validated a dynamic risk prediction model based on detailed and chronologically updated clinical information available in the EMR for 21,220 encounters to predict the risk of developing a HAPI greater than 24 hours postadmission among adult ICU patients. Our model could make acceptable predictions up to 7 days postadmission, further out from time of admission than a model using Braden Scale score categories alone.

Our finding of older age predicting HAPI development is consistent with previous literature (32). Multiple chronic diseases, such as cardiovascular diseases, diabetes, chronic pulmonary disease, renal diseases, and neurodegenerative disorders, are found in the older adult population and contribute to the development of HAPI (33). However, we found that increased risk in the elderly population persists despite adjustment for common comorbid conditions, demonstrating that older age is an independent risk factor for HAPI development.

BMI was previously found to be predictive of HAPI development in a U-shaped relationship. Two studies found that underweight and extremely obese patients were at higher risk for developing HAPI (34, 35). Our

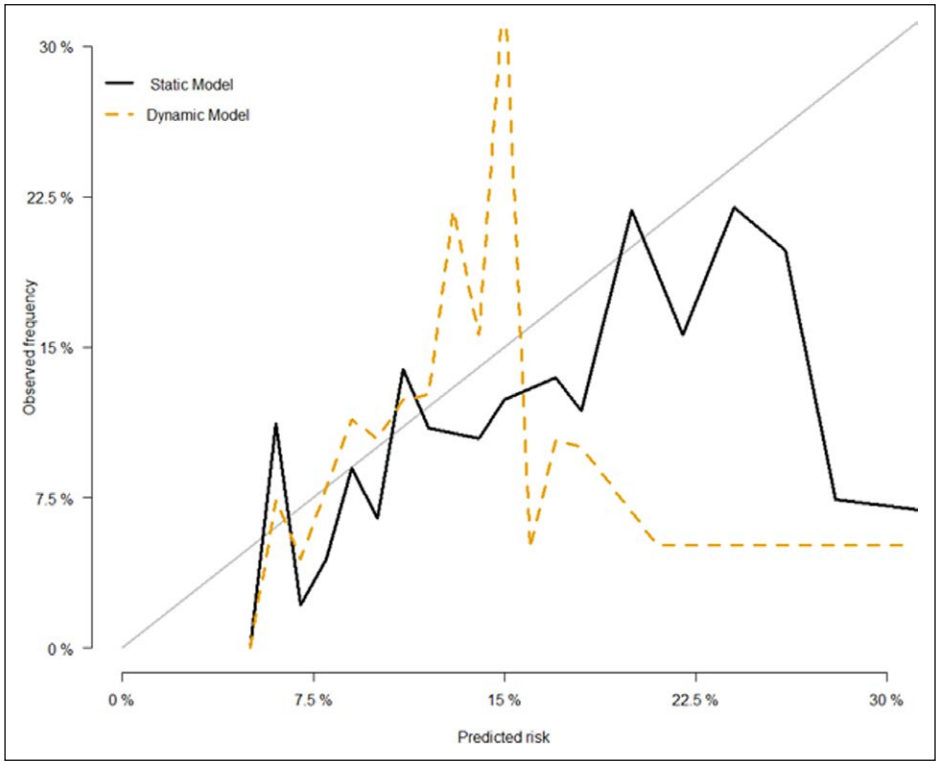

Figure 2. Calibration plots for 24-hr cohort prediction model risk scores.

study also confirmed class 3 obesity to be predictive of HAPI development. Obesity is thought to affect PI formation in several ways, including making visualization and hygiene of vulnerable areas more difficult, directly increasing pressure on vulnerable areas, and as a risk factor for metabolic disease.

Our finding that a maximum of lactate serum values of greater than 3 mg/dL predicts HAPI development is expected, as a higher lactate serum value often indicates tissue hypoperfusion and shock (36). The lack of lactate serum measurement being negatively associated with HAPI development is not surprising, as it likely reflects the scenario that the provider was less concerned about shock and organ perfusion in a patient.

