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Pressure Injury Risk Assessment and Prevention in Patients With COVID-19 in the Intensive Care Unit

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ABSTRACT

Background: Patients critically ill with COVID-19 are at risk for hospital-acquired pressure injury, including device-related pressure injury.
Methods: Braden Scale predictive validity was compared between patients with and without COVID-19, and a logistic regression model was developed to identify risk factors for device-related pressure injury.
Results: A total of 1920 patients were included in the study sample, including 407 with COVID-19. Among the latter group, at least 1 hospital-acquired pressure injury developed in each of 120 patients (29%); of those, device-related pressure injury developed in 55 patients (46%). The Braden Scale score area under the receiver operating characteristic curve was 0.72 in patients without COVID-19 and 0.71 in patients with COVID-19, indicating fair to poor discrimination.
Conclusions: Fragile skin and prone positioning during mechanical ventilatory support were risk factors for device-related pressure injury. Clinicians may consider incorporating factors not included in the Braden Scale (eg, oxygenation and perfusion) in routine risk assessment and should maintain vigilance in their efforts to protect patients with COVID-19 from device-related pressure injury.
Key words: COVID-19, pressure injury, risk assessment, prevention measures

Pressure injuries (PrIs) are defined as localized areas of damage to the skin and/or underlying tissue occurring due to pressure or pressure combined with shear. Pressure injuries are staged according to the degree of visible tissue damage. Pressure injuries usually occur over bony prominences but may also be associated with use of devices, termed device-related pressure injuries (DRPrI).[1] Hospital-acquired PrIs (HAPrIs) result in a longer length of hospital stay and human suffering, with care costs exceeding $26 billion annually.[2,3] In the acute care setting, patients admitted to the intensive care unit (ICU) are at 3.8 times greater risk for HAPrI development than are other acute care patients in the same hospital.[4] The increased HAPrI prevalence in patients in the ICU is thought to be the result of a combination of high severity of illness, the

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presence of invasive treatments that make repositioning more difficult (eg, mechanical ventilation), medical devices, and, especially, impaired oxygenation and perfusion.[1,5,6] Impaired oxygenation and/or perfusion result in inadequate blood flow to the tissue, tissue damage from ischemia and ischemia-reperfusion injury, and, ultimately, reduced tissue tolerance for pressure.[7,8]

Hospital-acquired PrIs are considered mostly preventable. Prevention begins with risk assessment. Risk assessment allows clinicians to identify risk factors associated with HAPrI development and intervene accordingly. The National Pressure Injury Advisory Panel (NPIAP) recommends the routine use of a combination of structured risk assessment and identifying any additional risk factors not encompassed in the structured risk assessment tool. In the United States, the Braden Scale for Predicting Pressure Sore Risk[9] (hereafter referred to as the Braden Scale) is the most commonly used risk assessment tool. The Braden Scale is used to measure PrI risk factors on 6 subscales, but it lacks other known risk factors of patients in the ICU (eg, impaired oxygenation and perfusion).

Patients critically ill with COVID-19 are likely at high risk for HAPrI formation because of their increased illness severity, impaired oxygenation and perfusion, and use of medical devices.[10] However, to our knowledge, the predictive validity of the Braden Scale has not been studied in a sample containing patients diagnosed with COVID-19. Furthermore, although PrI risk due to COVID-19 treatments, such as prone positioning,[11,12] has been addressed in recent studies, little is known about risk factors for DRPrIs in critically ill patients positive for COVID-19.

Background

The purpose of this study was to examine PrI risk in a sample of patients in the ICU. The specific aims were to (1) compare the Braden Scale's predictive validity for patients positive for COVID-19 with its predictive validity for those negative for COVID-19 and (2) identify risk factors for DRPrI in patients with COVID-19.

Pressure injuries are staged according to the degree of visible tissue damage.[1,13] Stage 1 injuries are intact skin with localized, nonblanchable redness or areas where the skin texture or temperature differs from the surrounding skin. Stage 1 injuries may be difficult to detect in darker skin tones. Stage 2 injuries are partial-thickness loss of dermis presenting as a shallow open ulcer, whereas stages 3 and 4 both indicate full-thickness tissue loss. Unstageable injuries are full-thickness injuries in which the extent of tissue damage cannot be visualized because of eschar or slough. Deep tissue PrIs are intact or nonintact skin with a localized area of maroon or purple discoloration, indicating deep tissue damage. Finally, mucosal-membrane PrIs occur on the mucous membranes and, therefore, cannot be staged using the current PrI staging system.

