



ORIGINAL ARTICLE

Stress hyperglycemia ratio outperforms admission blood glucose in predicting in-hospital mortality among type 2 diabetes patients in emergency department

Melvin Andrean¹  and Nabil Muhtadi Falah² ¹Emergency Department, Cengkareng General Hospital, Jakarta, Indonesia²Department of Internal Medicine, Cengkareng General Hospital, Jakarta, Indonesia*** Correspondence Author:** melvinandrea@gmail.com

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ABSTRACT

BACKGROUND

Admission blood glucose is routinely used to assess diabetic patients in the emergency department (ED), but it cannot distinguish chronic from acute stress-induced hyperglycemia. The stress hyperglycemia ratio (SHR), derived by dividing admission glucose by the HbA1c-estimated average glucose, adjusts for the patient's chronic glycemic baseline and may more accurately capture acute metabolic stress. This study aimed to determine the discriminative performance of SHR versus admission blood glucose in predicting mortality among type 2 diabetes patients.

METHODS

A retrospective cohort study was conducted involving 200 adults with type 2 diabetes and admission glucose ≥ 200 mg/dL. The primary outcome was mortality. Secondary outcomes were ICU/HCU admission, composite adverse outcome, and prolonged hospital length of stay. Multivariable logistic regression and ROC curve analyses were performed to evaluate the independent associations and compare the discriminative ability of SHR and admission blood glucose using AUC.

RESULTS

Among 200 participants (mean age 57.6 ± 10.2 years; mean SHR 1.5 ± 0.59), 41 (20.5%) died and 50 (25.0%) experienced composite adverse outcomes. SHR was independently associated with mortality (AOR 3.25; 95%CI 1.44–7.32; $p=0.004$) and composite adverse outcome (AOR 3.21; 95%CI 1.45–7.10; $p=0.004$), while admission glucose was not significantly associated with any outcome. SHR outperformed admission glucose in discriminating mortality (AUC 0.734 vs 0.567) and composite adverse outcome (AUC 0.714 vs 0.542). Neither predictor significantly discriminated ICU/HCU admission or prolonged LOS.

CONCLUSION

SHR outperforms admission blood glucose in predicting mortality and adverse outcomes in hyperglycemic ED patients with type 2 diabetes. Routine SHR calculation at ED admission may support early risk stratification in high-risk populations.

Keywords: Admission blood glucose, diabetes mellitus, emergency department, outcomes, stress hyperglycemia ratio

INTRODUCTION

Hyperglycemia is one of the most frequently encountered metabolic disturbances in the emergency department (ED), occurring in both diabetic and non-diabetic patients across a wide spectrum of acute illnesses. Studies have reported that hyperglycemia at ED presentation affects approximately 21% of acutely ill patients.⁽¹⁾ In patients with diabetes mellitus, acute hyperglycemia is particularly prevalent and has been consistently associated with adverse clinical outcomes, including increased in-hospital mortality, higher rates of ICU admission, and prolonged hospital length of stay.^(1,2) Emergency departments represent the critical first point of contact for acutely ill patients with metabolic comorbidities; the ability to rapidly identify high-risk individuals at initial presentation carries significant implications for clinical decision-making, resource allocation, and patient safety. Early and accurate risk stratification is therefore essential in this setting.⁽³⁾

Despite its widespread use, admission blood glucose as a standalone prognostic marker carries important limitations in the diabetic population. A single blood glucose measurement at ED admission cannot differentiate between chronic baseline hyperglycemia reflecting long standing poor glycemic control, and acute stress related hyperglycemia driven by the physiological response to illness severity, two metabolically distinct phenomena with fundamentally different prognostic implications.⁽⁴⁾ In patients with type 2 diabetes and chronically elevated baseline blood glucose, a markedly elevated admission blood glucose may represent only a modest acute rise above habitual glycemic levels, thereby underestimating the true severity of the acute metabolic stress response.^(4,5) This fundamental limitation means that admission blood glucose may systematically misclassify the degree of acute metabolic derangement: overestimating risk in patients with chronic poor control and underestimating it in those with tighter long-term management.^(4,5)

