Am J Transl Res 2026;18(8):6672-6681
www.ajtr.org /ISSN:1943-8141/AJTR0173681

Original Article

Prediction of acute metabolic complications in hospitalized elderly patients with type 2 diabetes using admission HbA1c and hypertension: a retrospective cohort study

Chunqiang Chen[1], Suxia Hu[2], Xiaoqin Zhu[1], Zhenzhen Xie[3], Yifei Zhu[3]

[1]Department of Clinical Laboratory, Susong County People's Hospital, Anqing 246500, Anhui, China; [2]Department of Clinical Laboratory, The First Hospital of Anhui University of Science and Technology, Huainan 232007, Anhui, China; [3]School of Medicine, Anhui University of Science and Technology, Huainan 232001, Anhui, China

Received April 29, 2026; Accepted July 29, 2026; Epub August 15, 2026; Published August 30, 2026

Abstract: Objective: To identify admission-based predictive indicators for in-hospital acute metabolic complications in elderly patients with type 2 diabetes mellitus (T2DM), and to develop and internally validate a simple predictive model for early risk stratification. Methods: A total of 203 elderly inpatients with T2DM were included in this retrospective cohort study. According to the occurrence of acute metabolic complications during hospitalization, patients were divided into a complication group (n = 40) and a non-complication group (n = 163). All candidate predictive indicators were collected from admission medical records and laboratory test results obtained within 24 to 48 hours after admission. Univariate and multivariate logistic regression analyses were performed to screen for independent predictive factors. The model's discriminatory power was evaluated using the area under the receiver operating characteristic curve (AUC), and 1000 bootstrap resamplings were adopted for internal validation. Results: The complication group had significantly higher admission HbA1c levels than the non-complication group (P<0.001), as well as a higher prevalence of hypertension (P = 0.002). Multivariate regression analysis verified that HbA1c (OR = 1.761, 95% CI: 1.44-2.15, P<0.001) and hypertension (OR = 3.904, 95% CI: 1.60-9.53, P = 0.003) were independent risk factors for in-hospital acute metabolic complications. The predictive model based on these two indicators exhibited good discriminatory performance, with an original AUC of 0.836. After bootstrap internal validation, the optimism-corrected AUC was 0.726, indicating acceptable stability of the model. Conclusion: Elevated admission HbA1c and comorbid hypertension are independent predictive factors for in-hospital acute metabolic complications in elderly T2DM patients. This novel simple model has favorable predictive efficacy and stability, and it can serve as a feasible clinical tool for early risk stratification in elderly T2DM inpatients.

Keywords: Type 2 diabetes mellitus, acute metabolic complications, elderly patients, risk prediction, glycated hemoglobin

Introduction

Global population aging is accelerating, and type 2 diabetes mellitus (T2DM) has become a major chronic disease threatening the health of elderly individuals [1, 2]. T2DM causes long-term metabolic disorders and multiple systemic complications, which severely impair the quality of life of elderly patients and shorten their survival time, bringing a heavy social and economic burden [3]. Elderly T2DM patients often have declined organ function and multiple comorbidities, which increase the difficulty of glycemic management. Acute metabolic complications, including severe hypoglycemia, diabetic ketoacidosis (DKA), and hyperosmolar hyperglycemic state (HHS), are common critical complications in elderly T2DM inpatients. These acute conditions are life-threatening and require urgent clinical intervention [4, 5].

The occurrence of acute metabolic complications in elderly T2DM patients results from the combined effects of metabolic instability and age-related organ fragility, with complex pathophysiologic mechanisms. Blood glucose fluctu-

https://doi.org/10.62347/QQCR9025

ations, decreased renal clearance, liver dysfunction, and persistent chronic inflammatory status can alter insulin sensitivity, hepatic glucose synthesis, and the pharmacokinetic and pharmacodynamic characteristics of hypoglycemic drugs. These changes further increase the risk of metabolic decompensation in patients [6]. Clinically, acute metabolic complications such as severe hypoglycemia, DKA, and HHS are closely associated with elevated short-term mortality. They can also induce secondary cardiovascular and renal adverse events, complicate acute-stage treatment and adversely affect long-term patient prognosis [7].

Previous studies have identified multiple factors associated with acute diabetic metabolic events, including poor long-term glycemic control, renal dysfunction, infection, dehydration, cardiovascular comorbidities, and irrational medication use. Glycated hemoglobin (HbA1c) reflects long-term cumulative glycemic exposure and can indicate chronic metabolic instability in patients. Hypertension and renal dysfunction can reflect vascular injury, decreased renal clearance, endothelial dysfunction, and systemic metabolic vulnerability. However, most existing studies focus on single complication outcomes, young and middle-aged populations, type 1 diabetes patients, or outpatient populations. High-quality clinical evidence targeting hospitalized elderly T2DM patients is still insufficient, and simple predictive models based on routine admission indicators are lacking.

Although the management of chronic diabetes has been continuously optimized, there is still a lack of systematic risk assessment and prevention strategies for in-hospital acute metabolic complications in elderly T2DM patients [8]. In particular, few predictive models combine routine laboratory indicators and comorbidity characteristics to screen for high-risk patients at admission [8].

Against this background, this retrospective cohort study analyzed the correlation between routine admission indicators (glycemic parameters, renal function, comorbidities) and in-hospital acute metabolic complications in elderly T2DM patients. We also constructed and internally validated a simple predictive model to provide an effective tool for early risk stratification and targeted intervention for this high-risk population.

Materials and methods

Study design and patient selection

This retrospective cohort study was conducted at Susong County People's Hospital. Clinical data of elderly T2DM inpatients admitted from January 2018 to December 2024 were retrieved from the hospital's electronic medical record system. The study protocol was approved by the Ethics Committee of Susong County People's Hospital. Since this study adopted anonymized retrospective clinical data, the requirement for informed consent was waived.