Pressure injuries are further differentiated as device related or non-device related. Device-related PrIs are HAPrIs primarily caused by medical devices; in the ICU, the most common devices causing DRPrIs are oxygen tubing, endotracheal tubes, and nasogastric tubes.[14] Patients in whom a DRPrI develops are more than twice as likely as patients without a DRPrI to go on to have a non–device-related HAPrI develop.[15]

The Braden Scale was developed in 1987 to predict PrI risk across all care settings, including most ICU settings in the United States.[9] The Braden Scale includes 6 subscales: moisture, mobility, activity, sensory perception, nutrition, and friction and shear. Nurses record information for each subscale, and the subscale scores are summed to indicate HAPrI risk. Possible Braden Scale scores range from 6 to 24, with lower scores indicating higher risk for PrI formation. Scores from 15 to 18 indicate mild risk, 13 or 14 indicate moderate risk, 10 to 12 indicate high risk, and scores of 9 or less indicate severe or very high risk. Predictive validity—defined as the ability to predict in which patients an HAPrI will develop—is measured against 4 criteria: sensitivity, specificity, positive predictive value, and negative predictive value (Figure 1[16]). In studies conducted before the COVID-19 pandemic, the Braden Scale demonstrated relatively poor predictive validity in patients in the ICU, because most patients are considered at high risk for pressure injury (ie, high sensitivity, low specificity).[17-20]

In addition to data obtained from structured risk assessment, the NPIAP recommends caregivers consider additional factors in HAPrI risk assessment and associated care planning.[1] Cox and Schallom's conceptual schema for

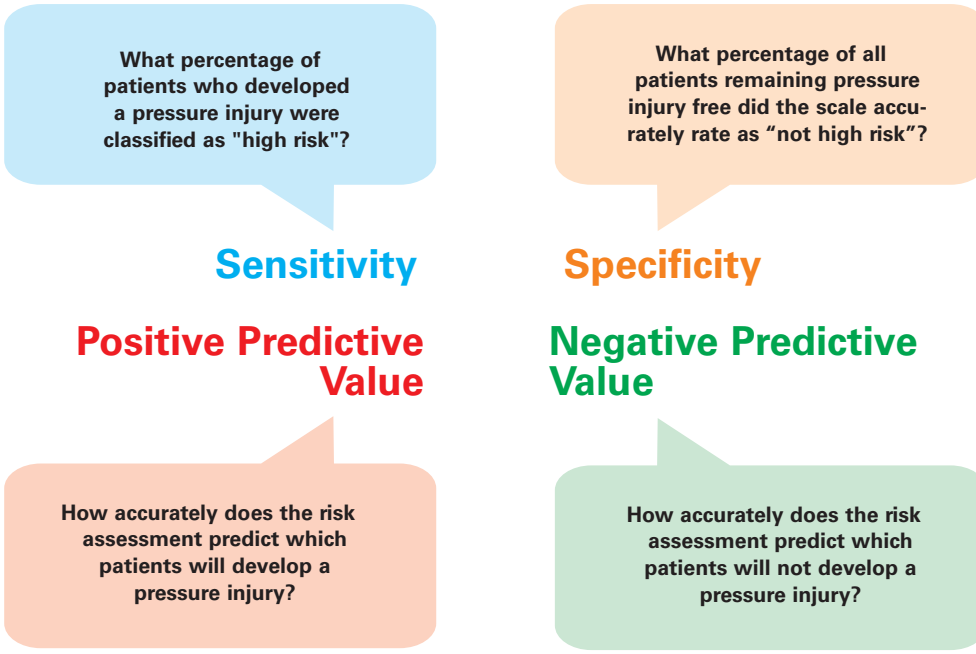

Figure 1: The 4 criteria of predictive validity.[16]

PrI development in critical care patients[7] (Figure 2) illustrates the complex relationships between risk factor exposure duration and HAPrI development. Hospital-acquired PrIs form when a dynamic combination of impaired tissue tolerance, impaired oxygenation and perfusion, and/or pressure, shear, and microclimate exceed the tissue's ability to withstand damage. Within the schema, risk factors may develop as a result of an intrinsic etiology (eg, hypotension) and/or an extrinsic etiology (eg, vasopressor infusion), all of which can influence the tissue's ability to tolerate pressure or shear. The conceptual model also includes evidence-based mitigation strategies (eg, support surfaces, repositioning, heel suspension, nutritional interventions, prophylactic dressing) shown to be effective in HAPrI prevention.[21-26]

Methods

Design

For this study, we used a retrospective cohort design using electronic health record (EHR) data from 2 ICUs within 1 institution. The study was approved by the University of Utah's institutional review board. The data were obtained via a query of the institution's enterprise data warehouse. A data architect conducted the query with a nurse informaticist, and the query results were verified for accuracy by 2 practicing ICU nurses. Data for variables not obtainable via query were obtained via manual review of the EHR.

Setting and Sample

Patients aged at least 18 years who were admitted to 1 of 2 medical ICUs at a teaching hospital in Utah were eligible for inclusion in the study. Patients were admitted to the ICU between April 2020 and April 2021. Patients who had a PrI present on admission were included in the study because a preexisting PrI is a risk factor for subsequent, additional HAPrIs.[27] A PrI was considered hospital acquired in patients with a PrI if it occurred 48 hours after admission in a different anatomic location than the community-acquired PrI.

A PrI prevention protocol was in effect at the time of this study and included the following prevention measures: repositioning every 2 hours, frequent skin inspection, keeping skin clean and dry, prophylactic sacral dressings, and padding and inspecting skin under devices. All patients in the study sample were cared for on low-air-loss critical care beds. A proning protocol was in place at the time of the study, including PrI prevention strategies to be implemented while the patient is in the prone position.

