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ORIGINAL RESEARCH

The Neutrophil Percentage-to-Albumin Ratio as a New Predictor of All-Cause Mortality in Maintenance Hemodialysis Patients

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Introduction: Neutrophil percentage-to-albumin ratio (NPAR), dually reflecting inflammation and malnutrition, is related to poor prognosis closely in a range of diseases. However, prognostic value of NPAR in maintenance hemodialysis (MHD) patients remains to be confirmed. This study aimed to investigate the association between NPAR and all-cause mortality in MHD patients.

Methods: Patients undergoing maintenance hemodialysis in the blood purification department of The First Affiliated Hospital of Guangxi Medical University from January 2021 to June 2021 were prospectively studied. NPAR was calculated by dividing neutrophils percentage by Albumin. Participants were followed for 36 months, with all-cause mortality as the primary endpoint.

Results: There were 146 male and 80 female MHD patients included in this study, with a median age of 56 years. 53 (23.5%) patients died during the follow-up period. Kaplan–Meier (K–M) analysis revealed significantly lower survival in patients with high NPAR (>16.96) compared to those with low NPAR (≤16.96) (log rank test p=0.000). Multivariate Cox regression has identified NPAR as an independent predictor of all-cause mortality (HR=1.346, 95% CI 1.192–1.521, p=0.000). Receiver operating characteristic (ROC) analysis demonstrated that the Area Under the Curve (AUC) of NPAR was 0.821 (95% CI: 0.759–0.882, p=0.000) and had a trend to be better than that of neutrophil-to-lymphocyte ratio (NLR; AUC=0.710), platelet-to-lymphocyte ratio (PLR; AUC=0.647), neutrophil count (AUC=0.606), albumin (ALB; AUC=0.752), and high-sensitivity C-reactive protein (hs-CRP; AUC=0.670).

Conclusion: NPAR is independently associated with all-cause mortality in MHD patients and may serve as an emerging indicator for risk stratification and prognostic management for this group of patients.

Keywords: hemodialysis, neutrophil percentage-to-albumin ratio, mortality

Introduction

Chronic kidney disease affects more than 10% of the population, a considerable number of whom progress to end-stage renal disease (ESRD) eventually and require renal replacement therapy to survive.[1] Hemodialysis is one of the most important renal replacement therapies. Although a large number of advances have been made in clinical and basic research over the past decades, maintenance hemodialysis (MHD) patients still face a high risk of mortality. Cardiovascular disease (CVD) is prevalent in MHD patients and is the most common cause of death.[2] Chronic inflammation and malnutrition are proved to play a key role in developing CVD in MHD patients and closely associate with mortality.[3–5]

Neutrophils, key mediators of innate immunity, serve as critical biomarkers of systemic inflammation. Elevated neutrophil percentage is independently associated with adverse outcomes in MHD cohorts.[6,7] Conversely, serum albumin, a well-established marker of nutritional status and anti-inflammatory capacity, demonstrates inverse correlations with

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morbidity and mortality in ESRD.[8,9] Mechanistically, hypoalbuminemia exacerbates inflammation, oxidative stress, and endothelial dysfunction, cementing its role as an independent risk factor for poor prognosis.[10]

Given the synergistic interplay between inflammation and malnutrition in MHD outcomes, recent research has prioritized integrated biomarkers capturing both pathophysiological axes. The neutrophil percentage-to-albumin ratio (NPAR), calculated by dividing neutrophil percentage by serum albumin concentration, theoretically quantifies pro-inflammatory activation (numerator) and nutritional/anti-inflammatory reserve (denominator). As a novel inflammation-nutrition composite index, NPAR has demonstrated robust prognostic utility across diverse conditions such as sepsis, acute coronary syndrome and heart failure.[11–14]

In recent years, several studies have been reported on the role of NPAR in CKD patients. High NPAR is not only associated with increased risk of CKD, but also a predictor for both all-cause and CVD deaths in advanced CKD patients.[15,16] Two studies indicated that NPAR is closely related to the prognosis of peritoneal dialysis (PD) patients, and also found NPAR exhibits superior discriminative performance compared to isolated markers (eg, albumin alone) and other inflammatory ratios (eg, neutrophil-to-lymphocyte ratio NLR, platelet-to-lymphocyte ratio PLR) in predicting PD mortality.[17,18] A study demonstrated that an elevated NPAR was correlated to adverse outcome in MHD patients.[19] However, data on the relationship between NPAR and mortality are limited. Therefore, in this study, we aimed to investigate the relationship between NPAR and all-cause mortality in MHD patients.

