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RESEARCH ARTICLE

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Deep sedation predicts pressure injury in patients admitted to intensive care units

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Abstract

Background: Patients in intensive care units (ICU) are frequently prescribed sedatives, which might increase the risk for pressure injury (PI). Although the association between sedation and incidence of PI has been noted, the adequate sedation level to prevent the incidence of PI in patients admitted to ICU is still unclear.

Aim: This study aimed to investigate the association between fluctuating sedation levels and the incidence of PI in patients admitted to ICU.

Study Design: We retrospectively reviewed the medical records of 104 patients admitted to ICU. Data regarding the length of ICU stay (LOS) and comorbid infection were abstracted from medical records. The Richmond Agitation-Sedation Scale (RASS) was scored twice per day, and the standardized RASS (S-RASS, summation of RASS values divided by the number of samples) was used to evaluate changes in sedation levels.

Results: Among the 104 included patients, 65 patients (62.5%) were male (median age: 68.0 years), and 13 patients (12.5%) had PI during ICU admission. S-RASS scores were lower in patients with PI than in those without PI ($P = .0001$) even after adjustment for confounders (OR [95%CI]: 0.14 [0.03–0.58], $P = .006$). The LOS and infections were higher in patients with PI than in those without PI ($P < .0001$ and $P = .005$, respectively). The cut-off value of S-RASS for PI incidence was −3.2 (sensitivity: 88%; specificity: 85%), and a significant predictor of PI incidence (HR [95%CI]: 20.07 [2.53–159.11], $P = .005$).

Conclusions: Deeper sedation levels based on S-RASS scores, which account for the effects of fluctuating sedation levels, were a strong, highly accurate predictor of PI incidence in patients admitted to ICU.

Relevance to Clinical Practice: Assessing fluctuations in the level of sedation using the S-RASS might help to identify sedative-induced PI in patients admitted to ICU.

KEYWORDS
intensive care units, pressure injury, sedation level

1 | INTRODUCTION

Pressure injury (PI) is a common adverse event in people who have been in the hospital.[1] A previous study estimated that the incidence of PI in patients who were admitted to general wards was 6.3%.[2] Further, the incidence of PI increased the cost of admission by 14 000–79 000 USD, which was required for its management.[3] The estimated incidence of PI was higher in patients admitted to intensive care units (ICU) than in those admitted to general wards, because in ICU, patients would be under physiological restraint, sedative condition, or have an unstable circulatory system for the management of their severe and unstable underlying disease.[4–6] Previous studies have shown that the predictors of the incidence of PI in patients admitted to ICU included lower mean aortic pressure, presence of dialysis treatment, and use of sedatives.[7]

It is well known that the incidence of PI is associated with adverse outcomes, such as a low quality of life (QOL), prolonged hospitalization, and higher in-hospital mortality.[8–11] Although the cause-effect association is still under debate, it indicates that the prevention of PI would be one of the mandatory requirements for the management of patients admitted to ICU. As mentioned above, the use of sedation could be one of the predictors of PI incidence, although the sedative levels would significantly fluctuate during admission. If assessments of the sedation level that account for such fluctuations were associated with an increased incidence of PI, they might aid in the development of preventive and early-intervention strategies for patients requiring deep sedation in the ICU.

WHAT IS KNOWN ABOUT THIS SUBJECT

- Pressure injury (PI) is a common adverse event in patients admitted to intensive care units (ICU).
- Although PIs are associated with worsening of quality of life and patients' prognosis, the association between fluctuating sedation level and incidence of PI in patients admitted to ICU is unclear.

WHAT THIS PAPER ADDS

- The standardized Richmond Agitation-Sedation Scale (S-RASS), which was calculated to include the effects of fluctuations in sedation levels, was significantly associated with the incidence of PI in patients admitted to ICU. The cutoff value for sedation level indexed using the S-RASS was −3.2 points (area under the curve: 0.85; sensitivity: 88%; specificity: 85%).
- Assessments that include the effects of fluctuating sedation levels might aid in preventing PI and improving safety for patients admitted to ICU.

