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Research Article

Predictors of pressure injury development in critically ill adults: A retrospective cohort study

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ABSTRACT

Objective: The purpose of this research was to identify predictors of pressure injury, using data from the electronic health records of critically ill adults.
Methodology: A retrospective cohort study was conducted using logistic regression models to examine risk factors adjusted for age, gender, race/ethnicity and length of stay.
Setting: The study cohort included 1587 adults in intensive care units within an urban academic medical centre.
Main Outcome Measures: The presence or absence of a hospital-acquired pressure injury was determined during monthly skin integrity prevalence surveys. All pressure injuries were independently confirmed by two Certified Wound Care Nurses.
Results: Eighty-one (5.1%) of the 1587 cohort patients developed pressure injuries. After adjusting for confounders, the clinical variables associated with pressure injury development included mean arterial pressure <60 mmHg and lowest Total Braden score up to two weeks prior to the date of HAPI development or date of prevalence survey for the comparison group.
Conclusions: This study provides a more comprehensive understanding about pressure injury risk in critically ill adults, identifying extrinsic and intrinsic factors associated with pressure injury development. Prospective multisite studies are needed to further examine these potential contributors to pressure injury development within the context of adherence to prevention interventions.

Implications for clinical practice

- HAPI continues to occur in critically ill patients, at rates higher than patients in general acute care, even when nurses adhere to clinical practice guidelines. Healthcare professionals need to more fully understand all risk factors for HAPI development.
- Patients with HAPI more often had vasopressor infusions, higher oxygen concentrations and larger fluid bolus volumes; lower MAP, PF ratios, platelet counts, GCS scores, and Braden scale scores; and longer LOS and sepsis/septic shock diagnoses.
- MAPs < 60 mmHg and lower total Braden scores were predictors of HAPI development.
- Using clinical predictors from the electronic health records of patients with expert-validated HAPI may contribute to the development of a more accurate real-time ICU risk assessment.

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Introduction

Though the Agency for Healthcare Research and Quality (AHRQ, 2019) reports a 13% reduction in overall hospital-acquired conditions since 2014, hospital-acquired pressure injuries (HAPI) increased 6% during this same time. Regarded as reflecting the quality of nursing care since the advent of national nursing-sensitive quality indicators (Gallagher and Rowell, 2003), HAPI can lead to serious health and economic consequences including: pain and discomfort, infection/sepsis, greater utilisation of healthcare resources, longer length of stay and higher healthcare and litigation costs (AHRQ, 2017; Bennett et al., 2000; NPUAP, 2014), among others.

For decades, clinical practice guidelines for HAPI prevention have focused on frequent skin assessment coupled with reducing the effects of pressure, moisture, friction/shear, immobility, inadequate nutrition, sensory impairment and inactivity (AHCPR, 1992; NPUAP and EPUAP, 2009; WOCN, 2016). In spite of compliance with these guidelines and advances in health technology to aid in prevention, patients in the intensive care unit (ICU) continue to develop pressure injuries (Cox, 2013, 2011; Cox and Roche, 2015; Schmitt et al., 2017) at rates higher than those in general acute care (Chaboyer et al., 2018; Coyer and Tayyib, 2017; NPUAP, 2014). A recent meta-analysis reported a cumulative pressure ulcer prevalence of 16.9–23.8% (95% CI) for adults in intensive care (Chaboyer et al., 2018).

Professional organisations including the National Pressure Ulcer Advisory Panel (NPUAP) and the Wound Ostomy and Continence Nurses Society acknowledge that there are multiple factors that potentially contribute to HAPI development and that current assessment tools do not incorporate all relevant risk factors (NPUAP, 2014; Schmitt et al., 2017). A need has been recognised for additional research examining risk factors most predictive of HAPI in the critically ill (Bly et al., 2016; Cox, Schallom, & Jung, 2020; Schmitt et al., 2017; Tschannen and Anderson, 2020). Of late, investigators have endorsed prioritising the evaluation of clinical variables already present in the electronic health record (EHR), as this knowledge could be used to develop electronic HAPI risk assessments that are more accurate and that can be provided in real-time (Cox, Schallom, & Jung, 2020).