Materials and Methods

Subjects

MHD patients in the Blood Purification Department of The First Affiliated Hospital of Guangxi Medical University from January 2021 to June 2021 were prospectively research. The inclusion criteria were as follows: (1) age >18 years, (2) HD treatment duration >3 months, 4h 2–3 times weekly using standard bicarbonate dialysate. The exclusion criteria were: (1) recent heart failure and acute coronary syndrome within 1 month, (2) recent infections within 1 month, (3) history of kidney transplantation or peritoneal dialysis, (4) hematological disorders, (5) autoimmune diseases, (6) tumors, (7) taking steroids or immunosuppressive drugs.

Follow-Up and Endpoints

All subjects included were followed up for 36 months. The primary endpoint was defined as all-cause mortality. Deaths were confirmed by hospital records for inpatients and death certificates for outpatients. Follow-up terminated if the patient died, received a kidney transplant, transferred to another dialysis center, voluntarily gave up treatment during the follow-up period.

Ethics

This research follows the Helsinki Declaration and has been approved by the Ethics Committee of The First Affiliated Hospital of Guangxi Medical University (NO. 2023-K282-01). Informed consent was obtained from each subject prior to study commencement.

Data Collection

Data were all collected when the patients were included from the hospital's multi-source database integrated from electronic health records (EHR), laboratory information system (LIS), hemodialysis information system, imaging system and treatment records, and defined as baseline data, including age, sex, etiology of CKD, history of diabetes, Duration of HD, type of vascular access for dialysis, dry weight, body mass index (BMI) and laboratory data. Laboratory data included white blood cell count (WBC), neutrophil count, lymphocyte count, platelet count, percentage of neutrophils, hemoglobin, serum albumin, blood urea nitrogen, creatinine, uric acid, potassium, chloride, calcium, phosphorus, serum ferritin, hypersensitive C-reactive protein (hs-CRP), intact parathyroid hormone (iPTH). When serum albumin was lower than 40 g/L, serum calcium was corrected using the formula: Ca^{2+} (mmol/L) = total serum Ca^{2+}(mmol/L) + 0.2 × [4-Alb (g/dL)]. All laboratory indicators were tested using blood samples collected before initiation of dialysis. iPTH was measured using immunoradiometric assay, while the rest were analyzed using standard laboratory methods with an autoanalyzer. NPAR was calculated by dividing neutrophils percentage by serum

albumin with a formula as: neutrophil percentage (%) ×100/serum albumin (g/dL).[20] NLR, and PLR were calculated by dividing neutrophils and platelets by lymphocytes, respectively.

Statistical Analysis

SPSS22.0 statistical software (IBM) was used for the statistical analysis. Regression imputation was used for missing value filling. The normality of distribution of continuous data was evaluated by the Kolmogorov–Smirnov test. Normally distributed continuous variables were expressed as mean ± standard deviation and compared using the *t*-test of independent samples. Non-normally distributed data were recorded as medians and interquartile range and compared using the Mann–Whitney *U*-tests. The categorical data were described using counts and percentages and compared using Chi-square test. All research subjects were classified based on the median NPAR. The Pearson's correlation was used for the normally distributed data, and the Spearman correlation was used for the nonparametric data. The receiver–operating characteristic (ROC) curve was used to identify the predictive ability of NPAR. The Youden index = sensitivity + specificity −1. The optimal cutoff, sensitivity, and specificity of the indicators were obtained by using and the maximum value of the Youden index. All-cause mortality rates were evaluated by Kaplan–Meier analysis with the use of the Log rank test. Univariate and multivariate Cox proportional hazards regression models were used to explore the associations between NPAR and the prognosis of MHD patients. Factors that were significantly related to all-cause mortality were included in multivariate COX regression analysis. Values of $p < 0.05$ were considered statistically significant.