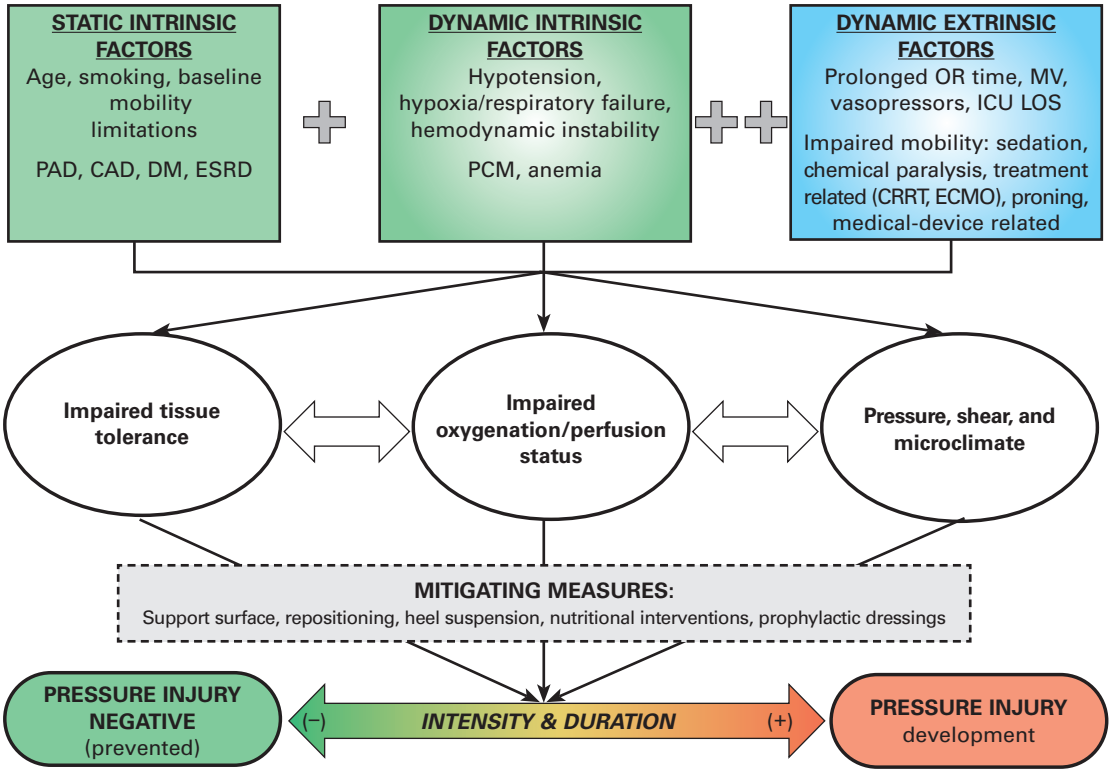

Figure 2: Conceptual schema for development of pressure injuries in critically ill patients. Reproduced with permission from Dr Jill Cox and Dr Marilyn Schallom. + indicates increased risk; ++, cumulative increased risk; CAD, coronary artery disease; CRRT, continuous renal replacement therapy; DM, diabetes mellitus; ECMO, extracorporeal membrane oxygenation; ESRD, end-stage renal disease; ICU, intensive care unit; LOS, length of stay; MV, mechanical ventilation; OR, operating room; PAD, peripheral artery disease; PCM, protein K-calorie malnutrition.

Measures

The HAPrI outcome variable was defined according to the NPIAP staging definitions (stages 2-4: unstageable, deep tissue PrI, or mucous membrane PrI).[1] Stage 1 HAPrIs were not included because stage 1 injuries are reversible and considered less severe.[28,29] The study-hospital protocol dictates a certified wound care nurse must evaluate an HAPrI to stage the injury and determine whether the PrI is device related. However, because of the COVID-19 surge and staffing limitations, it was not always possible for a wound care nurse to evaluate the PrI. When a wound care nurse was not available, the bedside critical care registered nurse staged the PrI and indicated whether the PrI was device related.

The lowest (ie, most severe) Braden Scale score determined the Braden Scale's predictive validity. The lowest Braden Scale score was defined as the lowest score throughout the ICU stay (or the lowest score at any point before HAPrI development in patients with HAPrI).

Potential risk factors for DRPrI were identified from the relevant literature.[30,31] Data for potential risk factors were limited to items available in the EHR. Variables included demographic factors (eg, age, length of stay, race/ethnicity, death in the ICU); Braden Scale scores; treatments and devices (eg, urinary catheters, nasogastric tubes, mechanical ventilatory support, proning); laboratory values (eg, blood gas values; levels of creatinine, lactate, hemoglobin, albumin); nursing skin assessments (as documented in the EHR; eg, fragile skin, pitting edema, moist skin); nutritional factors (eg, weight loss >4.5 kg [10 pounds]; any 3 days during the admission with recorded no dietary intake); a clinical deterioration score (namely, the Modified Early Warning Score); and diagnosis and

comorbidities (eg, acute respiratory failure, Charlson Comorbidity Index, diabetes mellitus, chronic obstructive pulmonary disease, heart failure, spinal cord injury).[6,32-34] Information on these risk factors was collected for the entire ICU stay for patients without DRPrI and during the period before DRPrI formation for patients who subsequently had a DRPrI.