The inclusion criteria were as follows: (1) Age ≥65 years; (2) Clinically confirmed diagnosis of T2DM [9]; (3) Hospital stay ≥24 hours; (4) Complete in-hospital laboratory examination records; (5) Complete routine laboratory data detected within 24-48 hours after admission.

The exclusion criteria were as follows: (1) Diagnosis of type 1 diabetes, gestational diabetes, or secondary diabetes; (2) Admission due to initial onset of acute metabolic complications; (3) Repeated hospitalization during the study period (only the first eligible hospitalization record was included); (4) Missing key predictive indicator or outcome data; (5) Receipt of continuous renal replacement therapy or other treatments that may interfere with baseline metabolic indicators; (6) Having end-stage diseases, advanced malignant tumors, severe liver failure, or other severe non-diabetic diseases affecting the interpretation of laboratory results; (7) Incomplete medical records or unclear outcome documentation.

The patient screening flow chart is shown in Supplementary Figure 1. A total of 286 hospitalization records were initially screened. After excluding ineligible cases, 203 elderly T2DM inpatients were finally enrolled, including 40 patients (19.7%) with in-hospital acute metabolic complications (complication group) and 163 patients (80.3%) without complications (non-complication group).

Data extraction

We extracted complete clinical data from electronic medical records, including patients' demographic characteristics, comorbidities, medication history, admission laboratory indicators, and in-hospital clinical outcomes.

Demographic indicators wereage and sex. Comorbidities included hypertension, cardiovascular disease, heart failure, dementia, and chronic kidney disease (CKD) stage. Medication information included the use of dipeptidyl peptidase-4 inhibitors (DPP-4i), sodium-glucose cotransporter-2 inhibitors (SGLT2i), glucagon-like peptide-1 receptor agonists (GLP-1RA), glucocorticoids, sympathomimetic agents, and diuretics.

Admission laboratory indicators included HbA1c, random blood glucose (RBG), fasting blood glucose (FBG), serum creatinine, estimated glomerular filtration rate (eGFR), alanine aminotransferase (ALT), aspartate aminotransferase (AST), total cholesterol, triglycerides, and C-reactive protein (CRP). All laboratory data were obtained from tests completed within 24-48 hours after admission. For patients with multiple test results within the time window, the first valid result was adopted for analysis. Two researchers independently completed data extraction and cross-checking. Any discrepancies were resolved by reviewing original medical records and consulting a senior researcher.

Outcome measures

The primary outcome was the occurrence of any acute metabolic complication during hospitalization, including severe hypoglycemia, DKA, and HHS. Severe hypoglycemia was defined as a hypoglycemic episode requiring clinical intervention or intravenous glucose supplementation. DKA and HHS were diagnosed based on standard clinical manifestations, laboratory test results, and final discharge diagnoses. Secondary outcomes included the incidence of individual acute metabolic complications and the predictive performance of the model (discrimination, calibration, reclassification ability, and clinical utility).

Statistical analysis

SPSS 26.0 and R 4.2.1 software were used for all statistical analyses. Continuous variables were compared using the Student's t-test or Mann-Whitney U test according to data distribution. Categorical variables were compared using the chi-square test.

First, univariate logistic regression analysis was performed for all candidate indicators. Variables with $P<0.05$ by univariate analysis and clinically relevant variables were included in the initial multivariate logistic regression model. Non-significant variables after multivariate adjustment were excluded to establish the final parsimonious predictive model. The OR value, 95% CI, and *P* value of each independent predictor were calculated. A visual nomogram was constructed based on the final regression model.

The AUC was used to evaluate the model's discriminatory ability. A total of 1000 bootstrap resamplings were performed for internal validation to correct model overfitting and evaluate stability. In each bootstrap sample, the model was refitted and its predictive performance was re-evaluated to obtain the optimism-corrected AUC value. Bootstrap-corrected calibration curves, calibration slope, calibration intercept, and Brier score were used to assess the calibration degree and overall predictive accuracy of the model.

Net reclassification improvement (NRI) and integrated discrimination improvement (IDI) were used to evaluate the incremental predictive value of combining hypertension with HbA1c. Decision curve analysis (DCA) was performed to calculate the net clinical benefit of the model at different risk threshold probabilities. All statistical tests were two-tailed, and $P<0.05$ was considered significant.

Results

Baseline characteristics

Baseline characteristics of the two groups are summarized in **Table 1**. The complication group had a significantly higher prevalence of hypertension than the non-complication group (42.50% vs. 19.02%, $\chi^2 = 9.809$, $P = 0.002$). Among the 40 patients in the complication group, 20 developed severe hypoglycemia, 17 developed DKA, and 13 developed HHS, and a small number of patients had multiple concurrent acute metabolic complications. There were no significant differences between the two groups in age, sex, hospital stay, CKD stage, or the use of hypoglycemic drugs (DPP-4i, SGLT2i, GLP-1RA) (all $P>0.05$).