Results

Baseline Patient Characteristics

As shown in Figure 1 and Table 1, a total of 226 MHD patients were included in the study, with a median age of 56 years, including 146 males (64.6%). Patients were stratified into lower NPAR (≤16.96) and higher NPAR (>16.96) groups according to the median NPAR. Baseline characteristics are summarized in Table 1. Patients in the higher NPAR group were significantly older (median age 58 vs 54 years, p=0.003) and exhibited higher WBC count (7.02×10^9/L vs 6.27×10^9/L, p<0.001), higher neutrophil percentages (74.19% vs 63.47%, p<0.001), lower hemoglobin (101.04 g/L vs 112.46 g/L, p<0.001), lower Albumin (3.86 g/dl vs 4.19 g/dl, p=0.000), lower Creatinine (974.37μmol/L vs 1098.97μmol/L, p=0.003), higher Chloride (100.4 mmol/L vs 99.00 mmol/L, p=0.016) and elevated inflammatory markers (NLR: 4.78 vs 2.99, p<0.001; PLR: 199.91vs 153.28, p<0.001; hs-CRP: 8.40 mg/L vs 4.77 mg/L, p<0.001). Non-autogenous arteriovenous

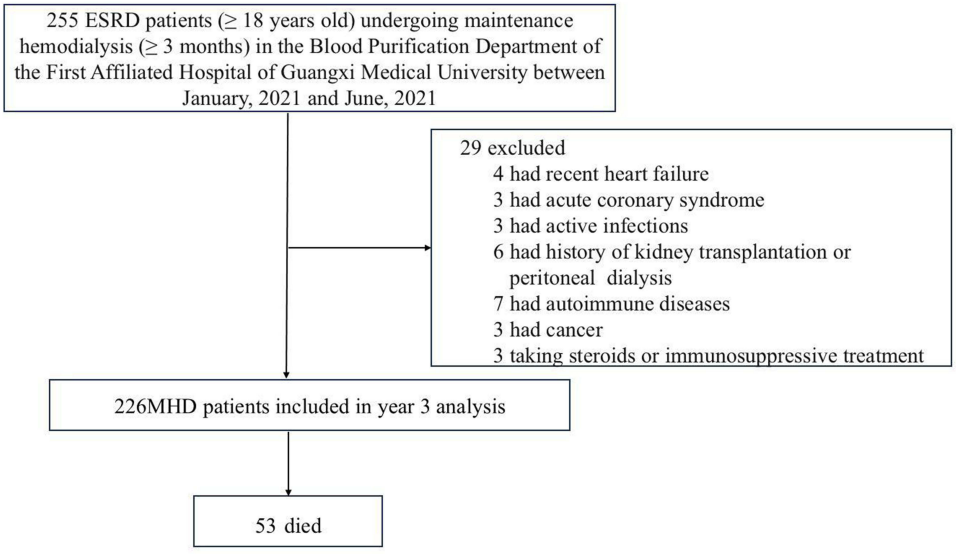

Figure 1 Study flow chart.

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Table 1 Characteristics of 226 Maintenance Hemodialysis Patients by NPAR Groups