Though investigators have examined a multitude of extrinsic (e.g. moisture, shear) and intrinsic risk factors (e.g., hypotension, comorbidities) experienced by critically ill adults (Bly et al., 2016;, Cox, 2011; Cox and Roche, 2015; Kirkland-Kyhn, et al., 2017; Slowikowski and Funk, 2010; Sookyung et al., 2014; Tschannen et al., 2012) the factors significant in past multivariable models of ICU HAPI development have varied. Inconsistencies in findings may be partially attributed to methods of past research. In a systematic review of risk factors for pressure injuries in critically ill adults, the majority of studies contained multiple potential sources of bias (Alderden et al., 2017). For example, more than half of the studies reviewed did not use wound nurses/nurses who were specially trained to identify the pressure injuries (Alderden et al., 2017).

Methods

Objective

The purpose of this retrospective cohort study was to identify predictors of HAPI in adult intensive care units, using clinical variables from the EHRs of patients examined by nurses specially trained in the identification and staging of pressure injuries.

Study setting, ethical approval, and participants

The research was conducted using data from a cohort of adult patients admitted to the intensive care unit (ICU) at [X], an urban academic medical centre. This study was approved by the University's Institutional Review Board and a waiver of consent was obtained (IRB16-1034-CR001). We used a convenience sample of patients admitted to five ICUs (medical, surgical, cardiothoracic, cardiac and neurology), comprised of 76 patient beds. Inclusion criteria consisted of all intensive care patients physically examined during the hospital's monthly HAPI prevalence surveys between 1/1/2014 and 14/7/2016. Patients were excluded if they had a pressure injury upon ICU admission (community acquired or hospital acquired prior to ICU admission), were under the age of 18 years, or admitted to ICU for<24 hours.

Per hospital guidelines, all patients received current evidence-based practices to prevent pressure injury (Berlowitz et al., 2014; NPUAP and EPUAP, 2009; Ratliff and Tomaselli, 2010; Walsh et al., 2012). Further description of the prevention practices in the intensive care units and medical surgical floors are reported elsewhere (Solmos et al., 2019). Nursing staff received HAPI prevention education upon hire and on an ongoing basis. A hospital-wide registered nurse Skin Care Team, staff nurses who receive more extensive ongoing skin care education, assisted in the integration of evidence-based practices into patient care.

Data collection and variables

Data were collected from quality improvement records and EHRs. The study cohort was identified using records from monthly HAPI prevalence surveys conducted during the study period. During prevalence surveys, all ICU patients were evaluated for presence or absence of skin injury by a Skin Care Team nurse; two Certified Wound Care Nurses independently examined all skin injuries and if the injury was confirmed as a HAPI, verified its staging. All stages of pressure injury were included; the HAPI were staged using criteria from the NPUAP staging system (Black et al., 2007; Edsberg et al., 2016). Patients without HAPI throughout their ICU stay were included in the comparison group.

The Clinical Research Data Warehouse maintained by the Center for Research Informatics at [X] provided all EHR data. Demographic variables included age, race and gender. The clinical variables were: mean arterial blood pressure (MAP), vasopressor use, fluid bolus administration, total Glasgow coma scale (GCS) scores, fraction of inspired oxygen (FiO2), oxygen saturation (SaO2), partial pressure arterial oxygen and fraction of inspired oxygen (PF) ratio, total bilirubin, platelet count and creatinine. Total Braden scores were also collected. The possible Braden score range is from 6 to 23, with lower scores indicating higher risk of developing pressure injury (Bergstrom et al., 1987). Scores on the Glasgow Coma Scale range from 3 to 15 with lower scores indicating decreased level of consciousness and a score of $\leq$ 8 indicating severe unconsciousness (Teasdale et al., 2014). Additional clinical variables included length of stay (LOS) (total hospital and hospital to event date), diagnose of sepsis (sepsis, septicaemia, severe sepsis or septic shock), and comorbid conditions of myocardial infarction, congestive heart failure, cerebrovascular accident, chronic pulmonary disease and diabetes mellitus; diagnoses were identified using International Classification of Diseases (ICD) codes at hospital discharge. Diagnosis codes (ICD 9/10) for comorbidity categories in the Charlson Comorbidity Index were used to classify comorbid conditions (Charlson et al, 1987).

