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CLINICAL STUDY

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Use of the systemic inflammation response index (SIRI) as a novel prognostic marker for patients on peritoneal dialysis

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ABSTRACT

Background: The systemic inflammatory response index (SIRI), a novel inflammation maker, has proven to be associated with prognostic outcomes in various diseases. However, few studies have been conducted assessing how SIRI may influence outcomes of patients on peritoneal dialysis (PD). Herein, we assessed the predictive value of SIRI on mortality all-cause mortality, including cardiovascular disease (CVD) in PD patients.

Methods: A total of 646 PD patients were enrolled in this study. PD patients received regular PD treatments at the Zhujiang Hospital from 1 January 2011 to 31 December 2018. SIRI values could be computed as follows: neutrophil count × monocyte count/lymphocyte count. Patients were divided into two groups according to the median level of SIRI. Cox regression analysis and Kaplan–Meier methods were applied to analyze the relationship between SIRI and mortality outcomes in PD patients.

Results: During the median 31-month follow-up period, 97 (15.0%) PD patients died from all-causes, and 47 (49.0%) died of CVD. Kaplan–Meier analyses revealed that a high SIRI corresponded to the high mortality of all-cause deaths, including CVD (both $p < 0.001$) in patients on PD. After adjusting for potential confounders, the higher SIRI level was significantly associated with an increased all-cause mortality (HR: 2.007, 95% CI: 1.304–3.088, $p = 0.002$) and cardiovascular mortality (HR: 2.847, 95% CI: 1.445–5.608, $p = 0.002$).

Conclusions: SIRI was a promising predictor of mortality in PD patients, with a higher SIRI corresponding to increased risk of mortality.

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Systemic inflammatory response index; microinflammation; peritoneal dialysis; all-cause mortality; cardiovascular disease

Introduction

Kidney failure has gradually become a major public health problem globally. Based on the 2019 Annual Data Report of US Renal Data System, the population of kidney failure patients reached 746,557 in 2017 [1]. Peritoneal dialysis (PD) is one of the most effective therapies for kidney failure patients, with an estimated 272,000 patients in the world currently undergoing PD treatment [2,3]. Although there are many advantages of PD treatment including ease of operation, it is reported that the 5-year cumulative survival for PD patients is only 60% in 2012 in Asia [4]. Specifically, cardiovascular disease (CVD)-associated deaths account for 40–60% of total deaths worldwide, and is the main cause of death in PD patients [5]. Therefore, the early identification of high-risk patients on PD is extremely important to reduce mortality.

Microinflammation, a well-known risk factor associated with PD treatment, plays an important role in promoting poor outcomes [6]. Oxidative stress, inflammatory cytokines, gut dysbiosis, metabolic acidosis, and vitamin D deficiency can aggravate and promote inflammation in patients with chronic kidney disease (CKD). Inflammation can exacerbate the occurrence of CVD, malnutrition, and anemia, which ultimately puts CKD patients at higher risk of death [7]. Previous studies have identified a strong relationship between microinflammation and cardiac valve calcification and ventricular hypertrophy that could increase CVD mortality in PD

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patients [8,9]. Malnutrition is another important factor affecting the poor prognosis of PD patients. The state of microinflammation can influence malnutrition by reducing patient appetites and exacerbating protein-energy wasting (PEW) [10]. The identification of inflammation mainly relies on measuring inflammatory markers, such as C-reactive protein (CRP), interleukin-6, and tumor necrosis factor-α, which play significant roles in promoting outcomes in PD patients. However, owing to the high price and inconvenience of these tests, inflammatory markers have not been widely used clinically. Therefore, exploring novel inflammation makers to predict the prognosis of PD patients require further exploration and identification.

Complete blood cell counts can be easily obtained and are routinely collected and tested in hospitalized patients. Previous research has shown that blood cell counts and associated ratios can predict poor prognosis in a variety of diseases [11–13]. The systemic inflammatory response index (SIRI) has recently been shown to reflect the microinflammation status, and is a predictive risk factor in the prognosis of malignant tumors [14–17]. However, there have been no reports to date regarding the association between SIRI and PD patient outcomes. For this reason, the goal of our study was to investigate the predictive value of SIRI and mortality in PD patients.

