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Outcomes Associated With Stage 1 Pressure Injuries: A Retrospective Cohort Study

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Abstract

Background—Approximately half of hospital-acquired pressure injuries identified among critical care patients are stage 1. Although stage 1 injuries are common, outcomes associated with them among critical care patients have not been examined.

Objectives—To examine the outcomes of stage 1 pressure injuries among critical care patients and to identify factors associated with worsening of pressure injuries.

Methods—Electronic health records were used to determine which surgical critical care patients at a level I trauma center and academic medical center had stage 1 pressure injuries. Competing risk survival analysis was used to identify factors associated with worsening of pressure injuries.

Results—Review of 6377 patient records indicated that 259 patients (4.1%) experienced stage 1 injuries. The injuries persisted until discharge from the hospital in 92 patients (35.5%), worsened into injuries of stage 2 or greater in 84 (32.4%), and healed in 83 (32.0%). Patients whose pressure injuries worsened were more likely to be older (subdistribution hazard ratio [SHR], 1.02; 95% CI,

1.01–1.03; $P = .002$), or to have higher levels of serum lactate (SHR, 1.06; 95% CI, 1.02–1.10; $P = .007$), lower levels of hemoglobin (SHR, 0.82; 95% CI, 0.71–0.96; $P = .01$), or decreased oxygen saturation by pulse oximetry ($< 90\%$; SHR, 1.50; 95% CI, 1.00–2.25; $P = .05$).

Conclusions—Stage 1 pressure injuries worsen in about one-third of patients (32.4%). Nurses should consider maximal treatment for patients who are older or who experience alterations in oxygen delivery or perfusion. (*American Journal of Critical Care*. 2018; 27:471–476)

Hospital-acquired pressure injuries occur in 8% to 10% of surgical intensive care unit patients,^{1,2} and patients with these injuries have longer stays, increased costs, and more human suffering than do patients without such injuries.³ Pressure injuries are categorized as stages 1 to 4, suspected deep tissue injury, and unstageable.⁴ A stage 1 pressure injury is a localized area of nonblanchable redness and/or a change in sensation, skin texture, or temperature. In contrast to stage 1, all other stages are associated with at least partial-thickness tissue injury. Although stage 1 pressure injuries usually indicate only superficial skin injury, they may be more extensive than initially apparent and can deteriorate into a more severe stage (stage 2–stage 4, unstageable, or deep tissue injury). In a study by Halfens et al,⁵ stage 1 pressure injuries worsened in 9% of long-term care patients and 22% of acute care patients.

Stage 1 pressure injuries are common; about half of hospital-acquired pressure injuries reported among surgical critical care patients are stage 1,² yet no researchers have examined the clinical course or outcomes of stage 1 pressure injuries among critical care patients. In a recent systematic review² of studies of risk factors for pressure injuries, only half of the articles with staging information (8 of 16) included stage 1 pressure injuries as the outcome variable. This situation is unfortunate, 1, disproportionately affect critical care patients.⁶

Because of the prevalence of stage 1 pressure injuries in surgical critical care patients and associated adverse consequences, more information on the clinical relevance of these injuries is needed. The purpose of our study was to examine the outcomes of stage 1 pressure injuries among surgical critical care patients. Our specific aims were to examine outcomes associated with stage 1 pressure injuries at the time of discharge from the hospital (healed, stayed the same, or worsened) and to identify factors associated with worsening of pressure injuries.

Methods

We retrospectively reviewed medical records to identify surgical critical care patients with hospital-acquired stage 1 pressure injuries at a level I trauma center and academic medical center.

Patients were eligible if they were admitted to the adult surgical critical care unit or adult cardiovascular surgical critical care unit at the University of Utah Hospital, Salt Lake City, Utah, between September 1, 2008, and May 1, 2013. We included patients who were younger than 18 years if they were admitted to the adult unit. We excluded patients who had pressure injuries at the time of admission.

Measures

Data pertaining to stage 1 pressure injuries were obtained from electronic health records. In an effort to exclude patients with only transient redness, we recorded a patient's condition as a stage 1 pressure injury only if the condition persisted for at least 24 hours. Nurses conducted and documented a head-to-toe skin assessment once per shift (twice per day). The nurses received annual training on recognition and staging of pressure injuries. We identified predictor variables of interest by using the National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel, Pan Pacific Pressure Injury Alliance conceptual framework,⁷ a literature review,² and interviews with clinicians. Data for potential predictor variables were obtained from electronic health records in partnership with a bioinformatics team, and data for each variable were validated via the clinicians' view of the electronic health record for 15 cases. Data were considered valid when the values and the dates and times matched with 100% agreement for the 15 cases, with date/time stamp agreement within 10 minutes.