2 | AIM

The aim of this study was to investigate the association between sedation levels and PI incidence in patients admitted to ICU using an assessment that accounted for fluctuations in such levels.

3 | METHODS

3.1 | Subjects

This retrospective study was performed at a university-affiliated tertiary emergency care center with 22 beds in a 724-bed hospital in Hiroshima City, Japan. We retrospectively abstracted data from the medical records of patients admitted to the general ICU. The inclusion criteria for patients were as follows: (a) > 20 years of age, and (b) admitted in an ICU for over 48 hours. The exclusion criteria were as follows: (a) patients with PI before ICU admission, (b) admitted for management post-surgery, and (c) with any severe skin injury or skin-related diseases, since we could not assess whether the patients had PI after ICU admission. Based on these criteria, we identified 104 patients who were admitted between April 2016 and May 2017 (Figure S1). The study protocol was approved by the Ethical Committee for Epidemiology of Hiroshima University (#E-1224). As this was a retrospective study, an opt-out statement was used for patient consent.

3.2 | Data collection

We abstracted data related to the following from each patient's medical record: demographics (e.g., age, sex, body weight, and body height), length of ICU stay, the reason for ICU admission, comorbidities, medications and devices used for treatment during ICU admission. Data regarding infection and comorbidities were abstracted by collected information regarding diseases diagnosed by a medical doctor. With respect to laboratory data, serum albumin level, haemoglobin level, haematocrit level, white blood cell count, and C-reactive protein (CRP) levels were extracted. The reasons for ICU admission were categorized according to the Japanese Intensive Care Patient Database.[12] We also extracted data on the use of organ support devices such as intra-aortic balloon pumping, extracorporeal membrane oxygenation or blood purification (i.e., intermittent renal replacement therapy [IRRT], continuous renal replacement therapy [CRRT], or plasma exchange). To evaluate the severity of disease in the patients, the Acute Physiology and Chronic Health Evaluation II (APACHE-II) score were calculated by the lowest scores within 24 hours after ICU admission was used.[13]

3.3 | Pressure injury

With respect to PI-related data, the incidence of PI was assessed every day during ICU admission, and the severity of PI was assessed according to the National Pressure Ulcer Advisory Panel (NPUAP) pressure injury stages when the patients had PI.[14] We regarded the

patients with PI when the patients had stage 1 to 4 pressure injury including unstageable stage on NPUAP classification. Briefly, the severity of PI could be classified as follows: stage 1: non-blanchable erythema of intact skin; stage 2: partial-thickness skin loss with exposed dermis; stage 3: full-thickness skin loss; stage 4: full-thickness skin and tissue loss; unstageable: obscured full-thickness skin and tissue loss; and deep-tissue pressure injury: persistent non-blanchable deep red, maroon, or purple discoloration. In this study, we determined the severity of PI based on the most severe stage of PI during ICU admission.

In addition to the incidence of PI, the history of PI, presence of physical restraint, days from ICU admission to the incidence of PI, and