The stress hyperglycemia ratio (SHR) was developed specifically to address this limitation by contextualizing acute blood glucose levels against the patient's own chronic glycemic baseline as reflected by glycated hemoglobin (HbA1c).^(4,6) The SHR is calculated as admission blood glucose (mg/dL) divided by $(28.7 \times \text{HbA1c} - 46.7)$, where

the denominator estimates the patient's long-term average glucose equivalent derived from HbA1c.⁽⁶⁾ A higher SHR reflects a greater degree of acute glucose elevation relative to the patient's own baseline, more accurately representing the acute metabolic stress burden than admission glucose alone.⁽⁴⁾ This conceptual advantage has been supported by growing evidence from critical care and cardiovascular medicine. Meta-analyses and large cohort studies have demonstrated that elevated SHR is independently associated with increased mortality in critically ill patients with sepsis, acute myocardial infarction, and atrial fibrillation.⁽⁷⁻⁹⁾ A systematic review and dose-response meta-analysis including 39 studies and over 100,000 patients further confirmed that SHR is an independent predictor of 30-day and one-year mortality across diverse critically ill populations.⁽¹⁰⁾

More recently, evidence has begun extending SHR evaluation to emergency department settings; Ou et al.⁽¹¹⁾ demonstrated that SHR independently predicted adverse outcomes in ED patients presenting with upper gastrointestinal bleeding, while Lai et al.⁽¹²⁾ confirmed that SHR outperformed glycemic variability indices in predicting in-hospital and one-year mortality among AMI patients with reduced ejection fraction, with effects more pronounced in non-diabetic patients. However, these studies evaluated specific emergency subpopulations — gastrointestinal bleeding and cardiac patients — rather than a broader general population of hyperglycemic type 2 diabetes patients presenting with diverse acute illnesses. In contrast to these prior studies, which evaluated SHR in ICU-based or specific emergency subpopulations without direct head-to-head comparison against admission blood glucose in a general hyperglycemic type 2 diabetes cohort, the present study uniquely addresses this gap by directly comparing the prognostic association and discriminative performance of SHR versus admission blood glucose across multiple clinical outcomes in a broad hyperglycemic type 2 diabetes population presenting in the emergency department.

Therefore, this study aimed to assess the association between SHR and admission glucose with key clinical outcomes, including mortality, ICU/HCU admission, composite adverse outcome, and hospital length of stay among hyperglycemic ED patients with type 2 diabetes and compare the discriminative performance of

SHR against admission glucose using ROC curve analysis.

METHODS

Research design

A retrospective observational cohort study was conducted at the Emergency Department of Cengkareng General Hospital, West Jakarta, Indonesia, covering the period from January to August 2025.

Study subjects

Adults aged ≥ 18 years with a confirmed diagnosis of type 2 diabetes mellitus and an admission blood glucose of ≥ 200 mg/dL presenting to the emergency department during the study period were eligible for inclusion. Patients were identified through a systematic review of the hospital electronic medical record (EMR) system. Total sampling was applied, all eligible patients presenting to the ED during the study period were included without further selection beyond the specified eligibility criteria, ensuring that all qualifying cases were captured without selection bias. Patients were excluded if they had a diagnosis of type 1 diabetes mellitus, a history of pancreatic disease affecting endogenous insulin secretion, chronic glucocorticoid therapy, active pregnancy, dialysis, and history of renal transplantation. Patients with missing HbA1c values within three months of admission were also excluded, as SHR calculation requires a valid concurrent or recent HbA1c measurement. The patient selection process is summarized in Figure 1.

Sample size determination

As this study was retrospective and post-hoc in design, formal a priori sample size calculation was not performed. The final cohort of 200 patients yielded 41 primary outcome events, corresponding to an events-per-variable (EPV) ratio of approximately 8.2 for the five-covariate multivariable model. Because this falls below the conventionally recommended EPV threshold of 10,⁽¹³⁾ the results should therefore be interpreted with this limitation in mind.