Admission laboratory indicators

As shown in **Table 2**, patients in the complication group had poorer baseline metabolic status and renal function at admission. The HbA1c level in the complication group was significantly

Table 1. Baseline characteristics of patients with and without complications

Characteristic	Complications (n = 40)	No complications (n = 163)	t/Z/χ^2	*P* value
Age (years)	73.00 (68.00-78.75)	74.00 (68.00-77.00)	-0.093	0.926
Sex			0.062	0.804
Male	19 (47.50%)	81 (49.69%)		
Female	21 (52.50%)	82 (50.31%)		
Length of hospital stay (days)	11.50 (10.00-13.75)	10.00 (10.00-13.00)	-1.525	0.127
Chronic kidney disease (CKD) stage			0.027	0.987
Stage 1	8 (20.00%)	34 (20.86%)		
Stage 2	20 (50.00%)	82 (50.31%)		
Stage 3	12 (30.00%)	47 (28.83%)		
Comorbidities				
Cardiovascular disease	10 (25.00%)	45 (27.61%)	0.111	0.740
Hypertension	17 (42.50%)	31 (19.02%)	9.809	0.002
Heart failure	12 (30.00%)	38 (23.31%)	0.774	0.379
Dementia	9 (22.50%)	27 (16.56%)	0.776	0.378
Medications				
DPP-4 inhibitor (DPP-4i)	12 (30.00%)	49 (30.06%)	0.000	0.994
SGLT2 inhibitor (SGLT2i)	7 (17.50%)	31 (19.02%)	0.049	0.825
GLP-1 receptor agonist (GLP-1RA)	8 (20.00%)	32 (19.63%)	0.003	0.958
Glucocorticoids	10 (25.00%)	39 (23.93%)	0.020	0.887
Sympathomimetic agents	12 (30.00%)	37 (22.70%)	0.579	0.447
Diuretics	8 (20.00%)	46 (28.22%)	0.731	0.393
Acute metabolic complications				
Hypoglycemia	20 (50.00%)	0 (0.00%)	90.407	<0.001
Diabetic ketoacidosis (DKA)	17 (42.50%)	0 (0.00%)	75.607	<0.001
Hyperosmolar hyperglycemic state (HHS)	13 (32.50%)	0 (0.00%)	56.600	<0.001

Note: Data are presented as median (interquartile range) or n (%). CKD, chronic kidney disease; DPP-4i, dipeptidyl peptidase-4 inhibitor; SGLT2i, sodium-glucose cotransporter-2 inhibitor; GLP-1RA, glucagon-like peptide-1 receptor agonist; DKA, diabetic ketoacidosis; HHS, hyperosmolar hyperglycemic state. Some patients experienced more than one type of acute metabolic complication; therefore, the sum of individual events exceeded 40.

higher than that of the non-complication group [10.11% (8.82-13.25) vs. 7.87% (6.97-9.15), Z = -6.530, P<0.001]. The admission RBG level was also significantly higher in the complication group (P = 0.007). In terms of renal function, the complication group had a lower eGFR level [66.93 (42.86-79.14) mL/min/1.73 m^2] compared to the non-complication group [75.74 (55.93-87.50) mL/min/1.73 m^2] (P = 0.043). No significant differences were observed in FBG, ALT, AST, blood lipid levels, and CRP between the two groups (all P>0.05).

Screening of independent predictive factors

The results of univariate logistic regression analysis for all candidate indicators are presented in Supplementary Table 1. All indicators with significance in univariate analysis were included in the multivariate regression model. After multivariate adjustment, RBG and eGFR lost significance (P = 0.351). Finally, HbA1c (OR = 1.761, 95% CI: 1.444-2.148, P<0.001) and hypertension (OR = 3.904, 95% CI: 1.600-9.525, P = 0.003) were confirmed as independent risk factors for in-hospital acute metabolic complications in elderly T2DM patients (**Table 3**).

Construction and validation of the predictive model

Based on the regression coefficients by multivariate logistic regression analysis, we established a predictive model for in-hospital acute metabolic complications in elderly T2DM patients. The prediction formula is as follows: P = 1/[1 + e^(-(-7.051 + 0.566HbA1c + 1.362Hypertension))].

Table 2. Laboratory values in patients with and without complications

Characteristic	Complications (n = 40)	No complications (n = 163)	Z	*P* value
HbA1c (%)	10.11 (8.82-13.25)	7.87 (6.97-9.15)	-6.53	<0.001
Random Blood Glucose (mmol/L)	16.96 (12.16-18.54)	14.83 (11.23-18.34)	-2.706	0.007
eGFR (mL/min/1.73 m^2)	66.93 (42.86-79.14)	75.74 (55.93-87.50)	-2.206	0.043
Fasting Blood Glucose (mmol/L)	9.03 (5.35-11.18)	8.56 (5.92-11.52)	-0.415	0.678
Serum Creatinine (μmol/L)	132.39 (114.38-143.78)	125.38 (92.80-152.22)	-1.195	0.232
ALT (U/L)	18.70 (12.79-22.25)	17.70 (13.20-22.09)	-0.174	0.862
AST (U/L)	22.98 (18.87-26.54)	21.79 (17.79-26.23)	-0.03	0.976
Total Cholesterol (mmol/L)	5.28 (3.35-7.03)	4.77 (3.34-6.35)	-0.512	0.609
Triglycerides (mmol/L)	1.54 (1.20-1.89)	1.52 (1.16-1.81)	-0.315	0.752
C-reactive protein (mg/L)	5.33 (3.38-6.35)	5.33 (3.39-6.58)	-0.465	0.642

Note: Data are presented as median (interquartile range). HbA1c, glycated hemoglobin; eGFR, estimated glomerular filtration rate; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein.

Table 3. Multivariable logistic regression analysis of predictors of acute metabolic complications

Variable	β	SE	Wald χ^2	*P* value	OR (95% CI)
HbA1c	0.566	0.101	31.15	<0.001	1.761 (1.444-2.148)
Hypertension (Yes vs. No)	1.362	0.455	8.96	0.003	3.904 (1.600-9.525)
Constant	-7.051	1.027	47.10	<0.001	-

Note: β indicates the regression coefficient. SE, standard error; OR, odds ratio; CI, confidence interval; HbA1c, glycated hemoglobin. Acute metabolic complications were coded as yes = 1 and no = 0. Hypertension was coded as yes = 1 and no = 0. HbA1c was entered as a continuous variable. Random blood glucose entered the initial model but was not retained after adjustment (P = 0.351).