TABLE 1 Clinical characteristics

	Total	w/o PI	with PI	*P*-value
Number, N (%)	104 (100.0)	91 (87.5)	13 (12.5)	—
Age, y.o.	68.0 (62.0, 76.0)	69.0 (62.0, 77.0)	65.0 (62.0, 74.0)	.43
Male, N (%)	65 (62.5)	54 (59.3)	11 (84.6)	.08
BMI, kg/m^2	22.2 (19.4, 26.4)	22.5 (19.4, 26.6)	22.1 (19.8, 23.6)	.41
Length of ICU stay, days	5.0 (3.0, 9.0)	4.0 (3.0, 8.0)	15.0 (12.0, 27.0)	<.0001
S-RASS, points	−1.4 (−0.5, −2.9)	−1.2 (−0.4, −2.4)	−4.0 (−3.4, −4.4)	.0001
APACHE-II score, point	24.6 ± 9.0	23.5 ± 8.4	32.5 ± 9.6	.0008
Infection	43 (41.4)	33 (36.3)	10 (76.9)	.005
PI				
Days from ICUs admission to incidence, days	—	—	7.9 ± 7.0	—
Braden scale, points	13.0 (11.0, 14.0)	13.0 (12.0, 15.0)	11.0 (10.0, 11.0)	.0001
Hx of PI, N (%)	0 (0.0)	0 (0.0)	0 (0.0)	—
Braden scale based PI risk, N (%)				
No risk	5 (4.8)	5 (5.5)	0 (0.0)	.01
Mild risk	19 (18.3)	19 (20.9)	0 (0.0)	
Moderate risk	30 (28.9)	29 (31.9)	1 (7.7)	
High risk	42 (40.4)	33 (36.3)	9 (69.2)	
Very high risk	8 (7.7)	5 (5.5)	3 (23.1)	
Reasons for admission to ICUs, N (%)				
Respiratory diseases	44 (42.3)	36 (39.6)	8 (61.5)	.74
Cardiovascular diseases	20 (19.2)	17 (18.7)	3 (23.1)	
Digestive disease	18 (17.3)	17 (18.7)	1 (7.7)	
Sepsis	7 (6.7)	6 (6.6)	1 (7.7)	
Nervous system disease	7 (6.7)	7 (7.7)	0 (0.0)	
Musculoskeletal/Skin diseases	4 (3.8)	4 (4.4)	0 (0.0)	
Genitourinary disorders	3 (2.9)	3 (3.3)	0 (0.0)	
Metabolic diseases	1 (1.0)	1 (1.1)	0 (0.0)	
Comorbidities, N (%)				
Diabetes	21 (20.2)	18 (19.8)	3 (23.1)	.78
Dyslipidemia	10 (9.6)	10 (11.0)	0 (0.0)	.21
Malignant tumour	45 (43.3)	40 (44.0)	5 (38.5)	.71
Cardiac diseases	38 (36.5)	37 (40.7)	1 (7.7)	.02
Laboratory data				
Albumin, g/dL	2.6 ± 0.7	2.6 ± 0.7	2.2 ± 0.5	.06
Haemoglobin, g/dL	9.9 (8.6, 11.5)	10.0 (8.6, 11.7)	9.6 (8.3, 10.3)	.22
Haematocrit, %	30.1 (25.1, 34.6)	30.3 (25.1, 34.8)	28.6 (24.1, 30.5)	.21
WBC, $\times 10^3/\mu L$	10.1 (6.7, 14.3)	9.8 (6.8, 14.3)	10.4 (6.6, 15.5)	.97
CRP, mg/dL	8.7 (3.0, 16.5)	7.5 (2.8, 15.3)	10.0 (8.9, 21.1)	.10

Abbreviations: APACHE, acute physiology and chronic health evaluation; BMI, body mass index; CRP, C-reactive protein; Hx, History; ICU, intensive care units; N, mumber; PI, pressure injury; S-RASS, standardized richmond agitation-sedation scale; WBC, white blood cell.

PI incidence area were evaluated. The risk for the incidence of PI was scored using the Braden Scale, which includes six subscales: sensory perception, skin moisture, activity, mobility, nutritional status, and friction/shear.[15] Total scores on the Braden scale range from 6 to 23 points, with lower scores indicating a higher risk of PI. Braden scale was assessed upon ICU admission, and the risk of PI was categorized into the following five stages: very high risk (<10 points), high risk (10-12 points), moderate risk (13-14 points), mild risk (15-18 points), and no risk (>18 points).

3.4 | Richmond Agitation-Sedation Scale

The Richmond Agitation-Sedation Scale (RASS) was developed for evaluation of sedation and agitation levels in the patients who are admitted to ICU, and the RASS ranged from −5 (Unarousable) to +4 (Combative).[16,17] The sedation and agitation levels of patients were evaluated at 8:00 AM and 8:00 PM every day during ICU admission, and the ICU staff attempted to maintain the RASS score of patients between 0 and −2. Furthermore, the points of RASS were standardized, referred to as the standardized RASS (S-RASS; formula: summation of RASS points during ICU admission divided by the times of sampling), as the sedation and agitation levels usually fluctuated during admission. In patients with PI during the follow-up, the formula used for S-RASS calculation was as follows: S-RASS = summation of RASS points based on PI incidence divided by the times of sampling. A lower S-RASS value indicated that the patients were under deeper sedation.

