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ORIGINAL ARTICLES

Geriatric nutritional risk index predicts in-hospital mortality in elderly patients with cerebral infarction

[1]Hang Su, [2]Xiaoyong Huang, [2]Yunpao Xiao, [3]Xiaoman Xu, [1]Zhezhe Sun, [1]Shuihong Yu, [1]Qinghua Cao

[1]Department of Cerebrovascular Diseases, The First Affiliated Hospital of Ningbo University, Ningbo, China; [2]Department of Neurology, Beilun District Second People's Hospital, Ningbo, China; [3]Ningbo University, China

Abstract

Objective: This study aimed to evaluate the predictive value of the Geriatric Nutritional Risk Index (GNRI) for in-hospital mortality among elderly patients with cerebral infarction. *Methods:* Patients aged ≥60 years with a diagnosis of cerebral infarction were extracted from the MIMIC-IV database. GNRI was calculated using height, weight, and serum albumin. The primary outcome was in-hospital mortality. Restricted cubic spline regression, univariate and multivariate logistic regression, and forest plot visualization were performed to assess the prognostic significance of GNRI. *Results:* A total of 746 patients were included, with an in-hospital mortality rate of 30.2%. GNRI was significantly lower in non-survivors (86.43) than survivors (88.00), p<0.001. Multivariable logistic regression demonstrated GNRI as an independent predictor of mortality (OR per 1-unit increase, 0.91; 95% CI: 0.87–0.96). The restricted cubic spline showed a near-linear inverse relationship (p overall <0.001, p nonlinear = 0.654). *Conclusion:* GNRI is a significant and independent predictor of in-hospital mortality in elderly cerebral infarction patients and may aid clinical risk stratification.

Keywords: Geriatric nutritional risk index, cerebral infarction, in-hospital mortality, prognosis; MIMIC-IV

INTRODUCTION

Cerebral infarction, commonly known as ischemic stroke, represents a substantial global health challenge, affecting millions of individuals worldwide. The burden of ischemic stroke is particularly pronounced due to its high incidence, mortality, and the long-term disability it often causes.[1] According to the Global Burden of Disease Study 2021, ischemic stroke continues to be a leading cause of death and disability-adjusted life years (DALYs) globally. In 2021, approximately 7.8 million individuals were affected by ischemic stroke, resulting in over 3.5 million deaths and more than 70 million DALYs. These figures highlight an increase of 88% in incidence, 55% in mortality, and 52.4% in DALYs since 1990, underscoring the growing impact of this condition on public health.[2]

Malnutrition, which affects 30–50% of elderly patients hospitalized, significantly deteriorates the prognosis of cerebral infarction through various mechanisms.[3] Deficiencies in protein and energy compromise immune function, hinder tissue repair, and exacerbate sarcopenia, thereby increasing the risk of infections (odds ratio = 2.1–3.5), extending hospitalization duration, and impairing functional recovery.[4] Importantly, nutritional status plays a critical role in neuroplasticity and rehabilitation potential, with hypoalbuminemia (defined as serum albumin levels <3.5 g/dL) independently associated with a three-month mortality risk (hazard ratio = 1.8, 95% confidence interval 1.3–2.5) in stroke patients.[5] The Geriatric Nutritional Risk Index (GNRI), which combines serum albumin levels and body weight, has emerged as a valuable tool for assessing nutritional risk; however, its prognostic significance specifically within the elderly stroke population remains insufficiently investigated.[6] Although there is an increasing acknowledgment of the detrimental

Address correspondence to: Xiaoyong Huang, Department of Neurology, Beilun District Second People's Hospital, Ningbo, China. Email: subenyuan890808@163.com

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effects of malnutrition, the majority of existing research has concentrated on general stroke cohorts rather than the particularly vulnerable elderly subgroup.[7] Current nutritional assessment methods frequently depend on complex tools that are impractical for routine clinical application, while simpler biomarkers, such as albumin alone, lack the necessary specificity.[8] This study aims to evaluate the GNRI as an accessible and objective predictor of in-hospital outcomes in this demographic.

METHODS

Data source

The study data were extracted from a publicly accessible database, the MIMIC-IV (version: v1.0). The database contains comprehensive clinical data for ICU patients at Beth Israel Deaconess Medical Center from 2008 to 2019.[9] Data collected by BIDMC were deidentified, transformed and made available to researchers who completed human research training and signed data use agreements. The Institutional Review Board at BIDMC granted a waiver for informed consent and approved the sharing of research resources.[10] Accessing this database involved completing required courses and application processes, passing specified exams and obtaining appropriate data access permissions.

Patient and public involvement

The MIMIC-IV data used in this retrospective analysis are accurate medical data that can be accessed for free. All personal information in the database has been deidentified, replaced with random codes instead of patient identifiers, ensuring anonymity. As such, publicly available databases do not require patient-informed consent or ethical approval.

Patient selection

We identified patients with cerebral infarction from the MIMIC-IV database using International Classification of Diseases (ICD) codes (ICD-9: 433-434; ICD-10: I63). From an initial cohort of 4,545 patients meeting the diagnostic criteria, we applied the following exclusion criteria: (1) non-first ICU admissions, (2) age <60 years, (3) missing anthropometric measurements (height/weight) or serum albumin data, and (4) absence of other key clinical variables. After exclusions, 746 eligible elderly patients were included in the final analysis. The patient selection process is plotted in Figure 1.