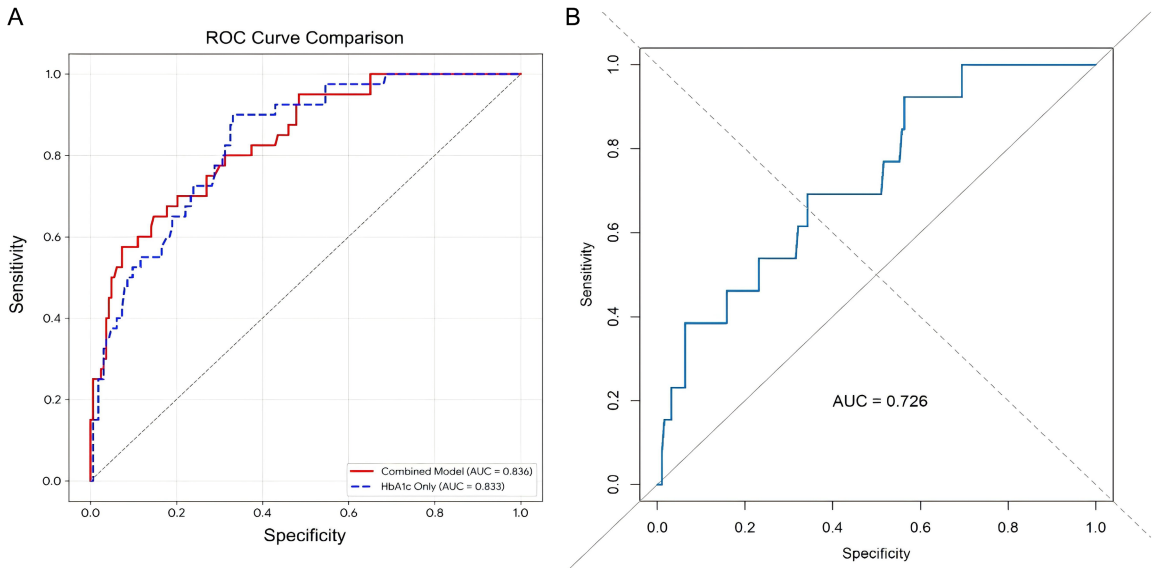


Figure 1. Receiver Operating Characteristic (ROC) curves of the prediction models. A. The ROC curve of the combined model [glycated hemoglobin (HbA1c) + hypertension] in the full study cohort (red solid line) demonstrates its significant discriminative performance in the original dataset. The area under the curve (AUC) is 0.836, while the AUC of the model using only HbA1c (blue dashed line) is 0.833. B. The internal validation using bootstrap resampling (1,000 iterations) shows that the AUC after optimism correction is 0.726. The diagonal of the gray dashed line represents the performance of a random classifier.

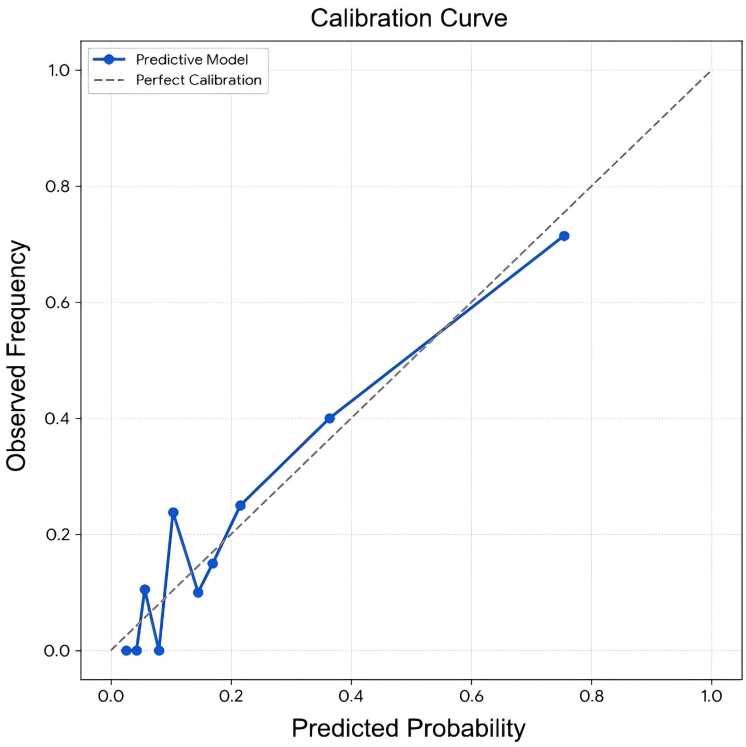


Figure 2. Calibration curve of the prediction model. This graph shows the relationship between the predicted probability (on the x-axis) and the observed frequency (on the y-axis). The blue solid line represents the prediction model, and the gray dotted line indicates the perfect calibration state, meaning that the predicted probability is exactly the same as the observed result.

In this model, hypertension was assigned a value of 1 for positive cases and 0 for negative cases.

Model performance and internal validation

The original AUC of the model in the overall cohort was 0.836 (95% CI: 0.769-0.903) (**Figure 1A**), indicating good discriminatory ability in the original dataset. After 1000-bootstrap internal validation and overfitting correction, the optimism-corrected AUC was 0.726 (**Figure 1B**), verifying good stability of the model.

The bootstrap-corrected calibration curve showed favorable consistency between the model's predicted risk probability and actual clinical outcome (**Figure 2**). The calibration slope was 0.955 and the calibration intercept was -0.042. The model's Brier score was 0.113, further confirming the high overall predictive accuracy.

Clinical utility of the model

According to the Youden index, the optimal risk threshold of the model was 0.218. At this threshold, the model's sensitivity was 65.0%, specificity was 85.3%, and negative predictive value (NPV) was 90.9% (**Table 4**). A predicted risk lower than 0.218 can accurately screen low-risk patients, avoiding unnecessary intensive clinical monitoring and excessive treatment.

