Risk Factors for Hospital-Acquired Pressure Injury in Surgical Critical Care Patients

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Background Hospital-acquired pressure injuries disproportionately affect critical care patients. Although risk factors such as moisture, illness severity, and inadequate perfusion have been recognized, nursing skin assessment data remain unexamined in relation to the risk for hospital-acquired pressure injuries.

Objective To identify factors associated with hospital-acquired pressure injuries among surgical critical care patients. The specific aim was to analyze data obtained from routine nursing skin assessments alongside other potential risk factors identified in the literature.

Methods This retrospective cohort study included 5101 surgical critical care patients at a level I trauma center and academic medical center. Multivariate logistic regression using the least absolute shrinkage and selection operator method identified important predictors with parsimonious representation. Use of specialty pressure redistribution beds was included in the model as a known predictive factor because specialty beds are a common preventive intervention.

Results Independent risk factors identified by logistic regression were skin irritation (rash or diffuse, nonlocalized redness) (odds ratio, 1.788; 95% CI, 1.404-2.274; $P<.001$), minimum Braden Scale score (odds ratio, 0.858; 95% CI, 0.818-0.899; $P<.001$), and duration of intensive care unit stay before the hospital-acquired pressure injury developed (odds ratio, 1.003; 95% CI, 1.003-1.004; $P<.001$).

Conclusions The strongest predictor was irritated skin, a potentially modifiable risk factor. Irritated skin should be treated and closely monitored, and the cause should be eliminated to allow the skin to heal.(*American Journal of Critical Care.* 2020;29:e128-e134)

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Patients admitted to the intensive care unit (ICU) are twice as likely as other acute care patients to have a hospital-acquired pressure injury (HAPI) develop.[1] A pressure injury (PI) is defined as localized damage of the skin or underlying tissue as a result of pressure or pressure in combination with shear.[2] Patients who undergo surgery and who are older than 65 years have a higher risk than younger patients of acquiring a PI in the hospital.[3,4] In the United States, PI costs attributed to patients exceed $26.8 billion annually,[5] and having a HAPI develop results in a median 4-day increase in the length of stay.[6]

Determining the factors associated with HAPI development in critical care patients is necessary to enable risk-based preventive measures. Although HAPIs are associated with known risk factors such as decreased mobility, surgery duration, vasopressor infusion, excessive moisture, altered perfusion, and history of a prior PI, the relationship between HAPIs and skin status remains mostly unexamined in the critical care population.[4,7-18] Assessing skin status (including turgor, excessive dryness, irritation, skin tears, and the loss of subcutaneous tissue) to identify potential HAPI prevention interventions is particularly essential when caring for older patients because of age-related changes. Such changes include thinning skin, decreased subcutaneous tissue, flattening of the dermal-epidermal junction (decrease in rete ridges), structural disorganization of collagen fibers in the dermis, loss of vertical capillary loops, and loss of elasticity.[2]

Using informatics to analyze the vast amounts of electronic health record (EHR) data, such as skin assessment data, routinely produced during care delivery is an excellent way to identify risk factors for HAPI development. Critical care nurses routinely conduct head-to-toe skin assessments every 12 hours and document changes in condition in the EHR. However, these large-scale real-world data have not been fully examined in relation to HAPIs in the surgical critical care setting.

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The unprecedented quantities and diverse sources of data collected during care delivery make this an opportune time to conduct HAPI research. The purpose of our study was to identify factors associated with HAPI development among surgical critical care patients. Our specific aim was to examine data obtained from routine nursing skin assessments along with other previously reported HAPI risk factors.