In an effort to include the dynamic physiological status of critical care patients, we recorded data on predictor values daily, from the time of identification of a stage 1 pressure injury until the outcome of the injury (healed, worsened, or the patient died or was discharged with the injury). For the Braden Scale, we recorded the lowest (most severe) score recorded by the patient's bedside nurse. If different values were recorded on the day shift and the night shift, we recorded the mean of the 2 as the daily score. We used data from patients' monitors to obtain measures of blood pressure and oxygen saturation by pulse oximetry (SpO₂). We defined hypotension as 3 consecutive systolic blood pressure values less than 90 mm Hg, in an effort to exclude the spurious values sometimes generated by an automatic blood pressure cuff or dampening of an arterial catheter. Similarly, we defined low SpO₂ as 3 SpO₂ values less than 90%. We defined a fever as at least 1 temperature greater than 100.94°F (38.3°C). Laboratory data were also recorded daily and included the highest lactate, creatinine, and glucose levels and the lowest hemoglobin and albumin levels. If no laboratory value was recorded on a given day, we used the most recent value ("nearest neighbor").

Statistical Analysis

We analyzed the data by using Stata 15 software (StataCorp LLC). Statistical significance was set at $\alpha = 0.05$. We included only variables with less than 15% missing data; missing data are described in Table 1. We used descriptive statistics to summarize the distribution of patient characteristics and the health outcomes at the time of discharge from the hospital. We used Fine-Gray model analyses for the survival outcome in the presence of competing risk to conduct univariate analyses and explore the association between factors and the outcome of the stage 1 pressure injury (healed, worsened into > stage 2, or stayed the same). We used univariate competing risks regressions to assess factors related to worsening of pressure injuries. In our analysis, patients who had stage 1 injuries at the time of discharge were considered censored, worsening of pressure injury was the event, and healing of pressure injury was the competing risk to the event.

Results

In a review of 7218 records, we excluded 841 records because of problems with the patient identification number (eg, a date or single-digit number). The final sample consisted of 6377 patients; among those, a stage 1 pressure injury that persisted for at least 24 hours developed in 259 (4.1%); in 1 patient, a stage 1 pressure injury persisted for less than 24 hours, and that patient's data were excluded from the analysis. The mean age among patients with stage 1 injury was 60.4 years (SD, 17.2), and 59.8% of those with stage 1 injuries were men (n = 155). Most of the patients were white (85.7%; n = 222). A total of 5860 patients in the sample had no pressure injury, and 257 patients had pressure injuries of stage 2 or greater, for an 8.1% incidence of global hospital-acquired pressure injury.

Among the 259 patients with stage 1 pressure injuries, the injury stayed at stage 1 in 92 patients (35.5%), healed in 83 (32.0%), and worsened to greater than stage 2 in 84 (32.4%). The mean time from hospital admission to diagnosis of stage 1 pressure injury was 8 days (range, 3–15 days).

Factors associated with worsening of stage 1 pressure injuries are given in Tables 1 and 2. Among the variables we examined, the following were significantly related to increased risk for worsening of a pressure injury: older age, higher lactate level, higher creatinine level, lower hemoglobin level, and lower SpO₂. We were unable to calculate a subdistribution hazard ratio for the variable fever because 252 patients in the sample (97.3%) had fever.

Discussion

The finding that nearly one-third (32.4%) of critical care patients with stage 1 pressure injuries experienced worsening of their injury was unexpected. In an earlier study,⁵ about 9% of long-term care patients and 22% of acute care patients experienced worsening of stage 1 pressure injury. We speculate that critical illness and its attendant physiological burden are responsible for the increased incidence of worsening of stage 1 pressure injury.

The finding that older patients were more likely than younger ones to experience worsening of a pressure injury was expected. In another study⁸ on outcomes of pressure injuries (primarily stages > 2), older patients were less likely than younger patients to have healing of a pressure injury. However, in that study, age was dichotomized into 2 categories: more than 40 years and less than 40 years. Patients more than 40 years old were considered older. In our sample, the mean age was 60.4 (SD, 17.2) years, and therefore the majority of our patients were more than 40 years old. The mechanism of increased risk for worsening of pressure injury among older patients is most likely a combination of increased comorbid conditions among older patients,⁹ aging-related skin changes that pre-dispose older people to development of pressure injuries (particularly related to elasticity),¹⁰ and aging-related vascular changes that affect the delivery of oxygen-rich blood to tissue.¹⁰