DCA results showed that compared to the "universal intervention" and "no intervention" strategies, this model maintained a higher net clinical benefit within the threshold probability range of 10%-80% (**Figure 3**). The nomogram of the final predictive model is shown in **Figure 4**, which can directly and visually evaluate the individual risk of acute metabolic complications in elderly T2DM inpatients.

Discussion

This retrospective cohort study confirmed that elevated admission HbA1c and comorbid hypertension were independent risk factors for in-hospital acute metabolic complications in elderly T2DM patients. We further constructed and internally validated a simple predictive model based on these two routine admission indicators. The model had good predictive efficacy and stability, so it may be a targeted risk assessment tool for elderly T2DM inpatients and support clinical individualized diabetes management.

As a core indicator reflecting long-term glycemic control, HbA1c has stable predictive value for diabetic metabolic risk, which is consistent with the metabolic memory theory and existing clinical research conclusions [10, 11]. This study found that the risk of life-threatening acute metabolic complications (severe hypoglycemia, DKA, HHS) increased significantly with the elevation of HbA1c. Elevated HbA1c indicates persistent long-term hyperglycemia, insufficient islet β-cell reserve, severe insulin resistance, poor outpatient treatment adherence, and suboptimal long-term glycemic management [12]. Elderly T2DM patients are vulnerable to adverse events caused by excessive hypoglycemia, so clinical guidelines usually rec-

Table 4. Performance metrics of the multivariable model for predicting acute metabolic complications

Metric	Value/estimate
Discrimination	
Apparent AUC	0.836 (0.769-0.903)
Optimism-corrected AUC	0.726
Calibration	
Slope	0.955
Intercept	-0.042
Brier Score	0.113
Reclassification (vs. HbA1c alone)	
Net Reclassification Improvement (NRI)	0.470 (P = 0.002)
Integrated Discrimination Improvement (IDI)	0.049 (P<0.001)
Clinical Utility (at cutoff 0.218)	
Sensitivity	65.0%
Specificity	85.3%
Positive Predictive Value (PPV)	52.0%
Negative Predictive Value (NPV)	90.9%

Note: AUC, area under the curve; CI, confidence interval; NRI, net reclassification improvement; IDI, integrated discrimination improvement; PPV, positive predictive value; NPV, negative predictive value. Bootstrap validation used resamples of the full cohort; no independent validation cohort was available.

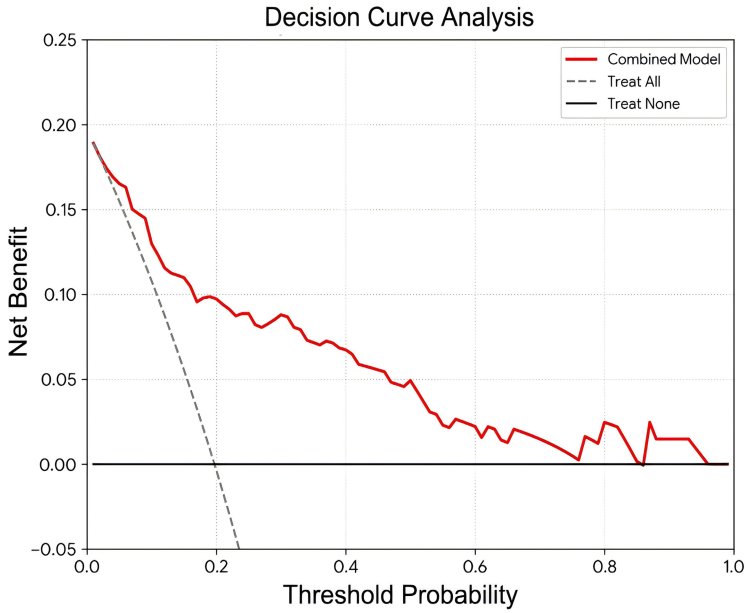


Figure 3. Decision Curve Analysis (DCA) of the combined model. The combined model (red solid line) achieved a higher net benefit within a wider range of threshold probabilities compared to the "all treatment" (gray dotted line) and "all no treatment" (black horizontal line) strategies, suggesting its good clinical practicability.

ommend individualized and relaxed glycemic targets for elderly populations. However, this study found that poor pre-admission glycemic control (elevated HbA1c) was an important inducement for in-hospital acute metabolic complications. This may be attributed to the rapid adjustment of hypoglycemic regimens after admission or unstable blood glucose fluctuations caused by long-term metabolic disorders [13].

Notably, RBG was correlated with acute metabolic complications in univariate analysis but failed to become an independent predictive factor after multivariate adjustment. Unlike HbA1c, a single RBG detection result is easily affected by recent diet, acute stress, infection, and pre-admission hypoglycemic treatment. It reflects only transient glycemic status, while HbA1c can stably reflect long-term glycemic burden and basic metabolic vulnerability of patients, thus having higher clinical predictive value.