Statistical Analysis

All analyses were performed in R, version 3.4.4 (R Foundation for Statistical Computing).[35] Sample characteristics were described in terms of mean and standard deviation for continuous or ordinal variables, or number and percentage for nominal-level variables.

Braden Scale predictive validity[36] was calculated and compared between patients with COVID-19 and those without COVID-19. A score of 12 was chosen as the cutoff score for assessing the Braden Scale score's predictive validity, because scores of 12 or lower are considered to indicate high risk for pressure injury.[9] The Braden Scale score's area under the receiver operating characteristic curve was also compared between patients with and without COVID-19.

Bivariate relationships between patient characteristics and DRPrI status in patients with COVID-19 were compared using a *t* test or the nonparametric equivalent for continuous or ordinal level variables or a χ^2 test for nominal-level variables. Least absolute shrinkage and selection operator (LASSO) logistic regression[37] was used to identify the most important risk factors for DRPrI in patients with COVID-19. Predictors from the LASSO regression were selected if their coefficients were nonzero, and then regression coefficients from unpenalized logistic regression were reported using the LASSO-selected coefficients.

Results

Sample Characteristics

A total of 1920 patients were included in the study sample. The mean age (SD) of the sample was 56 (17) years. The patients were primarily White (n = 1359; 71%), non-Hispanic (n = 1532; 80%), and male (n = 1100, 57%). Of the 1920 patients in the study sample, 407 (21%) were diagnosed with COVID-19.

Pressure Injury Outcome

At least 1 HAPrI developed in each of 354 patients (18%); of those patients, at least 1 PrI that was considered a DRPrI developed in 117 patients (33%). In 42 patients, both DRPrI and non–device-related HAPrIs developed.

Among the 407 patients with COVID-19, at least 1 HAPrI developed in each of 120 patients (29%); of those, a total of 75 DRPrIs developed in each of 55 patients (46%). The anatomic locations of the DRPrIs in patients with COVID-19 are listed in Table 1. Stage 2 (n = 35; 47%) was the most common category of PrI, followed by mucosal-membrane PrI (n = 34; 45%), deep tissue PrI (n = 4; 5%), and stage 3 PrI (n = 2; 3%).

Braden Scale Predictive Validity in Patients With Versus Those Without COVID-19

At a cutoff score of 12 (high risk), the Braden Scale had high sensitivity and low specificity for predicting DRPrI in patients with and without COVID-19 (Table 2). The Braden Scale score's area under the receiver operating characteristic curve was 0.72 for patients without COVID-19 and 0.71 for patients with COVID-19, indicating fair to poor discrimination in both groups.

Risk Factors for Device-Related Pressure Injury in Patients With COVID-19

The relationships between potential risk factors and DRPrI formation in patients with COVID-19 are presented in Table 3. Two variables, fragile skin (defined in our study site's EHR as thin epidermis with subcutaneous tissue loss) and prone positioning during mechanical ventilation, emerged from the LASSO multivariable logistic regression. The multivariate model is presented in Table 4.

Discussion

Data regarding HAPrI risk in patients in the ICU with COVID-19 are limited. Still, emerging data and results from this study show that patients with COVID-19 are at even greater risk for PrIs than is the general ICU population.[38] The finding that patients with COVID-19 experience extremely high risk for HAPrI is logical given that the pathophysiology of severe COVID-19 includes impaired oxygenation and perfusion status—a known risk factor for HAPrI—along with treatment-related factors such as prone positioning, possibly increasing the risk for DRPrI.[38-40]

Table 1: Device-Related Pressure Injury Anatomic Locations in Patients With COVID-19 (N = 75 Pressure Injuries in 55 Patients)

Anatomic Location	No. (%)	Device (No.)
Cheek	7 (9%)	Oxygen tubing (2) ETT securement device (2) Device information not documented (3)
Eye area	4 (5%)	Telemetry lead (used for train-of-4 sensor) (2) Device information not documented (2)
Forehead	4 (5%)	Bispectral index monitor (2) Device information not documented (2)
Extremity (wrist, hand/finger, arm, toe)	16 (21%)	Arterial line board (1) Intravenous or arterial line tubing (6) Pulse oximeter probe (4) Bed footboard (1) Device information not documented (4)
Ear	9 (12%)	Oxygen tubing (6) Pulse oximeter (2) Device information not documented (1)
Nare	15 (20%)	Nasogastric tube (7) Pulse oximeter (1) Nasal cannula (5) Device information not documented (2)
Mouth or lip	11 (15%)	ETT (10) Orogastric tube (1)
Back	2 (3%)	Telemetery lead (1) Device information not documented (1)
Chest or neck	3 (4%)	Telemetry lead (2) Device information not documented (1)
Rectal/genital	4 (5%)	Fecal management system (1) Urinary catheter (1) Device information not documented (2)

Abbreviation: ETT, endotracheal tube.