	Total	Lower NPAR Group (≤16.96)	Higher NPAR Group (>16.96)	p value
Age (years)	56(44–65)	54(40–64)	58(48–68)	0.003
Duration of HD (months)	51(24–72)	50(26–70)	49(24–82)	0.692
Male (%)	146 (64.6%)	78(69%)	68(60.2%)	0.164
Etiology of CKD n, (%)				0.079
Chronic glomerulonephritis	128 (56.6%)	73(64.6%)	55(48.7%)	
Diabetic nephropathy	37 (16.4%)	15(13.3%)	22(19.5%)	
Hypertensive nephropathy	32 (14.2%)	15(13.3%)	17(15%)	
Other	29 (12.8%)	10(8.8%)	19(16.8%)	
Diabetes	40 (17.8%)	17(15%)	23(20.5%)	0.300
Vascular access type n, (%)				0.006
Autogenous AVF	191 (84.5%)	103 (91.2%)	88 (77.9%)	
Non autogenous AVF	35 (15.5%)	10 (8.8%)	25 (22.1%)	
Dry weight	58(50–65.15)	57.5(50.2–64.5)	58.0(49.8–65.85)	0.716
BMI (kg/m ^{2})	21.47(19.26–23.84)	20.98 (19.13–24.70)	22.14 (19.77–23.73)	0.464
Laboratory variables				
WBC (10^9/L)	6.52(5.34–8.26)	6.27(5.26–7.62)	7.02(5.67–9.06)	0.001
Platelet (10^9/L)	193.50(162.68–235.4)	189.2(161.55–227.55)	204.9(165–248.45)	0.181
Hemoglobin (g/L)	106.75±21.18	112.46±20.02	101.04±20.85	0.000
Neutrophil (%)	68.83±8.88	63.47±6.47	74.19±7.64	0.000
NLR	3.74(2.84–5.27)	2.99(2.39–3.77)	4.78(3.70–6.93)	0.000
PLR	170.83(131.55–227.15)	153.28(116.73–187.92)	199.91(145.64–259.44)	0.000
Albumin (g/dL)	4.01(3.77–4.27)	4.19(3.97–4.42)	3.86(3.60–4.08)	0.000
BUN (mmol/L)	23.76±7.43	23.53±6.56	24.00±8.23	0.636
Creatinine (μmol/L)	1036.67±312.15	1098.97±276.82	974.37±333.60	0.003
Uric acid (mmol/L)	449.08±116.83	448.96±110.93	449.21±122.95	0.988
Potassium (mmol/L)	4.80±0.78	4.82±0.72	4.78±0.85	0.753
Chloride (mmol/L)	99.7(97.15–102.43)	99.00(96.90–101.8)	100.4(97.55–103.15)	0.016
Calcium (mmol/L)	2.20(2.05–2.38)	2.17(2.03–2.39)	2.22(2.08–2.36)	0.329
Phosphorus (mmol/L)	1.89±0.56	1.90±0.59	1.88±0.54	0.787
iPTH (pg/mL)	583.4(312.05–998.08)	592.6(344.35–976.85)	535.2(264.18–1029.90)	0.490
Ferritin (ng/mL)	505.95(191.88–882.61)	526.29(174.39–815.0)	481.82(217.96–910.24)	0.787
Hs-CRP (mg/L)	5.00 (2.08–14.69)	4.77 (2.05–13.69)	8.40 (3.54–21.93)	0.000
Triglyceride (mmol/L)	1.29(0.82–2.27)	1.30(0.89–2.29)	1.26(0.76–2.19)	0.563
Cholesterol (mmol/L)	4.09(3.31–5.00)	4.14(3.38–4.97)	4.04(3.26–5.13)	0.634
β2-MG (mg/L)	37.01(30.96–43.89)	36.49(29.63–45.07)	37.16(32.00–43.46)	0.771
Outcome				0.000
Survival n, (%)	173 (76.5%)	105 (92.9%)	68 (60.2%)	
Death n, (%)	53 (23.5%)	8 (7.1%)	45 (39.8%)	

Note: Non autogenous AVF including AV graft and permanent catheter.

Abbreviations: NPAR, neutrophil percentage to albumin ratio; HD, hemodialysis; CKD, chronic kidney disease; AVF, arteriovenous fistula; BMI, body mass index; WBC, white blood cell; NLR, neutrophils to lymphocytes ratio; PLR, platelets to lymphocytes ratio; BUN, blood urea nitrogen; hs-CRP, hypersensitive C-reactive protein; iPTH, intact parathyroid hormone. β_2-MG: β_2-Microglobulin.

fistula (AVF) usage was more prevalent in the higher NPAR group (22.1% vs 8.8%, p=0.006). No significant differences were observed in dialysis duration, BMI, or most biochemical parameters (eg, BUN, potassium, phosphorus).