3.5 | Statistical analyses

The values are represented as means ± SD, median (interquartile range: IQR), number (%), odds ratio (OR) [95% confidence interval; 95%CI], and hazard ratio (HR) [95%CI]. We used the Kolmogorov–Smirnov test to evaluate the normality of the data before assessments using the paired t-test or Mann–Whitney U test. Fisher's exact test was used to assess the differences between two groups in the binary data. To assess the predictors of PI incidence, we used stepwise logistic regression analysis with forward selection (*P*-value for the forward selection: $P < .10$). To estimate the cut-off values for PI incidence, the receiver operating characteristic (ROC) curve with the Youden index method was used. After the estimation of the cut-off point, the patients were stratified based on this cut-off point, and the risk for PI was compared using the Kaplan–Meier curve with the log-rank test and Cox proportional hazards model. With regard to the Cox proportional hazards model, we used potential confounders as variables that showed significant associations in the stepwise logistic regression analysis for predictors of PI incidence. If the *P*-value was <.05, the two-sided tests were considered statistically significant. All statistical tests were performed using STATA version 15.1 (Stata-Corp, TX, USA).

TABLE 2 Treatments during the Admission

	Total	w/o PI	PI	*P*-value
Physical restraint, N (%)	68 (65.4)	60 (65.9)	8 (61.5)	.76
Medications				
Propofol, N (%)	23 (22.1)	18 (19.8)	5 (38.5)	.13
Dexmedetomidine, N (%)	42 (40.4)	36 (39.6)	6 (46.2)	.65
Midazolam, N (%)	15 (14.4)	8 (8.8)	7 (53.9)	<.0001
Rocuronium, N (%)	4 (3.9)	2 (2.2)	2 (15.4)	.02
Dobutamine, N (%)	11 (10.6)	8 (8.8)	3 (23.1)	.12
Dopamine, N (%)	5 (4.8)	5 (5.5)	0 (0.0)	.39
Adrenaline, N (%)	8 (7.7)	3 (3.3)	5 (38.5)	<.0001
Noradrenaline, N (%)	48 (46.2)	37 (40.7)	11 (84.6)	.003
Vasopressin, N (%)	6 (5.8)	3 (3.3)	3 (23.1)	.004
Treatments				
IABP, N (%)	4 (3.8)	3 (3.3)	1 (7.7)	.44
VA-ECMO, N (%)	4 (3.8)	2 (2.2)	2 (15.4)	.02
VV-ECMO, N (%)	5 (4.8)	2 (2.2)	3 (23.1)	.001
IRRT, N (%)	20 (19.2)	13 (14.3)	7 (53.9)	.001
CRRT, N (%)	21 (20.2)	5 (16.5)	6 (46.2)	.02
PE, N (%)	5 (4.8)	4 (4.4)	1 (7.7)	.60

Abbreviations: CRRT, continuous renal replacement therapy; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pumping; IRRT, Intermittent renal replacement therapy; N, number; PE, plasma exchange; PI, pressure injury; VA, veno-arterial; VV, veno-venous.