To our knowledge, this is the first study in which the predictive validity of the Braden Scale was examined in patients in the ICU with COVID-19. The results add to a growing body of literature showing the Braden Scale lacks predictive validity in the ICU population, because of low specificity.[18,41] Findings from this study show nurses should also consider risk factors not included in the Braden Scale, including the risk for DRPrI posed by treatments and devices, particularly in patients with fragile skin or those undergoing invasive mechanical ventilatory support during prone positioning.

Non–Device-Related Pressure Injury Risk Assessment and Prevention

The first step in successful PrI prevention is accurately determining PrI risk. Although the Braden Scale is the most widely used tool in the United States to determine PrI risk, its predictive validity in the ICU population has been suboptimal, as demonstrated by this study and previous research.[42,43]

PrI risk determination in patients in the ICU is a complex process because it is multifactorial and grounded in many sources. To comprehensively determine PrI risk, assessment must include the pathophysiologic impacts associated with critical illness, the concomitant preexisting conditions present at the time of a critical illness, as well as the treatment-related effects during a critical illness.[7] Severely ill patients with COVID-19 represent a unique subset of critically ill patients who have profound physiologic impacts associated with illness and require complex treatment strategies to improve survival.

Table 2: Braden Scale Predictive Validity[a]

Measure	Sensitivity, %	Specificity, %
COVID-19 negative (n=1513)	88	20
COVID-19 positive (n=407)	82	33

[a] Cutoff score was 12.

Cox and Schallom[7] proposed a conceptual schema to explain PrI development in the critically ill population (Figure 2).The schema describes an interplay of empirically derived risk factors driving PrI development in this population. Factors such as preexisting comorbidities (static intrinsic factors); conditions associated with critical illness, such as hypotension or respiratory failure (dynamic intrinsic factors); as well as treatment-related factors, such as mechanical ventilation (dynamic extrinsic factor), all synergistically increase PrI risk in this population. The challenge is that the majority of these factors are not included in formalized PrI risk assessment; therefore, the ability of caregivers to objectively quantify the impact of these factors is presently not feasible.

Evidence-based preventive interventions should be assigned on the basis of an understanding of a patient's risk level and risk factors. Interventions are generally aimed at reducing exposure to pressure (ie, repositioning, support surfaces), managing microclimate (ie, temperature, airflow, humidity next to the skin surface),[1] maintaining skin integrity (ie, skin inspection and care), and nutritional screening and support.

Exposure to prolonged pressure is a key factor in PrI formation. Therefore, PrI prevention mainstays include routine repositioning to reduce and redistribute pressure and use of pressure-reducing surfaces. The NPIAP does not recommend a specific support surface for patients in the ICU, instead indicating that an appropriate surface should be selected on the basis of the patient's size, tissue tolerance, and risk for PrI development, with attention to pressure reduction and microclimate.[1] Note that no support surface negates the need for routine positioning and that the lateral rotation support-surface function is not considered a PrI prevention intervention.[1]

Similarly, the NPIAP does not designate a repositioning interval and calls for frequency based on the individual patient's tissue tolerance, general health and severity of illness, level of activity and mobility, skin condition, and comfort.[1] Whenever possible, avoid positioning patients on devices and avoid prolonged pressure to bony prominences such as the ischium or heels (eg, periodically "floating" the heels is a way to reduce pressure).[44] It is also essential to consider the effects of moving and handling equipment on risk for PrI formation. For example, the material used in overhead lifts may interfere with support-surface pressure redistribution properties.[1]

Malnutrition is associated with numerous poor outcomes,[22] including PrI formation. Characteristics of protein-calorie malnutrition in patients in the ICU include insufficient energy intake, loss of muscle mass or subcutaneous fat, fluid accumulation, or intended weight loss.[45] The NPIAP recommends routine nutrition screening using a validated tool for all patients in the ICU considered at risk for PrI, followed by expert nutrition consultation for individuals found to be at nutritional risk. Note that serum albumin level and the Braden Scale nutrition subscore are not considered reliable indicators of nutritional status for patients in the ICU.[1,46]

Routine skin inspection and associated preventive skin care should be applied to all patients in the ICU. A head-to-toe skin assessment must be conducted regularly to identify potential problem areas requiring pressure offloading or skin care. Skin assessments should extend beyond visual inspection because changes in texture and temperature may be the first signs of a PrI in patients with darker skin tones.[47] The foundations of preventive skin care include keeping the skin clean and appropriately hydrated, cleansing after exposure to urine or feces, and protecting the skin from moisture.[1] Prophylactic sacral and heel dressings are effective for preventing HAPrIs[48-50]; nurses should peel back the dressing to assess the skin if the product instructions support this use.[1]