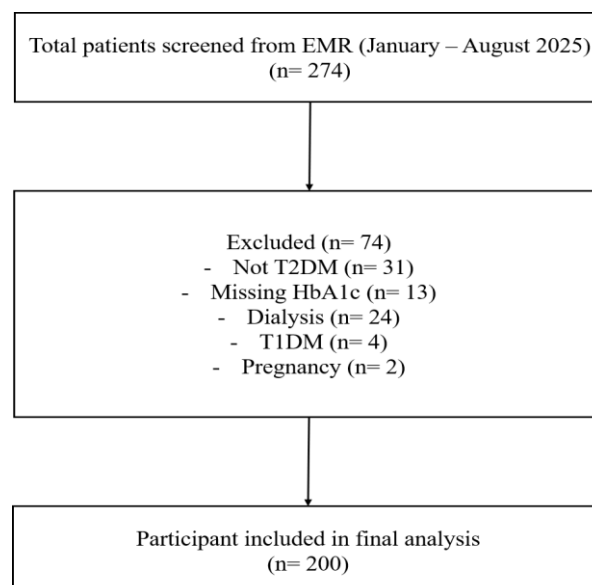
Measurements

All data were extracted retrospectively from the hospital EMR following ethics committee approval. The primary exposure variables were admission blood glucose (mg/dL), measured at the

time of ED presentation, and the stress hyperglycemia ratio (SHR), calculated as $SHR = \text{Admission Glucose} \div (28.7 \times \text{HbA1c} - 46.7)$.⁽⁶⁾ HbA1c values were obtained from measurements taken within three months prior to or at the time of ED admission, consistent with the approach used in prior SHR literature. Baseline clinical data extracted included age, sex, admission vital signs, National Early Warning Score 2 (NEWS2)⁽¹⁴⁾ at ED presentation, principal diagnosis, infection status, presence of diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic state (HHS), and documented comorbidities.

Study outcomes

The primary outcome of this study was in-hospital mortality, defined as death from any cause ascertained through systematic EMR review capturing in-hospital deaths. Secondary outcomes were: (i) ICU or high care unit (ICU/HCU) admission during the index hospitalization, (ii) composite adverse outcome, defined as the occurrence of death, ICU/HCU admission, or major in-hospital complication within the index admission, and (iii) prolonged hospital length of stay, defined as ≥ 5 days from the date of ED admission, the cut-off being made using the median LOS observed in the study cohort. The third outcome was the comparison of discriminative performance of SHR against that of admission glucose for each outcome using ROC curve analysis.



Note :T2DM: type 2 diabetes mellitus; T1DM: type 1 diabetes mellitus; HbA1c: glycated hemoglobin; EMR: electronic medical record

Figure 1. Flowchart of patients' selection

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics version 25.0. Categorical variables were summarized as frequencies and percentages. Continuous variables were presented as mean $\pm$ standard deviation (SD). For descriptive purposes, SHR was additionally categorized as high (≥ 1.14) or low (< 1.14) based on the optimal threshold to characterize the distribution of the study population.⁽⁴⁾

Multivariable binary logistic regression analyses were performed to evaluate the independent associations of SHR and admission blood glucose with each outcome, with adjustment for age, sex, infection status, presence of DKA or HHS, and NEWS2 score. Results were expressed as adjusted odds ratios (AOR) with 95% confidence intervals (CI). Receiver operating characteristic (ROC) curve analyses were subsequently performed to evaluate and compare the discriminative performance of SHR and admission blood glucose for each outcome. For individual AUC values, statistical significance was assessed against the null hypothesis of no discrimination (AUC=0.5). Differences in AUC between SHR and admission blood glucose were evaluated using DeLong's method for paired ROC curves. Statistical significance was set at $p < 0.05$ for all analyses. SHR and admission blood glucose were entered as continuous variables in all multivariable logistic regression and ROC curve analyses. For continuous predictors, results are expressed as AOR per 1-unit increase for SHR and NEWS2 score, and per 1 mg/dL increase for admission blood glucose.

Ethical Clearance

The study protocol was approved by the Ethics Committee of Cengkareng General Hospital (Approval No. 001/KEP/RSUDCGK/2025). Given the retrospective nature of this study and the use of de-identified medical record data, individual informed consent was waived in accordance with institutional policy.