Methods

Participants

The retrospective cohort study was conducted in kidney failure patients who began PD treatment at Zhujiang Hospital from 1 January 2011 to 31 December 2018. Patients 18 years of age and older undergoing PD therapy for greater than 3 months were enrolled in our study. Exclusion criteria included: 1) patients transferred from hemodialysis (HD) or with a history of renal transplantation; 2) patients with acute or chronic infection, malignant tumors, hematological, or rheumatic diseases; and 3) patients who received immunosuppressive treatment regimens within 3 months of the study period (Figure 1).

Data collection

Demographic and laboratory data collected within 1–3 months of PD treatment were acquired and used as baseline data measurements. Demographic data included age, sex, cause of kidney failure, body mass index (BMI), CVD, hypertension, and diabetes mellitus status. Baseline laboratory data involved white blood cell count, neutrophil count, lymphocyte count, monocyte count, hemoglobin, red blood cell distribution width (RDW), serum urea nitrogen, serum creatinine, phosphorus, corrected serum calcium, cholesterol, triglyceride, serum albumin, and serum uric acid levels. The above data parameters were collected and analyzed at Zhujiang Hospital. SIRI values were computed as follows: neutrophil count × monocyte count/lymphocyte count [16]. The neutrophil-to-lymphocyte (NLR) ratio and monocyte-to-lymphocyte ratio were also calculated [12,13]. BMI was computed as weight/height^2 (kg/m^2)[18]. Kt/V was obtained by PD Adequest software version 2.0 (Baxter Healthcare Crop. Deerfield, IL, USA). The diagnostic criteria for diabetes were based on

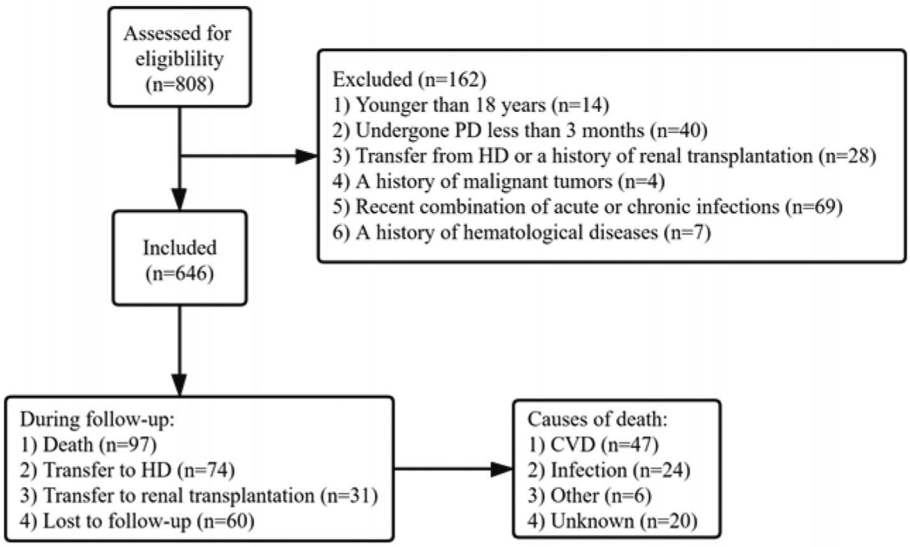


Figure 1. Study procedures, including how patients were selected and their outcomes. CVD: cardiovascular disease; PD: peritoneal dialysis; HD: hemodialysis.

those laid out by the American Diabetes Association [19]. The diagnostic criteria for hypertension were determined by having two independent blood pressure readings ≥140/90 mmHg or by the use of antihypertensive drugs [20]. CVD diagnoses were considered for patients with one of the following conditions including cardiac arrhythmia, heart failure, coronary heart disease, cardiac arrest, stroke, or peripheral vascular disease [21].

Outcomes

The primary outcome in this study was all-cause death and the second outcome was CVD-related deaths. The end-point of the study was death, discontinued PD treatment or 31 December 2019. During follow up, the cause of death was recorded for respective patients. If the patient died in the hospital, the cause of death recorded on the death certificate was collected. If death occurred outside of the hospital, medical records were reviewed by medical professionals along with a detailed description of the death provided by patient family members to determine cause of death.