Correlation Results Between NPAR and Other Significant Parameters in MHD Patients

NPAR demonstrated significant correlations with inflammatory, nutritional, and clinical parameters (Table 2). It was positively associated with age (r=0.257, p<0.001), WBC count (r=0.303, p<0.001), hs-CRP (r=0.383, p<0.001), NLR (r=0.708, p<0.001), and PLR (r=0.445, p<0.001). Conversely, NPAR showed negative correlations with hemoglobin (r=−0.313, p<0.001), lymphocyte (r=−0.449, p<0.001) and creatinine (r=−0.340, p<0.001).

Table 2 Bivariate Correlation Results Between NPAR and Other Significant Parameters in MHD Patients

Variables	r	p value
Age	0.257	0.000
WBC	0.303	0.000
Lymphocyte	−0.449	0.000
Hs-CRP	0.383	0.000
Hemoglobin	−0.313	0.000
Creatinine	−0.340	0.000
NLR	0.708	0.000
PLR	0.445	0.000
Dry weight	−0.036	0.593
BMI	−0.012	0.860

Abbreviations: NPAR, neutrophil percentage to albumin ratio; MHD, maintenance hemodialysis; WBC, white blood cell; hs-CRP, hypersensitive C-reactive protein; NLR, neutrophils to lymphocytes ratio; PLR, platelets to lymphocytes ratio; BMI, body mass index.

Relationship Between NPAR and All-Cause Mortality in MHD Patients

During the follow-up period, 53 (23.5%) patients died. The mortality rate in the higher NPAR group was significantly higher than that in the lower NPAR group (39.8% vs 7.1%, p=0.000), as shown in Table 1. The Kaplan–Meier survival analysis curve showed that the survival rate of lower NPAR group was significantly higher than that of higher NPAR group (log rank test P=0.000) (Figure 2).

In the univariate Cox regression analysis, all-cause mortality was associated with age, diabetes mellitus, type of vascular access, WBC, hemoglobin, creatinine, uric acid, phosphorus, NPAR, calcium, and hs-CRP. In multivariate analysis, NPAR