For patients with HAPI confirmed at a prevalence survey, clinical variables were collected for up to two weeks of ICU admission prior to the date the patient's HAPI was initially documented. If a patient developed multiple HAPI during ICU admission, the date of the first HAPI was used. For patients without HAPI, clinical variables were collected for up to two weeks of ICU admission prior to the ICU prevalence survey date in which it was confirmed that no HAPI were present. If a patient without HAPI was in the ICU for

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more than one prevalence survey, the date of the last survey was used. These dates for patients with and without HAPI are from here forward referred to as the date of the "event." For clinical variables preceding the event, in order to capture patient acuity/occurrences of instability, either the value that indicated the greatest severity of illness (e.g. lowest arterial oxygen saturation) or the occurrence of values above or below a score/threshold of clinical significance (e.g. GCS $\leq$ 8) was used.

Data analysis

Descriptive statistics were used to summarize participant characteristics and HAPI rates. Continuous variables for patients with and without HAPI were compared using t-tests for comparing means and Wilcoxon Rank Sum test for comparing medians. Differences in categorical variables were assessed using chi-square tests. Individual logistic regression models were used to determine if the association between risk factors and development of HAPI persisted after adjusting for potential confounders: age, gender, race/ethnicity, and hospital LOS to event (HAPI or prevalence day). The final regression model contained all predictors. All analyses were performed in R version 3.3 (R Project for Statistical Computing, Free Software Foundation, Inc. Boston, MA) with two-sided p-value $\leq$ 0.01 denoting statistical significance.

Results

Participant characteristics

The final cohort included 1587 ICU patients. Patients in the cohort had a mean age of 59.6 years, and 56% were male. Approximately half (51.5%) of the patients were Black/African American, 41.8% were White and 6.6% were Other. Nearly half (47%) of the participants had a total Braden Scale score documented on admission that indicated an increased risk of developing pressure injury (score of 18 or less). Eighty-one patients (5.1%) developed 114 HAPI during ICU admission; characteristics of the pressure injuries, including stage and location, are presented in Table 1. Statistically significant differences in demographics and comorbidities between patients with and without HAPI were present (Table 2); patients with HAPI had longer hospital LOS and more often had a sepsis diagnosis during hospitalisation.

Table 1
Characteristics of intensive care acquired pressure injuries (*N* = 114).

Characteristic	*N* (% of HAPI)
HAPI Stage*	
Stage I	2 (1.8)
Stage II	12 (10.5)
Stage III	5 (4.4)
Stage IV	1 (0.9)
Suspected deep tissue injury	35 (31.8)
Unstageable	38 (33.3)
Mucous membrane	21 (18.4)
Anatomical location	
Sacrococcygeal	21 (18.4)
Coccyx	14 (12.3)
Medical device	45 (39.5)
Heel	12 (10.5)
Ear	9 (7.9)
All other locations	12 (10.5)
Incontinence associated dermatitis also present	10 (8.8)

* Of the 81 patients with pressure injuries, 21 developed multiple (two or more) pressure injuries while in the intensive care unit.

Table 2
Demographics and comorbid conditions of study cohort by patient group (*N* = 1587).