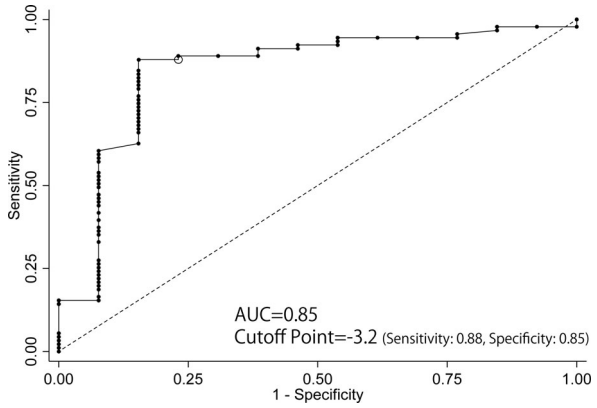

FIGURE 1 Receiver operating characteristic carve. The open circle indicates the cut-off point, which was calculated using the Youden index. AUC, Area under the curve

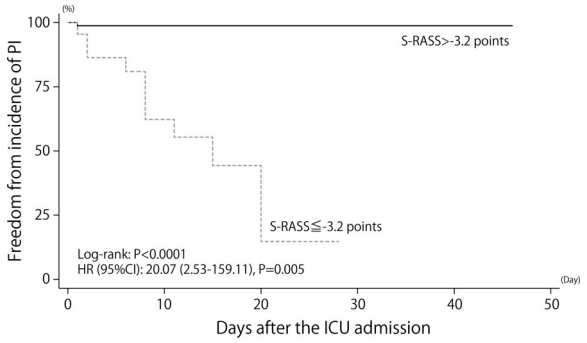

FIGURE 2 Kaplan–Meier curve and hazard ratio for the estimated cut-off value. PI, pressure injury; S-RASS, standardized richmond agitation-sedation scale; HR, hazard ratio; CI, confidence interval; ICU, intensive care units

4 | RESULTS

Of the identified patients who were admitted to ICU, the rate of PI incidence during ICU admission was 12.5% (13 patients). The median (IQR) age and length of ICU stay were 68.0 (62.0, 76.0) years and 5.0 (3.0, 9.0) days, respectively (male: 65 patients [62.5%], body mass index [BMI]: 22.2 [19.4, 26.4] kg/m^2) (Table 1). The mean ± SD of the APACHE-II score was 24.6 ± 9.0 points. The most frequent reason for ICU admission was respiratory disease (44 patients; 42.3%), followed by cardiovascular diseases (20 patients; 19.2%), digestive diseases (18 patients; 17.3%), and sepsis (7 patients; 6.7%).

Regarding the differences in clinical characteristics between patients with PI and those without PI, significant differences were not observed for age, the proportion of male sex, BMI, the proportion of reason for ICU admission, presence of diabetes mellitus, dyslipidemia, malignant tumour, or laboratory data, including serum albumin level and CRP. However, the length of ICU stay, APACHE-II score, and presence of infection were significantly higher in patients with PI than in those without PI ($P < .0001$, $P = .0008$ and $P = .005$, respectively). On the other hand, S-RASS and the presence of cardiac diseases in patients with PI were significantly lower than those without PI ($P = .0001$ and $P = .02$, respectively). Furthermore, the rates of use of IRRT and CRRT were significantly higher in patients with PI than in those without PI ($P = .001$ and $P = .02$, respectively) while no significant difference was observed in the rate of a physical restraint ($P = .76$) (Table 2).

Among patients with PI, 38.5% (5/13 patients) had PI by 3 days after ICU admission Table S1). The peak time for the incidence of PI was observed 9 days after ICU admission. The severity of PI was assessed based on the NPUAP pressure injury stages, ranging from stage II to stage III; 53.8% of patients with PI (7/13 patients) had stage III PI. PI was frequently observed in the sacral region (8/13 patients, 61.5%) and calcaneal region (6/13 patients, 46.2%), which suggests that PI might occur when patients spent in the supine position.

Next, we evaluated the factors associated with the incidence of PI using the logistic regression analysis. S-RASS and presence of infection were significant predictors of PI incidence during ICU admission (OR [95% CI]: 0.14 [0.03–0.58], $P = .006$; OR [95% CI]: 18.77 [1.52–232.19], $P = .02$, respectively) (Table S2). In addition, with respect to S-RASS, ROC analysis showed that the cut-off point for PI incidence was −3.2 (area under the curve: 0.85; sensitivity: 88%, specificity: 85%) (Figure 1). Furthermore, Kaplan–Meier curve and cox proportional hazards analysis using the cut-off value of S-RASS showed that the patients who had values below the cut-off value of S-RASS had significantly higher PI than those who did not have values below the cut-off value, even after adjustment for potential confounders, as shown in Figure 2 (log-rank: $P < .0001$; HR [95%CI]: 20.07 [2.53–159.11], $P = .005$).

