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Improving the Reliability of Continuous Glucose Monitoring in Critically Ill Patients Using AI-Assisted Calibration

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Key Words

Continuous glucose monitoring; Critically ill patients; Glucose variability; Model-based calibration

Abstract:

Background

Dysglycemia and increased glucose variability are common in critically ill patients and are associated with adverse outcomes. Continuous glucose monitoring (CGM) offers real-time assessment of glucose trends but its reliability in the intensive care unit (ICU) remains uncertain. We therefore evaluated whether a model-based calibration strategy could improve the accuracy, agreement, and stability of CGM measurements in critically ill patients.

Methods

In this single-center prospective observational study, adult ICU patients with paired point-of-care fingerstick glucose and synchronous CGM measurements were enrolled. A temporal calibration model combining liquid time-constant networks and gated recurrent units was applied to correct raw CGM signals, incorporating key clinical features including continuous renal replacement therapy (CRRT), vasoactive drug use, diabetes, and surgical status. CGM performance before and after calibration was assessed using mean absolute error (MAE), root mean square error (RMSE), mean absolute relative difference (MARD), Bland–Altman analysis, and glycemic variability indices. Subgroup analyses were conducted to explore factors influencing calibration performance.

Results

One hundred critically ill patients were included. Calibration markedly improved CGM accuracy, with MAE decreasing from 1.87 to 1.13 mmol/L, RMSE from 2.29 to 1.53 mmol/L, and MARD from 21.1% to 12.7%. Bland–Altman analysis after calibration showed minimal bias (−0.03 mmol/L) with 95% limits of agreement from −3.05 to 2.98 mmol/L. Glycemic stability improved following calibration, with reductions in standard deviation, coefficient of variation, and mean amplitude of glycemic excursions. Calibration benefits were consistent across clinical subgroups, although CRRT and vasoactive drug use contributed modestly to residual error.

Conclusions

Model-based calibration significantly improved the accuracy, agreement, and stability of CGM in critically ill patients. The calibrated CGM signal demonstrated clinically acceptable performance for continuous glucose trend monitoring under complex ICU conditions. Calibrated CGM may serve as a reliable continuous glycemic signal, complementing intermittent measurements by providing dynamic glucose information and potentially reducing reliance on intermittent monitoring.

Introduction

Critically ill patients in ICU frequently experience profound metabolic disturbances, among which dysglycemia and glucose variability are common and clinically relevant[1]. Abnormal glucose levels and increased glycemic variability have been consistently associated with adverse outcomes in ICU, including increased morbidity and mortality[2, 3]. Accurate and timely assessment of blood glucose is therefore an essential component of metabolic monitoring in critically ill patients.

In current clinical practice, blood glucose monitoring in the ICU primarily relies on intermittent point-of-care fingerstick measurements or arterial blood sampling[4-6]. Although these methods are widely used and clinically accepted, they are inherently intermittent and invasive, and may be insufficient to capture rapid glucose fluctuations in critically ill patients with unstable metabolic states[6-8]. Frequent sampling also increases nursing workload and patient discomfort, highlighting the need for alternative monitoring strategies that provide more comprehensive glycemic information.

Continuous glucose monitoring (CGM) offers the potential advantage of providing real-time, continuous glucose data without the need for repeated blood sampling.

CGM has been widely adopted in outpatient diabetes management and in non-critical care settings, where it has demonstrated the ability to detect early glucose trends and variability[9-11]. However, its application in the ICU remains limited. In critically ill patients, factors such as vasopressor use, microcirculatory dysfunction, and organ support therapies may compromise CGM accuracy and reliability. As a result, the accuracy, agreement with standard glucose measurements, and stability of CGM under ICU conditions have not been fully established, which limits its clinical applicability in this setting[11-14].

