NIU Journal of Health Sciences Copyright©2026
Nexus International University ISSN: 3136-6449; 12(2) 197-203

Serum Albumin Levels and Risk of Pressure Injuries in Intensive Care Unit Patients: A Single-Center Prospective Cohort Study

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Abstract.

Background: Pressure ulcers (PUs) / pressure injuries are a significant complication that can occur and be prevented in intensive care units (ICUs). Given the common association between hypo-albuminemia and PU development, unadjusted associations are found to exaggerate its independent predictive potential by confounding with severity of illness and systemic inflammation. We prospectively studied the association of baseline/2-week serum albumin levels with PU incidence in a cohort of critically ill patients.
Methods: This is a STROBE-compliant, prospective observational study carried out in the Al-Hussain Teaching Hospital (Samawa, Iraq) from October 2023 until October 2025. Seventy patients aged 40–80 years with a ICU length of stay ≥ 30 days were included and. The serum albumin was assessed at admission and 2 weeks post-discharge using a standard BCG spectrophotometric assay (hypoalbuminemia < 3.5 g/dL). The primary outcome was development of stage 2+ PU during follow-up. Adjusted odds ratios (aOR) were determined using univariate tests and multivariable logistic regression.
Results: PUs occurred in 40/70 patients (57.1%), all <55–80 years of age. Highest incidence was observed in stroke/CVA (72.7%) and postoperative (60.0%) patients, while the lowest incidence was detected in pancreatitis (0.0%). Admission hypoalbuminemia (< 3.5 g/dL) was seen in 20 patients (28·6%) based on cut-off levels, whereas a mean of only 60 days after admission had hypoalbuminemia at two weeks to reflect an acute depletion trajectory among these patients with support for 1500 human risks [85.7%]. Hypoalbuminemia was present in 35/40 (87.5%) PU patients versus 5/30 (16.7%) non-PU patients at PU onset / 2-week follow-up (unadjusted OR 35·0,95% CI 9·2–133·9; p < 0·001). In multivariable logistic regression, controlling for age and admission category, low serum albumin remained a powerful independent predictor of PU development in the orthopedic group (aOR 18.4, 95% CI 3.8–89.2; p < 0.001). For PU, there was a complete separation between people who required non-oral tube feeding (100%) compared to those that did not (0%), indicating immobility and illness severity.
Conclusion: Hypoalbuminemia, an independent and powerful predictor of pressure ulcer risk in long-stay ICU patients. Incorporating standardized serum albumin tracking with clinical risk scales and early mobility protocols can advance PU risk stratification in critical care.

Keywords: Pressure ulcer, Pressure injury, Hypoalbuminemia, Serum albumin, Critical care, Intensive care unit, Risk stratification

1. Introduction

Background: Pressure ulcers (PUs), otherwise known as pressure injuries or bed sores, are defined as localized damage of the skin and underlying subcutaneous tissues arising from sustained pressure, shear forces and friction in

tissue over bony prominences. PU in intensive care units (ICUs) are one of the most common and severe preventable adverse events, with incidence ranging from 1% to >50% depending on population case mix, severity of illness, and diagnostic vigilance [1, 6]. PUs does cause physical harm, but they also prolong length of ICU stay, increase risk of secondary bacterial sepsis, hospital mortality and healthcare costs [1, 7].

The pathogenesis by which pressure injuries develop is a dynamic interplay of extrinsic (interface pressure, shear, microclimate) and intrinsic patient factors that decrease tissue tolerance [2]. Critically-ill patients, marked by extensive immobility, mechanical ventilation, deep sedation and peripheral vasoconstriction induced by high doses of vasopressors are especially prone to acute tissue hypoperfusion which can occur in the absence of load mitigation within hours [12].

Of metabolic and nutritional biomarkers, serum albumin has been studied extensively. Albumin represents the major factor of plasma colloid oncotic pressure. Profound decrease in serum albumin causes interstitial fluid redistribution and dermal edema, which increases diffusion distance for oxygen and metabolic substrates at the skin microvascular level [15]. In addition, hypoalbuminemia is important as a negative acute-phase reactant in time of systemic inflammation, it is both an index of metabolism and a strong surrogate for severity of critical illness [15, 16]. Although low serum albumin has consistently been associated with both PU development and impaired wound healing in clinical studies [13, 14], unadjusted observational studies often report extremely high effect sizes that likely represent confounding due to overall severity of illness and patient immobility.