Data collection

This retrospective cohort study utilized data from the MIMIC-IV database. Variables extracted included demographics (age, gender), lifestyle factors (smoking and alcohol abuse), nutritional status (GNRI), and a range of comorbidities (including AKI, sepsis, hypertension, diabetes, CHF, chronic pulmonary disease, malignancy, etc.). GNRI was calculated as: GNRI = (1.489 × albumin [g/L]) + (41.7 × actual body weight / ideal body weight), with ideal body weight computed using the Lorentz formula.[11–14]

Disease severity was assessed using several scoring systems: Acute Physiology Score III (APSIII), Simplified Acute Physiology Score II (SAPSII), Sequential Organ Failure Assessment (SOFA), Glasgow Coma Scale (GCS), Systemic Inflammatory Response Syndrome (SIRS), Logistic Organ Dysfunction System (LODS), Charlson index, Model for End-Stage Liver Disease (MELD), and Oxford Acute Severity of Illness Score (OASIS). First recorded vital signs (temperature, heart rate, respiratory rate, systolic and diastolic blood pressure, mean blood pressure) and initial laboratory test values within 24 hours of ICU admission were extracted. Interventions such as invasive mechanical ventilation, Cardiopulmonary Resuscitation (CPR), and renal replacement therapy were also recorded.

In addition, custom-defined complications were included (e.g., pneumonia, urinary tract infection, gastrointestinal bleeding, intracerebral hemorrhage, atrial fibrillation, intravenous tissue plasminogen activator (tPA), endovascular thrombectomy (EVT), percutaneous transluminal angioplasty/stenting (PTA/PTAS) procedures). For laboratory values with multiple entries, the first measurement within 24 hours was used to reflect baseline physiological status. The primary outcome was in-hospital mortality.

Statistical analysis

Continuous variables were expressed as medians with interquartile ranges (IQRs) due to non-normal distributions, while categorical variables were presented as frequencies and percentages. Group differences for continuous variables were assessed using the Mann-Whitney U test, and categorical variables were compared using the Chi-square test. A two-sided p-value < 0.05 was considered statistically significant. Patients

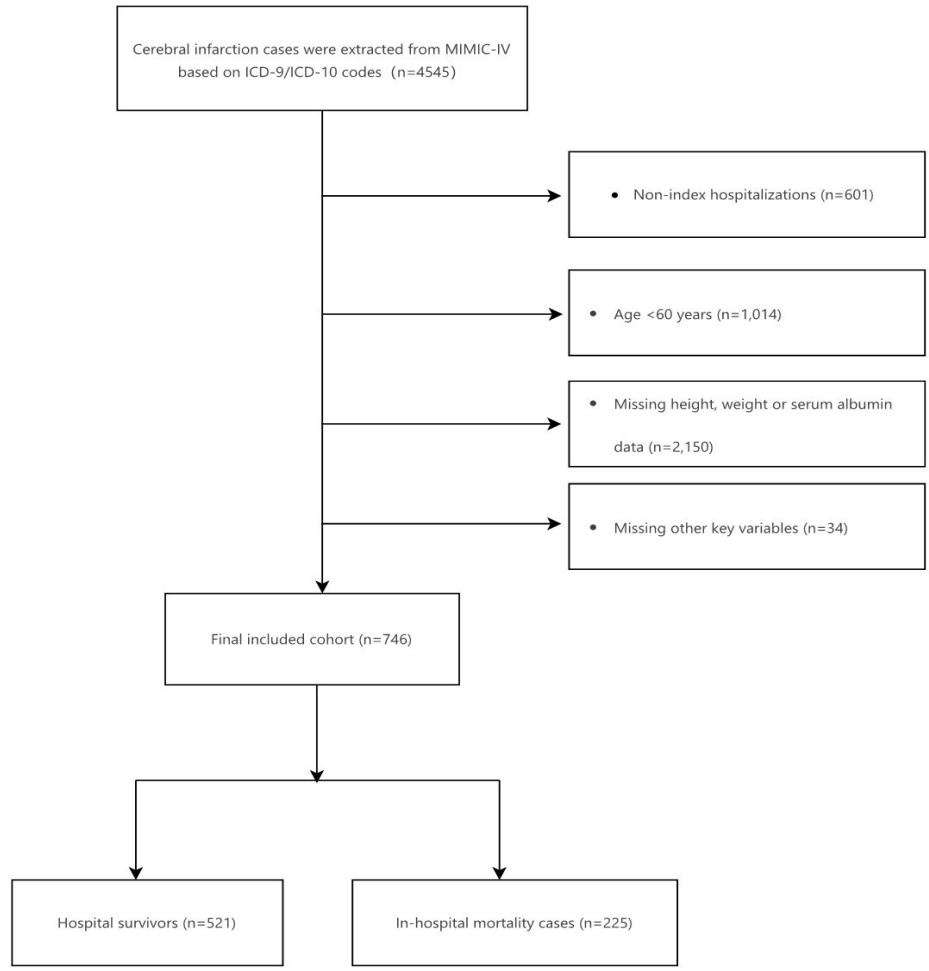

Figure 1. Flow chart of selection

were randomly divided into a training set and a validation set in a 7:3 ratio. Feature selection was performed using the Boruta algorithm, a random forest-based method, to identify potentially important predictors through univariate screening. In particular, Boruta (Version: 8.0.0) is executed to perform feature selection, where the algorithm iteratively compares the importance of each original variable with its shadow variable, and determines the importance of each variable over 500 iterations or until all variables are stable. Importance results are extracted with the attStats function and formatted with a customized adjust data function.[15] The selected variables were subsequently entered into a multivariable logistic regression model to identify independent prognostic factors, retaining variables with $p < 0.05$. Model performance was evaluated through receiver operating characteristic (ROC) curve analysis for discrimination, calibration plots and the Hosmer-Lemeshow test for calibration, and decision curve analysis (DCA) to assess clinical utility. The relationship between GNRI and mortality was explored using restricted cubic spline (RCS) regression. All statistical analyses and visualizations were conducted using R software (version 4.4.2) and JD_DCPM (version 6.03, Jingding Medical Technology Co., Ltd.).

RESULTS

Baseline characteristics

A total of 746 elderly patients with cerebral infarction were included in the study, of whom 521 survived and 225 died during hospitalization (Table 1). The median age of the cohort was 73.56 years (interquartile range [IQR], 66.88–80.85), with no significant difference between the survival and non-survival groups ($p = 0.051$). The gender

distribution was nearly equal, with 51% male and 49% female participants.