To address these limitations, we evaluated whether a model-based calibration approach could improve the accuracy, agreement, and stability of CGM in critically ill patients. In this single-center prospective observational study, we applied a temporal calibration model to correct CGM signals using paired point-of-care glucose measurements as reference. We further explored clinical factors influencing calibration performance through subgroup analyses, aiming to provide evidence to support the use of CGM for more reliable glycemic monitoring in the ICU。

Methods

Research Objects and Data Sources

This study collected data from blood glucose monitoring records and clinical records of patients admitted to ICU, including three types of core data. CGM device used was the Abbott FreeStyle Libre H continuous glucose monitoring system:

- Baseline clinical data of patients: including demographic information (age, sex), diagnosis, and clinical status (CRRT use, vasoactive drug use, diabetes history, and surgical status).

- Paired fingertip blood glucose and CGM data: including multi-time point fingertip blood glucose values (peripheral blood glucose) and corresponding synchronous CGM values, recording key information such as wearing time, monitoring time, and placement site;
- CGM data: including continuous blood glucose measurements of patients and corresponding timestamps, completely recording the dynamic changes of blood glucose over time.

The inclusion criteria for research objects were: 1. having complete paired fingertip blood glucose and CGM records; 2. complete clinical baseline characteristics (including CRRT use status, vasoactive drug use, diabetes history, surgical status). Finally, the included samples covered different clinical subgroups to ensure the generalizability of the results.

Data Preprocessing

A multi-source data fusion and cleaning process was designed for the heterogeneity of the three types of data, with specific steps as follows:

Multi-source Data Parsing and Merging

Using the patient’s name as the key for association, merge the baseline clinical data of patients, paired fingertip blood glucose and synchronous CGM data, and continuous CGM data. Deduplicate based on timestamps (removing duplicate records at the same time point), sort in ascending order of time, and construct an integrated "clinical features-continuous blood glucose-fingertip blood glucose" monitoring sequence for each patient.

Data Cleaning and Feature Extraction

- Outlier filtering: Deleting samples with invalid timestamps, missing key clinical features or blood glucose values;

- Temporal feature engineering: Calculate the number of hours (Hours) of each record relative to the start time of CGM wearing as the temporal input feature of the model; encode the patient's baseline subgroup features (CRRT status, vasoactive drug use, diabetes history, surgical status) as numerical variables (Yes=1, No=0, Unknown=0.5);

- Sequence sample construction: Using two consecutive fingertip blood glucose measurements as the time window, intercept the CGM sequence during this period to form training sample pairs of "input CGM sequence-target fingertip blood glucose value", and simultaneously associate the patient's clinical baseline features during the corresponding period to ensure the model learns the mapping relationship between continuous CGM, clinical features and single-point fingertip blood glucose.

CGM Blood Glucose Calibration Model

CGM signals were treated as physiological time-series signals requiring calibration under dynamic ICU conditions. A CGM calibration model integrating long-term temporal dependence and subgroup feature influence was designed, consisting of two parts: a core calibration network and a subgroup feature influence weight network.

Core Calibration Network (LTC-GRU)

Aiming at the nonlinearity and temporal dependence of blood glucose fluctuations in ICU patients, a hybrid structure of Liquid Time-constant (LTC) network and Gated Recurrent Unit (GRU) was adopted:

- LTC Cell: Captures the dynamic temporal characteristics of blood glucose changes, modeling the decay and feedback of blood glucose over time through learnable parameters. The calculation formulas are as follows:

$$\tau = \tau_{\min} + \mathrm{softplus}(\tau_{\mathrm{raw}})$$

$$A = \text{sigmoid}(A_{\text{raw}})$$

$$\tau_{\text{inv}} = 1/(\tau + 10^{-8})$$

$$dh = -h_{\text{prev}} \cdot \tau_{\text{inv}} + A \cdot W_{\text{rec}}(h_{\text{prev}})$$

$$h_{\text{new}} = h_{\text{prev}} + dh \cdot dt$$

- Where $\tau_{\min} = 10^{-3}$ is the lower limit of the time constant, dt is the time interval between adjacent records, W_{rec} is the weight matrix of the recurrent layer, and h_{prev} is the hidden state at the previous moment;

- GRU Cell: Fuses the dynamic features output by the LTC, capturing long-term sequential dependencies and mitigating the limitation of LTC in modeling long sequences;

- Output layer: Concatenates the hidden states of LTC and GRU, and maps them to calibrated blood glucose values through a fully connected layer to achieve full fusion of multiple features.