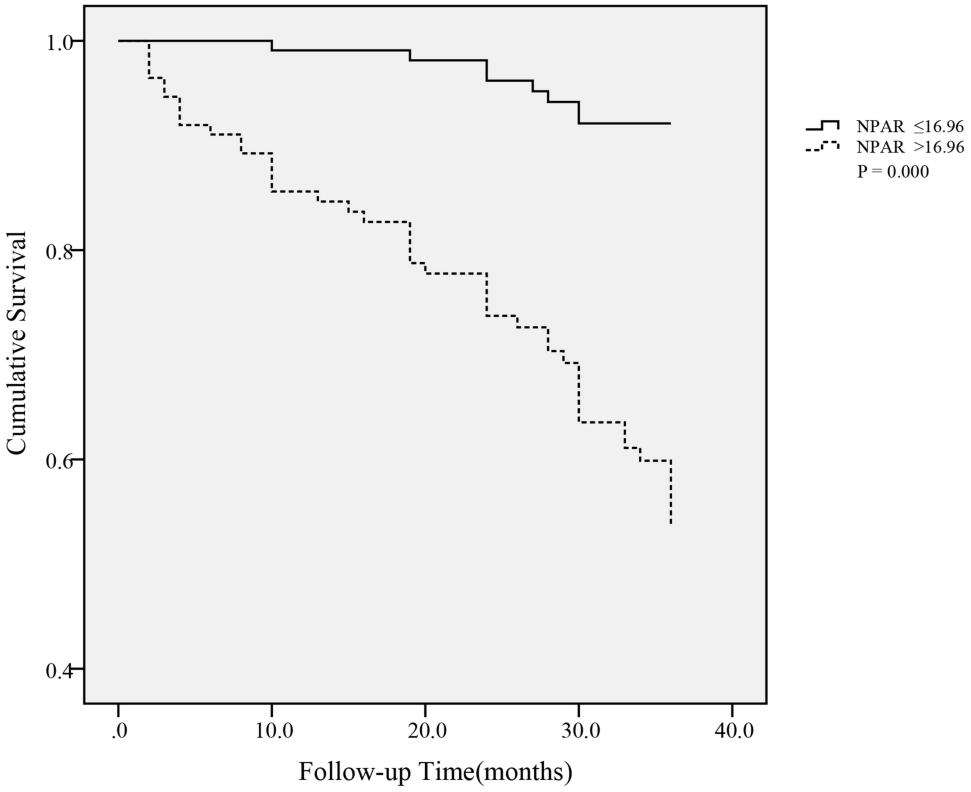

Figure 2 K–M survival curve analysis of MHD patients.

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remained an independent risk factor (adjusted HR=1.346, 95% CI: 1.192–1.521, p<0.001), alongside age (HR=1.051, 95% CI: 1.024–1.079, p<0.001), non-autogenous AVF (HR=2.564, 95% CI: 1.283–5.123, p=0.008), phosphorus (HR=2.258, 95% CI: 1.176–4.336, p<0.05), and serum calcium (HR=1.080, 95% CI: 1.043–1.118, p<0.001) (Table 3).

Prediction of Mortality

Receiver Operating Characteristic (ROC) analysis indicated that the Area Under the Curve (AUC) of NPAR was 0.821 (95% CI: 0.759–0.882, p=0.000), the optimal cutoff was 17.14, with a sensitivity of 0.849 and a specificity of 0.647. The AUC of NPAR had a trend to be better than that of NLR (AUC= 0.710, 95% CI: 0.627–0.793, p=0.000), PLR (AUC=0.647, 95% CI: 0.553–0.741, p=0.001), neutrophil count (AUC=0.606, 95% CI: 0.512–0.700, p=0.019), albumin (AUC=0.752, 95% CI: 0.677–0.826, p=0.000), and hs-CRP (AUC=0.670, 95% CI: 0.583–0.756, p=0.000) (Table 4 and Figure 3).

Table 3 Univariate and Multivariate Cox Proportional Hazards Regression Analyses of Factors Associated with Mortality

	HR	95.0% CI	p value	HR[a]	95.0% CI	p value
Male	1.585	0.871–2.882	0.131	1.915	0.898–4.082	0.093
Age	1.067	1.043–1.092	0.000	1.051	1.024–1.079	0.000
Duration of HD	1.001	0.995–1.001	0.720			
Diabetes	2.340	1.286–4.260	0.005	1.251	0.625–2.501	0.527
Non autogenous AVF	3.719	2.078–6.654	0.000	2.564	1.283–5.123	0.008
WBC	1.201	1.088–1.326	0.000	0.889	0.775–1.021	0.095
Hemoglobin	0.981	0.968–0.994	0.004	0.998	0.984–1.013	0.841
Platelet	1.001	0.998–1.005	0.379			
NPAR	1.243	1.185–1.304	0.000	1.346	1.192–1.521	0.000
iPTH	1.000	0.999–1.000	0.285			
Creatinine	0.997	0.996–0.998	0.000	0.999	0.997–1.000	0.076
Potassium	0.801	0.561–1.144	0.221			
Phosphorus	0.548	0.332 0.905	0.019	2.258	1.176–4.336	0.014
Chloride	1.009	0.973–1.045	0.632			
BUN	0.976	0.938–1.016	0.244			
Uric acid	0.995	0.992–0.997	0.000	0.998	0.994–1.001	0.216
Ferritin	1.000	1.000–1.001	0.185			
Calcium	1.067	1.033–1.102	0.000	1.080	1.043–1.118	0.000
Hs-CRP	1.014	1.009–1.020	0.000	0.991	0.981–1.001	0.093

Note: [a]HR value corrected by multiple factors.

Abbreviations: HR, hazard ratio; CI, confidence interval; HD, hemodialysis; AVF, arteriovenous fistula; WBC, white blood cell; NLR, neutrophils to lymphocytes ratio; PLR, platelets to lymphocytes ratio; NPAR, neutrophil percentage to albumin ratio; iPTH, intact parathyroid hormone; BUN, blood urea nitrogen.

Table 4 ROC Curves for the Prediction of All-Cause Mortality in MHD Patients of Various Parameters

Parameters	AUC	95% CI	p value
NPAR	0.821	0.759–0.882	0.000
NLR	0.710	0.627–0.793	0.000
PLR	0.647	0.553–0.741	0.001
Neutrophil	0.606	0.512–0.700	0.019
Albumin	0.752	0.677–0.826	0.000
Hs-CRP	0.670	0.583–0.756	0.000

Abbreviations: NPAR, neutrophil percentage to albumin ratio; NLR, neutrophils to lymphocytes ratio; PLR, platelets to lymphocytes ratio; hs-CRP, hypersensitive C-reactive protein.

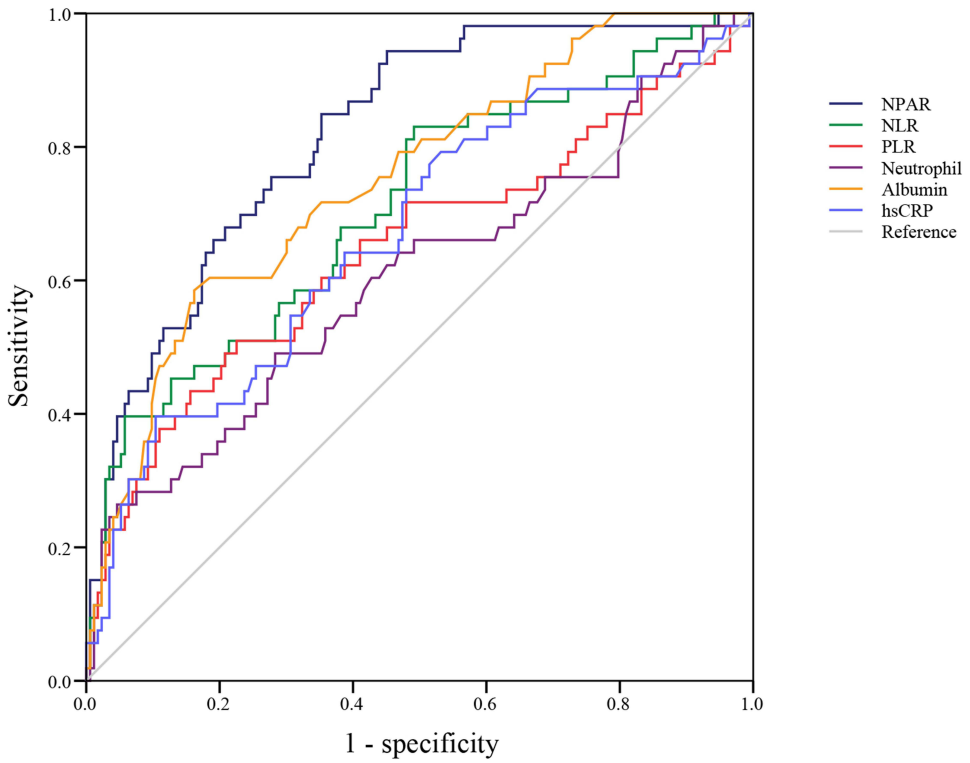

Figure 3 ROC curves for the prediction of all-cause mortality in MHD patients of various parameters.

Discussion

In this prospective study, NPAR was found to be associated with all-cause mortality in MHD patients, and as an independent predictor of mortality, NPAR had a trend to be superior to those traditional inflammation and nutrition markers.[21–25] NPAR was also found to be correlated with inflammatory markers, age, and anemia-related parameters in this study. A multicenter study reported recently also discovered high NPAR level was independently associated with a higher increased risk of death in MHD patients.[19] These findings provide novel insights into risk stratification and prognostic management for MHD patients.