Characteristic	HAPI (*n* = 81)	No HAPI (*n* = 1506)	*P*
Age, mean (SD), years	59.4 (14.9)	59.7 (15.7)	0.86
Race, n (%)			
African American	33 (40.7)	776 (51.5)	0.13
White	43 (53.1)	630 (41.8)	
Other	5 (6.2)	100 (6.6)	
Male Gender n (%)	48 (59.3)	841 (55.8)	0.63
Total Braden at ICU Admit	16.7 (4.65)	17.4 (4.27)	0.17
Hospital LOS, median (IQR), days	33 (21, 56)	15 (8, 27)	<0.001
Comorbid Conditions			
Sepsis	40 (49.4)	329 (21.85)	<0.001
Myocardial Infarction	2 (2.5)	29 (1.9)	1.00
Congestive Heart Failure	27 (33.3)	418 (27.8)	0.34
Cerebral Vascular Disease	3 (3.7)	139 (9.2)	0.13
Chronic Pulmonary Disease	14 (17.3)	371 (24.6)	0.17
Diabetes with complication	3 (3.7)	48 (3.2)	1.00
Diabetes without complications	15 (18.5)	239 (15.9)	0.63

Abbreviations: HAPI, hospital acquired pressure injury; SD, standard deviation; ICU, intensive care unit; LOS, length of stay; IQR, interquartile range.

Differences in groups two weeks prior to the event

Several statistically significant differences were found between patients with and without HAPI in the two weeks prior to the event (Table 3). Patients with HAPI had a significantly longer hospital LOS to the time of the event. Among perfusion variables, patients with HAPI more often had a MAP < 60 mmHg, received vasopressors and received a larger volume (litres) of isotonic fluid boluses. In terms of oxygenation, more patients with HAPI were administered a FiO2 > 50% and had a PF ratio < 100. Comparison of the neurological variable found that the lowest GCS score was significantly lower in patients with HAPI. In addition, total Braden score and platelet count in the two weeks prior to the event was lowest for patients with HAPI. No statistically significant differences were present between groups by remaining demographic and clinical variables (Table 3).

Logistic regression models predicting HAPI

We independently determined the association between occurrences of HAPI and the following clinical variables: MAP < 60 mmHg, GCS $\leq$ 8, lowest total Braden score, fluid bolus volume and administration of FiO2 > 50%, while adjusting for age, gender, race, and hospital duration until event. Individual predictors of HAPI were: MAP < 60 mmHg (OR 9.88, CI 3.07–60.43), administration of vasopressors (OR: 2.92, CI 1.82–4.75), FiO2 > 50% (OR 3.06, CI 1.58–6.67), lowest Braden score (OR 0.71, CI 0.65–0.77), and GCS $\leq$ 8 (OR 5.16, CI 3.05–9.23) (Table 4, individual models).

The association between all risk factors and development of HAPI is also depicted in Table 4 (complete model). As can be seen, MAP < 60 mmHg and lowest Total Braden score were associated with increased risk of developing HAPI (MAP < 60 OR: 8.22, CI 1.74–147.07, Braden OR 0.79, CI 0.70–0.88). All other risk factors were not associated with development of HAPI after full adjustment.

Discussion

This study examined extrinsic and intrinsic EHR variables thought to be predictors of HAPI in critically ill patients. The greatest predictor for HAPI was a MAP < 60 mmHg; patients with a MAP < 60 mmHg within two weeks prior to the event (HAPI devel-

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Table 3
Comparison of clinical characteristics within two weeks of event date*

Characteristic	Measurement	HAPI		No HAPI		*P*
		n	Mean (SD) or Median (IQR)	*n*	Mean (SD) or Median (IQR)	
Fluid bolus volume	Litres, total	81	2 (2–14)	1506	1 (0–14)	0.001
SaO2	%, lowest value	81	84 (73–89)	1506	88 (80–91)	<0.001
Total bilirubin	Mg/dl, highest value	71	1.5 (0.7–4.0)	1134	0.7 (0.4–1.6)	<0.001
Platelet count	10^3 µl, lowest value	81	94 (48–153)	1503	150 (95–205.5)	<0.001
Creatinine	Mg/dl, highest value	81	2.4 (1.4–3.9)	1503	1.4 (1.0–2.7)	<0.001
Braden Scale	Total Score, lowest value	81	10 (8–11)	1506	13 (11–16)	<0.001
Hospital LOS to event*	Days, total	81	11.0 (6, 23)	1506	5.0 (2, 12)	<0.001
Characteristic	**Variables**	***n***	**%**	***n***	**%**	
Glascow Coma Scale	≤ 8	64	79	622	41.3	<0.001
	> 8	17	21	883	58.7	
Mean Arterial Pressure	< 60 mmHg	79	97.5	1167	77.5	<0.001
	≥ 60 mmHg	2	2.5	339	22.5	
Vasopressors	Yes	52	64.2	537	33.8	<0.001
	No	29	35.8	969	64.3	
FiO2 > 50%	> 50%	68	88.3	731	70.9	0.002
	≤ 50%	9	11.7	300	29	
PF ratio	< 100	41	56.2	286	33.5	<0.001
	> 100	32	43.8	567	66.5	