Subgroup Feature Influence Weight Network

To quantify the influence of clinical subgroup features on calibration accuracy, a single-layer linear weight network was constructed:

- Input: 4-dimensional subgroup feature vector (CRRT status, vasoactive drug use, diabetes history, surgical status);

- Output: Contribution coefficient of subgroup features to calibration error. The absolute value of the weight reflects the significance of the feature's influence, and the sign indicates the direction of influence (positive for increasing error, negative for decreasing error);

- Training objective: Minimize the mean squared error (MSE) between the output coefficient and the actual relative calibration error ($|true\ value -$

$calibrated\ value|/true\ value$) to ensure the weight is consistent with the actual clinical influence.

Evaluation Index System

A multi-dimensional evaluation system was constructed, covering accuracy, clinical applicability, stability, and subgroup specificity:

Core Accuracy Indicators

- Mean Absolute Error (MAE): Measures the average absolute deviation between predicted values and true values, with the formula:

$$MAE = \frac{1}{N}\sum_{i=1}^{N} |\, y_i - \hat{y}_i|$$

- Root Mean Square Error (RMSE): Amplifies the impact of larger errors, with the formula:

$$RMSE = \sqrt{\frac{1}{N}\sum_{i=1}^{N} (\, y_i - \hat{y}_i)^2}$$

- Mean Absolute Relative Difference (MARD): Eliminates the influence of differences in blood glucose levels, with the formula:

$$MARD = \frac{1}{N}\sum_{i=1}^{N} \frac{|y_i - \hat{y}_i|}{y_i} \times 100\%$$

- Correlation Coefficients: Calculate Pearson correlation coefficient (linear correlation) and Spearman rank correlation coefficient (nonlinear correlation) to measure the correlation strength between calibrated values and true values.

Clinical Applicability Evaluation

Bland-Altman analysis was used to evaluate the consistency between calibrated CGM values and fingertip blood glucose: calculate the bias (mean of predicted values minus true values), standard deviation of bias, and 95% confidence interval ($bias \pm 1.96 \times standard\ deviation\ of\ bias$) to reflect the systematic error and dispersion between the two.

Blood Glucose Fluctuation and Stability Evaluation

- Standard Deviation (SD): Standard deviation of blood glucose calibration errors, reflecting the dispersion of errors;
- Coefficient of Variation (CV): Eliminates the interference of blood glucose levels on fluctuation evaluation, with the formula:

$$CV = \frac{SD}{\text{mean of true values}} \times 100\%$$

- Mean Amplitude of Glycemic Excursions (MAGE): Identifies peak-valley fluctuations exceeding the standard deviation of the mean blood glucose, and calculates their average amplitude to reflect the severity of blood glucose fluctuations.

Subgroup Analysis

Patients were divided into subgroups according to CRRT status, vasoactive drug use, diabetes history, and surgical status. Core accuracy indicators and clinical applicability indicators were calculated for each subgroup to analyze the performance differences of the model in different clinical scenarios.

Results

A total of 100 participants were enrolled in the calibration study. Baseline demographic and clinical characteristics are presented in Table 1. The mean age was 62.7 ± 16.0 years, and 64.0% (64/100) of participants were male. The mean BMI was 22.9 ± 4.3 kg/m². CRRT was used in 19.0% of participants, 18.0% were postoperative, and 22.0% had diabetes. Vasopressors were administered in 80.0% of participants at the time of monitoring. The mean APACHE II score was 16.0 ± 6.1 (Table 1).