This study verified that hypertension is a powerful independent risk factor for acute metabolic complications. In elderly patients, hypertension is often accompanied by endothelial dysfunction, renal microvascular injury, and systemic vascular fragility [14]. Although eGFR was lower in the complication group, it was not retained in the final model as an independent predictor. Previous studies have shown that the combination of diabetes and hypertension can impair vascular regulation and neurological perception function [15, 16]. These abnormalities reduce the physiologic reserve of elderly patients, weaken their ability to perceive blood glucose fluctuations, and delay the early identification of metabolic deterioration. Additionally, common antihypertensive drugs such as diuretics may cause dehy-

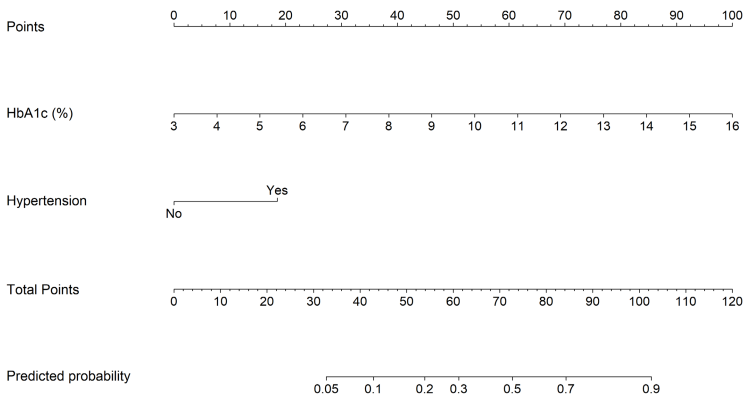


Figure 4. Nomogram for predicting acute metabolic complications in hospitalized elderly patients with type 2 diabetes mellitus. The nomogram was constructed using HbA1c and hypertension from the final multivariable logistic regression model. The points assigned to the two predictors are summed to obtain the total points, which are then mapped to the predicted probability of acute metabolic complications.

dration and electrolyte disorders, further disrupting glycemic balance [17]. It is worth emphasizing that this observational study can only confirm hypertension as a clinical marker of cardiorenal and vascular vulnerability, rather than a direct causal factor of acute metabolic complications.

The model's original AUC was 0.836, and the bootstrap-corrected AUC was 0.726, indicating moderate and stable discriminatory ability. Most existing diabetic complication prediction models focus on single complications, young populations, or outpatient patients [18, 19]. In contrast, this model was specially designed for elderly T2DM inpatients with multiple comorbidities and complex metabolic status, giving a stronger targeted clinical applicability. The model has good calibration, and DCA verifies its stable net clinical benefit. Importantly, the NPV reaches 90.9% at the optimal threshold, which can accurately screen low-risk patients. For these low-risk patients, clinicians can appropriately reduce monitoring frequency and treatment intensity, so as to optimize medical resource allocation and reduce medical costs [20]. It should be noted that the corrected AUC is more objective and reliable than the original value. Limited by the lack of external validation, this model is more suitable for preliminary clinical risk stratification rather than definitive clinical decision-making.

This study had several strengths, but also had inherent limitations. First, the retrospective single-center design cannot confirm the causal relationship between indicators and complications. Second, this study failed to fully include potential confounding factors such as infection severity, nutritional status, and pre-admission medication adherence, which have been proven to affect diabetic metabolic outcomes [21, 22]. Third, the enrolled patients were all from a single hospital, which may limit the generalizability of the model [23]. Fourth, the model did not incorporate geriatric syndrome indicators such as frailty and cognitive impairment, which are closely related to severe hypoglycemia and adverse diabetes events [24, 25]. Fifth, this study only completed internal bootstrap validation without external multicenter validation. In addition, the small number of individual complication cases cannot support subtype-specific modeling, and early in-hospital treatment may interfere with admission laboratory test results.

Future research should enroll multicenter prospective cohorts to complete external validation of the model and verify its clinical applicability in different medical environments. Incorporating continuous glucose monitoring data can dynamically reflect blood glucose fluctuations and further improve the model's predictive accuracy [26, 27]. Subsequent studies will also explore the predictive efficacy of the model for different types of acute metabolic complications and evaluate the clinical outcome improvement brought by model-assisted risk management.

In conclusion, admission elevated HbA1c and comorbid hypertension were independent predictive factors for in-hospital acute metabolic complications in elderly T2DM patients. The simple prediction model constructed based on these two routine indicators has good stability and clinical utility. It can realize early risk stratification of elderly T2DM inpatients, help clinicians formulate individualized intervention strategies, and improve the safety and quality of clinical diabetes management for elderly patients.

Acknowledgements

This work was supported by the Open Fund of the Joint Research Center for Occupational Medicine and Health, Institute of Health and Medicine, Hefei Comprehensive National Science Center (No. OMH-2024-034).

Disclosure of conflict of interest

None.

Address correspondence to: Suxia Hu, Department of Clinical Laboratory, The First Hospital of Anhui University of Science and Technology, Huainan 232007, Anhui, China. Tel: +86-15305548917; E-mail: 18054009029@163.com

References

[1] Thipsawat S. Early detection of diabetic nephropathy in patient with type 2 diabetes mellitus: a review of the literature. Diab Vasc Dis Res 2021; 18: 14791641211058856.

[2] Wang L, Li L, Liu J, Sheng C, Yang M, Hu Z and Yue R. Associated factors and principal pathophysiological mechanisms of type 2 diabetes mellitus. Front Endocrinol (Lausanne) 2025; 16: 1499565.

[3] Cadena Sandoval M and Haeusler RA. Bile acid metabolism in type 2 diabetes mellitus. Nat Rev Endocrinol 2025; 21: 203-213.

[4] Wysham C, Bindal A, Levrat-Guillen F, Kostadinova D and Poon Y. A systematic literature review on the burden of diabetic ketoacidosis in type 2 diabetes mellitus. Diabetes Obes Metab 2025; 27: 2750-2767.

[5] Jancev M, Vissers TACM, Visseren FLJ, van Bon AC, Serné EH, DeVries JH, de Valk HW and van Sloten TT. Continuous glucose monitoring in adults with type 2 diabetes: a systematic review and meta-analysis. Diabetologia 2024; 67: 798-810.

[6] Wang X, Li Q, Liu Y, Jiang H and Chen W. Intermittent fasting versus continuous energy-restricted diet for patients with type 2 diabetes mellitus and metabolic syndrome for glycemic control: a systematic review and meta-analysis of randomized controlled trials. Diabetes Res Clin Pract 2021; 179: 109003.