5 | DISCUSSION

In this study, we investigated the association between sedation levels and PI incidence in patients admitted to ICU using the S-RASS score, which accounts for fluctuations in such levels. Our findings indicated that deeper sedation levels as indexed by S-RASS scores were associated with the incidence of PI, and that the cut-off value for PI incidence was −3.2 (sensitivity: 88%, specificity: 85%). The presence of infection was also identified as a significant predictor of PI incidence. These findings indicate that assessments that account for fluctuations in sedation levels such as the S-RASS might represent useful indicators of PI incidence and that greater attention to the potential for PI would be necessary for patients admitted to ICU with infection.

In addition to sedative use, we speculated that patients admitted to ICU had poor physical activity status when compared to those admitted to general wards due to the presence of acute or unstable underlying diseases. General ward-based studies reported that the predictors of PI incidence were edema, diabetes mellitus and low activity levels in daily life.[18] On the other hand, in the case of patients who were admitted to ICU, the reported predictors of PI incidence included the use of sedatives, coma stage, and cardiovascular instability owing to the use of dopamine, intermittent haemodialysis, and continuous veno-venous hemofiltration.[19–21] Regarding the

relationships between PI incidence and sedation levels, previous studies reported that the predictor of PI incidence in ICU was only the usage of sedation, not sedation levels. In this study, the S-RASS score of −3.2 points was a significant predictor of PI incidence, which might indicate that deep sedation (i.e., S-RASS ≤ −3.2) could be one of the indicators for PI incidence in patients admitted to ICU. When the S-RASS score was below the cut-off value, it was highly speculated that the patients could not remove their weight pressure by themselves, and the blood flow in the pressured skin area might be chronically restricted. Some previous studies indicated that insufficient blood flow to the pressed skin tissue might lead to PI due to insufficient oxygen supply.[22] One of the mechanisms underlying the association between insufficient oxygen supply and the PI incidence is that insufficient oxygen supply leads to an increase in hypoxia-inducible factors (HIF) and the HIF leads to a disruption in c-Myc which promotes cell growth signal and is a transcriptional activator. When the oxygen supply is insufficient and hypoxemia develops, binding of c-Myc to deoxyribonucleic acid is inhibited, which leads to poor relieving of repression of gene expression. The poor relieving of repression of gene expression leads to cell apoptosis. These reactions from insufficient blood flow might cause PI through reduced tissue tolerance due to cell apoptosis.

For the relationship between infection and PI incidence, some previous studies indicated the association between PI incidence and pneumonia, which is a common disease among patients admitted to ICU.[23,24] Although the pathophysiological relationships between PI incidence and pneumonia remain unclear, pneumonia in the ICU might be one of the causes of PI incidence because of impaired gas exchange in the lungs and the consequent insufficient oxygen supply to the skin. Under this situation, it was also speculated that oxygen supply to the skin and its surroundings were severely limited but the blood flow to the skin was sufficiently supplied. As mentioned before, the insufficient oxygen supply to the skin might lead to cell apoptosis through an increase in the HIF.[22] In patients with infections such as pneumonia, the risk of PI incidence might be high even when the medical staff removed their pressure to the skin for preventing PI.

Previous studies showed that the estimated incidence rate of PI in patients who were in ICU ranged from 8% to 9%, which is comparable to the incidence rate of PI in this study (12.5%), although the incidence rates of PI in general wards and nursing homes were reported to be 6.3% and 2.1%, respectively.[2,4–6,25] These differences in the incidence rates of PI may be affected by differences in nutritional status, use of medicines, the severity of underlying diseases, and prevalence of comorbidities including edema, hypoxemia, and hypotension among patients who were in nursing homes, general wards, and ICU.[18] For differences between patients who were in general wards and those in ICU, the elevation of inflammatory agents such as CRP is a well-known predictor of PI incidence in patients in general wards; however, the relationship between the incidence of PI and CRP was not observed in this study.[26] The reason for the differences may be the masked effects of inflammation, which may be confounded by severe and acute underlying diseases or the presence of infections.