Characteristic	Value
Number of participants	N=100
Age, years	62.7±16.0
Gender	
Male	64.0% (64)
Female	36.0% (36)
BMI (kg/m²)	22.9±4.3
CRRT use	19.0% (19)
Surgical status	18.0% (18)
Diabetes	22.0% (22)
Vasopressor use	80.0% (80)
APACHE II Score	16.0±6.1

Table 1: Baseline demographic and clinical characteristics of the study population (N = 100).

Accuracy improvement after calibration

Calibration reduced absolute measurement error relative to raw CGM values. MAE, RMSE and MARD decreased from 1.873 mmol/L, 2.286 mmol/L and 21.08% to 1.125 mmol/L, 1.534 mmol/L and 12.72%, respectively. The relative reduction in MARD following calibration was 39.63%. Figure 1 illustrates the calibration

workflow and the improvement in CGM measurement accuracy after calibration （Fig 1）.

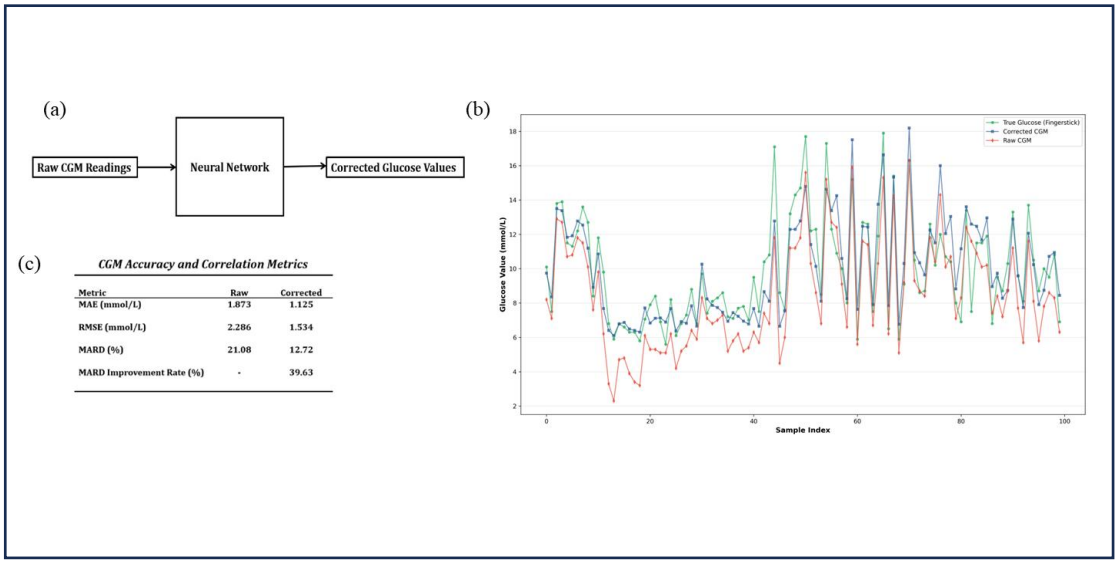


Figure 1. Neural network–based calibration of CGM measurements. Neural network–based calibration of continuous glucose monitoring (CGM). (a) Schematic workflow illustrating the calibration process in which raw CGM readings are processed by a neural network using fingerstick glucose measurements as reference to generate corrected glucose values. (b) Temporal comparison of fingerstick glucose, raw CGM readings, and neural network–corrected CGM values. (c) Comparison of glucose measurement accuracy metrics between raw and corrected CGM, including mean absolute error (MAE), root mean square error (RMSE), mean absolute relative difference (MARD), and MARD improvement rate.