Using evidence-based measures to mitigate PrI development is foundational to any

Table 3: Factors Associated With Device-Related Pressure Injury in Patients With COVID-19

Variable	All Patients With COVID-19 (N=407)	DRPrI (n=55)	No DRPrI (n=352)	*P* Value
Demographic Characteristics and Length of Stay				
Age, mean (SD), y	59 (15)	59 (14)	59 (15)	.82
Male sex, n (%)	256 (63)	38 (69)	218 (62)	.38
Hospital length of stay, mean (SD), d	16 (16)	29 (33)	13 (10)	<.001
Died during hospitalization, n (%)	99 (24)	16 (29)	83 (24)	.47
Race, n (%)				.09
Native American or Alaska native	22 (5)	1 (2)	21 (6)	
Asian	11 (3)	4 (4)	7 (2)	
Black	10 (2)	1 (2)	9 (3)	
Native Hawaiian or other Pacific Islander	19 (5)	0 (0)	19 (5)	
White	229 (56)	33 (60)	196 (56)	
Other, unknown, or choose not to disclose	116 (29)	16 (29)	100 (28)	
Hispanic ethnicity	98 (24)	13 (24)	85 (24)	1.0
Braden Scale Score				
Lowest Braden Scale total score, mean (SD)	11.3 (3.8)	11.1 (3.8)	11.4 (3.7)	.07
Treatment				
Invasive mechanical ventilation days, mean (SD)	5 (10)	16 (19)	3 (6)	<.001
Dialysis, n (%)	89 (22)	20 (36)	69 (20)	.008
Vasopressor infusion, n (%)	49 (12)	14 (25)	35 (10)	.002
Prone positioning (awake or sedated), n (%)	333 (83)	52 (95)	281 (80)	<.001
Prone positioning during invasive mechanical ventilation, n (%)	155 (38)	40 (72)	115 (33)	<.001
Nasogastric tube, n (%)	347 (85)	51 (93)	296 (84)	.07
Urinary catheter, n (%)	402 (99)	55 (100)	347 (99)	1.0
Laboratory Test				
Maximum lactate, mean (SD), mg/dL	3.81 (3.87)	5.42 (4.62)	3.56 (3.68)	.006
Maximum serum creatinine, mean (SD), mg/dL	2.16 (2.22)	2.88 (3.04)	2.05 (2.05)	.053
Minimum hemoglobin, mean (SD), g/dL	10.46 (3.00)	7.95 (2.54)	10.84 (2.88)	.001
Minimum albumin, mean (SD), mg/dL	2.68 (0.52)	2.36 (0.44)	2.73 (0.52)	<.001
Min Pao_2, mm Hg	64 (62)	57 (56)	67 (77)	.10
Maximum $Paco_2$, mm Hg	53 (21)	68 (28)	51 (18)	<.001
Nursing Skin Assessment				
Fragile skin (thin epidermis with subcutaneous tissue loss), n (%)	198 (49)	41 (75)	157 (44)	<.001
Excessively moist skin, n (%)	81 (20)	18 (33)	63 (18)	<.001
Pitting edema, n (%)	84 (21)	16 (29)	68 (19)	<.001
Nutritional Factor				
Unplanned weight loss >4.5 kg (10 lb) prior to admission, n (%)	43 (11)	11 (20)	32 (9)	.02
No intake >3 d, n (%)	28 (7)	4 (7)	24 (7)	1.0

Continued

Table 3: *Continued*

Variable	All Patients With COVID-19 (N=407)	DRPrI (n=55)	No DRPrI (n=352)	*P* Value
Clinical Deterioration Score				
Maximum MEWS, mean (SD)	5.39 (2.01)	6.67 (1.60)	5.28 (2.0)	<.001
Diagnosis and Comorbidities				
Acute respiratory failure, n (%)	386 (95)	55 (100)	334 (98)	.98
Charleson Comorbidity Index, mean (SD)	3.38 (3.32)	3.43 (3.13)	3.37 (3.35)	.89
Diabetes, n (%)	222 (55)	28 (51)	194 (55)	.66
Spinal cord injury, n (%)	27 (7)	6 (11)	21 (6)	.28
Heart failure, n (%)	95 (23)	12 (22)	83 (23)	.91
COPD, n (%)	128 (31)	18 (33)	110 (31)	.83

Abbreviations: COPD, chronic obstructive pulmonary disease; DRPrI, device-related pressure injury; MEWS, modified early warning score.

Table 4: Logistic Regression Model for Device-Related Pressure Injury After LASSO in Patients With COVID-19

Predictor Variable	Odds Ratio (95% CI)	*P* Value
Fragile skin (thin epidermis with subcutaneous tissue loss)	2.79 (1.04-4.34)	<.001
Prone positioning during mechanical ventilation	4.28 (1.02-8.99)	<.001

Abbreviation: LASSO, least absolute shrinkage and selection operator.

successful PrI prevention program. However, in the critical care population, the awareness of certain clinical scenarios beyond the compensatory abilities of skin and underlying tissues—or the preventive capacity of caregivers—strengthens our understanding of why PrIs continue to occur despite best practice.[7]

In the context of DRPrIs, determining the devices posing the greatest risk for PrI development is paramount to successful DRPrI prevention. However, most medical devices used in treating critical illnesses (eg, endotracheal tubes, nasogastric tubes, extracorporeal membrane oxygenation) are nonnegotiable; thus, in certain circumstances, their use may result in an unavoidable PrI.