On the other hand, it was well known that factors contributing to PI development are mechanical boundary conditions (i.e., magnitude, duration, and types of mechanical load to the skin) as well as the susceptibility and tolerance of the patient (i.e., mechanical properties of the tissue, geometry of the tissue and bone, transport-related and thermal properties, physiological properties, and repair properties).[14] In the current study, infection and S-RASS were significant risk factors for PI incidence, reflecting the susceptibility and tolerance of the person, even though the main causes of PI are the application of pressure and shearing force to the skin. We should therefore remain aware that reducing pressure and shear to the skin is important for preventing PI incidence even in patients without infection and in those who are not under deep sedation.

6 | LIMITATIONS

This study has some limitations. First, since this was a retrospective study, we cannot deny any selection bias in this study. Second, we could not mention other emergency medical facilities (i.e., first and second emergency medical facilities), as the setting of this study was a single third emergency medical facility. Third, although localized infection and infection sites might be significant risk factors for PI incidence, we could not abstract detailed data related to these variables from medical records due to the retrospective nature of the study. However, since patients with skin-related diseases were excluded from this study, we strongly speculated that no patients had skin infections in any specific area. Fourth, no relationships between PI incidence and Braden scale were observed in this study population, although the Braden scale includes some assessment items recommended in the NPUAP guidelines (i.e., sensory perception, skin moisture, activity, mobility, nutritional status, and friction/shear), and the tool has been validated in a previous study as mentioned before. This might be because most patients in this study had low sensory perception ability, activity, and mobility levels (i.e., under sedation) and poor nutritional status (i.e., low serum albumin level; Table 1), which would lead to difficulties in risk stratification for PI using Braden scale.[27] That is, the status of patients typically admitted to ICU might have influenced our findings. Lastly, all patients were assessed for the risk of PI and the medical staff performed some preventive measures regardless of risk for PI. However, the frequency of the preventive measures for PI is unknown, but there was less bias regarding the frequency of preventive measures for PI as the study was retrospective. Further studies are required to overcome these limitations.

7 | IMPLICATIONS FOR PRACTICE

Assessing fluctuations in the level of sedation using the S-RASS might help to identify sedative-induced PI in patients admitted to ICU. This might aid in the development of preventive and early-intervention

strategies for sedative-induced PI, which will in turn help to improve patient safety and QOL.

8 | CONCLUSION

In conclusion, our results indicated that the presence of infections was a significant predictor of PI incidence in patients admitted to ICU. Furthermore, S-RASS scores, which were calculated to eliminate the effects of fluctuations in sedation levels, were identified as a strong, highly accurate predictor of PI incidence (sensitivity: 88%; specificity: 85%) in patients admitted to ICU. Performing such assessments in patients who require deep sedation in the ICU might help to prevent PI.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest

AUTHOR CONTRIBUTIONS

Satoshi Yamaga, Mayumi Niitani, Kazuaki Tanabe, and Hiroyuki Sawatari contributed to the study design. Yayoi Sasabe, Mayumi Niitani, Satoshi Yamaga, and Nobuaki Shime contributed to data collection. Satoshi Yamaga, Mayumi Niitani, Tsuyoshi Kataoka, and Hiroyuki Sawatari contributed to analysing data/manuscript drafting. All authors contributed to the interpretation of data and critical revision of the manuscript for important intellectual content.

DATA AVAILABILITY STATEMENT

Data sharing not applicable.

ETHICS STATEMENT

The study protocol was approved by the Ethical Committee for Epidemiology of Hiroshima University.

INFORMED CONSENT

Opt-out statement had done since this study is retrospective study.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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