RESULTS

Clinical characteristics and outcomes

A total of 200 patients were included in this study. The mean age of participants was 57.6 ± 10.2 years, and most participants were female (58.5%). The mean HbA1c of participants was 10.4 ± 2.80 %, and admission blood glucose of

participants was 352.5 ± 140.57 mg/dL. High SHR (≥ 1.14) was found in 140 patients (70%). During their hospitalization, 41 participants (20.5%) died. Admission to the intensive care unit or high care unit occurred in 34 (17.0%) patients, prolonged hospital length of stay (≥ 5 days) was observed in 125 (62.5%), and adverse outcome was observed in 50 (25.0%) patients. Baseline clinical characteristics and outcomes of this study are presented in Table 1.

Table 1. Characteristics and clinical features of study participants (n=200)

Characteristics	n (%)
Age (years)	57.6 ± 10.20
Gender	
Male	83 (41.5)
Female	117 (58.5)
Infection status	
Yes	79 (39.5)
No	121 (60.5)
DKA/HHS status	
Yes	13 (6.5)
No	187 (93.5)
NEWS2 score	3.3 ± 3.23
HbA1c (%)	10.4 ± 2.80
Admission blood glucose (mg/dL)	352.5 ± 140.57
SHR	1.5 ± 0.59
≥ 1.14	140 (70.0)
< 1.14	60 (30.0)
Mortality status	
Yes	41 (20.5)
No	159 (79.5)
Admission to ICU/HCU	
Yes	34 (17.0)
No	166 (83.0)
Length of stay (LOS)	
< 5 Days	75 (37.5)
≥ 5 Days	125 (62.5)
Adverse outcome	
Yes	50 (25.0)
No	150 (75.0)

Note: Data presented as n (%), except for age, NEWS2 score, HbA1c, and admission blood glucose as mean $\pm$ standard deviation; DKA/HHS: diabetic ketoacidosis/hyperosmolar hyperglycemic state; HbA1c: glycated hemoglobin/hemoglobin A1C; ICU/HCU: intensive care unit/high care unit; NEWS2: National Early Warning Score 2; SHR: stress hyperglycemia ratio.

Association of SHR and admission blood glucose with clinical outcomes

On multivariable logistic regression analysis, admission glucose was not associated with mortality, ICU/HCU admission, LOS ≥ 5 days, and adverse outcome after adjustment for age, sex, infection status, DKA/HHS status, and NEWS2

score. Although SHR was not associated with ICU/HCU admission and LOS ≥ 5 days, it was associated with mortality (adjusted OR 3.25, $p=0.004$) and adverse outcome (adjusted OR 3.21, $p = 0.004$). The results of the multivariable analysis are shown in Table 2.

Discriminative performance of SHR and admission blood glucose for clinical outcomes

To further evaluate the clinical utility of SHR, receiver operating characteristic (ROC) curve analyses were performed comparing the discriminative performance of SHR and admission glucose for each outcome. SHR demonstrated acceptable discriminative ability for mortality (AUC 0.734; 0.656 – 0.812; $p<0.01$), and composite adverse outcome (AUC 0.714; 0.639 – 0.789; $p<0.01$). SHR showed fair discriminative ability for ICU/HCU admission (AUC 0.679; 0.594 – 0.764; $p<0.01$) (Figure 2).

In contrast, admission blood glucose did not achieve statistically significant discrimination for any outcome: AUC 0.567 for mortality, 0.542 for composite adverse outcome, and 0.469 for ICU/HCU admission (all $p>0.05$). Neither SHR nor admission blood glucose demonstrated significant discriminative performance for prolonged hospital length of stay. Across all outcomes where discrimination was statistically significant, SHR consistently outperformed admission blood glucose. DeLong's method for paired ROC curves demonstrated that SHR had significantly superior discriminative performance compared to admission blood glucose for all three significant outcomes: mortality (DeLong $p=0.002$), ICU/HCU admission (DeLong $p<0.001$), and composite adverse outcome (DeLong $p<0.001$). These results are summarized in Table 3, and ROC curves for mortality, ICU/HCU admission, and composite adverse outcome are presented in Figures 2.