The median Geriatric Nutritional Risk Index (GNRI) was 87.54 (IQR, 84.09–89.8), and it was significantly lower in non-survivors (86.43 [82.65–88.92]) than in survivors (88.00 [84.8–90.31], $p < 0.001$). Other clinical variables, including comorbidities, severity scores, and laboratory values, are detailed in. Significant differences were observed in several parameters between groups, suggesting their potential association with in-hospital mortality.

This study divided 746 patients into a training set (n=522) and a test set (n=224). Baseline characteristic analysis demonstrated high comparability between the two cohorts. Demographic features (gender: 51% female in the training set vs. 51% in the test set, *p* = 0.949; median age: 73.94 vs. 73.08 years, *p* = 0.685) and major comorbidities (hypertension, diabetes, chronic pulmonary disease, etc.) showed no statistically significant differences (all *p* > 0.05). Disease severity scores (APSIII, SOFA, SAPSII) remained balanced between groups (median differences ≤1 point, *p* = 0.185–0.815), with key laboratory parameters (WBC: 11.05 vs. 10.9 $\times 10^3/\mu L$; creatinine: 1.0 vs. 1.1 mg/dL; albumin: 3.1 vs. 3.1 g/dL) and therapeutic interventions (invasive ventilation: 73% vs. 78%, *p* = 0.136; CRRT: 11% vs. 15%, *p* = 0.179) also exhibiting no significant heterogeneity. Notably, tissue plasminogen activator (tPA) usage was marginally lower in the test set (1% vs. 4%, *p* = 0.075) but did not reach statistical significance. Overall, baseline characteristics between the training and test sets were well-matched (96% of variables and confirming suitability for machine learning model development and validation.

Boruta-driven feature selection and multivariable logistic regression

To reduce dimensionality and enhance model robustness, we applied the Boruta algorithm—a random forest-based, all-relevant feature selection technique—to screen for predictors of in-hospital mortality among elderly patients with cerebral infarction. The algorithm iteratively compares the importance of each real feature with its randomized shadow counterparts to identify variables that are strongly, weakly, or not at all associated with the outcome.

In the Boruta importance plot (Figure 2), variables are displayed in descending order of their Z-score importance. Green bars represent features that were confirmed as significantly predictive, while red bars indicate features rejected due to lack of contribution. Yellow bars denote variables with intermediate importance, classified as "tentative" by the algorithm. These variables are often borderline and may require further statistical verification. Considering both predictive strength and clinical relevance, we selected all confirmed (green) and tentative (yellow) variables for inclusion in the multivariable logistic regression analysis. Notably, variables such as GNRI, APSIII, SOFA, GCS, sepsis, and invasive ventilation were among the top-ranking confirmed features. Tentative variables—while statistically inconclusive in Boruta—may still hold clinical value and were retained to ensure a comprehensive assessment.

This inclusive approach allowed us to balance statistical rigor with clinical insight, minimizing the risk of prematurely excluding variables with potential interactions or nonlinear associations. The subsequent multivariate model was therefore built upon a refined and clinically relevant feature set derived from both robust statistical screening and expert judgment.

Multivariable logistic regression analysis

Independent predictors of in-hospital mortality were assessed through multivariable logistic regression, incorporating variables with $p < 0.05$ in the univariate analysis. Invasive mechanical ventilation was the strongest risk factor (OR = 4.09, 95% CI: 2.18–>8.10, $p < 0.05$; Figure 3). Other significant risk factors included higher SOFA score (OR = 1.10, 95% CI: 1.02–1.18, $p < 0.05$), older age at admission (OR = 1.06, 95% CI: 1.03–1.09, $p < 0.05$), elevated white blood cell count (WBC) (OR = 1.02, 95% CI: 1.00–1.03, $p < 0.05$), and blood urea nitrogen (BUN) (OR = 1.01, 95% CI: 1.00–1.03, $p < 0.05$). Conversely, higher GNRI scores (OR = 0.94, 95% CI: 0.90–0.98, $p < 0.05$) and serum magnesium levels (OR = 0.47, 95% CI: 0.28–0.77, $p < 0.05$) were independently associated with a reduced risk of in-hospital mortality.

All statistically significant variables ($p < 0.05$) were highlighted in yellow in the forest plot, with red indicating risk factors (OR > 1) and green indicating protective factors (OR < 1). The arrow symbol denotes odds ratios exceeding the upper limit of the x-axis (OR > 3.0).

ROC curves of training and validation sets

The receiver operating characteristic (ROC) curves

Table 1: Baseline clinical characteristics of patients

Variables	Total (n=746)	Survival (n=521)	Death (n=225)	p
Demographic and lifestyle characteristics				
Gender, n (%)				0.763
Female	380 (51)	263 (50)	117 (52)	
Male	366 (49)	258 (50)	108 (48)	
Admission age, median (Q1,Q3)	73.56 (66.88, 80.85)	73.12 (66.59, 80.49)	74.45 (68.13, 81.27)	0.051
Smoker, n (%)				0.824
NO	716 (96)	499 (96)	217 (96)	
YES	30 (4)	22 (4)	8 (4)	
Alcohol abuse n (%)				1
NO	742 (99)	518 (99)	224 (100)	
YES	4 (1)	3 (1)	1 (0)	
Nutrition				
GNRI, median (Q1,Q3)	87.54 (84.09, 89.8)	88 (84.8, 90.31)	86.43 (82.65, 88.92)	< 0.001
Comorbidities				
AF, n (%)				0.127
NO	499 (67)	358 (69)	141 (63)	
YES	247 (33)	163 (31)	84 (37)	
Pneumonia, n (%)				1
NO	694 (93)	485 (93)	209 (93)	
YES	52 (7)	36 (7)	16 (7)	
UTI, n (%)				0.287
NO	675 (90)	467 (90)	208 (92)	
YES	71 (10)	54 (10)	17 (8)	
GIB, n (%)				0.02
NO	701 (94)	497 (95)	204 (91)	
YES	45 (6)	24 (5)	21 (9)	
ICH, n (%)				1
NO	644 (86)	450 (86)	194 (86)	
YES	102 (14)	71 (14)	31 (14)	
AKI, n (%)				0.909
NO	66 (9)	47 (9)	19 (8)	
YES	680 (91)	474 (91)	206 (92)	
Sepsis, n (%)				< 0.001
NO	207 (28)	170 (33)	37 (16)	
YES	539 (72)	351 (67)	188 (84)	