Agreement between calibrated CGM and fingerstick glucose

Agreement analyses demonstrated reduced measurement deviation following calibration. The bias between calibrated CGM and reference glucose was −0.034 mmol/L with SD 1.53 mmol/L. The 95% limits of agreement ranged from −3.05 to 2.98 mmol/L (Fig 2). Bland–Altman plots of calibrated CGM versus reference values are shown in Fig 2a.

Stability of corrected glucose values

Glucose variability decreased following calibration. SD was reduced from 2.29 mmol/L to 1.53 mmol/L, CV from 24.97% to 16.76%, and MAGE from 6.26 mmol/L to 5.77 mmol/L (Fig 2).

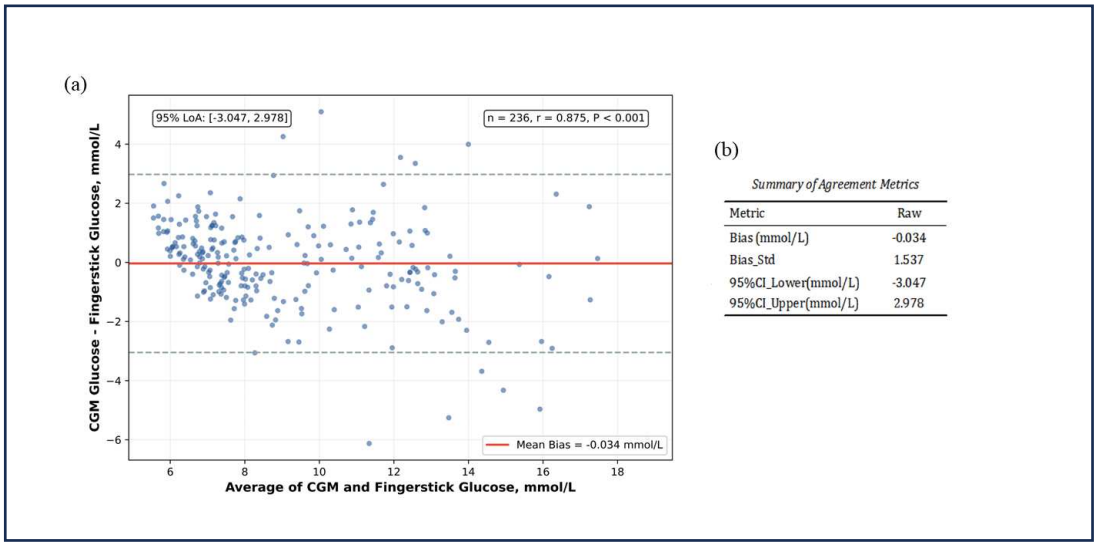


Figure 2. Agreement analysis and glycemic variability comparison between neural network‐corrected CGM and fingerstick glucose measurements. (a) Bland‐Altman plot showing the agreement between corrected CGM values and fingerstick glucose measurements. The solid line represents the mean bias, and the dashed lines indicate the 95% limits of agreement. (b) Summary of agreement statistics corresponding to the Bland‐Altman analysis, including mean bias, standard deviation of the bias, and 95% limits of agreement. (c) Comparison of glycemic variability metrics between raw and corrected CGM measurements, including SD, CV, and MAGE.

Subgroup analyses

Calibration consistently reduced MARD across all predefined clinical subgroups (Fig 3). After calibration, MARD values ranged from 12.2% to 14.7% across CRRT, vasoactive drug use, diabetes, and surgical status subgroups, compared with 19.3% to 22.3% for raw CGM signals. Although calibration benefits were observed in all subgroups, residual error contributions differed by clinical condition. Model-derived feature weights indicated higher residual error influence from CRRT status

(absolute weight 0.042) and vasoactive drug use (absolute weight 0.036), whereas diabetes and surgical status showed smaller contributions.