Table 2. Multiple logistic regression for association of admission blood glucose and SHR with clinical outcomes

Predictors	Mortality (AOR, 95% CI)	ICU/HCU admission (AOR, 95% CI)	LOS ≥ 5 days (AOR, 95% CI)	Adverse outcome (AOR, 95% CI)
Admission blood glucose	1.00 (1.00 – 1.01) $p = 0.227$	1.00 (1.00 – 1.01) $p = 0.376$	1.00 (1.00 – 1.01) $p = 0.681$	1.00 (1.00 – 1.01) $p = 0.377$
Continuous SHR index per 1 unit	3.25 (1.44 - 7.32) $p = 0.004$	1.60 (0.76 – 3.36) $p = 0.219$	1.00 (0.58 – 1.74) $p = 0.987$	3.21 (1.45 – 7.10) $p = 0.004$

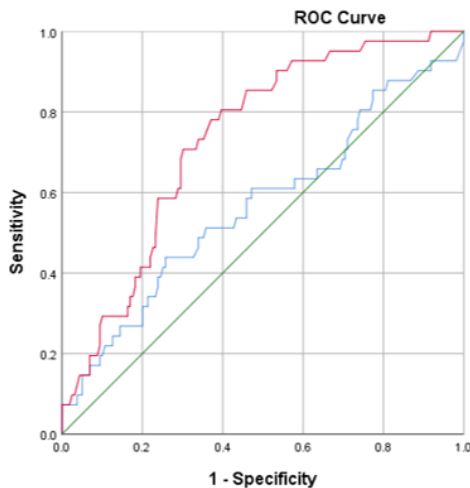
Note ; AOR: adjusted odds ratio; CI: confidence interval; ICU/HCU: intensive care unit/high care unit; LOS: length of stay; SHR: stress hyperglycemia ratio. *Adjusted by age, sex, infection status, DKA/HHS, and NEWS2 score

Table 3. Comparison of discriminative performance of AUC between SHR and admission blood glucose for clinical outcomes

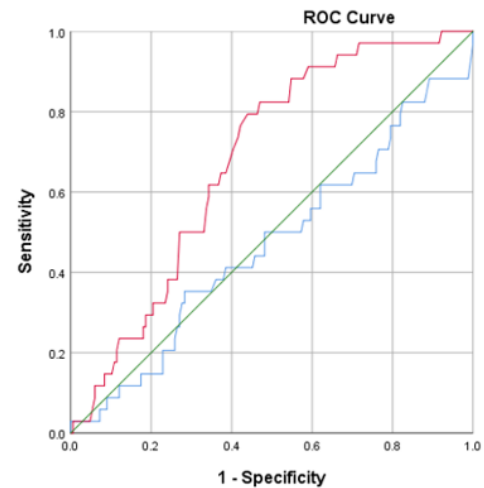
Outcome	Predictor	AUC	95% CI	p-value	DeLong p
Mortality	SHR	0.734	0.656 – 0.812	0.001	0.002
	Admission blood glucose	0.567	0.461 – 0.672	0.189	-
ICU/HCU Admission	SHR	0.679	0.594 – 0.764	0.001	<0.001
	Admission blood glucose	0.469	0.359 – 0.580	0.575	-
Adverse outcome	SHR	0.714	0.639 – 0.789	0.001	<0.001
	Admission blood glucose	0.542	0.444 – 0.639	0.379	-
LOS ≥ 5 days	SHR	0.518	0.436 – 0.601	0.664	-
	Admission blood glucose	0.511	0.429 – 0.594	0.786	-

Note ; AUC: area under the curve; CI: confidence interval; DeLong p: p-value from DeLong's method for paired ROC curve comparison; ICU/HCU: intensive care unit/high care unit; LOS: length of stay; SHR: stress hyperglycemia ratio; —: not applicable.