Hypertension, n (%)				0.108
NO	406 (54)	273 (52)	133 (59)	
YES	340 (46)	248 (48)	92 (41)	
Diabetes, n (%)				0.091
NO	457 (61)	330 (63)	127 (56)	
YES	289 (39)	191 (37)	98 (44)	
Congestive heart failure, n (%)				0.026
NO	477 (64)	347 (67)	130 (58)	
YES	269 (36)	174 (33)	95 (42)	
Renal disease, n (%)				0.018
NO	552 (74)	399 (77)	153 (68)	
YES	194 (26)	122 (23)	72 (32)	
Chronic pulmonary disease, n (%)				0.561
NO	592 (79)	410 (79)	182 (81)	
YES	154 (21)	111 (21)	43 (19)	
Peripheral vascular disease, n (%)				0.498
NO	619 (83)	436 (84)	183 (81)	
YES	127 (17)	85 (16)	42 (19)	
Malignant cancer, n (%)				0.995
NO	668 (90)	466 (89)	202 (90)	
YES	78 (10)	55 (11)	23 (10)	
Rheumatic disease, n (%)				0.39
NO	715 (96)	502 (96)	213 (95)	
YES	31 (4)	19 (4)	12 (5)	
Scores				
APSIII, Median (Q1,Q3)	49 (36, 65)	46 (34, 62)	55 (44, 73)	< 0.001
SAPSII, Median (Q1,Q3)	42 (34, 52.75)	41 (32, 50)	46 (39, 59)	< 0.001
SOFA, Median (Q1,Q3)	7 (5, 10)	7 (4, 9)	9 (6, 12)	< 0.001
GCS, Median (Q1,Q3)	9 (5, 13)	9 (6, 13)	6 (3, 12)	< 0.001
SIRS, Median (Q1,Q3)	3 (2, 3)	3 (2, 3)	3 (2, 4)	< 0.001
LODS, Median (Q1,Q3)	6 (4, 8)	5 (3, 8)	7 (5, 9)	< 0.001
Charlson, Median (Q1,Q3)	7 (6, 9)	7 (5, 9)	7 (6, 9)	0.208
MELD, Median (Q1,Q3)	13 (9, 20)	12 (9, 18)	16 (10, 23)	< 0.001
OASIS, Median (Q1,Q3)	37 (31, 43)	36 (30, 42)	39 (33, 44)	< 0.001
AKI Score, n (%)				< 0.001
0	66 (9)	47 (9)	19 (8)	
1	99 (13)	80 (15)	19 (8)	
2	294 (39)	220 (42)	74 (33)	
3	287 (38)	174 (33)	113 (50)	

Interventions				
Invasive Vent, n (%)				< 0.001
NO	192 (26)	168 (32)	24 (11)	
YES	554 (74)	353 (68)	201 (89)	
CPR, n (%)				0.465
NO	737 (99)	516 (99)	221 (98)	
YES	9 (1)	5 (1)	4 (2)	
CRRT, n (%)				< 0.001
NO	656 (88)	479 (92)	177 (79)	
YES	90 (12)	42 (8)	48 (21)	
RRT, n (%)				< 0.001
NO	631 (85)	462 (89)	169 (75)	
YES	115 (15)	59 (11)	56 (25)	
tPA, n (%)				1
NO	721 (97)	504 (97)	217 (96)	
YES	25 (3)	17 (3)	8 (4)	
EVT, n (%)				0.261
NO	723 (97)	502 (96)	221 (98)	
YES	23 (3)	19 (4)	4 (2)	
PTA or PTAS, n (%)				0.674
NO	740 (99)	516 (99)	224 (100)	
YES	6 (1)	5 (1)	1 (0)	
Laboratory tests				
Temperature first, median (Q1, Q3)	36.72 (36.44, 37.06)	36.72 (36.44, 37.06)	36.78 (36.44, 37.11)	0.484
Heart rate first, median (Q1, Q3)	84 (72, 98)	83 (72, 97)	87 (73, 101)	0.08
Resp rate first, median (Q1, Q3)	18 (16, 22)	18 (16, 22)	19 (16, 23)	0.037
SBP first, median (Q1, Q3)	130 (111, 150)	131 (112, 151)	129 (109, 149)	0.384
DBP first, median (Q1, Q3)	68 (58, 81.75)	69 (58, 84)	67 (57, 79)	0.076
MBP first, median (Q1, Q3)	87 (75, 100)	87 (76, 101)	85 (72, 100)	0.164
WBC first, median (Q1, Q3)	11 (8, 15.38)	10.8 (C7.9, 14.5)	12.2 (8.4, 17.7)	< 0.001
Platelet first, median (Q1, Q3)	194 (142, 262.5)	196 (143, 258)	187 (133, 267)	0.478
RBC first, median (Q1, Q3)	3.73 (3.09, 4.25)	3.78 (3.16, 4.28)	3.56 (2.87, 4.14)	0.016
Albumin first, median (Q1, Q3)	3.1 (2.7, 3.6)	3.2 (2.8, 3.7)	3 (2.5, 3.4)	< 0.001
Creatinine first, median (Q1, Q3)	1.1 (0.8, 1.6)	1 (0.8, 1.4)	1.2 (0.9, 2)	< 0.001
BUN first, median (Q1, Q3)	21 (15, 35)	20 (14, 31)	24 (18, 43)	< 0.001
Anion gap first, median (Q1, Q3)	14 (12, 17)	14 (12, 17)	15 (12, 18)	0.007
Calcium total, first, median (Q1, Q3)	8.45 (8, 9)	8.5 (8, 9)	8.4 (7.8, 8.9)	0.047
PT, first, median (Q1, Q3)	13.8 (12.2, 16.2)	13.6 (12.1, 15.7)	14.3 (12.4, 17.5)	< 0.001
PTT, first, median (Q1, Q3)	29.85 (26.13, 36.4)	29.3 (26, 35.4)	31.6 (26.5, 38.5)	0.007