* For patients with HAPI confirmed at a prevalence survey day, event was the date the HAPI was first documented in the EHR. For patients without HAPI, the event was the date of the patient's last prevalence survey day in ICU when it was confirmed no pressure injuries were present.
Abbreviations: HAPI, hospital acquired pressure injury; IQR, interquartile range; SD, standard deviation; SaO2, arterial oxygen saturation; LOS, length of stay; FiO2, fraction of inspired oxygen; PF ratio, partial pressure arterial oxygen and fraction of inspired oxygen

Table 4
Individual and complete logistic regression models examining risk factors/predictors of pressure injury in intensive care unit patients after adjusting for control variables.

Risk Factor/Predictor	Individual Models		Complete Model	
	Odds Ratio	95% CI	Odds Ratio	95% CI
MAP < 60	9.88	3.07–60.43	8.22	1.74–147.07
FIO2 > 50%	3.06	1.58–6.67	1.68	0.82–3.80
Vasopressor administered	2.92	1.82–4.75	1.37	0.80–2.38
Total Braden*	0.71	0.65–0.77	0.79	0.70–0.88
Glasgow Coma Scale ≤ 8	5.16	3.05–9.23	1.29	0.63–2.77
Fluid bolus volume	1.00	1.00–1.00	1.00	1.00–1.00

Note: Control variables included in all models are age, gender, ethnicity/race, and hospital length of stay to event. For patients with HAPI, event was the date the HAPI was first identified. For patients without HAPI, the event was the date of the patient's last prevalence day in ICU.
*For the total Braden score the lowest value in the two weeks prior to the event was used.
Abbreviations: MAP, mean arterial pressure; FiO2, fraction of inspired oxygen.

opment or prevalence survey day) were eight times as likely to develop HAPI. Our results are consistent with past investigations, despite differing operational definitions of low blood pressure (e.g. lowest diastolic blood pressure, systolic blood pressure < 90 mmHg, prolonged MAP < 70 mmHg) (Bly et al., 2016; Cox and Roche, 2015; El-Marsi et al., 2018; Kirkland-Kyhn et al., 2017; Wilczweski, et al., 2012).

For over 30 years, conceptual models for pressure injury development have included physiologic alterations in perfusion/oxygen delivery (Benoit and Mion, 2012; Braden and Bergstrom, 1987; Coleman et al., 2014; Tschannen and Anderson, 2020). More recently, investigators have proposed that poor perfusion is a direct causal factor for HAPI, as it reduces a patient's tolerance to pressure (Coleman et al., 2014; Tschannen and Anderson, 2020). It is not uncommon for patients in the ICU to experience hypotension from their critical illness and/or the effects of treatments provided (Cecconi et al., 2015; Kane-Gill et al., 2014; Yapps et al., 2017) and may in part explain the higher rates of HAPI seen in this population. Additional research is needed to expound the role of altered perfusion in HAPI development; a consistent approach to measuring the degree and duration of hypotension would improve the generalisability and comparability of findings across studies.