Methods

Design and Sample

This was a retrospective cohort study. We included data from surgical critical care patients admitted consecutively to the surgical ICU (SICU) or cardiovascular surgical ICU (CVICU) at our study site, an urban level I trauma center and academic medical center, from 2014 through 2018. We included patients with a PI present on admission to the hospital because patients with prior PIs are at increased risk for subsequent HAPIs.[16] We did not count community-acquired PIs as HAPIs because they were not acquired in the hospital. However, if patients with a community-acquired ulcer had a HAPI develop, that subsequent PI was included in the analysis because it was hospital acquired. The exclusion criterion was a stay of less than 24 hours because of inadequate time for a HAPI to be considered a facility-acquired PI.

The relationship between hospital-acquired pressure injuries and skin status remains mostly unexamined in the critical care population.

Data Collection

Data were obtained via EHR query and retrieved from our institution's enterprise data warehouse for critical care data. For patients with multiple hospital admissions, we limited data collection to the first SICU or CVICU admission. A biomedical informatics team performed the query. Query results were validated by a critical care nurse who verified information obtained (including date and time stamps)

via the human-readable system EHR for 30 patients, including 15 patients with HAPIs. A practicing critical care nurse and a certified wound nurse also manually reviewed medical records, including data from the notes and images, to obtain data that were missing or unclear in the query.

Outcome Variable

The outcome variable was the development of a HAPI of any stage (stages 1 through 4, deep tissue injury, or unstageable) according to the National Pressure Injury Advisory Panel staging guidelines.[2] We included stage 1 HAPIs in our outcome because prior studies showed that one-third of stage 1 HAPIs detected among surgical critical care patients worsen to stage 2 or greater.[19] A certified wound nurse verified the PIs in our sample to differentiate potential cases of moisture-related skin breakdown from true HAPIs. In cases in which a HAPI might be confused with another source of injury, the certified wound nurse made the final decision as to the presence or absence of the HAPI. We were able to differentiate between community-acquired PIs and HAPIs because each PI in our EHR has a unique identification number with a date and time stamp.

Data from more than 5000 consecutive surgical critical care patients were analyzed retrospectively.

Predictor Variables

We conducted a systematic review of the literature to identify predictor variables of interest.[4] Possible predictor variables included vasopressor infusions and their durations,[17] blood gas and laboratory values,[18,19] surgical time,[20] levels of sedation and agitation,[21] and total score on the Braden Scale (a common tool used by nursing staff to assess the risk of PI development by examining moisture, mobility, sensory perception, and friction/shear).[22]

We included comprehensive nursing skin assessment data. At our facility, nurses undergo annual training in head-to-toe skin assessment and PI staging. Nurses at our facility conduct a global head-to-toe skin assessment twice daily and document the following changes: excessively moist skin, excessively dry skin, thin epidermis with loss of subcutaneous tissue, and the presence of irritation (defined as a rash or diffuse, nonlocalized, blanchable redness). Nurses also document the presence of a skin tear. Table 1 lists the predictor variables included in our analysis.

For patients who had a HAPI develop, we collected data only for events occurring at least 24 hours before HAPI detection. We chose this time frame to capture events predictive of a HAPI rather than events occurring at the same time as a HAPI.

Analysis

Analysis was conducted with R, version 3.6.1 (R Foundation for Statistical Computing).[23] We summarized and compared the distributions of potential prediction factors by HAPI status with a χ^2 test for categorical factors and a 2-sample *t* test (or its nonparametric alternative, the Mann-Whitney *U* test) for continuous and ordinal variables. We performed multivariable logistic regression analysis with the least absolute shrinkage and selection operator (LASSO)[24] to identify the subset of potential predictors most informative for predicting the likelihood of a HAPI developing. The final model for outcomes was based on the optimal penalty term using 10-fold cross-validation criteria.

By imposing some penalty in the regression model fitting, the LASSO approach can shrink the coefficients of unimportant predictors to 0 while retaining prominent predictors. A predictor has predictability on the outcome only if its coefficient is nonzero. The final models, therefore, include all important predictors with parsimonious representation, enhanced interpretability, and improved prediction precision. In this study, the variable *specialty bed* was forced into the model as a known prediction factor (even though our general SICU and CVICU bed is a low-air-loss mattress) because some of our patients were placed on other types of specialty rental beds (eg, bariatric beds or specialty prone positioning beds) because of body habitus or clinical condition.[25]

Results

Sample

The initial query produced 5102 patients. We excluded 1 patient from the analysis because of incomplete demographic data, so the final sample size was 5101. Demographic data are shown in Table 1.