INR, first, median (Q1, Q3)	1.2 (1.1, 1.5)	1.2 (1.1, 1.4)	1.3 (1.1, 1.6)	< 0.001
Chloride, first, median (Q1,Q3)	104 (100, 107)	104 (100, 107)	103 (99, 107)	0.065
Glucose, first, median (Q1, Q3)	138 (109, 183)	135 (107, 176)	144 (114, 198)	0.038
Fasting blood glucose, first, median (Q1, Q3)	136(108,175.75)	132 (106, 169)	144 (117, 194)	0.001
Potassium, first, median (Q1, Q3)	4.1 (3.7, 4.57)	4 (3.7, 4.5)	4.1 (3.8, 4.6)	0.021
Sodium, first, median (Q1, Q3)	139 (136, 142)	139 (136, 142)	139 (136, 142)	0.789
Hemoglobin, first, median (Q1, Q3)	11.3 (9.4, 12.9)	11.5 (9.7, 13)	10.6 (8.9, 12.6)	< 0.001
Magnesium, first, median (Q1,Q3)	2 (1.7, 2.2)	1.9 (1.7, 2.2)	2 (1.7, 2.2)	0.46
Phosphate, first, median (Q1,Q3)	3.6 (3, 4.4)	3.6 (2.9, 4.3)	3.8 (3.1, 4.8)	0.002

GNRI, Geriatric Nutritional Risk Index; AF, Atrial Fibrillation; UTI, Urinary Tract Infection; GIB, Gastrointestinal Bleeding; ICH, Intracerebral Hemorrhage; AKI, Acute Kidney Injury; APSIII, Acute Physiology Score III; SAPSII, Simplified Acute Physiology Score II; SOFA, Sequential Organ Failure Assessment; GCS, Glasgow Coma Scale; SIRS, Systemic Inflammatory Response Syndrome; LODS, Logistic Organ Dysfunction System; Charlson index, Charlson Comorbidity Index; MELD, Model for End-Stage Liver Disease; OASIS, Oxford Acute Severity of Illness Score; Vent, Invasive Mechanical Ventilation; CPR, Cardiopulmonary Resuscitation; CRRT, Continuous Renal Replacement Therapy; RRT, Renal Replacement Therapy; tPA, Tissue Plasminogen Activator; EVT, Endovascular Thrombectomy; PTA/PTAS, Percutaneous Transluminal Angioplasty/Stenting; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; MBP, Mean Blood Pressure; WBC, White Blood Cell Count; RBC, Red Blood Cell Count; BUN, Blood Urea Nitrogen; PT, Prothrombin Time; PTT, Partial Thromboplastin Time; INR, International Normalized Ratio.

for both the training and validation sets (Figure 4) demonstrate the discriminative performance of the predictive model in distinguishing between positive and negative outcomes. The area under the curve (AUC) for the model is 0.774 (95% CI: 0.733–0.816), indicating moderate to good predictive accuracy. An AUC value in this range suggests that the model effectively differentiates between individuals with and without the outcome of interest, though there remains room

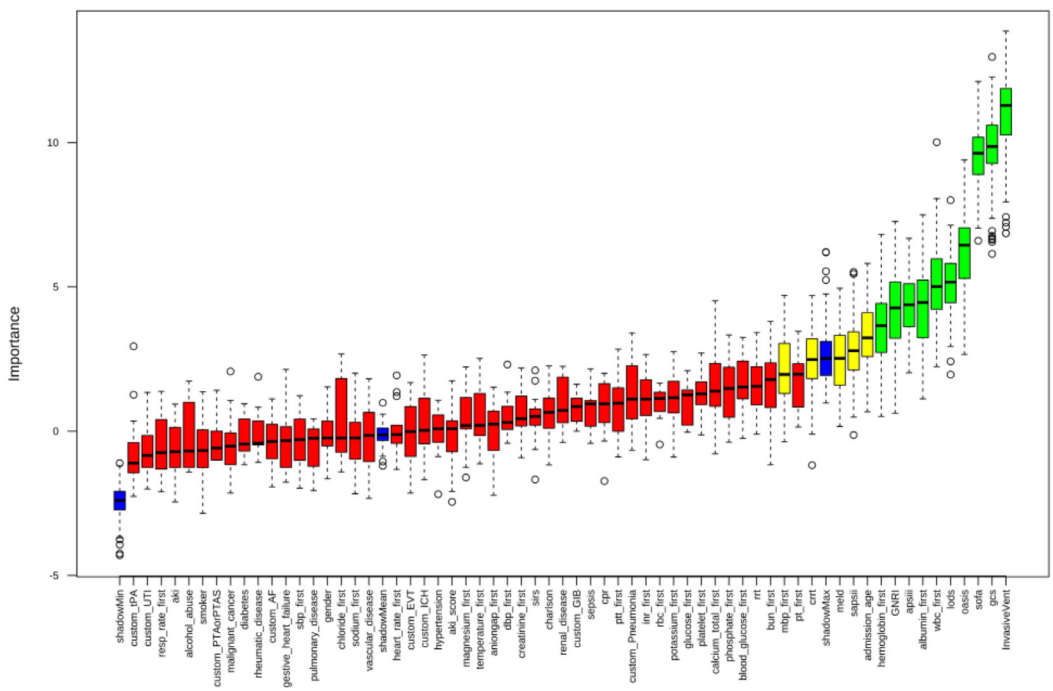

Figure 2. Boruta feature selection plot
Variables are ranked by importance score (Z-score); green = confirmed important, red = rejected, yellow = tentative, blue=shadow features.