[7] Kadowaki T, Maegawa H, Watada H, Yabe D, Node K, Murohara T and Wada J. Interconnection between cardiovascular, renal and metabolic disorders: a narrative review with a focus on Japan. Diabetes Obes Metab 2022; 24: 2283-2296.

[8] Ma CX, Ma XN, Guan CH, Li YD, Mauricio D and Fu SB. Cardiovascular disease in type 2 diabetes mellitus: progress toward personalized management. Cardiovasc Diabetol 2022; 21: 74.

[9] Adeva-Andany MM, Fernández-Fernández C, Funcasta-Calderón R, Ameneiros-Rodríguez E, Adeva-Contreras L and Castro-Quintela E. Insulin resistance is associated with clinical manifestations of diabetic kidney disease (Glomerular Hyperfiltration, Albuminuria, and Kidney Function Decline). Curr Diabetes Rev 2022; 18: e171121197998.

[10] Khunti K, Zaccardi F, Amod A, Aroda VR, Aschner P, Colagiuri S, Mohan V and Chan JCN. Glycaemic control is still central in the hierarchy of priorities in type 2 diabetes management. Diabetologia 2025; 68: 17-28.

[11] Chen J, Yin D and Dou K. Intensified glycemic control by HbA1c for patients with coronary heart disease and type 2 diabetes: a review of findings and conclusions. Cardiovasc Diabetol 2023; 22: 146.

[12] Ding J, Shi Q, Dong L, Su H, Du Y, Pan T and Zhong X. Association of HbA1c variability with vibrating perception threshold in middle-aged and elderly patients with type 2 diabetes mellitus: a retrospective cohort study. Diabetes Metab Syndr Obes 2024; 17: 193-202.

[13] Zhang KX, Kan CX and Sun XD. Balancing act: the dilemma of rapid hyperglycemia correction in diabetes management. World J Diabetes 2024; 15: 129-132.

[14] Ellis PA and Cairns HS. Renal impairment in elderly patients with hypertension and diabetes. QJM 2001; 94: 261-265.

[15] Marmarelis VZ, Shin DC, Kang Y and Novak V. Data-based modeling of cerebral hemodynamics quantifies impairment of cerebral blood flow regulation in type-2 diabetes. J Cereb Blood Flow Metab 2024; 44: 1288-1301.

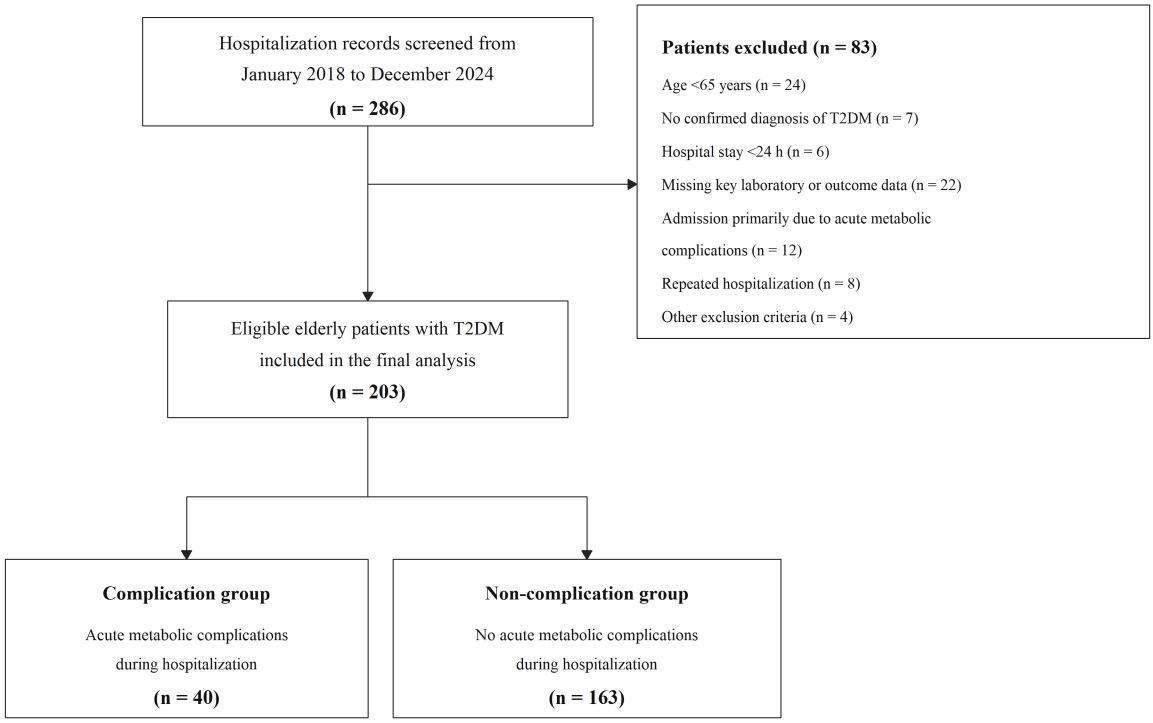
[16] Li M, Li Y, Zhao K, Qin C, Chen Y, Liu Y, Qiu S, Tan X and Liang Y. Abnormal cerebral blood flow and brain function in type 2 diabetes mellitus. Endocrine 2024; 85: 433-442.

[17] Wu J, Han X, Sun D, Zhang J, Li J, Qin G, Deng W, Yu Y and Xu H. Age-specific association of stage of hypertension at diagnosis with cardiovascular and all-cause mortality among elderly patients with hypertension: a cohort study. BMC Cardiovasc Disord 2023; 23: 270.