Pressure Injury Outcomes

Of the 5101 patients in our sample, 399 (8%) had at least 1 HAPI develop. Of the 399 patients with a HAPI, 110 (28%) had a stage 1 HAPI develop; 182 (46%), stage 2 HAPI; 6 (2%), stage 3 HAPI; 1 (<1%), stage 4 HAPI; 33 (8%), unstageable HAPI; 62 (16%), deep tissue injury; and 5 (1%), mucosal PI. Of the 110 stage 1 HAPIs, 44 (40%) worsened to a more severe stage during the SICU or CVICU stay. The most common PI location was the coccyx (n = 153

Table 1
Potential predictor variables and development of hospital-acquired pressure injury

Variable	No. (%) of patients[a] All (N=5101)	With no HAPI (n=4702)	With a HAPI (n=399)	*P*
Demographic data				
Age, mean (SD), y	58 (17)	59 (16)	58 (16)	.24
Sex, male	3302 (65)	3040 (65)	262 (66)	.73
Race, White	4256 (83)	3934 (84)	322 (81)	.14
Ethnicity, non-Hispanic	4452 (87)	4112 (87)	340 (85)	.17
Length of hospital stay, mean (SD), d	12 (11)	11 (9)	28 (20)	<.001
Length of ICU stay before HAPI, mean (SD), d	5 (7)	5 (6)	13 (13)	<.001
Laboratory data, mean (SD)				
Maximum lactate, mg/dL	4.0 (3.7)	3.9 (3.6)	5.6 (4.8)	<.001
Maximum serum creatinine, mg/dL	1.9 (1.9)	1.8 (1.9)	2.7 (2.1)	<.001
Maximum serum glucose, mg/dL	231 (148)	227 (141)	280 (210)	<.001
Minimum hemoglobin, g/dL	8.9 (2.6)	9.1 (2.6)	7.7 (2.2)	<.001
Minimum albumin, g/dL	3.1 (0.8)	3.2 (0.8)	2.7 (0.7)	<.001
Minimum Pao_2, mm Hg	54 (40)	55 (41)	47 (32)	<.001
Minimum arterial pH	7.27 (0.11)	7.27 (0.10)	7.23 (0.13)	<.001
Maximum $Paco_2$, mm Hg	52 (14)	52 (13)	55 (16)	<.001
Skin status				
Thin epidermis/subcutaneous tissue loss	888 (17)	792 (17)	96 (24)	<.001
Excessively dry skin	351 (7)	296 (6)	55 (14)	<.001
Skin tear	641 (13)	534 (11)	107 (27)	<.001
Excessively moist skin	816 (16)	712 (15)	104 (26)	<.001
Irritated skin[b]	1394 (27)	1176 (25)	218 (55)	<.001
Community-acquired pressure injury present at admission	167 (3)	120 (3)	47 (12)	<.001
Duration of surgery, mean (SD), h				
Longest single surgery	3.0 (2.6)	3.0 (3.2)	3.3 (2.5)	.08
Total surgical time	3.7 (3.4)	3.6 (3.3)	4.6 (4.7)	<.001
Duration of vasopressor infusion, mean (SD), h				
Norepinephrine	9 (36)	7 (33)	30 (62)	<.001
Epinephrine	8 (35)	7 (31)	23 (61)	<.001
Phenylephrine	1 (8)	1 (14)	2 (20)	.01
Dopamine	1 (14)	6 (13)	23 (19)	.12
Vasopressin	11 (55)	9 (51)	37 (86)	<.001
Other potential predictors				
Minimum Braden Scale score, mean (SD)	13 (3)	13 (3)	12 (3)	<.001
Minimum Riker score,[c] mean (SD)	2.8 (1.2)	2.87 (1.19)	2.15 (1.22)	<.001
Admission body mass index,[d] mean (SD)	30.1 (12.4)	30.1 (12.5)	30.2 (10.7)	.89
Nonstandard bed (eg, bariatric bed or other)	1390 (27)	1234 (26)	156 (39)	.73
Comorbid diabetes	1756 (34)	1579 (34)	177 (44)	<.001