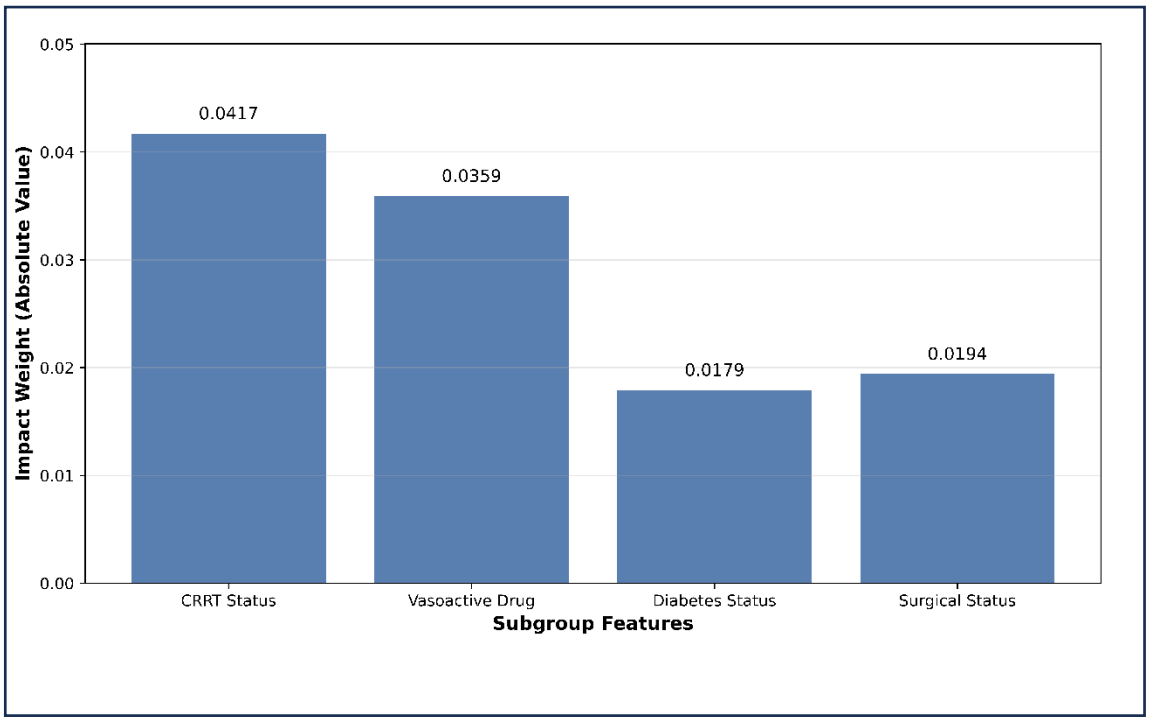


Figure 3. Impact of clinical subgroup characteristics on CGM calibration performance. Bar chart illustrating the absolute impact weights of selected clinical variables on calibration accuracy. Features include CRRT status, vasopressor use, diabetes status, and surgical status. Larger values indicate greater influence on CGM measurement accuracy.

Discussion

This study demonstrates that model-based calibration significantly improves the accuracy, agreement, and stability of continuous glucose monitoring in critically ill patients. Uncalibrated CGM exhibited clinically meaningful deviations from reference measurements, whereas calibration substantially reduced measurement error and enhanced signal reliability. These findings suggest that key limitations of CGM performance in the ICU may be partially mitigated through calibration,

supporting more reliable use of CGM for glycemic monitoring in critically ill patients.

In our ICU cohort, model-based calibration substantially reduced CGM measurement error, with MARD decreasing from 21.1% to 12.7% and MAE from 1.87 to 1.13 mmol/L (approximately 40% reduction). Post-calibration MARD fell within the clinically acceptable range (10–15%) for continuous glucose trend monitoring, consistent with previous studies in hospitalized and critically ill populations[15, 16]. These improvements indicate that calibrated CGM can achieve a level of accuracy suitable for clinically meaningful glucose trend assessment. Reduced absolute error and narrower limits of agreement further suggest a lower risk of clinically significant measurement deviation. The observed improvement may be partly explained by the model's ability to capture temporal dynamics and incorporate relevant clinical features, thereby reducing signal drift under complex ICU conditions. Together, these findings support the use of model-based calibration to enhance the reliability of CGM measurements and facilitate more robust glucose monitoring in critically ill patients.