First, the predictive advantage of NPAR may stem from its dual reflection of systemic inflammation and nutritional status. Albumin, a key hepatic protein, serves as both a sensitive marker of nutritional status and a negative acute-phase reactant in chronic inflammation,[26] while elevated neutrophil percentage directly indicates systemic inflammatory activation and activated neutrophil induces and aggravates atherosclerotic cardiovascular disease.[27–31] By integrating the interplay between inflammation and nutrition, NPAR may comprehensively capture the pathophysiological features of the "Malnutrition-Inflammation-Atherosclerosis (MIA)" syndrome. MIA is a common condition and associated with poor outcome in ESRD patients.[32–34] Unlike hs-CRP reflecting only inflammation or NLR/PLR lacking nutritional assessment, NPAR combines both aspects, potentially explaining its superior predictive efficacy. Interestingly, although BMI as a nutritional indicator and low BMI predicts poor prognosis in MHD patients, we observed no significant linear correlation between NPAR and dry weight/BMI. The reasons may be as follows: (1) Dry weight primarily reflects volume status, while BMI fails to differentiate body composition and predict nutrition status accurately.[35,36] (2) Most subjects included in this study had normal BMI, with insufficient high/low-BMI subgroups for stratified analysis. (3) Mortality in hemodialysis population is 10 times higher than that of general population, which is driven more by inflammation and malnutrition than BMI. [37] This underscores unique value of NPAR as a composite marker of the "inflammation-malnutrition axis", providing prognostic insight independent of weight metrics.

Second, the positive correlations between NPAR and CRP, PLR, and NLR further validate its role as an inflammatory marker. MHD patients often exhibit a microinflammatory state, and persistent inflammation accelerates cardiovascular

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events and infectious complications by promoting endothelial injury, oxidative stress, and protein-energy wasting.[38] Additionally, the negative correlation between NPAR and hemoglobin may suggest inflammation-mediated erythropoietin resistance or iron metabolism dysregulation.[39,40] The positive association with age likely reflects the heightened risks of immunosenescence and malnutrition in elderly patients, a phenomenon increasingly recognized in geriatric nephrology.[41] These relationships highlight NPAR's ability to integrate multidimensional pathophysiological processes, thereby enhancing its prognostic value.

From a clinical perspective, NPAR measurement offers distinct advantages: it requires only routine blood tests and biochemical data, incurs no additional costs, and provides immediate results. This makes it particularly suitable for resource-limited settings, aligning with the growing emphasis on cost-effective biomarkers in global nephrology practice[42] Dynamic monitoring of NPAR could enable clinicians to identify high-risk patients earlier and tailor anti-inflammatory therapies, nutritional support, or anemia management strategies, potentially improving outcomes. Critically, our identification of an optimal NPAR cutoff at 17.14 provides a clinically actionable threshold for mortality risk stratification in MHD patients.

However, this study has limitations. First, as a single-center analysis, potential selection bias exists, necessitating validation through multicenter prospective cohorts. Second, we measured NPAR for only one time, the optimal cutoff value of NPAR and the prognostic implications of its dynamic changes require further investigation. Third, comparisons with other emerging biomarkers (eg, Klotho protein) were not included, limiting direct benchmarking against cutting-edge prognostic tools. Fourth, the indicators included in this study were limited and may not include all possible prognostic indicators such as residual renal function, therapeutic model, dialysis adequacy, medication, etc. In addition, this is a clinical observational study, the potential mechanisms and their practicality in guiding interventions still need further exploration.

In conclusion, NPAR, dually reflecting inflammation and nutrition, was independently correlated with all-cause mortality in MHD patients. As a simple, cost-effective and available indicator, NPAR can be a routine test to serve the clinic. However, future research should focus on exploring mechanisms, conducting multicenter prospective studies, developing NPAR-based risk scoring systems and validating its role in guiding individualized therapies through interventional trials, ultimately advancing precision management for MHD patients.

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Disclosure

The authors declare no conflicts of interest in this work.

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