Abbreviations: HAPI, hospital-acquired pressure injury; ICU, intensive care unit.

[a] Unless otherwise indicated in first column.
[b] Irritated skin is defined as a rash or diffuse, nonlocalized, blanchable redness, not over a bony prominence.
[c] Riker Sedation-Agitation Scale.
[d] Calculated as weight in kilograms divided by height in meters squared.

[38%]), followed by the buttocks (n = 62 [16%]), sacrum (n = 47 [12%]), extremity excluding heel (eg, arms or legs; n = 46 [12%]), head or face (n = 40 [10%]), other location (n = 32 [8%]), back (n = 10 [3%]), and heel (n = 9 [2%]).

Pressure Injury Predictors

Univariate relationships between potential predictor variables and HAPI development are presented in Table 1. From the soft-thresholding property of the LASSO in linear models, the estimated regression coefficient is biased toward 0. To mitigate these bias problems, we report a more unbiased estimation of regression coefficients from unpenalized multivariate logistic regression using the selected factors in the LASSO (Table 2).

Discussion

The purpose of our study was to identify risk factors for HAPI development among SICU and CVICU

Table 2
Results of LASSO logistic regression[a]

Predictor variable	Odds ratio (95% CI)	*P*
Intercept	0.278 (0.147-0.523)	<.001
Irritated skin[b]	1.788 (1.404-2.274)	<.001
Minimum Braden Scale score	0.858 (0.818-0.899)	<.001
Duration of stay in intensive care unit before hospital-acquired pressure injury	1.003 (1.003-1.004)	<.001
Specialty bed[c]	0.816 (0.634-1.044)	.11

Abbreviation: LASSO, least absolute shrinkage and selection operator.

[a] A total of 5019 patients (98%) were included in the logistic regression; 82 patients' data were excluded from the analysis because of missing data.
[b] Irritated skin is defined as a rash or diffuse, nonlocalized, blanchable redness, not over a bony prominence.
[c] Included in the model as a control factor because specialty beds were used inconsistently.

patients. Identifying risk factors is useful to improve our understanding and care planning for patients considered high risk and to recognize factors that are potentially modifiable. In our study, candidate predictor variables included the duration of vasopressor infusion, blood gas values, surgery duration, Braden Scale scores, nursing skin assessment data, and laboratory values. In multivariable LASSO regression, the most informative predictors for HAPI risk were length of SICU or CVICU stay, the minimum Braden Scale score, and skin irritation (defined as a rash or diffuse, nonlocalized, blanchable redness).

A longer hospital stay is an established risk factor for HAPI because patients with longer stays generally experience a higher severity of illness and longer exposure times than do patients with shorter stays.[9,10,14] Consistent with the results of prior studies, in our study the duration of ICU stay before HAPI was an independent predictor for HAPI development, although the effect was small.[7,17,26]

The Braden Scale, developed in 1987 for residents of long-term care facilities,[22] was found in a recent meta-analysis to be a poor predictor of HAPI among surgical patients.[27] In our study, patients with lower Braden Scale scores (ie, at greater risk) were 14% more likely to have a HAPI develop than were patients with higher Braden Scale scores. The clinical relevance of this finding is uncertain because the mean (SD) minimum Braden Scale score was 13 (3) in patients without a HAPI and was 12 (3) in patients with a HAPI. On a scale with possible scores ranging from 6 to 23, this absolute difference is relatively small and the corresponding standard deviation is large, so this finding may not be actionable at a clinical level.[28] Black[29] speculated that the lack of clinical utility of the Braden Scale in this population is because of the dynamic and evolving nature of critical care patients' physiological status. In the critical care population, a risk assessment would need to be completed contemporaneously with changes in patient condition, which would be difficult because of time and workflow constraints.