The post-calibration Bland–Altman analysis demonstrated negligible mean bias (≈0 mmol/L) with 95% limits of agreement (LoA) of approximately −3.0 to 3.0 mmol/L. These limits indicate that, for most measurements, deviations remained within a clinically acceptable range for trend-based monitoring in the ICU, consistent with recent CGM validation studies reporting similarly narrow biases but broad LoA bands in critically ill cohorts[17, 18]. The persistent LoA width likely reflects physiological rather than algorithmic limitations. Vasopressor-induced peripheral vasoconstriction and microcirculatory shunting can impair subcutaneous perfusion, increasing interstitial – blood glucose gradients and widening CGM – reference discrepancies. Furthermore, rapid glycemic variations

driven by insulin titration, fluctuating nutrition, and acute stress dysregulation accentuate the intrinsic ISF – blood glucose lag[19], producing transient deviations despite accurate overall trajectories. Therefore, although calibration improved agreement, CGM in the ICU may remain more suitable for continuous trend surveillance and detection of large excursions rather than definitive replacement of capillary/arterial measurements at therapeutic decision thresholds.

Calibration improved CGM stability in our ICU cohort, reflected by reductions in SD (2.29→1.53 mmol/L), CV (25.0%→16.8%) and MAGE (6.26→5.77 mmol/L), indicating mitigation of excessive glycemic excursions. These changes suggest improved capacity of calibrated CGM to track physiologic glucose dynamics and dampen noise-driven fluctuations, consistent with prior inpatient findings that algorithm-enhanced CGM can reduce variability-related uncertainty in glucose interpretation. Lower CV and MAGE have been linked to reduced hypoglycemia and mortality risk in critically ill patients[20], highlighting the clinical significance of stabilizing continuous glucose trajectories. Improved signal stability may support tighter glucose control protocols, facilitate semi-automated insulin infusion adjustments, and reduce confirmatory sampling frequency when integrated into closed-loop systems. Further validation is warranted to determine whether reduced variability following calibration translates into meaningful clinical outcomes.

Subgroup analyses provided insight into clinical conditions that may differentially influence CGM calibration performance in the ICU. Although calibration reduced MARD across all predefined subgroups, higher residual error contributions were observed in patients receiving CRRT and vasoactive agents. These findings are clinically plausible, as both conditions are closely associated with acute hemodynamic instability, microcirculatory dysfunction, and increased vascular permeability, which can alter interstitial fluid dynamics and exacerbate the

physiological mismatch between interstitial and blood glucose measurements[21, 22]. Mechanistic research has highlighted that critical illness-related microcirculatory dysfunction, including regional hypoperfusion, capillary shunting, and endothelial barrier disruption, is common in shock and sepsis and contributes to impaired nutrient and solute exchange at the tissue level, supporting the notion that altered perfusion states can affect interstitial glucose kinetics and sensing accuracy[23]. In contrast, diabetes history and surgical status showed relatively smaller residual effects after calibration, suggesting that acute disturbances in perfusion and microcirculation may exert a greater influence on CGM signal fidelity in critically ill patients than chronic metabolic status alone. Real-world studies of CGM in hospitalized settings have similarly noted that physiological factors influencing tissue perfusion may affect CGM performance more than underlying metabolic diagnoses. Importantly, the consistency of post-calibration performance across heterogeneous subgroups supports the robustness of the context-aware calibration strategy under diverse ICU conditions, while highlighting clinical scenarios—such as CRRT use or vasopressor therapy—where additional caution or adaptive calibration strategies may be warranted.