Higher serum levels of creatinine were also related to worsening of pressure injuries. Although creatinine levels have not been examined in relation to worsening of pressure injuries, Frankel et al¹¹ found that creatinine level was an independent risk factor for development of pressure injuries. Similarly, both renal failure¹ and dialysis¹² were identified

in previous studies as independent risk factors for pressure injury. The finding that increased levels of creatinine are associated with worsening of pressure injuries is unsurprising; renal impairment is known to affect wound healing, most likely because of the effects of uremic toxins.¹³

In our study, low serum levels of lactate, low hemoglobin concentrations, and decreased oxygenation (SpO₂) were also related to worsening of pressure injuries. These findings were expected: delivery of oxygen-rich blood is essential to wound healing, higher lactate levels indicate altered perfusion, and low SpO₂ and low hemoglobin concentrations indicate impaired oxygen-carrying capacity. We anticipated that low blood pressure would be statistically associated with worsening of pressure injuries, but it was not (SHR, 1.54; 95% CI, 0.93–2.55; *P* = .09). Possibly, some degree of hypotension was masked by vasopressor infusions; we were unable to obtain information on infusion of these medications.

In our sample of 259 patients with stage 1 pressure injury, 85.7% (*n* = 222) were white. Among the 6736 patients whose records we examined, only 71.8% (*n* = 4838) were white. This information is interesting, because stage 1 pressure injuries may be under-recognized in patients with darker skin tones. The National Pressure Ulcer Advisory Panel⁴ recommends that nurses caring for patients with darker skin tones assess the patients carefully for changes in temperature, sensation, or skin texture/firmness to identify stage 1 pressure injuries.

Increased body temperature was identified as a potential indirect causal factor for development of pressure injury in the theoretical schema of Coleman et al⁹ and has been identified as an independent risk factor for development of pressure injuries among critical care patients.¹⁴ Therefore, we anticipated that patients with fever would have increased risk for worsening of pressure injuries; however, we were unable to test that relationship because 252 of the 259 patients in our sample (97.3%) had a fever. The finding that most of the patients in our sample experienced a fever warrants future study, particularly because in the general population from which we drew our sample (*N* = 6376), only 767 (12.0%) experienced a fever.

The variables examined by using the Braden Scale and its subscales were not related to healing of pressure injuries. Earlier studies^{15–17} indicated that the Braden Scale lacks predictive validity among critical care patients, and therefore the scale is sometimes considered a care-planning tool in the intensive care unit rather than a risk-assessment tool. However, our findings suggest that the Braden Scale is not a useful tool for guiding care planning for patients who already have a stage 1 pressure injury.

Limitations

Limitations of our study include the retrospective design; we were limited to the accuracy and availability of the data in the electronic health records. To avoid misclassifying community-acquired pressure injuries as hospital-acquired, we excluded patients who had pressure injuries at the time of admission. This exclusion may have introduced bias, because patients with preexisting pressure injuries are at increased risk for development of pressure injuries.⁹ In addition, we were unable to access treatment-related variables, such as the

presence of prophylactic sacral dressings. Finally, although we sought to include variables related to oxygen and perfusion, we were not able to obtain data on infusion of vasopressors.

Conclusion

Worsening of stage 1 pressure injuries into more severe stages occurred among 32.4% (n = 84) of the surgical critical care patients in our sample. Patients who experienced worsening of pressure injury were more likely to be older, have higher serum levels of lactate and creatinine, and lower concentrations of hemoglobin and SpO₂. Because of the relatively high percentage of patients (32.4%) who experienced worsening of pressure injury, critical care nurses should consider aggressive offloading and other preventive measures for patients with a stage 1 pressure injury to prevent worsening of the injury. Aggressive measures are particularly important for patients with stage 1 injury who are older or who have alterations in oxygen delivery or perfusion as indicated by higher levels of serum lactate, lower oxygen-carrying capacity (hemoglobin), lower percentage of hemoglobin saturated with oxygen, or elevated serum levels of creatinine.

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About half of all hospital-acquired pressure injuries identified among critical care patients are stage 1.

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Patients with stage 1 pressure injuries are at high risk for pressure injury worsening.