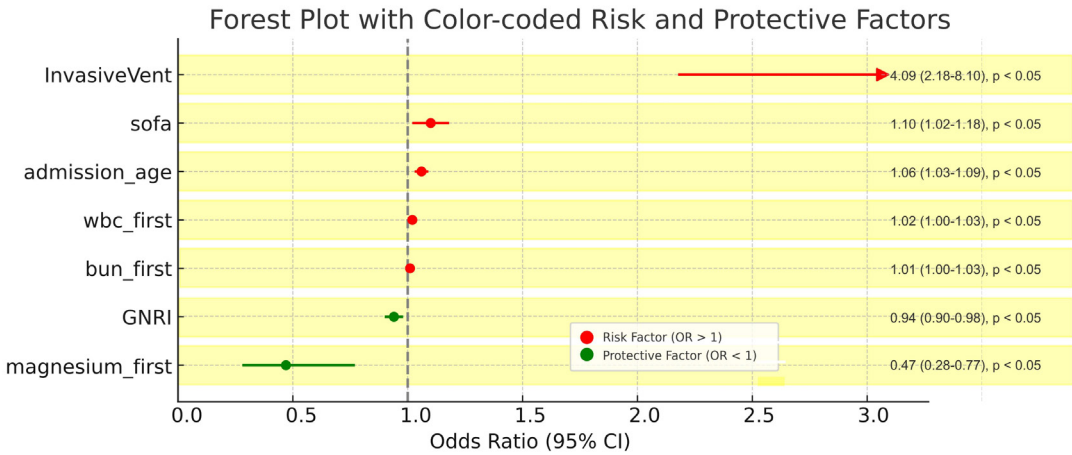

Figure 3. Forest plot illustrating the independent predictors of in-hospital mortality in elderly patients with cerebral infarction.

for improvement. The 95% confidence interval, which does not cross the null threshold of 0.5, confirms the statistical significance of the model's performance ($P < 0.001$). Notably, the sensitivity of 0.667 (95% CI: 0.590–0.744) highlights the model's ability to correctly identify approximately two-thirds of true positive cases, while the specificity (inferred from 1 – specificity values) reflects a balanced trade-off between minimizing false positives and maximizing true positives across varying thresholds.

The overlapping or comparable confidence intervals between the training and validation ROC curves suggest that the model generalizes well to unseen data, with no substantial evidence of overfitting. This consistency underscores the robustness of the model's predictive capacity. However, the relatively wide confidence intervals for both the AUC and sensitivity indicate some variability in performance, which may arise from sample heterogeneity or limited dataset size. Clinically, the model's moderate sensitivity and specificity support its potential utility as a screening tool, though further optimization or integration with additional biomarkers could enhance its diagnostic precision. These findings align with the need for reliable risk stratification tools in clinical settings, emphasizing the model's value in guiding early interventions for high-risk populations.

Calibration performance of training and validation sets

The calibration analysis for the training and

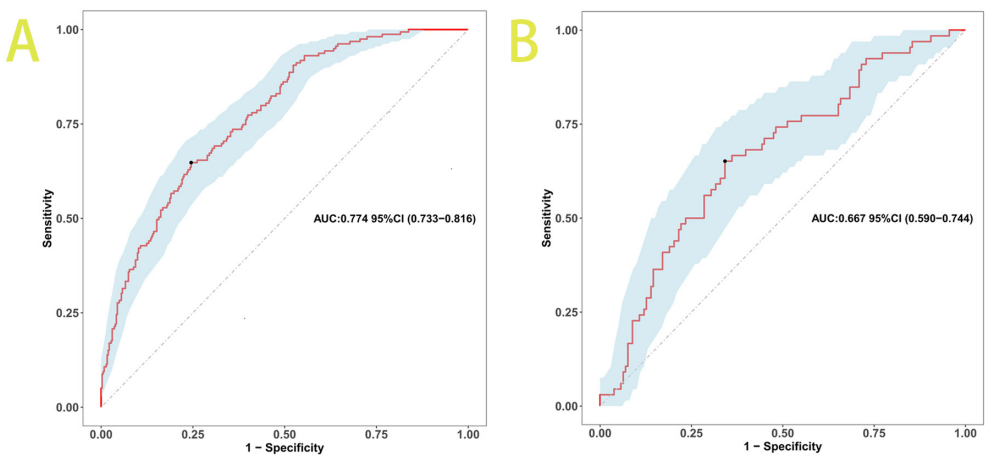

Figure 4. ROC curves of the mortality prediction model in elderly cerebral infarction patients: Training set A vs. Validation set B

validation sets (Figure 5) demonstrates the model's ability to align predicted probabilities with observed outcomes. For the training set (n=522), the mean absolute error (MAE) of 0.035 indicates a high level of calibration accuracy, with predictions deviating from actual probabilities by only 3.5% on average. This suggests that the model captures the underlying risk patterns effectively within the training cohort. In contrast, the validation set (n=224) exhibits a slightly higher MAE of 0.054, reflecting a moderate increase in prediction error (5.4%) when applied to unseen data. The divergence between training and validation MAE values may indicate subtle overfitting or differences in data distribution between the two cohorts. However, the relatively small magnitude of this discrepancy (ΔMAE=0.019) underscores the model's generalizability, particularly given the validation set's smaller sample size (n=224), which inherently amplifies variability in performance metrics.