(A). Mortality



(B). ICU/HCU Admission



(C). Composite Adverse Outcome

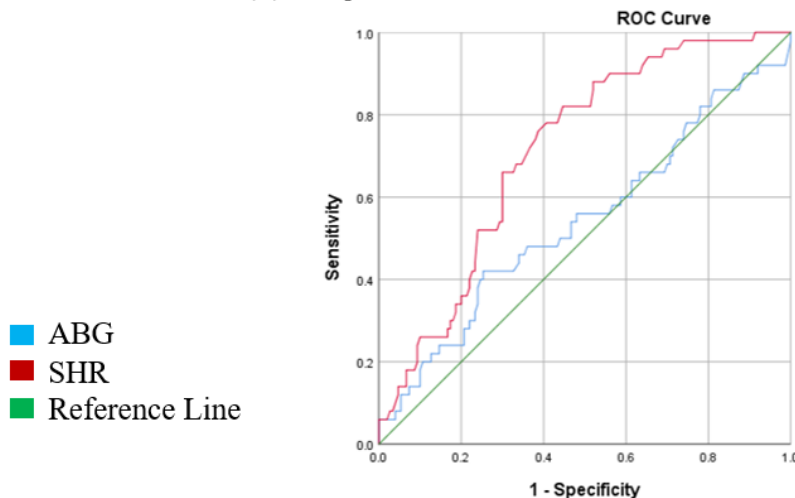


Figure 2. Receiver operating characteristic (ROC) curves comparing discriminative performance of stress hyperglycemia ratio (SHR) and admission blood glucose (ABG) for clinical outcomes. Panel A: Mortality — SHR AUC 0.734 (95% CI 0.656–0.812) vs admission blood glucose AUC 0.567 (95% CI 0.461–0.672), DeLong $p=0.002$. Panel B: ICU/HCU admission — SHR AUC 0.679 (95% CI 0.594–0.764) vs admission blood glucose AUC 0.469 (95% CI 0.359–0.580), DeLong $p<0.001$. Panel C: Composite adverse outcome — SHR AUC 0.714 (95% CI 0.639–0.789) vs admission blood glucose AUC 0.542 (95% CI 0.444–0.639), DeLong $p<0.001$.

DeLong's method was used to compare paired ROC curves

DISCUSSION

This retrospective cohort study evaluated the comparative utility of the stress hyperglycemia ratio and admission blood glucose for predicting clinical outcomes in 200 hyperglycemic ED patients with type 2 diabetes. On multivariable logistic regression, SHR was independently associated with mortality (AOR 3.25; 95% CI 1.44–7.32) and composite adverse outcome (AOR 3.21; 95% CI 1.45–7.10) after adjustment for age,

sex, infection status, DKA/HHS, and NEWS2 score. In contrast, SHR was not significantly associated with ICU/HCU admission (AOR 1.60; 95% CI 0.76–3.36) or prolonged length of stay (AOR 1.00; 95% CI 0.58–1.74). Importantly, admission blood glucose demonstrated no significant association with any of the four outcomes following multivariable adjustment. ROC curve analysis further confirmed the superiority of SHR over admission blood glucose in terms of discriminative performance, 0.734

versus 0.567 for mortality, 0.714 versus 0.542 for composite adverse outcome, and 0.679 versus 0.469 for ICU/HCU admission, a difference that reached statistical significance on DeLong's paired comparison for each endpoint. Neither predictor showed significant discriminative ability for prolonged LOS. These findings showed that SHR more accurately captures the prognostic significance of acute glycemic stress than admission blood glucose alone in this population.

The superior prognostic performance of SHR over admission blood glucose can be explained by its underlying physiological rationale. In acute illness, stress hyperglycemia arises from the coordinated release of counter-regulatory hormones including cortisol, catecholamines, glucagon, and growth hormone in response to tissue injury, infection, and systemic inflammation.^(4,15) These hormones promote hepatic gluconeogenesis and glycogenolysis while simultaneously inducing peripheral insulin resistance, resulting in a rapid elevation of blood glucose above the patient's own chronic baseline.^(4,15) This acute glucose surge is quantitatively distinct from the chronic hyperglycemia driven by progressive beta-cell dysfunction and insulin resistance that characterizes poorly controlled type 2 diabetes. The SHR directly captures this distinction by expressing admission blood glucose as a ratio relative to the patient's long-term glycemic average as estimated from HbA1c, thereby providing a more accurate representation of the acute metabolic stress burden than admission blood glucose in isolation.^(4,6) A patient with a high SHR has a glucose level that is disproportionately elevated relative to their own baseline, signaling a more intense acute stress response and, by inference, a more severe physiological derangement, an informational advantage that the absolute glucose value alone cannot provide.⁽⁴⁾