[18] Williams DD, Ferro D, Mullaney C, Skrabonja L, Barnes MS, Patton SR, Lockee B, Tallon EM, Vandervelden CA, Schweisberger C, Mehta S, McDonough R, Lind M, D'Avolio L and Clements MA. An "All-Data-on-Hand" deep learning model to predict hospitalization for diabetic ketoacidosis in youth with type 1 diabetes: development and validation study. JMIR Diabetes 2023; 8: e47592.

[19] Subramanian D, Sonabend R and Singh I. A machine learning model for risk stratification

of postdiagnosis diabetic ketoacidosis hospitalization in pediatric type 1 diabetes: retrospective study. JMIR Diabetes 2024; 9: e53338.
[20] Schwartz DD, Banuelos R, Uysal S, Vakharia M, Hendrix KR, Fegan-Bohm K, Lyons SK, Sonabend R, Gunn SK and Dei-Tutu S. An automated risk index for diabetic ketoacidosis in pediatric patients with type 1 diabetes: the RIDKA. Clin Diabetes 2022; 40: 204-210.
[21] Lombardo M, Feraco A, Bellia C, Prisco L, D'Ippolito I, Padua E, Storz MA, Lauro D, Caprio M and Bellia A. Influence of nutritional status and physical exercise on immune response in metabolic syndrome. Nutrients 2022; 14: 2054.
[22] Yu XL, Zhou LY, Huang X, Li XY, Wang MK and Yang JS. Role of nutrition in diabetes mellitus and infections. World J Clin Cases 2025; 13: 94389.
[23] Gong JY, Sajjadi SF, Motala AA, Shaw JE and Magliano DJ. Variation in type 2 diabetes prevalence across different populations: the key drivers. Diabetologia 2025; 68: 2327-2339.
[24] Kan W, Qu M, Wang Y, Zhang X and Xu L. A review of type 2 diabetes mellitus and cognitive impairment. Front Endocrinol (Lausanne) 2025; 16: 1624472.
[25] Dao L, Choi S and Freeby M. Type 2 diabetes mellitus and cognitive function: understanding the connections. Curr Opin Endocrinol Diabetes Obes 2023; 30: 7-13.
[26] Jancev M, Vissers TACM, Visseren FLJ, van Bon AC, Serné EH, DeVries JH, de Valk HW and van Sloten TT. Continuous glucose monitoring in adults with type 2 diabetes: a systematic review and meta-analysis. Diabetologia 2024; 67: 798-810.
[27] Ajjan RA, Battelino T, Cos X, Del Prato S, Philips JC, Meyer L, Seufert J and Seidu S. Continuous glucose monitoring for the routine care of type 2 diabetes mellitus. Nat Rev Endocrinol 2024; 20: 426-440.



Supplementary Figure 1. Patient selection flow diagram. A total of 286 hospitalization records were initially screened from January 2018 to December 2024. After excluding 83 records according to the predefined inclusion and exclusion criteria, 203 eligible elderly patients with type 2 diabetes mellitus (T2DM) were included in the final analysis, including 40 patients with acute metabolic complications and 163 without acute metabolic complications.

Supplementary Table 1. Univariate logistic regression analyses of all candidate variables

Variable	Assignment	Univariate OR (95% CI)	*P* value
Age	Continuous	0.993 (0.935-1.054)	0.811
Sex	Male = 1, Female = 0	0.916 (0.458-1.830)	0.804
Length of hospital stay	Continuous	1.094 (0.947-1.263)	0.222
CKD stage	Stage 1 as reference	-	0.987
CKD stage 2 vs. 1	2 vs. 1	1.037 (0.416-2.581)	0.938
CKD stage 3 vs. 1	3 vs. 1	1.085 (0.400-2.942)	0.872
Cardiovascular disease	Yes = 1, No = 0	0.874 (0.395-1.933)	0.740
Hypertension	Yes = 1, No = 0	3.147 (1.503-6.589)	0.002
Heart failure	Yes = 1, No = 0	1.410 (0.654-3.037)	0.381
Dementia	Yes = 1, No = 0	1.462 (0.625-3.419)	0.380
DPP-4 inhibitor	Yes = 1, No = 0	0.997 (0.469-2.121)	0.994
SGLT2 inhibitor	Yes = 1, No = 0	0.903 (0.366-2.232)	0.825
GLP-1 receptor agonist	Yes = 1, No = 0	1.023 (0.431-2.433)	0.958
Glucocorticoids	Yes = 1, No = 0	1.060 (0.476-2.361)	0.887
Sympathomimetic agents	Yes = 1, No = 0	1.459 (0.676-3.149)	0.335
Diuretics	Yes = 1, No = 0	0.636 (0.273-1.483)	0.294
HbA1c	Continuous	1.719 (1.427-2.072)	<0.001
Random blood glucose	Continuous	1.049 (1.001-1.100)	0.047
eGFR	Continuous	0.986 (0.971-1.001)	0.074
Fasting blood glucose	Continuous	0.993 (0.892-1.105)	0.891
Serum creatinine	Continuous	1.001 (0.997-1.004)	0.673
ALT	Continuous	0.984 (0.939-1.031)	0.500
AST	Continuous	0.988 (0.942-1.036)	0.619
Total cholesterol	Continuous	1.054 (0.875-1.270)	0.577
Triglycerides	Continuous	1.018 (0.652-1.590)	0.936
CRP	Continuous	0.963 (0.897-1.033)	0.294

Note: Acute metabolic complications were coded as yes = 1 and no = 0. Sex was coded as male = 1 and female = 0. Hypertension, comorbidities, and medication use were coded as yes = 1 and no = 0. Continuous variables were entered as continuous variables. CKD stage was analyzed as a categorical variable, with stage 1 as the reference category. OR, odds ratio; CI, confidence interval; CKD, chronic kidney disease; HbA1c, glycated hemoglobin; eGFR, estimated glomerular filtration rate; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein.