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ORIGINAL RESEARCH

Neutrophil-Percentage-to-Albumin Ratio and Monocyte-to-Albumin Ratio: Utility of the Combined Biomarkers in the Prediction of 30-Day Mortality in Male Diabetes Patients Over 80 Years Old with Bloodstream Infections

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Background: Elderly patients with diabetes mellitus (DM) are highly vulnerable to bloodstream infections (BSI), which lead to a significantly high risk of mortality. Novel inflammatory markers that attach to albumin, such as Neutrophil-percentage-to-albumin ratio (NPAR), C-reactive protein-to-albumin ratio (CAR), Monocyte-to-albumin ratio (MAR), Red blood cell distribution width-to-albumin ratio (RAR), Prognostic Nutritional Index (PNI) and C-reactive protein-albumin-lymphocyte index (CALLY index), have proven to be effective in predicting infections. However, the association between these factors and BSI in diabetes patients over 80 years old still remains to be fully elucidated.

Purpose: To investigate the predictive value of the novel inflammatory markers NPAR, CAR, MAR, RAR, PNI and CALLY index for the prognosis in male diabetes patients over 80 years old with bloodstream Infections of 30-day mortality.

Patients and Methods: Male diabetes patients over 80 years old who diagnosed with BSI were recruited between January 2012 and January 2025. Receiver operating characteristic (ROC) curves, Kaplan-Meier curves (K-M curves) and Cox regression analysis were used to investigate the association between these factors and 30-day mortality.

Results: A total of 350 diabetes patients with BSI were recruited in the study, with an average age of 89.77±8.62 years. There are 55 deaths within 30 days, with an all-cause mortality rate of 15.71%. After adjusting for multiple factors, the NPAR2 and MAR2 groups still correlated with 30 days mortality. (NPAR2 HR, 95% CI: 2.738, 1.058–7.084, P= 0.038; MAR2 HR, 95% CI: 1.871, 1.012–3.458, P=0.046). The CAR, RAR, PNI and the CALLY index showed no significant differences in COX regression.

Conclusion: NPAR and MAR are more sensitive markers in male diabetes patients over 80 years old with BSI and can provide a reference for predicting 30-day mortality in patients with BSI.

Keywords: inflammatory markers, neutrophil percentage-to-albumin ratio, monocyte-to-albumin ratio, bloodstream infections, elder, mortality

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Introduction

With the accelerating global aging trend and rising prevalence of diabetes, elderly patients with diabetes have become a key population susceptible to bloodstream infections (BSI). The risk of BSI in patients with diabetes is significantly higher owing to immune dysfunction, microcirculation disorders, and complications with multiple chronic diseases.[1,2]

BSI in patients with diabetes are associated with poor clinical outcomes and high mortality rates. A study involving 2551 patients showed that diabetes significantly increased the risk of bloodstream infections (OR=1.42).[3] Hyperglycemia is associated with all-cause ICU mortality in patients with sepsis, and the mean blood glucose level is significantly correlated with higher mortality of BSI in critically ill patients.[4,5] Research also suggests that patients with diabetes have a higher 30-day mortality rate of BSI compared to patients without diabetes.[6] These data highlight the vulnerability of mortality in elderly patients with diabetes to BSI.

Serum albumin, the most abundant plasma protein in the human body, not only maintains colloid osmotic pressure but also possesses multiple biological activities, including anti-inflammatory, antioxidant, anticoagulant, and anti-aggregation.[7] Increasing evidence in recent years indicates that hypoalbuminemia is closely associated with the severity and prognosis of various inflammatory diseases. A multicenter cohort study further indicated that dynamic changes in serum albumin levels have greater clinical significance than a single measurement.[8] Recently, studies have found that novel inflammatory indicators which combine of traditional inflammatory indicators and serum albumin have emerged as promising predictors for inflammatory diseases. For instance, the Neutrophil percentage-to-albumin ratio (NPAR) has been used as a predictive indicator for systemic inflammation in conditions such as acute kidney injury, septic shock, and stroke-related infections.[9,10] Research on hospitalized patients with bacteremic bloodstream infections has indicated that the C-reactive protein-to-albumin ratio (CAR) was associated with higher mortality.[11] Prognostic Nutritional Index (PNI) and its derived indicators have also been reported as important prognostic predictors for bloodstream infections in patients over 65 years old.[12] Other markers, such as Monocyte-to-albumin ratio (MAR), Red blood cell distribution width to albumin ratio (RAR), CRP-albumin-lymphocyte index (CALLY index), have also shown significant associations with infectious diseases.[13–16] Our research team has previously demonstrated the prognostic role of NPAR in elderly patients aged over 60 with BSI.[17]

The above indicators include common inflammatory markers such as neutrophils, lymphocytes, monocytes, red blood cell distribution width, and CRP (C-reactive protein), as well as a comprehensive indicator. These factors can comprehensively represent different types of inflammatory conditions associated with albumin. However, the comparative studies of the predictive value of these novel albumin-related inflammatory markers for BSI remain unclear, and there is a lack of research evidence in patients over 80 years old with diabetes.

This study aimed to systematically investigate the predictive value of novel inflammatory markers, such as NPAR, CAR, MAR, RAR, PNI and the CALLY index, for the prognosis of BSI in hospitalized diabetes patients over 80 years old. For predicting prognosis, we chose 30-day mortality rate as it can effectively reflect the acute phase outcome of BSI and aligns with previous research choices. This study is the first to explore the predictive value of inflammation-related albumin indicators for 30-day mortality of BSI in patients with diabetes aged over 80 years old. We also sought to develop a risk stratification tool for this specific population to facilitate early identification of high-risk patients. The ultimate goal is to provide clinicians with simple, cost-effective, and reliable prognostic assessment indicators to support clinical decision making and optimize treatment strategies.

Material and Methods

Study Population and Definition

This study recruited elderly male patients with diabetes mellitus and concomitant bloodstream infections who were hospitalized at the Second Medical Center of the PLA General Hospital between January 2012 and January 2025. Informed consent was waived by ethic committee for this study was a retrospective analysis and the patients' private information was not included. This study was approved by the ethics committee of the Chinese PLA General Hospital (S2024-359-02). This study was conducted in accordance with the principles of the Declaration of Helsinki.

Inclusion criteria: 1) age ≥ 80 years; 2) conforming to the diagnostic criteria for diabetes: having a history of diabetes or a blood test meeting the WHO diagnostic criteria for diabetes; 3) diagnosis of bloodstream infections, clear pathogenic evidence, and elimination of contamination; and 4) due to the characteristics of the population and the gender imbalance, only male patients were included in the screening.

The exclusion criteria were as follows: 1) contamination based on clinical data; 2) severely incomplete clinical data; 3) female sex.

Blood flow infection: 1) Patients present with one or more clinical symptoms or signs, such as fever (body temperature>38 °C), hypothermia (body temperature<36 °C), chills, hypotension, or hyperprolactinemia. (2) At least one blood culture result is positive.

Definition of the Markers

All novel inflammatory markers are grouped according to the third quartile.

(1) NPAR (Neutrophil-percentage-to-albumin ratio)

NAPR=Neutrophil percentage * 100/albumin
NPAR0: <2.18, NPAR1: 2.18–2.61, NPAR2: ≥ 2.61

(2) CAR (C-reactive protein-to-albumin ratio)

CAR=C-reactive protein/albumin
CAR0: <0.07, CAR1: 0.07–0.23, CAR2: ≥ 0.23

(3) MAR (Monocyte-to-albumin ratio)

MAR= Monocyte count * 100/albumin
MAR0: <0.90, MAR1: 0.90–1.75, MAR2: ≥ 1.75

(4) RAR (Red blood cell distribution width-to-albumin ratio)

RAR=Red blood cell distribution width/albumin
RAR0: <0.43, RAR1: 0.43–0.51, RAR2: ≥ 0.51

(5) CALLY index (CRP-albumin-lymphocyte index)

CALLY index =Albumin * lymphocyte count/ (C-reactive protein * 10)
CALLY0: <0.35, CALLY1: 0.35–1.38, CALLY2: ≥ 1.38

(6) PNI (Prognostic Nutritional Index)

PNI= Albumin + 5* lymphocyte count
PNI0: <36.94, PNI1: 36.94–41.81, PNI2: ≥ 41.81

Data Collection

Data were obtained from the hospital`s electronic medical records. Demographic, clinical and laboratory data as well as information on comorbidities and deaths were extracted. Laboratory data included measurements of neutrophils, albumin, serum creatinine (Scr), blood urea nitrogen (BUN), brain natriuretic peptide (BNP), C-reactive protein (CRP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood urea nitrogen (BUN), lactate, platelets (PLT), white

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blood cells (WBC), and red cell distribution width (RDW), all of which were obtained on the first day of BSI. The endpoint of the study was the 30-day mortality, namely the death occurring from any cause during hospitalization.

Statistical Analysis

All analyses were performed using SPSS 23.0 software. All tests were two-sided, and statistical significance was set at $P < 0.05$. Continuous variables are expressed as mean±SD or medians and interquartile ranges (IQR), and differences were analyzed using one-way ANOVA (data with normal distributions) and Kruskal–Wallis tests (data with skewed distributions). Categorical variables are presented as numbers and percentages and were analyzed using the $\chi 2$ test. Cox proportional hazard models were used to examine the association between factors-related mortality. Models included Model 1 adjusted for none variables; Model 2 adjusted for age, smoking, and drinking; Model 3 was further adjusted for multiple variables that were statistically significant in the univariate analysis based on Model 2. Multivariate analysis results are shown as hazard ratios (HRs) with 95% confidence intervals (CI), with tertile 1 serving as the reference group. The Kaplan-Meier method was used for survival curve plotting. Differences in survival between the groups were compared using the Log rank test. Interactions and subgroup analyses were also performed.

Results

Patient Characteristics

A total of 350 participants were included in this study. The average age was 89.77±8.62 years, with an average duration of diabetes 10.44±9.21 years and an average of length of hospital stay of 84.55±32.6 days. Among them, 122 patients (34.9%) had multiple infections. There were 55 deaths within 30 days, resulting in an all-cause mortality rate of 15.71%.

Most participants had more than one type of comorbidity, including tracheal cannula (178 patients, 50.9%), deep vein catheterization (DVC) (310 patients, 88.6%), and surgical intervention (148 patients, 42.3%). Frequently observed comorbidities included hypertension (272 patients, 77.7%), coronary heart disease (CHD) (174 patients,49.7%), chronic kidney disease (CKD) (87 patients, 24.9%), congestive heart failure (CHF) (59 patients, 16.9%), and chronic obstructive pulmonary disease (COPD) (83 patients, 23.7%).

Comparison Between Survival and Non-Survival Groups

Baseline characteristics revealed statistically significant differences between survivors and non-survivors in age; use of tracheal cannula; length of hospital stay; whether surgery was performed; multiple infections; and laboratory parameters such as WBC, neutrophils, monocytes, potassium, albumin, amylase, lipase, NPAR, and MAR. There were no statistical differences in average age and the duration of diabetes between the survivors and non- survivor group. No significant differences were found in indicators of CAR, RAR, CALLY indices or PNI between the two groups, indicating that these indicators may not be correlated with mortality. (Table 1)

Table 1 Comparison of Baseline Characteristics Between Survivor and Non-Survivor Patients with BSI at 30-Day

Characteristics	**All Patients (n=350)**	**Survivors (n=295)**	**Non-Survivors (n=55)**	***P* value**
Age, years	89.77±8.62	86.81±10.80	90.31±8.06	0.007
Diabetes duration, years	10.44±9.21	10.47±9.27	10.29±8.98	>0.05
Tracheal cannula, %	178(50.9)	161(54.6)	17(30.9)	0.005
DVC, %	310(88.6)	265(89.8)	45(81.8)	>0.05
Hospitalization, days	84.55±32.6	89.43±31.51	58.38±25.10	<0.001
Surgery, %	148(42.3)	115(39.0)	33(60.0)	0.003
Multiple infections, %	122(34.9)	111(37.6)	11(20.0)	0.007

(*Continued*)

Table 1 (Continued).

Characteristics	All Patients (n=350)	Survivors (n=295)	Non-Survivors (n=55)	*P* value
Hypertension, %	272(77.7)	229(77.6)	43(78.2)	>0.05
CHD, %	174(49.7)	142(48.1)	32(58.2)	>0.05
CKD, %	87(24.9)	73(24.7)	14(25.5)	>0.05
CHF, %	59(16.9)	49(16.6)	10(18.2)	>0.05
COPD, %	83(23.7)	69(23.4)	14(25.5)	>0.05
WBC, $\times 10^9$ /L	10.61±7.22	10.00±6.60	13.87±9.30	<0.001
Neutrophil, %	0.81±0.14	0.80±0.14	0.84±0.10	0.022
RDW, %	16.14±2.42	16.12±2.46	15.95±2.22	>0.05
Lymphocytes, $\times 10^9$ /L	0.86(0.52,1.47)	0.86(0.52,1.47)	0.90(0.54,1.22)	>0.05
Monocytes, $\times 10^9$ /L	0.86(0.52,1.47)	0.42(0.25,0.62)	0.57(0.29,0.87)	0.038
Hemoglobin, g/dL	110.78±20.13	110.68±20.00	111.27±21.00	>0.05
PLT, $\times 10^9$ /L	147.00(101.75,202.25)	147.00(105.00,202.00)	165.00(108.00,200.00)	>0.05
CRP, mg/L	4.62(1.92,9.45)	4.35(1.51,9.39)	5.59(5.04,9.09)	>0.05
Potassium, mmol/L	4.33±0.57	4.36±0.59	4.16±0.4	0.016
Sodium, mmol/L	137.27±6,72	137.34±6.80	136.87±6.34	>0.05
Glucose, mg/dL	9.30±3.93	9.09±4.21	9.27±3.97	>0.05
Albumin, g/dL	34.31±5.09	34.57±5.28	34.31±5.09	0.029
BUN, mg/dL	10.20(6.80,14.95)	10.0(6.65,14.55)	9.7(7.4,16.6)	>0.05
Scr, μmol/L	84.0(59.0,123.0)	85.0(59.5,122.5)	80.0(55.0,127.0)	>0.05
ALT, mmol/L	17.00(11.00,31.58)	17.00(10.85,30.30)	15.00(12.30,28.00)	>0.05
AST, mmol/L	26.70(18.02,52.75)	26.3(18.0,52.5)	26.4(18.6,46.1)	>0.05
Amylase, mmol/L	43.0(31.0,69.5)	43.0(31.0,64.0)	51.0(33.0,81.0)	0.010
Lipase, mmol/L	27.1(16.0,47.2)	28.0(16.0,46.9)	25.6(16.3,39.0)	<0.001
CAR	0.142(0.057,0.297)	0.122(0.044,0.296)	0.166(0.101,0.287)	>0.05
NPAR	2.40(2.06,2.73)	2.35(2.03,2.70)	2.65(2.28,2.80)	0.003
CALLY	0.69(0.26,2.15)	0.74(0.27,2.60)	0.40(0.22,1.25)	>0.05
MAR	1.25(0.75,2.02)	1.21(0.73,1.90)	1.81(0.87,2.64)	0.028
RAR	0.464(0.403,0.541)	0.464(0.399,0.540)	0.465(0.425,0.539)	>0.05
PNI	39.26(35.20,43.82)	39.62(35.22,44.16)	37.84(35.30,40.52)	>0.05

Abbreviations: DVC, deep vein catheterization; CHD, coronary heart disease; CKD, chronic kidney disease; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; WBC, white blood cell; RDW, red cell distribution width; PLT, platelets; CRP, C-reactive protein; BUN, blood urea nitrogen; Scr, serum creatinine; ALT, alanine aminotransferase; AST, aspartate aminotransferase.

Stratification by NPAR and MAR Quartiles

Based on the results in Table 1, patients were stratified into quartiles according to their NPAR and MAR values. As shown in Table 2, significant differences were observed across NPAR quartiles in WBC, neutrophil, lymphocyte, hemoglobin, platelet, D-dimer, CRP, BUN, Scr, albumin, and 30-day survival time. Table 3 shows similar trends for MAR quartiles, especially 30-day survival time, indicating that both NPAR and MAR may serve as significant predictors of mortality.

Table 2 Comparison of Baseline Characteristics Between Different NAPR Group of Patients with BSI

Characteristics	NAPR0 (n=115)	NAPR1 (n=113)	NAPR2 (n=116)	*P* value
Age, years	90.81±7.60	89.15±9.39	89.28±8.95	>0.05
Diabetes duration, years	11.90±9.84	9.71±8.79	9.68±8.97	>0.05
Tracheal cannula, %	58(50.4)	58(51.3)	17(47.4)	>0.05
DVC, %	105(91.3)	110(88.5)	99(85.3)	>0.05

(*Continued*)

Table 2 (Continued).

Characteristics	NAPR0 (n=115)	NAPR1 (n=113)	NAPR2 (n=116)	*P* value
Hospitalization, days	82.07±26.76	87.36±31.93	83.34±38.41	>0.05
Surgery, %	49(42.6)	55(48.7)	43(37.1)	>0.05
Multiple infections, %	40(34.8)	39(34.5)	41(35.3)	>0.05
Hypertension, %	96(83.5)	89(78.8)	84(72.4)	>0.05
CHD, %	59(51.3)	60(53.1)	55(47.4)	>0.05
CKD, %	28(24.3)	15(15.0)	23(19.8)	>0.05
CHF, %	16(13.9)	22(19.5)	21(18.1)	>0.05
COPD, %	31(27.0)	21(18.6)	30(25.9)	>0.05
WBC, $\times 10^9$ /L	9.01±6.69	9.9±4.4	13.29±8.95	<0.001
Neutrophil, %	0.7±0.16	0.83±0.07	0.89±0.06	<0.001
RDW, %	15.95±2.29	16.17±2.33	16.37±2.67	>0.05
Lymphocytes, $\times 10^9$ /L	1.3(0.85,1.75)	0.8(0.44,1.2)	0.63(0.39,1)	0.005
Monocytes, $\times 10^9$ /L	0.44(0.31,0.68)	0.39(0.23,0.63)	0.45(0.25,0.72)	>0.05
Hemoglobin, g/dL	114.67±18.46	115.41±20.1	102.41±18.67	<0.001
Platelet, $\times 10^9$ /L	176(131.5,210.5)	147(106,209)	128(86,175)	0.001
D-dimer, ug/mL	2.63±3.11	3.47±4.03	4.34±3.72	0.003
C-pro, mg/L	2.67(0.97,5.02)	3.41(1.49,8.32)	8.94(4.95,15.2)	<0.001
Potassium, mmol/L	4.36±0.55	4.34±0.54	4.31±0.62	>0.05
Sodium, mmol/L	137.28±5.59	136.64±5.75	137.88±8.48	>0.05
Glucose, mg/dL	8.29±3.38	8.91±3.7	10.43±4.3	<0.001
Albumin, g/dL	38.35±4.63	34.79±3.09	30.22±3.2	<0.001
BUN, mg/dL	9.2(6.7,13.55)	8.95(6,12.45)	13.2(7.6,22.4)	<0.001
Scr, μmol/L	85(59,111)	80(59.5,113)	89(55,149)	0.008
ALT, mmol/L	15(10.75,25.75)	17(10,29.05)	18(12,40)	0.042
AST, mmol/L	24(18,35.1)	27.45(17,50.2)	30.8(20,70)	>0.05
Amylase, mmol/L	48(36.5,64.5)	41.5(31,63.5)	42(27,75)	>0.05
Lipase, mmol/L	30.5(17.75,46.65)	26.9(16.65,46.55)	25.6(14,46)	>0.05
30-day survival time, days	27.83±6.46	26.47±8.19	24.78±9.31	0.017

Abbreviations: DVC, deep vein catheterization; CHD, coronary heart disease; CKD, chronic kidney disease; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; WBC, white blood cell; RDW, red cell distribution width; PLT, platelets; CRP, C-reactive protein; BUN, blood urea nitrogen; Scr, serum creatinine; ALT, alanine aminotransferase; AST, aspartate aminotransferase.

Table 3 Comparison of Baseline Characteristics Between Different MAR Group of Patients with BSI

Characteristics	MAR0 (n=111)	MAR1 (n=116)	MAR2 (n=117)	*P* value
Age, years	89.12±8.5	90.03±8.81	90.15±8.58	>0.05
Diabetes duration, years	10.25±8.85	10.58±8.89	10.49±10.07	>0.05
Tracheal cannula, %	59(50.4)	57(50.9)	57(48.7)	>0.05
DVC, %	104(88.9)	101(87.1)	105(89.7)	>0.05
Hospitalization, days	86.32±36.4	87.97±29.51	79.39±31.09	>0.05
Surgery, %	51(43.6)	45(38.8)	52(44.4)	>0.05
Multiple infections, %	14(12.0)	15(12.9)	11(9.4)	>0.05
Hypertension, %	88(75.2)	89(76.)	95(81.2)	>0.05
CHD, %	46(39.3)	63(56.3)	65(55.6)	0.022
CKD, %	21(17.9)	24(20.7)	23(19.7)	>0.05
CHF, %	11(9.4)	24(20.7)	24(20.5)	0.031
COPD, %	24(20.5)	24(20.7)	35(29.9)	>0.05

(*Continued*)

Table 3 (Continued).

Characteristics	MAR0 (n=111)	MAR1 (n=116)	MAR2 (n=117)	*P* value
WBC, ×10^9 /L	8.38±7.02	9.47±3.74	13.93±8.7	<0.001
Neutrophil, %	0.82±0.15	0.79±0.11	0.8±0.15	0.022
RDW, %	16.07±2.56	16.27±2.3	16.1±2.42	>0.05
Lymphocytes, ×10^9 /L	0.61(0.39,1.08)	1(0.6,1.56)	0.99(0.56,1.57)	>0.05
Monocytes, ×10^9 /L	0.19(0.12,0.25)	0.43(0.36,0.51)	0.79(0.65,0.95)	<0.001
Hemoglobin, g/dL	112.32±23.68	110.3±18.1	109.71±18.17	>0.05
Platelet, ×10^9 /L	133(94,168)	144(106,192)	178.5(121.5,215.5)	<0.001
D-dimer, ug/mL	4.14±4.11	2.75±2.93	3.57±3.79	0.022
C-pro, mg/L	3.6(1.47,7.96)	3.56(1.81,8.14)	6.17(2.39,11.49)	0.002
Potassium, mmol/L	4.24±0.58	4.38±0.54	4.37±0.59	>0.05
Sodium, mmol/L	137.86±7.15	137.15±6.06	136.79±6.93	>0.05
Glucose, mg/dL	9.41±3.82	8.96±3.86	9.43±4.22	>0.05
Albumin, g/dL	35.67±5.72	34.85±4.79	32.36±4.09	<0.001
BUN, mg/dL	9(6.65,14.15)	10.1(6.4,14)	10.75(7.2,17.8)	>0.05
Scr, μmol/L	81(60.5,107)	83.5(61,120)	87.5(55,134.5)	0.023
ALT, mmol/L	20.7(12.9,40.75)	17(11,29)	14(9.6,22.7)	>0.05
AST, mmol/L	31(19,82.75)	27.2(18,49.2)	24(17.6,43.9)	>0.05
Amylase, mmol/L	48(31,81)	45.5(30,71)	41.5(31.5,54)	>0.05
Lipase, mmol/L	35(18.1,52)	27.1(15.5,43.9)	24.2(15.35,44.3)	>0.05
30-day survival time, days	27.08±7.32	27.45±6.7	24.73±9.72	0.020

Abbreviations: DVC, deep vein catheterization; CHD, coronary heart disease; CKD, chronic kidney disease; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; WBC, white blood cell; RDW, red cell distribution width; PLT, platelets; CRP, C-reactive protein; BUN, blood urea nitrogen; Scr, serum creatinine; ALT, alanine aminotransferase; AST, aspartate aminotransferase.

ROC Curves of Different Markers

Receiver operating characteristic (ROC) curves were used to evaluate and compare the predictive effects of the five indicators on 30-day mortality. As shown in Figure 1 and Table 4, CAR, NPAR, the CALLY index, and MAR showed

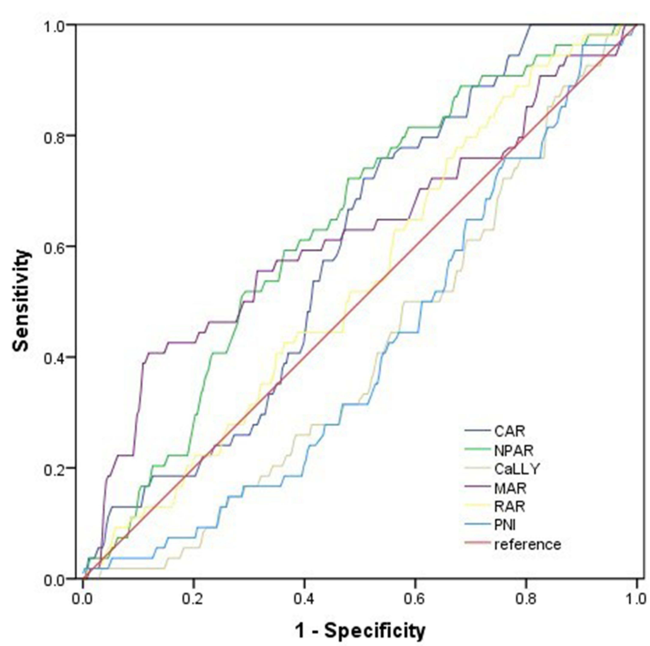

Figure 1 ROC curve for the predictive value of NPAR, CAR, MAR, RAR, PNI and CALLY index for 30-day mortality.

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Table 4 The Results of ROC Curve and Cut-off Value for CAR, NPAR, CALLY, MAR and RAR

	AUC	*P* value	Cut-off
CAR	0.592	0.031	0.101
NPAR	0.635	0.002	2.396
CALLY	0.409	0.033	0.084
MAR	0.620	0.005	2.437
RAR	0.538	0.372	0.384
PNI	0.411	0.038	31.245

statistically significant predictive abilities. NAPR and MAR were positively correlated with 30-day mortality (NPAR AUC=0.635, *P*=0.002, cut-off value=2.396; MAR AUC=0.620, *P*=0.005, cut-off value=2.437) and had a higher significant difference, while the predictive ability of CAR was relatively poor compared to NPAR and MAR (CAR AUC=0.592, *P*=0.031, cut-off value=0.101). The CALLY index and PNI were negatively correlated with 30-day mortality rate (CALLY AUC=0.408, *P*=0.031, cut-off value=0.084; PNI AUC=0.411, *P*=0.038, cut-off value=31.245). The remaining indicator, RAR, did not exhibit statistically significant predictive value.

Regression Analysis of Kaplan-Meier Curve

Kaplan-Meier (K-M) curves were plotted to assess the association between each indicator and 30-day mortality (Figure 2 and Table 5). NPAR, MAR, PNI and CALLY indices were statistically significant. The NPAR2 group exhibited a more pronounced association with mortality than NPAR1 group. (NPAR2 *P*= 0.001, NPAR1 *P*= 0.045). A similar trend was observed for MAR (MAR2, *P*= 0.014, MAR1 *P*= 0.039). NPAR and MAR were positively correlated with 30-day mortality, whereas the CALLY index and PNI showed a negative correlation. Neither CAR nor RAR reached statistical significance in the K-M analysis.

Cox Regression Analysis

Cox proportional hazards models were constructed for Model 1–3. The study showed similar conclusions regardless of the adjusted variables, including age, smoking, drinking, and multiple variables that were statistically significant in the univariate analysis. The NPAR2 group and MAR2 group remained significantly associated with 30-day mortality (NPAR2 HR, 95% CI: 2.738, 1.058–7.084, *P*= 0.038; MAR2 HR, 95% CI: 1.871, 1.012–3.458, *P*=0.046), whereas the NPAR1 group and MAR1 group showed no significant differences. None of the other markers, including CAR, PNI, RAR or CALLY index, showed a statistically significant association in the COX regression analysis. This indicates that NPAR and MAR are independent predictors of 30-day mortality. (Table 6)

Subgroup Analysis

Subgroup analysis further revealed that the correlation of NPAR and MAR with mortality exhibited a statistically significant variation across different patient groups. Blood glucose was divided into three groups based on the third quartile (<7.09 mmol/L, 7.09–10.3 mmol/L, >10.3 mmol/L). Elevated mortality risk was particularly prominent in subgroups that included patients aged ≥90 years, with blood glucose over than 10.3 mmol/L, those with a diagnosis of coronary heart disease or hypertension, and those who had undergone tracheal cannula, central venous catheterization, surgery, or multiple infections. NPAR showed a more consistent and robust association with mortality across these subgroups than MAR. (Table 7).

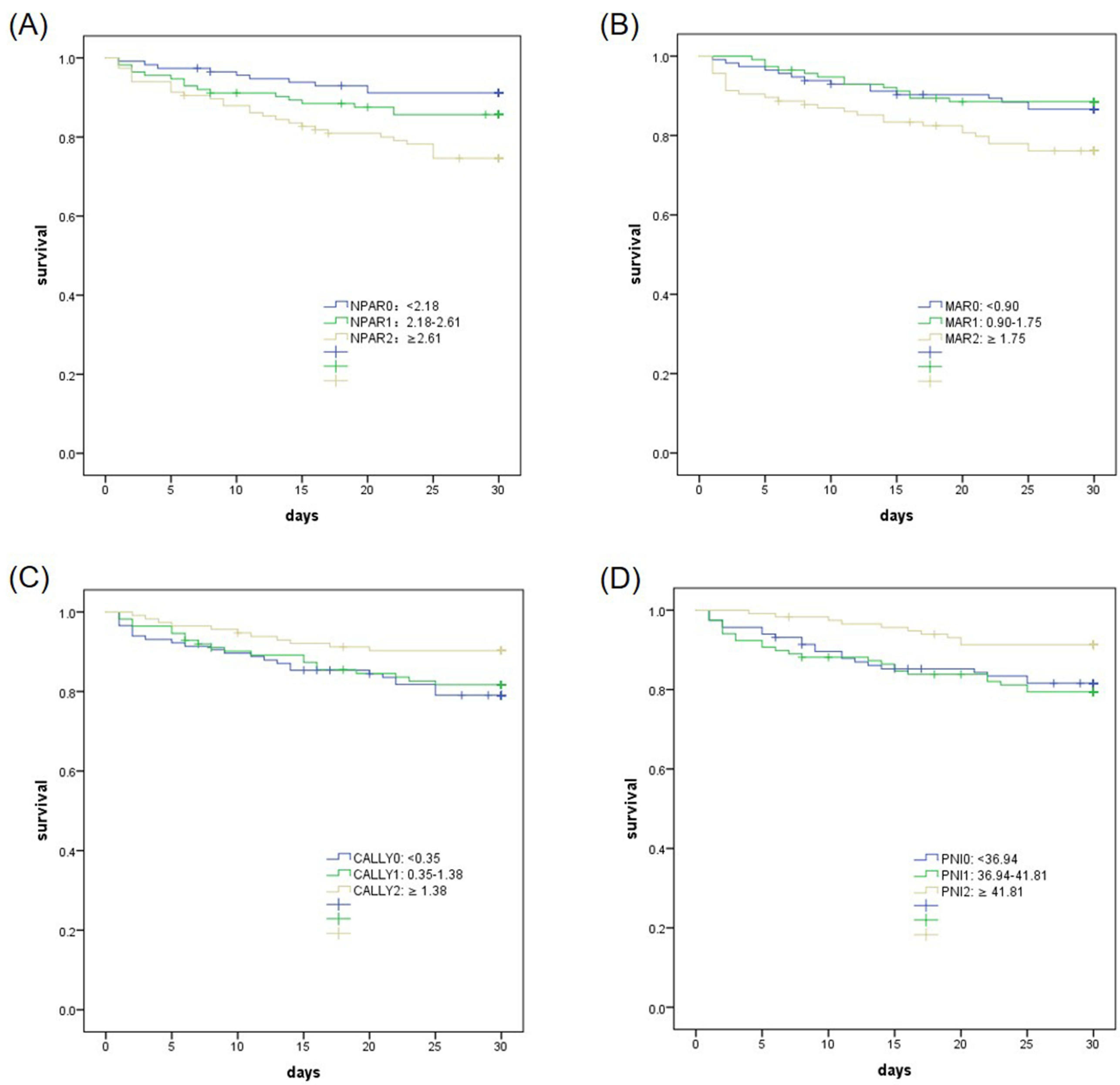

Figure 2 Kaplan-Meier curves for NPAR, MAR, PNI and CALLY index in 30-day mortality. (**A**) Kaplan-Meier curves for NPAR (NPAR0:<2.18, NPAR1:2.18–2.61, NPAR2:≥2.61); (**B**) Kaplan-Meier curves for MAR (MAR0: <0.90, MAR1: 0.90–1.75, MAR2: ≥ 1.75); (**C**) Kaplan-Meier curves for CALLY index (CALLY0: <0.35, CALLY1: 0.35–1.38, CALLY2: ≥ 1.38); (**D**) Kaplan-Meier curves for PNI (PNI0: <36.94, PNI1: 36.94–41.81, PNI2: ≥ 41.81).

Discussion

This study evaluated and compared the prognostic value of the NPAR, CAR, MAR, RAR, PNI and CALLY index in male diabetes patients over 80 years old with BSI. Among these, only NPAR and MAR demonstrated consistent and statistically significant associations with the 30-day mortality. This result differed in ROC, K-M curves, and Cox regression, while ROC and K-M curves were univariate analyses, and confounding factors were not corrected. Elevated NPAR (cut-off value=2.396) and MAR (cut-off value=2.437) levels were identified as independent risk factors for 30-day mortality in this patient population.

These findings are consistent with the growing body of evidence supporting the role of NPAR as a prognostic marker for critical illnesses. For instance, a 2023 study by Chunying et al involving 741 patients showed that high NPAR was significantly associated with 28-day mortality. (HR=1.42, and HR=1.35).[10] Similarly, Gong et al reported in a cohort of

Table 5 The Results of Log Rank Test of NPAR, MAR and CALLY

30-Day	*P* value
NAPR0	a
NAPR1	0.045
NAPR2	0.001
MAR0	a
MAR1	0.039
MAR2	0.014
CALLY0	a
CALLY1	0.067
CALLY2	0.002
PNI0	a
PNI1	0.670
PNI2	0.027

Notes: a: Reference; NPAR0: <2.18, NPAR1: 2.18–2.61, NPAR2: ≥2.61; MAR0: <0.90, MAR1: 0.90–1.75, MAR2: ≥ 1.75; CALLY0: <0.35, CALLY1: 0.35–1.38, CALLY2: ≥ 1.38; PNI0: <36.94, PNI1: 36.94–41.81, PNI2: ≥ 41.81.

Table 6 COX Regression Analysis of 30-Day Mortality of Diabetes Patients with BSI

	Model 1[b]		Model 2[c]		Model 3[d]	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value	HR (95% CI)	*P* value
NPAR						
NPAR0	a		a		a	
NPAR1	1.692(0.768,3.729)	0.192	1.249(0.547,2.853)	0.598	1.170(0.441,3.101)	0.753
NPAR2	3.116(1.518,6.395)	0.002	2.912(1.416,5.988)	0.004	2.738(1.058,7.084)	0.038
MAR						
MAR0	a		a		a	
MAR1	0.865(0.412,1.818)	0.702	0.993(0.439,1.986)	0.858	1.019(0.59,1.747)	0.946
MAR2	1.935(1.029,3.637)	0.040	1.995(1.036,3.842)	0.039	1.871(1.012,3.458)	0.046

Notes: [a]Reference. [b]NO adjusted. [c]Adjusted for age, smoking, drinking. [d]Adjusted for age, smoking, drinking, WBC, lymphocytes, hemoglobin, platelet D-dimer, c-pro, glucose, BUN, Scr, and ALT.

Table 7 Subgroup Analysis of COX Regression Analysis of 30-Day Mortality of Diabetes Patients with BSI

	NPAR		MAR	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Age, year				
80–90	1.564(0.985,2.554)	0.074	1.060(0.601,1.870)	0.841
≥90	2.467(1.294,4.703)	0.006	1.821(1.079,3.073)	0.025
Glucose, mmol/L				
<7.09	1.424(0.759,2.673)	0.271	0.923(0.530,1.607)	0.777
7.09–10.3	1.866(0.984,3.538)	0.056	1.836(0.997,3.378)	0.051
≥10.3	2.485(1.345,4.590)	0.004	1.870(0.989,3.535)	0.054

(*Continued*)

Table 7 (Continued).

	NPAR		MAR	
	HR (95% CI)	***P* value**	**HR (95% CI)**	***P* value**
CHD				
No	1.790(1.137,2.820)	0.012	1.292(0.821,2.033)	0.268
Yes	1.799(1.048,3.088)	0.033	1.809(1.083,3.023)	0.024
Hypertension				
No	1.642(1.123,2.401)	0.011	1.315(0.903,1.915)	0.154
Yes	2.810(1.077,7.329)	0.035	2.436(1.113,5.328)	0.026
CKD				
No	2.274(1.039,4.977)	0.040	1.185(0.558,2.520)	0.659
Yes	1.625(1.097,2.407)	0.015	1.567(1.075,2.283)	0.019
Tracheal cannula				
No	1.563(1.030,2.373)	0.036	1.454(0.969,2.181)	0.070
Yes	2.130(1.116,4.065)	0.022	1.527(0.845,2.760)	0.160
DVC				
No	2.463(0.897,6.760)	0.080	1.579(0.687,3.626)	0.282
Yes	1.643(1.126,2.397)	0.010	1.518(1.047,2.200)	0.028
Surgery				
No	1.786(1.023,3.116)	0.041	1.521(0.886,2.609)	0.128
Yes	1.867(1.177,2.961)	0.008	1.480(0.969,2.261)	0.070
Multiple infections				
No	1.752(0.756,4.058)	0.191	0.687(0.307,1.537)	0.361
Yes	1.779(1.206,2.626)	0.004	1.707(1.166,2.479)	0.006

Note: Glucose was divided based on the third quartile (<7.09 mmol/L, 7.09–10.3 mmol/L, >10.3 mmol/L).
Abbreviations: CHD, coronary heart disease, CKD, chronic kidney disease, DVC, deep vein catheterization.

2166 patients with severe sepsis or septic shock that elevated NPAR predicted increased mortality at 30, 90, and 365 days (HR=1.29, HR=1.41, HR=1.44, respectively).[18] More recently, Maside Ari's study (2025) showed that an NPAR > 0.286 was associated with higher mortality in patients aged ≥ 80 years who were followed up in the intensive care unit with a diagnosis of pneumonia. (HR=2.488).[19] Our research team has previously showed that NPAR was significantly associated with the risk of in-hospital mortality in elderly patients aged over 60 (HR = 3.36).[17] Our findings not only align with these reports but also suggest that a high NPAR may have an even stronger predictive value in male diabetes patients aged over 80 years with BSI (HR=2.738).

MAR has also been reported as a predictive indicator of chronic and inflammatory conditions. Cao et al observed in 1549 COPD patients that MAR was positively associated with the prevalence of COPD, and the highest MAR quartile (HR=1.66) was associated with an increased risk of all-cause mortality.[20] Jia et al further reported significant associations between elevated MAR and all-cause mortality in Parkinson's disease (HR=2.249)[21] and a higher stroke prevalence (adjusted OR=1.13), particularly among patients with diabetes.[22] To our knowledge, this study is the first to demonstrate that an elevated MAR predicts 30-day mortality in male diabetes patients with BSI (HR=1.871), supporting its potential utility in this high-risk population.

Subgroup analysis revealed that the association between NPAR/MAR and 30-day mortality was particularly pronounced in patients aged ≥90 years. Elderly patients with bloodstream infections have a higher likelihood of coexisting underlying diseases, atypical infection manifestations, a higher risk of multidrug-resistant bacterial infections, and differences in management compared to younger patients.[23] Laupland et al reported significantly higher mortality in patients aged 75–84 years (OR=1.66) and ≥ 85 years (OR=1.98) than in those aged 65–74 years.[24] Our study included a population with a higher baseline age, and for patients aged over 90, NPAR/MAR had higher predictive value for their 30-day mortality risk. This suggests that among the ultra-elderly population, these indicators have more significant clinical application value.

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Similarly, central venous catheterization is known to increase the risk of catheter-related BSI, prolonged hospital stay, and mortality,[25] often compounded by infections with resistant pathogens.[26] Therefore, for patients undergoing combined deep venous catheterization, avoiding catheter-related infections and performing NPAR/MAR index testing may help improve prognosis. However, there is still a lack of relevant clinical applications, and further research is needed.

Hyperglycemic can exacerbate infections, leading to poor prognosis. Subgroup analysis demonstrated that among individuals with blood glucose levels over 10.3 mmol/L, NPAR had the strongest correlation with 30-day mortality. This is consistent with previous studies, indicating that early prediction of mortality risk is particularly important for individuals with elevated blood glucose levels. Early control of blood glucose is also of great significance in the treatment of infections. To prevent the occurrence of hypoglycemia, strict blood glucose management may not be recommended.[27] Current guidelines recommend that the target for hyperglycemia regulation in critically ill patients should be 7.8~10.0 mmol/L.[28] Other factors include hypertension, CVD, and CKD, all of which may lead to a decline in overall health and result in poor prognosis.

Neutrophils and monocytes have been widely used in predicting prognosis of inflammation, as well as low nutritional status. Neutrophils and monocytes, the key cells in innate immunity, are activated during the early stages of bloodstream infection and differentiate into macrophages to clear pathogens. Moreover, they also secret various pro-inflammatory cytokines such as TNF-α, IL-1 and IL-6, promoting the inflammatory response.[29] In patients with diabetes, hyperglycemia induces a state of chronic inflammation and impairs neutrophil and monocyte functions, including reduced chemotaxis, phagocytosis, and bactericidal capacity. BSI aggravates this dysfunctional immune system, driven by pro-inflammatory cytokines, such as IL-6 and TNF-α,[30] triggering a “cytokine storm” and exacerbating tissue damage.[31]

Concurrently, the stress response and cytokine storm (notably IL-6) suppresses albumin synthesis in the liver.[32] Albumin is not merely a nutritional marker, but also possesses important anti-inflammatory, antioxidant, and endothelial-stabilizing properties. Thus, its decline represents a loss of endogenous protective capacity and a shift towards a pro-oxidant, pro-inflammatory state.[33,34] Lower serum albumin levels are an independent predictor of increased 30-day mortality in patients with single microbial bloodstream infections.[6] Our results reflect the convergence of inflammatory activation and metabolic-nutritional impairment inflammation and malnutrition. NPAR and MAR integrate these two pathways, and underscore the synergistic pathological effects of systemic inflammatory responses and nutritional metabolic imbalance.[35] This combination is particularly detrimental in patients with diabetes, who already have compromised vascular integrity and immune regulation. The predictive superiority of NPAR and MAR over isolated markers, such as neutrophil count, monocyte count or albumin alone, provides a more holistic view of a patient’s immune metabolic status. An elevated neutrophil and monocyte count reflects the degree of inflammatory activation, while low albumin levels indicate depletion of metabolic reserves and uncontrolled inflammation. Indicators of the interaction between the two pathways can better predict the prognosis of BSI in patients with diabetes.

This study has certain limitations. The actual clinical application still faces many obstacles. We found that NPAR over 2.396 and MAR over 2.437 were the suitable cut-off point, but the conclusions are inconsistent in multiple studies. It may relate to that most studies classify indicators into thirds or quartiles, largely depending on the sample size and disease type. At the same time, this study was a single-center retrospective study which only included male population, a selection bias was possible and there may be universality issues. Meanwhile, important clinical variables that may affect prognosis, such as the type of infectious pathogen and initial antibiotic treatment, were not included in the analysis. In future research, it is necessary to integrate multi-center samples, and include female patients to improve the generalizability of subsequent research conclusions, and incorporate more clinical factors into analysis. Moreover, retrospective studies may have limitations in clarifying causal relationships, so prospective studies can be improved in the future.

Conclusions

In conclusion, NPAR and MAR are easily measurable low-cost biomarkers that show significant prognostic value for 30-day mortality in male diabetes patients over 80 years old with BSI. Both markers integrate the inflammatory and nutritional dimensions of critical illness, with NPAR demonstrating a particularly strong predictive ability across key clinical subgroups. Especially for patients with multiple risk factors, early detection of NPAR and MAR may have significance in improving the prognosis of patients with BSI.

Abbreviations

BSI, bloodstream infections; NPAR, neutrophil-percentage-to-albumin ratio; CAR, c-reactive protein-to-albumin ratio; MAR, monocyte-to-albumin ratio; RAR, red blood cell distribution width-to-albumin ratio; PNI, Prognostic Nutritional Index; Scr, serum creatinine; BUN, blood urea nitrogen; BNP, brain natriuretic peptide; CRP, C-reactive protein; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; PLT, platelets; WBC, white blood cell; RDW, red cell distribution width; DVC, deep vein catheterization; CHD, coronary heart disease; CKD, chronic kidney disease; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease.

Data Sharing Statement

The datasets used and analyzed during the current study are available from the corresponding author (Yingzhen Du) upon reasonable request.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that there are no conflicts of interest related to this work.

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