The findings of this study are broadly consistent with the existing literature on SHR in critically ill and acutely ill populations. Yan et al.⁽⁷⁾ demonstrated in a retrospective cohort of critically ill patients with sepsis that higher SHR quartiles were significantly associated with increased 28-day all-cause mortality and in-hospital mortality. Similarly, Cheng et al.⁽⁸⁾ reported that elevated SHR was independently associated with all-cause mortality at 30, 90, 180, and 365 days in critically ill patients with atrial fibrillation. At the meta-analytic level, Chen et al.⁽¹⁰⁾ synthesized 39 studies encompassing over

100,000 patients and found SHR to be a significant independent predictor of 30-day and one-year mortality across diverse critically ill populations, with a dose-response relationship further supporting its prognostic validity. A recent systematic review by Song et al.⁽⁴⁾ similarly confirmed SHR as a broadly applicable and practical index across multiple critical illness subtypes. Similar findings have since been extended to acute coronary syndrome,⁽¹⁶⁾ chronic kidney disease,⁽¹⁷⁾ acute ischemic stroke,⁽¹⁸⁾ and myocardial infarction cohorts,⁽¹⁹⁾ collectively broadening the evidence base for SHR as a prognostic index. Notably, all of these studies were conducted in ICU-based or specialized cardiac populations and direct evidence from emergency department settings remains scarce, making the present findings a distinct contribution to the literature.

The absence of a significant association between SHR and ICU/HCU admission should be interpreted carefully and should not be regarded as a failure of the biomarker. This finding likely reflects the multifactorial nature of ICU admission decisions, as it is a complex clinical decision influenced by multiple factors beyond the patient's baseline metabolic profile, including acute illness severity, anticipated benefit from intensive care, resource availability, clinician judgment, and patient or family preferences.⁽²⁰⁾ In resource-limited tertiary care settings, critical care bed allocation decisions frequently reflect capacity constraints as much as clinical necessity, meaning that patients at high physiological risk may be managed in general wards due to non-availability of intensive care resources. This systematic disconnect between underlying risk and actual ICU disposition is well recognized in observational studies of acute care populations and represents a particular challenge in studies using ICU admission as a binary outcome variable. The fact that SHR nonetheless demonstrated a fair AUC of 0.679 for ICU/HCU admission in ROC analysis, despite non-significance in the regression model, further supports the view that SHR carries meaningful discriminative information for this endpoint, but that non-clinical factors in the admission decision introduce substantial noise in observational data.

The absence of a significant association between SHR and prolonged hospital length of stay in the present study contrasts with findings from some prior investigations. Tian et al.⁽²¹⁾ found that patients with higher SHR had longer

ICU and hospital stays in critically ill patients in non-resuscitation ICU, and Shen et al.⁽²²⁾ similarly reported a significant positive association between SHR and LOS in a large cohort of acute stroke patients. Several reasons may account for this divergence. Unlike mortality, binary endpoints are largely driven by disease severity. LOS reflects a mixture of clinical and non-clinical factors including discharge planning, bed availability, social circumstances, and local institutional policies, which in many healthcare settings influence the timing of discharge as much as clinical status does. In a diagnostically heterogeneous ED population such as ours, this variability is likely more pronounced than in the clinically homogeneous populations studied by Tian et al.⁽²¹⁾ and Shen et al.⁽²²⁾ A further consideration is survivorship, patients with the highest SHR face the greatest mortality risk, and early in-hospital deaths in this group would reduce their overall LOS, potentially masking an underlying association in logistic regression analysis. Finally, the ≥ 5 -day LOS threshold in this study was defined at the observed median, a practical choice, but not a clinically validated cut-point and may not have been sensitive enough to detect a meaningful signal in this setting.