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Table 1

Demographic and predictor variables

Variable	Total (N = 259)	Stayed at stage 1 (n = 92)	Progressed to stage 2 (n = 84)	Healed (n = 83)
Age, mean (SD), y	60.4 (17.2)	66.1 (16.9)	62.3 (15.3)	52.2 (16.5)
Male sex, No. (%) of patients	155 (59.8)	54 (58.7)	49 (58.3)	52 (62.7)
White race, No. (%) of patients	222 (85.7)	80 (87.0)	68 (81.0)	74 (89.2)
Score on Braden Scale, mean (SD)				
Total ^a	16.4 (3.0)	16.0 (2.7)	16.8 (3.3)	16.3 (3.0)
Activity subscale	1.8 (1.0)	1.8 (1.0)	1.9 (1.1)	1.8 (1.0)
Moisture subscale	3.6 (0.5)	3.6 (0.5)	3.7 (0.4)	3.7 (0.5)
Mobility subscale	2.8 (0.8)	2.7 (0.7)	2.8 (0.8)	2.8 (0.7)
Nutrition subscale ^b	2.4 (0.6)	2.4 (0.6)	2.5 (0.6)	2.4 (0.5)
Sensory subscale	3.2 (0.8)	3.2 (0.8)	3.3 (0.8)	3.2 (0.8)
Friction shear subscale ^a	2.5 (0.4)	2.4 (0.4)	2.6 (0.4)	2.5 (0.4)
Body mass index, ^{c,d} No. (%) of patients				
<19	23 (10.4)	10 (14.3)	4 (5.3)	9 (12.0)
19–30	128 (57.9)	47 (67.1)	41 (54.0)	40 (53.3)
>30	70 (31.7)	13 (18.6)	31 (40.8)	26 (34.7)
Hypotension, No. (%) of patients				
Lactate, ^e median (IQR), mg/dL	1.8 (1.3–2.8)	1.7 (1.2–2.2)	2.2 (1.5–3.9)	1.8 (1.3–2.2)
Creatinine, median (IQR), mg/dL	2.3 (2.0)	1.9 (1.6)	2.7 (2.2)	2.2 (2.0)
Glucose, mean (SD), mg/dL	212.3 (93.5)	203.4 (80.0)	225.9 (112.6)	208.5 (85.3)
Hemoglobin, mean (SD), g/dL	7.8 (1.6)	8.3 (1.6)	7.2 (1.3)	7.8 (1.5)
Low SpO ₂ , No. (%) of patients	100 (38.6)	26 (28.3)	43 (51.2)	31 (37.3)
Surgery time, mean (SD), min	455.0 (385.7)	366.6 (324.9)	555.1 (453.8)	444.0 (349.8)
Albumin, ^f mean (SD), mg/dL	2.4 (0.7)	2.4 (0.6)	2.2 (0.6)	2.4 (0.7)
Fever, No. (%) of patients	252 (97.3)	88 (95.7)	84 (100.0)	80 (96.4)

Abbreviations: IQR, interquartile range; SpO₂, oxygen saturation by pulse oximetry.

- ^bData were missing for 2 patients (0.7%).
- ^cData were missing for 1 patient (0.4%).
- ^dData were missing for 38 patients (14.7%).
- ^eCalculated as weight in kilograms divided by height in meters squared.
- ^fData were missing for 14 patients (5.4%).
- ^gData were missing for 32 patients (12.4%).

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Table 2

Competing risk survival analysis for patients with stage 1 pressure injury

Variable of interest	Subdistribution hazard ratios (95% CI) of worsening into a stage 2 pressure injury	P
Age	1.02 (1.01–1.03)	.002
Sex, female vs male	1.12 (0.74–1.69)	.60
Race, white vs nonwhite	0.65 (0.37–1.13)	.12
Total score on Braden Scale	1.05 (0.98–1.13)	.18
Braden activity subscale	1.06 (0.86–1.30)	.61
Braden moisture subscale	1.13 (0.73–1.74)	.60
Braden mobility subscale	1.13 (0.85–1.50)	.38
Braden nutrition subscale	1.37 (0.91–2.06)	.13
Braden sensitivity subscale	1.20 (0.91–1.58)	.19
Braden friction shear subscale	1.54 (0.94–2.50)	.08
Body mass index ^a		
< 19 vs 19–30	0.43 (0.17–1.07)	.07
> 30 vs 19–30	1.34 (0.85–2.12)	.21
Hypotension	1.54 (0.93–2.55)	.09
Lactate level	1.06 (1.02–1.10)	.007
Creatinine level	1.09 (1.01–1.18)	.03
Glucose level	1.00 (1.00–1.00)	.25
Hemoglobin level	0.82 (0.71–0.96)	.01
Low SpO ₂	1.50 (1.00–2.25)	.05
Surgery time, min	1.00 (1.00–1.00)	.20
Albumin level	0.84 (0.61–1.17)	.31

Abbreviations: SpO₂, oxygen saturation by pulse oximetry.^aCalculated as weight in kilograms divided by height in meters squared.