Device-Related Pressure Injury Assessment and Prevention

According to results of studies conducted before the COVID-19 pandemic, approximately 30% of ICU-acquired HAPrIs are DRPrIs, defined as an HAPrI caused by pressure from a device (or a combination of pressure from a device and pressure from body weight) against the skin.[1,15,51] Device-relate PrIs were even more common in the patients with COVID-19 in the present study, occurring in 46% of the patients with HAPrIs. The high proportion underscores the importance of DRPrI prevention in patients positive for COVID-19 in the ICU.

Many medical devices and even nondevice items such as furniture (eg, bedside tables, footboards) have been implicated as sources of device-related PrIs.[52] Any device exerting pressure against mucosal tissue (ie, the lining of the oral mucosa, nares, urinary and gastrointestinal tracts, and the vagina) should be considered especially high risk for mucosal-membrane DRPrIs because mucosal tissues have reduced tissue tolerance for pressure.[1] Examples of ICU devices contributing to pressure-related mucosal tissue damage include endotracheal tubes, bite blocks, nasogastric and orogastric tubes, urinary catheters, and fecal management systems. The considerable risk from devices pressing on mucous membranes was evident in this study: 45% of the DRPrIs were categorized as mucous-membrane PrIs and the device most commonly implicated was the endotracheal tube.

Device-related PrIs in this study most commonly occurred on the face (55%; inclusive of the cheek, nares, mouth, lip, and forehead) followed by extremities (21%; inclusive of the wrist, arm, hand or finger, and toe) and the ear (20%). This finding is consistent with findings from a recent multisite study in which researchers determined DRPrIs were most common in the facial area.[52] In contrast, in a recent study conducted across 66 adult ICUs in China, researchers identified 98 distinct anatomic locations where DRPrIs developed; in that study, the finger was the most common location of DRPrIs (33%) followed by the nose (18%). Findings across studies underscore the importance of head-to-toe skin inspection, because DRPrIs can and do occur in unexpected anatomic locations (eg, a toe pressing against a footboard).

Device-related PrIs develop when the tissue's innate ability to withstand mechanical forces is overcome. Cells in the soft tissue may become deformed or distorted under pressure from devices, resulting in cell damage or death. Deformation-inflicted cell damage results in secondary injury from edema and inflammation. The secondary injury leads to additional edema and inflammation—a vicious cycle between device pressure–induced cell deformation and tissue damage.[53] As in non–device-related HAPrI, the tissue's tolerance for pressure varies between individuals and is influenced by intrinsic and extrinsic risk factors.[7]

Intrinsic factors include impaired sensory perception, edema, moisture, and the presence of fragile skin may contribute to DRPrI risk. Patients who cannot communicate easily (eg, intubated patients) and patients with sensory impairment cannot alert the nurse if a device is pressing against their skin or causing discomfort.[54] Edema is also a DRPrI risk factor.[55] The presence of edema in combination with moisture under the device is a dangerous combination because edema increases the pressure against the device while the moisture reduces the tissue's tolerance for pressure.

In this study, the finding that fragile skin, a static intrinsic factor common in older people, was an independent risk factor for DRPrI, is additional evidence for the importance of including aging-related skin changes in DRPrI risk assessment.[26]

Other intrinsic risk factors for DRPrI previously identified in the literature are similar to the risk factors for non–device-related HAPrI, including decreased perfusion, altered skin status, and poor nutrition[15,56] The exception is risk directly imposed by specific treatments; the presence of an endotracheal tube, for example, is a risk factor for oral DRPrI.[56]

Prone positioning during invasive mechanical ventilatory support was an external independent risk factor for DRPrI in this study. This result is consistent with the observations from a recent case series conducted among 30 patients in an ICU who were positive for COVID-19.[40] The risks posed by prone positioning when a patient is receiving mechanical ventilatory support are greater because these factors are often compounded by high levels of sedation and, commonly, paralytic medication usually is required to assist the patient receiving mechanical ventilatory support in tolerating prone positioning. The result is a patient experiencing sensory impairment who cannot respond to painful or irritating foci. The presence of an endotracheal tube or tracheostomy in a patient in a prone position exerts pressure on the oral tissue or face. This risk may be mitigated, but not eliminated, with best-practice prevention.