This study has some limitations. First, it was conducted in a single surgical ICU with a limited sample size, which may restrict generalizability; prior ICU CGM studies have shown substantial center-to-center and population heterogeneity, underscoring the need for multicenter validation before widespread implementation[24]. Second, the observed improvement in accuracy and stability reflects a model-based calibration pipeline rather than intrinsic sensor upgrades, so our results represent algorithm-assisted performance and may not directly translate across platforms or settings. Recent work has emphasized that calibration strategy and POC sampling frequency can markedly influence CGM accuracy in the ICU[25]. Third, capillary reference measurements used for model training and

evaluation are themselves susceptible to error in states of impaired perfusion, sampling delay, or technical issues, which can bias agreement metrics and is a known limitation of ICU CGM trials[26]. Fourth, our cohort included heterogeneous critical illness phenotypes (e.g., vasopressor use, postoperative status, organ support), and model transportability to other ICUs or case mix remains uncertain, consistent with recent research calling for context-specific accuracy assessment[27]. Finally, we did not formally evaluate hypoglycemia detection sensitivity, false-alarm rates, or time-in-range-based endpoints; however, emerging hospital and ICU data suggest that these CGM-derived metrics, particularly hypoglycemia burden, are crucial for assessing the clinical utility and safety of CGM-guided insulin therapy[28].

In summary, calibration markedly improved CGM accuracy, agreement, and stability in our ICU cohort, suggesting that calibrated CGM may provide more reliable glucose trend information for critically ill patients. Improved monitoring accuracy may have important clinical implications. More reliable CGM signals may support glucose monitoring for clinical decision-making by reducing the risk of inappropriate interpretation of glucose readings. In addition, enhanced signal stability may facilitate earlier detection of dysglycemia. Importantly, under conditions of hemodynamic instability, including vasopressor use and CRRT, improved monitoring reliability may help mitigate the effects of impaired tissue perfusion on interstitial glucose measurements. These findings support further exploration of CGM-assisted glycemic monitoring strategies in the ICU. Future multicenter studies should validate performance across diverse hemodynamic states and treatment exposures, and assess whether CGM-guided approaches can improve glycemic control and clinical outcomes.

Abbreviation

1. CGM: continuous glucose monitoring
2. ICU: intensive care unit
3. CRRT: continuous renal replacement therapy
4. MAE: mean absolute error
5. RMSE: root mean square error
6. MARD: mean absolute relative difference
7. LTC: liquid time-constant network
8. GRU: gated recurrent unit
9. MAGE: mean amplitude of glycemic excursions
10. SD: standard deviation
11. CV: coefficient of variation

Declarations

1.Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Second Affiliated Hospital, Zhejiang University School of Medicine (Approval No. (2026) Ethics Review No. 0306). The requirement for informed consent was waived by the ethics committee because the study involved analysis of anonymized clinical data.

2. Consent for publication

Not applicable.

3. Availability of data and materials

The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request.

4. Competing interests

The authors declare that they have no competing interests.

5. Funding

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6. Authors' contributions

1）R.J., L.Z. (Long Zhang), and J.Y. contributed equally to this work.
2）R.J., L.Z. (Long Zhang), Z.Z., and H.L. conceived and designed the study.
3）R.J., J.Y., L.Z. (Liu Zhang), D.S., Y.C., and J.H. collected and curated the clinical data.
4）L.Z. (Long Zhang), B.W., and J.R. developed the calibration model and implemented the computational algorithms.
5）R.J., L.Z. (Long Zhang), and B.W. performed data analysis and statistical evaluation.
6）R.J. drafted the manuscript.
7）H.L., Z.Z., F.X., and L.L. critically revised the manuscript for important intellectual content.
8）All authors reviewed and approved the final manuscript.
9）Z.Z. and H.L. jointly supervised this work and contributed equally as corresponding authors.

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