Notably, the training set's "Apparent" curve shows minimal deviation from the bias-corrected line, reinforcing the model's stability.[16] Clinically, the MAE values (0.035–0.054) suggest acceptable calibration for risk stratification, though refinement may be warranted for applications requiring high precision (e.g., individualized treatment thresholds). Future work should prioritize external validation in larger, independent cohorts to confirm these results and explore strategies to further reduce validation set error, such as feature engineering or ensemble methods. Additionally, the bootstrap methodology (B=500) enhances confidence in the bias correction process, ensuring that the model's performance estimates are both reliable and reproducible.[17]

The association between GNRI and in-hospital mortality risk

The restricted cubic spline (RCS) analysis (Figure 6) elucidates the dose-response relationship between the Geriatric Nutritional Risk Index (GNRI) and in-hospital mortality risk. The results demonstrated a statistically significant nonlinear inverse association overall (P for overall <0.001), though the nonlinearity test did not reach significance (P=0.654), suggesting a predominantly linear trend. The mortality risk increased steeply with decreasing GNRI below the threshold of approximately 82.3, suggesting that even modest degrees of nutritional impairment may be clinically relevant in this population. Together, these findings underscore the independent and continuous prognostic value of GNRI in elderly patients with cerebral infarction. Nutritional status, as measured by GNRI, not only complements conventional severity scores but may serve as a modifiable target for early intervention.

DISCUSSION

Main findings

In this study, we identified several independent predictors of in-hospital mortality among elderly patients with cerebral infarction. Our multivariable logistic regression analysis revealed that higher SOFA scores, advanced age at admission, elevated white blood cell (WBC) counts, increased blood urea nitrogen (BUN) levels, and the requirement for invasive mechanical ventilation were all significantly associated with a heightened risk of in-hospital death. These findings are consistent with previous research highlighting the prognostic

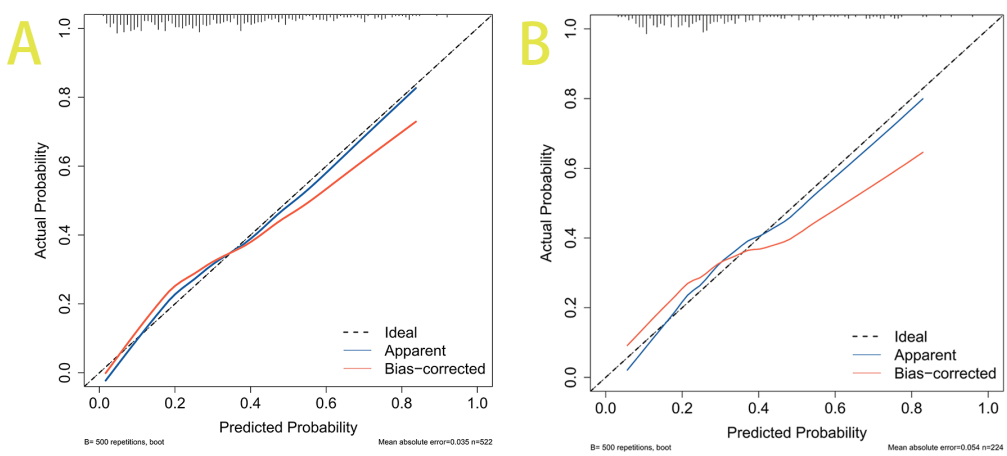

Figure 5. Calibration curves of the mortality prediction model in training set A and validation set B

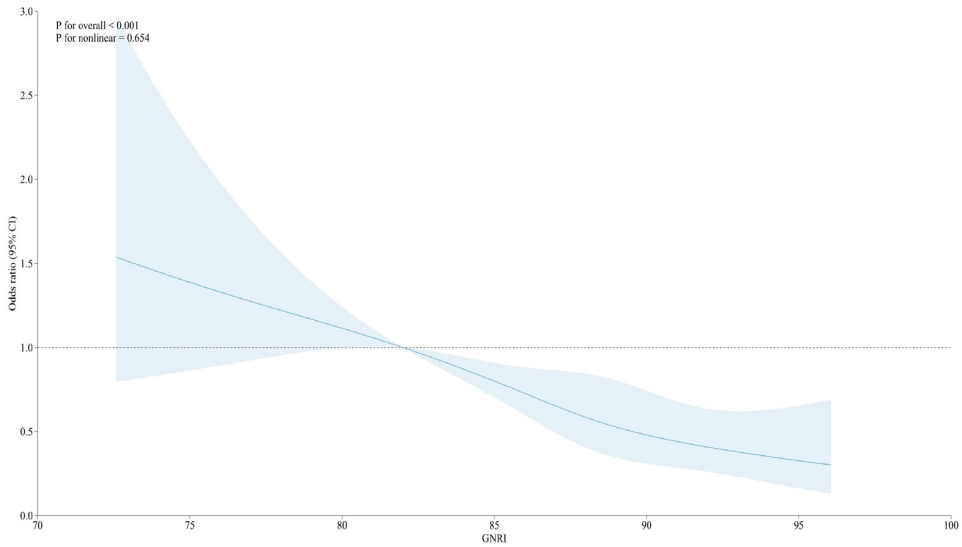

Figure 6. Association between GNRI and in-hospital mortality risk using restricted cubic spline analysis.

value of systemic illness severity, inflammatory burden, and organ dysfunction in the acute phase of ischemic stroke.[18,19]

Among all predictors, invasive ventilation exhibited the strongest association with mortality (OR: 4.09, 95% CI: 2.18–8.30, $p < 0.001$), suggesting that patients requiring ventilatory support are at markedly higher risk, potentially due to underlying respiratory failure or severe neurological impairment. Furthermore, age and SOFA score—both established indicators of frailty and systemic disease severity—demonstrated a graded relationship with mortality, where even modest increases were associated with worse outcomes. The inclusion of first-day laboratory parameters such as WBC and BUN further underscores the importance of early systemic assessment upon ICU admission.