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2. Methods

2.1 Study Design and Setting

Design: A two-year, prospective observational cohort study set in the Intensive Care Unit in Al-Hussain Teaching Hospital (Samawa, Al-Muthanna Governorate, Iraq) between October 2023 and October 2025. The protocol of this study has been approved by the Institutional Review Board of Al-Hussain Teaching Hospital (IRB Ref No: ATH-2023-0892). Patients, or their legally authorized representatives, provided written informed consent prior to enrollment. This manuscript was prepared according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

2.2 Study Population and Eligibility Criteria

Data were collected from seventy patients over 18 years old with an ICU admission between November 2016 and September 2020. Inclusion criteria were: (1) adult patients aged 40–80 years; (2) admission to the ICU because of any medical or surgical reason; and (3) ICU length of stay more than 30 days. Exclusions included patients with ICU stay≤ 30 days, transfer to another facility or death before 30-day of follow-up ICIU and the presence of existing pressure

injuries at admission. The final cohort included 40 postoperative surgical admissions and 30 medical admissions (22 stroke/cerebrovascular accident [CVA] and 8 acute pancreatitis patients).

2.3 Data collection and clinical variables

Inclusion criteria were prospective recording of demographic and clinical data at the time of admission such as age, sex, main diagnostic category on admission, height (cm), weight (kg), whole-body fat percentage (%) using Jackson-Pollock body composition equations and enteral/parenteral feeding route (non-oral packing tube vs oral feeding status). Non-oral feeding included total enteral nutrition through nasogastric/jejunal tubes complemented with total or partial parenteral nutrition. Certified critical care nurses performed daily physical skin examinations, which were verified by study investigators. Identification, location and categorization of PUs by the National Pressure injury advisory panel (NPIAP) clinical criteria [9] The main outcome measure was the development of a new stage 2 or higher pressure ulcer during follow-up.

2.4 Serum Albumin Assessment in the Laboratory

Venous blood was collected at two predetermined prospective time points: (1) Baseline admission (within 24 hours of ICU admission) and; (2) Follow-up at 14 days post-admission (or, at the exact time of PU diagnosis if prior to day 14). Serum albumin was measured with a standardized automated Bromocresol Green (BCG) dye-binding spectrophotometric colorimetric assay (Roche Cobas c501 analyzer, Roche Diagnostics, Mannheim, Germany). Hypoalbuminemia was defined quantitatively as a serum albumin concentration of < 3.5 g/dL (< 35 g/L), according to international laboratory standards. Serum albumin, both continuous-like and as a binary diagnostic classification (Normal ≥ 3.5 g/dL versus Reduced < 3.5 g/dL) were assessed in each study event on a random effect basis.

2.5 Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Categorical data were shown as absolute frequencies and percentages (n (%)). The Pearson Chi-Square (χ^2) test or Fisher's Exact test (depending on expected cell counts ≤ 5) were used for univariate comparisons between patients who developed PUs and those who did not. Primary exposure variables were analyzed by unadjusted Odds Ratios (OR), Relative Risks (RR) with 95% Confidence Intervals (95% CI).

A multivariable binary logistic regression model was created to isolate the independent predictive value of serum albumin and statistically control for critical demographic/clinical confounders. Analyses were adjusted for age (continuous) and admission diagnosis category (Postoperative vs Stroke vs Pancreatitis) and included serum albumin status (at follow-up/PU onset). Selection of variables was driven by biological plausibility and univariate significance. The feeding route variable (100% PU incidences in the nonoral fed group versus 0% in the oral fed group) could not be tested by parametric regression due to complete separation and it was assessed as a master clinical surrogate for immobility and illness severity. P values were considered statistically significant if <0.05 (two-sided).

3. Results

3.1 Baseline Characteristics

There were 70 ICU patients evaluated (45 males, 64.3% and 25 females, 35.7%) aged between 40–80 years old. Forty of those patients (57.1%) were postoperative surgical cases while the others included a total of 30 medical cases (42.9%), consisting of 22 stroke/CVA patients (31.4%) and eight acute pancreatitis patients (11.4%). In total, 30 patients (42.9%) were able to take nutrition orally and 40 patients (57.1%) required non-oral tube/parenteral feeding. Table 1 presents baseline characteristics.

Table 1: Baseline demographic and clinical characteristics of the study cohort (N = 70).

Baseline Characteristic	Sample Count (n)	Percentage (%)
Sex – Male	45	64.3%
Sex – Female	25	35.7%
Admission Category – Postoperative	40	57.1%
Admission Category – Cerebrovascular Accident (CVA)	22	31.4%
Admission Category – Acute Pancreatitis	8	11.4%
Feeding Route – Non-oral (Tube/Parenteral)	40	57.1%

3.2 The Overall Incidence and Subgroup Stratification

Forty of 70 (57.1%) patients developed pressure ulcers. Pus developed in the first 14 days after ICU admission exclusively among case patients aged 55 to 80 years. The incidence of PU between admission diagnoses was significantly different ($\chi^2 = 12.98$, df = 2, p= 0.002). The patients with highest incidence, as seen in Figure 1A along with the specific type of PU that developed are stroke/CVA (16/22, 72.7%), postoperative surgical patients (24/40:60.0%) while those admitted with acute pancreatitis had zero PU development (0/8, 0.0%) (Table 2 and Figure 1A).

Table 2: Pressure injuries incidence based on admission diagnosis category ($\chi^2 = 12.98$, p = 0.002).

Admission Diagnosis	Total Patients (n)	PUs Developed (n)	Incidence (%)
Cerebrovascular Accident (Stroke)	22	16	72.7%
Postoperative Surgical	40	24	60.0%
Acute Pancreatitis	8	0	0.0%
Total Cohort	70	40	57.1%

3.3 Incidence by Sex and Age

Example score of pressure ulcers in male patients 25/45 (55.6%) and female patients 15/25 (60.0%). There was no statistically significant difference regarding this (unadjusted OR 0.83, 95% CI 0.31–2.25; p = 0.72) (Table3). Out of the 40 patients involved, all were aged between 55 and 80 years confirming older age is a risk factor for cluster disease.

Table 3: Pressure injury incidence by patient gender (p = 0.72).

Sex Category	Total Patients (n)	PUs Developed (n)	Incidence (%)
Male	45	25	55.6%
Female	25	15	60.0%
Total Cohort	70	40	57.1%

3.4 Prediction of the Future Trends of Serum Albumin

At baseline admission, 50 (71·4%) patients had normal serum albumin (≥ 3·5 g/dL) and 20 (28·6%) presented with hypoalbuminemia (< 3·5 g/dL). All patients were stable at week 1:91 patients (88.6%) had a normal serum albumin (<3.5 g/dL), followed by a significant metabolic shift in week 2 of ICU stay, with reduced serum albumin observed in 60 patients (85.7%) vs10 patients (14.3%) who remained within the normal range (Table4). The acute depletion of circulating albumin that may occur during prolonged critical illness is illustrated in the longitudinal perspective shown in Figure 1B.

Table 4: Potential progression of serum albumin status between ICU admission and 2-week follow-up (N = 70).

Serum Albumin Category (< 3.5 g/dL)	Baseline Admission n (%)	2-Week Follow-up n (%)
Normal (≥ 3.5 g/dL)	50 (71.4%)	10 (14.3%)
Reduced / Hypoalbuminemia (< 3.5 g/dL)	20 (28.6%)	60 (85.7%)

Serum albumin and pressure injury (1)

Post-hoc analysis: Cross-tabulation of follow-up / PU evaluation serum albumin status with pressure injury onset Figure 1B shows that out of the 40 patients who developed PUs, 35 (87.5%) were found to have hypoalbuminemia (3.5 g/dL). In contrast, of the 30 PU-free patients, 25 (83.3%) had normal albumin levels and 5 (16.7%)

hypoalbuminemia ($\chi^2 = 35{\cdot}1$, $p < 0{\cdot}001$; Fisher's exact $p < 0{\cdot}001$) (Table √ in full-text PDF version for subscription link; Figure √ in full-text PDF). One in unadjusted univariate analysis gave an OR, 35.0 (95% CI 9.2–133.9; RR = 5.3).

The multivariable logistic regression model, adjusting for age and admission diagnosis, showed that hypoalbuminemia was an independent risk factor associated with PU development (aOR: 18.4 [95% CI 3.8–89.2; $p < 0.001$]).

Table 5: Cross-tabulation of follow-up serum albumin status and incident pressure injury ($\chi^2 = 35.1$, $p < 0.001$; Unadjusted OR = 35 & Multivariable aOR = 18.4).