Of the 110 stage 1 HAPIs, 44 (40%) worsened to a more severe stage during the patient's stay in the intensive care unit.

The strongest predictor of HAPI was skin irritation, a potentially modifiable risk factor. In our study, patients with skin irritation were 79% more likely than those with no skin irritation to have a HAPI develop. Skin irritation indicates an alteration in skin integrity and therefore a decrease in tissue tolerance to mechanical and shearing forces, such as those responsible for HAPI development.[16,30] Skin irritation may be caused by excessive skin dryness, allergic reactions to medications, or prolonged exposure to caustic substances acting as irritants, including urine, feces, strong soaps, laundry chemicals, and latex gloves. In all cases, skin irritation should be treated and closely monitored and the cause should be eliminated to allow the skin to heal.

Potential predictor variables not included in our LASSO model merit consideration as well. Clinically and statistically significant differences at the univariate level were noted in variables measuring aspects of perfusion, defined as the delivery of oxygen-rich blood to tissue. The mean serum lactate level in the HAPI group was markedly elevated, indicating tissue hypoperfusion and hypoxia.[31] Serum albumin (which affects perfusion via colloid osmotic pressure) and hemoglobin (oxygen-carrying capacity) were also decreased in the HAPI group. In addition, patients with HAPIs had clinically and statistically significantly longer infusion durations for all vasopressors than did patients without HAPIs.

Consistent with the results of a prior study,[32] patients with HAPIs in our study experienced longer surgical times, highlighting the importance of considering intraoperative events in HAPI risk. However, although surgical critical care patients are at elevated risk for HAPI,[3] little is known about intraoperative factors associated with HAPI risk in the surgical and cardiovascular surgical critical care population. In a study of patients undergoing urologic procedures, duration of anesthesia and a diastolic blood pressure of less than 50 mm Hg were predictive of HAPI development, indicating that perfusion during surgery may influence HAPI risk.[33,34] Research is urgently needed to identify intraoperative risk

factors in surgical critical care patients[33] and to identify potentially modifiable risk factors.

Limitations

Our study was limited by its retrospective design because we accessed only data available in the EHR. The subjectivity of clinician interpretation is also a limitation; individual nurses' definitions of *skin irritation* may not exactly coincide. Furthermore, we did not differentiate medical device–related HAPIs from other HAPIs. Other predictor variables that have been associated with HAPI in this population were not selected because these variables could not be obtained from the EHR. We did not include compliance with PI prevention protocols (eg, repositioning schedules) because the EHR is not a reliable source of information about preventive interventions. For instance, every 2 hours our EHR prompts nursing staff to document a position change. However, the changes might be faithfully documented every 2 hours but not always performed.[35] Finally, our sample was from a single site with a predominantly White population, which may also affect the generalizability of our results.[35,36]

Conclusions

Our results indicate that nursing staff should consider changes in the epidermal layer, especially skin irritation, to be influential risk factors for HAPI. Skin irritation should be promptly treated by eliminating the cause. The SICU and CVICU patients who had HAPI develop in our study also exhibited poor perfusion and longer surgical times. Future research is needed to elucidate the relationship between perfusion, intraoperative events, and HAPI risk.

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SEE ALSO

For more about hospital-acquired pressure injuries, visit the *Critical Care Nurse* website, **www.ccnonline.org**, and read the article by Schroeder and Sitzer, "Nursing Care Guidelines for Reducing Hospital-Acquired Nasogastric Tube–Related Pressure Injuries" (December 2019).

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