Statistical analysis

Patients were assigned to either the low SIRI group or high SIRI group based on the median SIRI values. For continuous variables, data were shown as the mean ± SD; in contrast, non-normal and categorical variable data were shown as the median value (interquartile range) and numbers (proportion). Appropriate statistical methods were used to compare two groups, including the independent-samples test, Mann–Whitney U-test, and Chi-squared test. Spearman correlation was applied to explore the relationship between SIRI and demographic and laboratory indexes. The survival curve of patients was plotted using standard Kaplan–Meier methods and analyzed by the log-rank test. COX regression analysis was used to analyze the hazard ratio and 95% CI. A significant variable by univariate analysis was further analyzed by multivariate analysis using forward stepwise regression for the independent prognostic value, with results shown in table 3/4. To evaluate the prognostic ability of SIRI, MLR, and NLR, the AUC was measured by plotting the ROC curve and verified by a Z test. All p values < 0.05 were considered to be statically significant. All data were analyzed using SPSS software version 25.0 (SPSS Inc., Chicago, IL) for Windows.

Results

A total of 646 PD patients were included in this study (Table 1). The average age of the patients was 50.05 ± 14.77, and 60.8% of them were male. The causes of kidney failure in the patient population were chronic glomerulonephritis (341, 52.8%), diabetic nephropathy (100, 15.5%), obstructive nephropathy (78, 12.1%), hypertension (70, 10.8%), and other causes (57, 8.8%).

According to the median level of SIRI (1.28), PD patients were assigned to either a low SIRI group ($n = 320$) or high SIRI group ($n = 326$). At baseline, there was a higher percentage of males, diabetes-related kidney failure, increased levels of white blood cell, neutrophil, monocyte, and RDW counts among patients in the high SIRI group. Furthermore, patients in the high SIRI group were on average older and had lower lymphocyte counts than individuals in the low SIRI group. Additionally, NLR and MLR (all $p < 0.05$, Table 1) were higher in the high SIRI group. Spearman rank correlation analysis indicated that SIRI levels were negatively correlated with lymphocyte count, but positively correlated with age, white blood cell count, neutrophil, monocyte, RDW counts, and cholesterol levels (all $p < 0.05$, Table 2) in PD patients.

During the follow-up period, 165 patients discontinued the study due to initiating HD (74, 11.5%), receiving a kidney transplantation (31, 4.8%), or loss to follow up (60, 9.3%). A total of 97 (15.0%) deaths occurred, with 47 (49.0%) attributed to CVD (Figure 1). We observed statistically significant differences in all-cause mortality (log-rank = 11.58, $p = 0.001$) and CVD mortality (log-rank = 12.74, $p < 0.001$) between the two groups (Figure 2).

According to univariate Cox regression analysis, PD patients with a high SIRI level at baseline correlated with a high incidence of all-cause death (HR: 2.065, 95% CI: 1.347–3.166, $p = 0.001$). After adjustment of the data for potential confounders including CVD, diabetes, age, RDW, serum creatinine, serum albumin, high NLR levels, and high MLR levels, this association remained significant (HR: 2.007, 95% CI: 1.304–3.088, $p = 0.002$) (Table 3). Similarly, PD patients with high SIRI levels had a significantly increased risk of CVD mortality (HR: 3.201, 95% CI: 1.628–6.292, $p = 0.001$). After adjusting potential confounders including CVD, age, RDW, serum albumin, total Kt/V, high NLR levels, and high MLR levels, multivariate Cox regression analysis indicated that high SIRI levels were still associated with CVD mortality (HR: 2.847, 95% CI: 1.445–5.608, $p = 0.002$) (Table 4).

Table 1. Baseline characteristics of the study grouped by the SIRI.