Best practice for DRPrI prevention includes carefully selecting and fitting medical devices, conducting frequent skin inspections under and around devices, and reducing and redistributing pressure at the device and skin interface whenever feasible and not medically contraindicated.[1] Device selection is an essential first step because ill-fitting devices (eg, too-small compression stockings) can dig into the skin.[57] Alternating different types of devices (eg, rotating between an oxygen mask and high-flow nasal cannula) or moving device sites (eg, alternating fingers on which to place the pulse oximeter; routinely repositioning the endotracheal tube) can also reduce tissue exposure to prolonged pressure.[1] Device selection should be based on the available empiric evidence regarding the risk for DRPrI from specific devices; for example, forehead oximetry was determined in a study to be less likely to result in a DRPrI than is nasal oximetry.[58]

Skin inspection is also critical for preventing DRPrI. The NPIAP recommends assessment of the skin under and around devices as a part of routine skin care.[1] When possible, medical devices should be temporarily removed for skin inspection. If a prophylactic dressing is in place, the skin under the dressing should be checked.[54] Skin inspection is particularly important in

patients who experience third spacing of fluid resulting in edema and overly tight devices. Clinicians should also check within skin folds and under breast tissue for medical devices because these devices, including telemetry wires, can become entrapped in skin folds.[1]

Preventing DRPrI in patients in prone position and receiving mechanical ventilatory support requires special consideration.[59] The NPIAP recommends the use of a pressure redistribution surface along with soft, silicone, prophylactic dressings on pressure points and under devices.[60] Commercially available endotracheal tube securement devices can contribute to skin breakdown and should be avoided if possible; tape should be used instead.[61] Securement devices should also be removed from urinary catheters and fecal management systems, and electrocardiogram leads should be placed on the patient's back during prone positioning.[60] The patient's head and body should be shifted every 2 hours and repositioned every 4 hours. If the patient cannot tolerate a more substantial repositioning event, small shifts may be used. The inclusion of a certified wound nurse on the prone team reduces the incidence of DRPrI[38] but may not be feasible given staffing constraints.

Future of Pressure Injury Risk Assessment and Prevention in the Intensive Care Unit

The results of this and prior studies show that more accurate HAPrI risk assessment is urgently needed.[18] The Braden Scale, first published in 1987,[9] tends to identify most patients in the ICU as being at high risk for PrI formation and, therefore, does not give nurses the information they need to differentiate among patients in terms of risk. Yet, risk differentiation is necessary because resources are limited (consider the COVID-19 pandemic) and patients do not all need the same level of care.

Studies conducted using EHR data from patients in the ICU show machine-learning methods can accurately classify patients in terms of HAPrI risk.[61-64] Machine-learning methods can detect complex relationships among variables, including correlated variables, not discernable to the human brain. Models developed using machine-learning methods can be calibrated and updated over time—an essential consideration because ICU populations tend to change over time as advances lead to increased survival rates. The model can be run in the background of the EHR without requiring clinicians to input data. Therefore, the model could be easily rerun with changes in patient status. The ability to dynamically update is critical because patients admitted to the ICU are, by definition, unstable, and HAPrI risk may change hour to hour. Finally, EHR-based models reduce documentation burden because they do not require nurses to manually input data (as with the Braden Scale).

Hospital-acquired PrI risk assessment, machine learning or otherwise, will never fully replace clinician judgment.[65] Nurses and other health care team members have access to the patient's entire story and may have information not visible to a risk prediction algorithm. For example, a nurse may be aware that a patient was discovered unconconsious prior to admission and, therefore, at risk for skin breakdown, but that critical factor will not be a variable in a risk algorithm. Furthermore, algorithms only represent the data they were trained on, so if a subpopulation is underrepresented in the training sample (eg, algorithms developed before the COVID-19 pandemic), the algorithm may not accurately discriminate risk in people within the subpopulation (eg, patients with COVID-19).[65] Explainable artificial intelligence is 1 way to increase transparency: with explainable artificial intelligence, clinicians can observe how the algorithm made its decision. The clinician can then apply that information when deciding whether the result is trustworthy for a given patient.

Limitations

This was a single-site, retrospective study conducted during the COVID-19 pandemic amid a regional surge. The study is limited by its single-site, retrospective design. Moreover, COVID-19 workload and staffing challenges resulted in sparse documentation at times and in some incongruence with established clinical protocols (eg, not all HAPrIs were staged and evaluated by a certified wound nurse despite protocol). Data for preventive interventions were also limited. Although we cite our facility's prevention protocol, it is impossible to verify whether all prescribed preventive interventions were in place at all times.

Conclusions

Patients in the ICU with COVID-19 are at exceptionally high risk for HAPrI, likely

because of a combination of intrinsic (eg, impaired oxygenation and perfusion) and extrinsic (eg, mechanical ventilation and prone positioning) factors. Results from this study show that the Braden Scale lacked predictive validity in patients in the ICU with and without COVID-19 because of low specificity. Device-related PrIs were common in patients with COVID-19; independent risk factors for DRPrI were fragile skin and concomitant mechanical ventilation and prone positioning. Clinicians may consider incorporating factors not included in the Braden Scale (eg, oxygenation and perfusion) in routine risk assessment and should maintain vigilance in their efforts to protect patients with COVID-19 from DRPrI, especially those with fragile skin or who are receiving mechanical ventilatory support during prone positioning. In the future, explainable artificial intelligence may improve the accuracy and interpretability of HAPrI risk assessment.

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