The clinical implications of this study are directly relevant to emergency department practice in Indonesia and analogous resource-limited settings. SHR is a simple, inexpensive, and rapidly calculable index requiring only two routine laboratory values, admission blood glucose and HbA1c,⁽⁴⁾ both of which are mostly available in most Indonesian referral hospitals. This makes SHR a practical prognostic tool that can be integrated into ED risk stratification workflows within minutes, without adding meaningful operational burden.⁽⁴⁾ Early warning tools such as NEWS2 are already established as valuable frameworks for risk stratification in the emergency department,⁽²³⁾ and SHR may represent a complementary glycemic parameter that could augment such existing frameworks for hyperglycemic diabetic patients specifically. In resource-constrained settings where bedside tools must balance simplicity with clinical utility, SHR represents a practical candidate for supporting early risk stratification, potentially informing timely goals-of-care discussions, decisions regarding specialist consultation, and allocation of monitoring resources in a clinically meaningful way.

Several limitations of this study should be acknowledged. First, the retrospective design is subject to inherent limitations including incomplete data capture and the potential for unmeasured confounding key clinical variables such as specific comorbidity burden, prior antidiabetic therapy, functional status, and nutritional markers were not available for inclusion in the multivariable models. Second, the study was conducted at a single tertiary referral hospital in West Jakarta, limiting the generalizability of findings to other Indonesian healthcare settings, particularly rural or primary care environments. Third, the study population was restricted to type 2 diabetic patients with significant hyperglycemia (≥ 200 mg/dL), while this is ensuring the internal validity of the SHR calculation, it precludes generalization to non-diabetic patients or those with lower levels of hyperglycemia. Fourth, with 41 mortality events and five covariates in the multivariable model, the events-per-variable ratio was approximately 8.2, approaching but not meeting the conventionally recommended threshold of 10. Therefore, the results should be interpreted with this limitation in mind. Finally, HbA1c values were drawn from records within three months prior to admission, which may not perfectly reflect the glycemic state at the exact time of presentation, although this approach is standard in the SHR literature.

Future studies should address the limitations of this work through multicenter prospective designs with larger, more diverse patient populations across Indonesian healthcare settings. Validation of optimal SHR cut-off values specific to the emergency hyperglycemic diabetic population, using prospectively collected data, would be particularly valuable for clinical translation. Studies incorporating glycemic variability indices alongside SHR may further refine early risk stratification in this group.

CONCLUSION

This study demonstrates that the stress hyperglycemia ratio is a superior predictor of mortality and composite adverse outcomes compared with admission blood glucose alone in hyperglycemic ED patients with type 2 diabetes. The mechanistic basis for this superiority lies in SHR's capacity to contextualize acute glucose elevation against the patient's own chronic glycemic baseline, making it more accurately capture the intensity of the acute stress response.

These findings constitute, to our knowledge, the first evidence supporting SHR utility in an Indonesian emergency department population, and suggest that routine SHR calculation at ED admission may meaningfully enhance early risk stratification in this high-risk group. Multicenter prospective studies with larger, more diverse samples and complete covariate data are needed to validate these findings and to establish optimal SHR thresholds for clinical decision support in Indonesian and Southeast Asian emergency care settings.

Conflict of Interest

No relevant disclosures.

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Authors' Contributions

MA contributed to conceptualization, data collection, statistical analyses, methodology, statistical methods, data interpretation, and manuscript writing. NBF contributed to supervised data collection, and manuscript supervision. All authors had read and approved the final manuscript.

Data Availability Statement

The data supporting the findings of this study are accessible from the corresponding author upon reasonable request.

Declaration of AI Usage in Scientific Writing

The authors used AI-assisted writing tools (Claude, Anthropic) to assist with manuscript drafting, structural editing, and reference verification. All scientific content, data, interpretations, and conclusions were reviewed, verified, and approved by the authors. The authors take full responsibility for the integrity and accuracy of the entire manuscript.

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