Consistent with our study's finding that vasopressor medication use is predictive of HAPI development, a previous study found that the addition of vasopressin administered alongside a first-line agent may represent the point at which the risk for HAPI escalates and may be an early warning to take preventative measures (37). In a literature review of HAPI development and vasopressor agents in adult critical care patients, Cox (38) found that seven of 10 studies identified reported statistically significant associations.

Longstanding HAPIs are frequently colonized by microorganisms in a biofilm, which may be composed of bacteria, fungi, or other organisms embedded in the underlying wound, preventing the wound from healing (39). Antimicrobial agents, such as antibacterial and antifungal medications, are often used to treat infected HAPIs (40). Furthermore, fungal infections in the critical care setting are often associated with significant morbidity and mortality; Aspergillus pneumonia is reported to have a mortality rate of 25%, with up to 90% in liver transplant recipients (41). Our finding that antifungal medication ordered prior to HAPI development is predictive of HAPI is likely reflected in patients being treated for systemic fungal infections.

Although the Braden Scale score has several limitations related to low specificity (42), in our study, only a minimum score less than or equal to 12 during the ICU encounter proved to be a significant risk factor. Although ostensibly an objective scale, the Braden Scale still represents a clinical judgment by a provider. Providers who feel that patients are at high risk for the HAPI may choose lower scores on the scale, and capturing that clinical judgment may be the primary benefit of the Braden Scale. Another benefit is that subscales of the Braden Scale aim to capture poor nutritional status. Serum albumin was missing for greater than 50% of patient encounters, so we could not assess albumin level as a marker of nutritional status. Moisture of a wound is another variable that is difficult to capture via EMR data, where the Braden Scale may provide value.

Overall, our model demonstrates that clinical predictors from the first 24 hours of ICU admission can predict development of HAPI up to 7 days after admission. Our dynamic prediction model allows the clinician to use the patient's characteristics and EMR data from the first 24 hours in the ICU to easily hand calculate the risk score and stratify patients based on risk of

HAPI. It must be emphasized that this model does not suggest causality. It is possible that some factors in the risk score can be directly modified, such as the nutrition subscore of the Braden Scale. However, the main function of the score is anticipated to be identifying high-risk patients that could benefit from increased monitoring and available preventative interventions (more frequent turning, padding, pressure reallocating beds, etc.) to improve outcomes. Last, our model can also be integrated into the EMR to automatically calculate dynamic HAPI risk scores for all the ICU patients, including those missing data for certain components of the model.

This study has several limitations. First, although our dynamic model predicting HAPI greater than 24 hours postadmission had adequate discrimination ability at 1 week, it became poor past 7 days postadmission. This suggests the need of real-time dynamic modeling updated per every hospital stay day elapsed. Second, although we developed a scoring system for clinical use, missing lactate serum was predictive of lower risk of HAPI greater than 24 hours postadmission. Only cases with complete data on all score components can be hand-calculated. Although the decision by a clinician not to order a lactate cannot be part of a risk score used directly by humans, the risk score that includes this missing category can be used by computers, that is, pulled directly from EMR data and calculated. Third, the retrospective nature of our study carries the risk of confounding. For example, the association of vasopressor medication use with HAPI may be a true effect from vasoconstriction and hypoxia of vulnerable tissue but may also result from concomitant sedation or mechanical ventilation. Finally, although our dataset was large, spanning 4 years of ICU patient encounters, our retrospective review is from a single institution; validation of the risk model with the EMR data from other institutions would allow confidence in expanding its use beyond our study population.

CONCLUSIONS

A dynamic risk prediction model predicting HAPI development greater than 24 hours postadmission in ICU patients for up to 7 days postadmission was developed and validated using a large dataset of clinical variables readily available in the EMR. This model can aid clinicians in selecting high-risk patients early in their ICU admission for increasing monitoring and focused interventions to prevent HAPI formation. Our risk score can also be implemented automatically in the EMR as a measure for clinicians to be aware of high-risk patients when considering interventions to reduce PI formation.

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