Serum Albumin Status	PU Developed (n = 40)	No PU (n = 30)	Total Cohort (N = 70)
Reduced / Hypoalbuminemia (< 3.5 g/dL)	35 (87.5%)	5 (16.7%)	40 (57.1%)
Normal Albumin (≥ 3.5 g/dL)	5 (12.5%)	25 (83.3%)	30 (42.9%)
Total	40 (100.0%)	30 (100.0%)	70 (100.0%)

3.6 Feeding Path and Fractionation Function

Statistical separation was absolute as to feeding route: absolutely none of the 30 orally-fed patients developed a PU (0%); while all 40 patients requiring non-oral tube/parenteral nutrition did develop a PU (100%) (Fisher's exact p 3.5 g/dL [blue]) and hypoalbuminemia (<3.5 g/dL [red]) The line indicates the gender-based cut-off (denoted by dashed and dotted lines) Y-axis corresponds to Serum Albumin Distribution (%) The left column depicts baseline pathway to admission (− 28.6% compared with ED only, while the middle and right columns depict cross-tabulation of follow-up vs PU evaluation. The prevalence of hypoalbuminemia was significantly higher in PU patients compared to non-PU patients (87.5% vs 16.7%; OR unadjusted = 35.0, 95% CI 9.2–133.9; multivariable aOR = 18.4, 95% CI 3.8–89.2; p <.001).

4. Discussion

This is a prospective single-center study of the incidence of pressure injuries in critically ill ICU patients with a length of stay longer than 30 days; 57.1% of patients developed PIs. This residency is at the upper end of reported international benchmarks found in critical care literature (1%–56%) [1, 6, 18] and reflects a high-risk cohort of long-stay patients with a considerable burden of baseline organ impairment.

The strongest result of this exploration is the independent association between hypoalbuminemia (< 3.5 g/dL) and PU development. Unadjusted OR by univariate analysis was very high (unadj OR 35.0), but this remained highly significant in multivariable regression adjusted for age and primary diagnostic category (aOR 18.4, 95% CI 3.8–89.2; $p < 0.001$). This confirms that hypoalbuminemia is not simply a coincidental consequence of age or diagnostic group but rather an independent correlate of tissue failure.

PU susceptibility was highly influenced by the diagnostic category (Figure 1A); tissue degradation occurred at a markedly high rate in stroke/CVA patients (72.7%) but also in the major postoperative admissions group (60.0%). In addition, a visual summary of the decline over time in serum albumin level and its historical separation between patients with PU (87.5% hypoalbuminemia) and those without (16.7% hypoalbuminemia) pre-to-follow-up is encapsulated in figure 1B.

Pathophysiological Process underlying this relationship are explained by several interlocking mechanisms. Eighty percent of plasma oncotic force is made by albumin. Interstitial fluid leaks into tissue spaces and cause localized edema due to severe hypoalbuminemia. The swelling of the skin is likely to increase the distance diffusion processes need to travel via capillaries through tissue into dermal cells for oxygen and essential nutrients, thus reducing microvascular ischemia thresholds during mechanical loading [15]. Second, serum albumin synthesis decreases markedly during systemic inflammation (mediated primarily by pro-inflammatory cytokines IL-6 and TNF-α), which have been known to simultaneously promote protein catabolism and increase vascular permeability [15, 16]. Thus, low serum albumin represents an amalgam of malnutrition, chronic metabolic input and prominent systemic inflammation [13, 14, 22].

The statistically significant results for feeding route, where 100% of patients fed with tubes developed pressure ulcers (PUs) against none who were fed orally, should be interpreted cautiously in clinical practice. In this cohort the dependency on enteral feeding tube was taken as a surrogate for severe neurological impairment (i.e. longstanding ischemic stroke), mechanical ventilation, and complete immobility in bed state. Again, tube feeding identifies an ultra-high risk population requiring very thorough preventive care [22, 24, 25] rather than enteral nutrition being the cause of PUs.

4.1 Study Limitations

Several limitations should be acknowledged. First, sample size was small (N = 70) and confined to a single tertiary care site which limits generalizability. Second, although our data were rich in clinical content, formal clinical illness severity scores (eg APACHE II or SOFA) could not be systematically collected at all baseline timepoints for our cohort hence the exclusion of such variables from the regression model. Third, the tube-feeding variable was treated using a complete separation method that blocked its parametric entry into multivariable modeling. These findings should be validated in a multicenter prospective study using high frequency daily monitoring of albumin and detailed severity scoring.

5. Conclusion

Serum albumin < 3.5 g/dL is a strong independent risk factor for pressure injury in long-stay intensive care patients. Against this background we demonstrated how routine serum albumin trajectories have strong prognostic potential for distinguishing at-risk phenotypes — in this case older adults pre- and post-stroke, and peri-operative tube-fed surgery patients. Implementing dynamic monitoring of albumin together with a universal assessment strategy of risk may substantially reinforce prevention protocols in intensive units for pressure sores.

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