Despite their clinical relevance, several complications were not retained in the final multivariable model:

1. *UTI (Urinary Tract Infection):* UTI was not significantly associated with mortality, likely due to its typically mild nature and good response to antibiotic treatment, especially when limited to the lower urinary tract. Unless complicated by sepsis or acute kidney injury, UTI alone contributes little to mortality. Additionally, its pathophysiological overlap with broader systemic indicators such as sepsis, SOFA score, or need for mechanical ventilation may have rendered it redundant in multivariate modeling.
2. *GIB (Gastrointestinal Bleeding):* The absence of GIB from the final model may be attributed to its low incidence in our cohort and to the mild severity of many events, such as subclinical bleeding or occult blood loss, which may not impact hemodynamic stability. Furthermore, GIB often results from antithrombotic therapy rather than the underlying disease burden and may therefore be outperformed by stronger indicators such as SOFA or GNRI in explaining mortality risk.
3. *ICH (In-hospital Intracerebral Hemorrhage):* ICH was a rare but serious complication in our cohort, with an incidence of less than 2%. Such low frequency limited its statistical power. Additionally, many ICH events occurred shortly before death, making them terminal rather than causative. Moreover, bleeding risk and neurological deterioration may have been sufficiently captured by variables like tPA use, SOFA, and GCS, thereby reducing the added value of including ICH in the model.[20]

Importantly, our findings highlight the critical role of nutritional status, as quantified by the Geriatric Nutritional Risk Index (GNRI), in determining short-term mortality. Restricted cubic spline analysis revealed a near-linear inverse relationship between GNRI and mortality, with each 10-point decrease in GNRI corresponding to a 200% increase in adjusted odds of death.[21] This dose-response relationship emphasizes the clinical value of early nutritional screening and intervention—particularly for patients with GNRI < 82.3, where mortality risk rises sharply.

The model demonstrated good discriminative performance across both the training and validation cohorts (AUC: 0.774; MAE: 0.035–0.054), supporting its potential utility as a clinical decision tool. Furthermore, decision curve analysis showed that the model provided a superior net benefit across a range of threshold probabilities (0.2–0.5), compared to treat-all or treat-none strategies. This suggests its potential for individualized risk stratification and resource allocation.[22]

Nevertheless, several limitations should be acknowledged. First, the retrospective design and reliance on secondary data from the MIMIC-IV database—common to many ICU-based studies—may introduce selection bias and limit the generalizability of our findings beyond the critical care setting. For example, in a study examining the association between furosemide administration and outcomes in patients with sepsis-associated acute kidney injury receiving renal replacement therapy, data were derived from the MIMIC-IV cohort, which exclusively represents patients admitted to the ICUs of the Beth Israel Deaconess Medical Center. This specific institutional and geographic context may not reflect the broader healthcare landscape, thereby affecting the external applicability of the results.[23]

Second, residual confounding remains a concern in studies of ischemic stroke outcomes, particularly when key variables are unavailable. For instance, stroke subtype is a critical determinant of both pathophysiology and treatment response, especially regarding interventions such as thrombolysis and thrombectomy. Likewise, infarct volume, often measured via diffusion-weighted MRI (DWI), is strongly associated with neurological prognosis; larger infarcts are consistently linked to worse functional recovery at 90 days post-stroke.[24]

Third, while baseline assessments of GNRI and magnesium levels offer valuable insights into nutritional and electrolyte status, they fail to capture the dynamic changes that may occur throughout hospitalization. In dialysis populations, for example, ionized magnesium levels are known to exhibit significant intra- and inter-individual variability influenced by age, nutritional intake, and even seasonal variation. This highlights the potential limitations of relying on a single time-point measurement and suggests that longitudinal monitoring could better inform prognosis.[25]

Finally, the observational nature of this study precludes any inference of causality between the identified predictors and in-hospital mortality. While associations are robust, prospective interventional studies are needed to validate these findings and establish causative pathways.

This study has several notable strengths. First, to the best of our knowledge, it is one of the few investigations to assess the prognostic value of the Geriatric Nutritional Risk Index (GNRI) in predicting in-hospital mortality among elderly patients with cerebral infarction using data from the MIMIC-IV database.[26] Second, by integrating nutritional indicators, vital signs, laboratory parameters, and critical care interventions into a single multivariable logistic regression model, we achieved strong predictive performance (AUC = 0.774) and good calibration in both the training and validation cohorts.

Third, the use of restricted cubic spline analysis revealed a nearly linear inverse association between GNRI and mortality, with each 10-point decrease in GNRI corresponding to an approximately threefold increase in the odds of in-hospital death. Fourth, decision curve analysis confirmed the model's clinical applicability by demonstrating a consistent net benefit across a wide range of threshold probabilities. Finally, our findings highlight that common ICU complications such as urinary tract infection, gastrointestinal bleeding, and in-hospital intracerebral hemorrhage were not independently associated with mortality in the multivariate context. This underscores the predominant role of early systemic illness severity and nutritional status in effective risk stratification for elderly stroke patients.

Further research is warranted to validate our findings in larger, prospective, multicenter cohorts, particularly those that include patients from general wards and more diverse clinical settings. In addition, interventional studies are needed to determine whether optimizing nutritional status or correcting electrolyte imbalances can effectively reduce in-hospital mortality among elderly patients with cerebral infarction. Moreover, the incorporation of dynamic nutritional and biochemical indices—rather than static baseline values—may enhance model accuracy and enable real-time clinical decision-making in acute care settings.

In conclusion, this study identified key predictors of in-hospital mortality in elderly patients with cerebral infarction. Invasive mechanical ventilation, advanced age, higher SOFA scores, and elevated levels of white blood cells and blood urea nitrogen were independently associated with increased mortality risk. In

contrast, better nutritional status, as measured by the Geriatric Nutritional Risk Index (GNRI), and higher baseline serum magnesium levels were protective factors. These findings underscore the importance of comprehensive clinical, nutritional, and biochemical assessment upon admission. Incorporating GNRI and magnesium into routine evaluation may offer practical and cost-effective strategies for early risk stratification and targeted intervention in this high-risk population.

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DISCLOSURE

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