Another significant predictor for this study was the lowest value for the total Braden Score in the two weeks prior to the event. Similarly to past investigations (Slowikowski and Funk, 2010; Tschannen et al., 2012) patients in our study with higher scores had decreased risk of HAPI. However, only the score during the two weeks before the HAPI was a predictor; there was no difference in total Braden score between groups on ICU admission. In past research, investigators collected Braden Scale scores at differing times, for example, upon ICU admission or on the day of HAPI discovery (Hyun et al., 2013; Kim et al., 2013; Kirkland-Kyhn et al., 2017; Tschannen et al., 2012). Future research should examine Braden scores in relationship to the timing of HAPI development, as a patients' condition in the ICU is often dynamic, and consequently pressure injury risk at a single point in time may not adequately reflect the actual risk for the patient in days leading up to the HAPI.

While low blood pressure was a predictor for HAPI in our study, treatments provided for low blood pressure (administration of vasopressors and isotonic fluid boluses) were not associated with HAPI development when included in a regression model with other variables. Vasopressor administration has been previously identified as a predictor of HAPI (Bly et al., 2016; Cox and Roche, 2015; Cox, 2011; Cox et al., 2020; Nijs et al., 2009; Theaker et al., 2000; Tschannen et al., 2012). This inconsistency might be related to differences in measures; some investigators have examined whether vasopressors were administered and the number of vasopressors given (Bly et al., 2016; Compton et al., 2008; Tschannen et al., 2012), while others examined the dose or length of use of specific vasopressor agents (Cox, 2011; Cox and Roche, 2015; Nijs et al., 2009). Differences in practice across study sites may also come into play, as various vasopressor agents (norepinephrine, vasopressin,

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dopamine) have been identified as independent predictors of HAPI (Cox, 2011; Cox & Roche, 2015; Cox et al., 2020; Nijs et al., 2009; Theaker et al., 2000).

Limitations

Our study had limitations. First, our use of EHR data resulted in the omission of potential confounding factors, including comorbidities at the time of the event (only diagnosis codes upon discharge were available), care provided to patients prior to admission to our medical centre, and the length of time patients were removed from the active support surface of their ICU bed for surgery, procedures or imaging. Second, because our data represented a single centre, findings might not be generalisable to centres with differing populations or resources. Lastly, while an evidence-based standard of care for all patients in the ICU is in place at our institution, we did not evaluate individual practices. Recent literature demonstrates the effectiveness of various HAPI prevention strategies in the critically ill (Kuniavsky et al., 2020; Lin et al., 2020; Powers et al., 2020; Schroeder and Sitzer, 2019; Tayyib and Coyer, 2016). Further examination of the relationship between patients' clinical risk factors and the prevention interventions provided is needed.

A major strength of our study was that all HAPI were validated in-person by two Certified Wound Care Nurses. Clinical nurses, even when educated regarding pressure injury assessment, frequently misclassify pressure injuries and have difficulty differentiating pressure lesions from moisture lesions (Ayello et al., 2005; Beeckman et al., 2010; Barnard and Copson, 2016); thus, the sole use of bedside nurses' EHR documentation to identify HAPI cases may result in research bias due to outcome misclassification (Pannucci and Wilkins, 2010). Another strength of this study is our lower prevalence of ICU HAPI (5.1%), suggesting that our institution's comprehensive pressure injury prevention practices on admission to ICU are consistently implemented and effective. Lastly, we examined all potential clinical predictors in the two weeks preceding HAPI development and LOS *prior* to HAPI development, avoiding the introduction of data that is potentially irrelevant to HAPI development.

Conclusion

It is vital that healthcare professionals understand all the factors that increase the risk of developing HAPI, including those that might not be modifiable by nursing practice in the context of critical illness. In this study, a key finding was that MAP < 60 mmHg within two weeks was the strongest predictor of HAPI. Understanding the causal relationship between low blood pressure and HAPI may inform future prevention efforts for critically ill patients. Our results provide a more comprehensive understanding about which critically ill patients are at risk and may contribute to the future development of a more accurate electronic risk assessment for this patient population. Differences in predictors of HAPI across studies are likely related to variances in patient subpopulations, the resources and delivery of prevention practices across institutions, and the measurement methods used (Chaboyer et al., 2018). Prospective multisite studies are needed to further examine these potential contributors to HAPI development within the context of adherence to prevention interventions.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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