Variables	Total ($N = 646$)	Low SIRI group (<1.28) ($N = 320$)	High SIRI group (≥1.28) ($N = 326$)	p
Demographics				
No. of men/women	393/253	180/140	213/113	0.018*
Age (years)	50.05 ± 14.77	47.63 ± 15.25	52.42 ± 13.90	**<0.001****
BMI (kg/cm^2)	21.93 ± 2.59	21.92 ± 2.59	21.94 ± 2.57	0.830
Causes of ESRD (*n*, %)				
Chronic glomerulonephritis	341 (52.8%)	179 (55.9%)	162 (49.7%)	–
Diabetic nephropathy	100 (15.5%)	45 (14.1%)	55 (16.9%)	–
Hypertension nephropathy	70 (10.8%)	32 (10.0%)	38 (11.7%)	–
Obstructive nephropathy	78 (12.1%)	39 (12.2%)	39 (12.0%)	–
Others	57 (8.8%)	25 (7.8%)	32 (9.8%)	–
Complication (*n*, %)				
Cardiovascular disease	203 (31.4%)	90 (28.1%)	113 (34.7%)	0.074
Diabetes mellitus	116 (25.7%)	69 (21.6%)	97 (29.8%)	**0.017***
Hypertension	618 (95.7%)	304 (95%)	314 (96.3%)	0.410
Laboratory data				
SIRI	1.28 (0.85–1.80)	0.85 (0.63–1.06)	1.79 (1.54–2.30)	**<0.001****
Neutrophil count (G/L)	3.91 (3.05–5.08)	3.14 (2.60–3.86)	4.83 (3.94–5.72)	**<0.001****
Monocyte count (G/L)	0.48 (0.38–0.62)	0.40 (0.33–0.49)	0.58 (0.46–0.71)	**<0.001****
Lymphocyte count (G/L)	1.52 (1.21–1.92)	1.62 (1.33–1.97)	1.44 (1.14–1.89)	**<0.001****
NLR	2.59 (1.96–2.40)	2.00 (1.56–2.47)	3.34 (2.70–3.97)	**<0.001****
MLR	0.31 (0.24–0.42)	0.25 (0.20–0.31)	0.40 (0.32–0.49)	**<0.001****
White blood cell count (G/L)	6.44 (5.28–7.82)	5.54 (4.76–6.66)	7.26 (6.18–8.72)	**<0.001****
Hemoglobin (g/L)	103.51 ± 18.09	103.83 ± 18.78	103.20 ± 17.42	0.655
RDW (%)	14.60 (13.60–16.03)	14.40 (13.50–15.80)	14.80 (13.70–16.20)	**0.023***
Serum urea nitrogen (mmol/L)	17.20 (13.78–20.92)	17.30 (13.93–20.30)	17.00 (13.48–22.00)	0.559
Serum creatinine (μmol/L)	712.80 (576.78–902.25)	707.00 (587.00–907.75)	714.50 (565.50–898.00)	0.992
Corrected calcium (mmol/L)	2.29 (2.19–2.40)	2.30 (2.20–2.40)	2.29 (2.17–2.42)	0.867
Phosphorus (mmol/L)	1.54 (1.28–1.81)	1.54 (1.31–1.80)	1.55 (1.27–1.86)	0.740
Serum uric acid (μmol/L)	407.00 (354.00–480.25)	404.50 (359.25–468.75)	409.00 (347.00–489.50)	0.504
Triglyceride (mmol/L)	1.29 (0.92–1.89)	1.33 (0.92–1.82)	1.29 (0.93–2.03)	0.544
Cholesterol (mmol/L)	4.30 (3.71–5.09)	4.23 (3.69–5.03)	4.37 (3.75–5.16)	0.112
Serum albumin (g/L)	31.87 ± 4.76	31.67 ± 4.57	32.05 ± 4.86	0.303
Total Kt/V	2.13 (1.79–2.45)	2.15 (1.78–2.48)	2.12 (1.79–2.43)	0.346

Bold values are when p is less than 0.05 or 0.01.
*$p < 0.05$; **$p < 0.01$.
SIRI: systemic inflammation response index; NLR: neutrophil count/monocyte count; MLR: monocyte count/lymphocyte count; RDW: red blood cell distribution width.

Table 2. Correlation between baseline SIRI and clinical, laboratory parameters.

SIRI	r	p
Age (years)	0.207	**<0.001****
BMI (kg/m^2)	−0.008	0.841
White blood cell count (G/L)	0.556	**<0.001****
Neutrophil count (G/L)	0.708	**<0.001****
Monocyte count (G/L)	0.659	**<0.001****
Lymphocyte count (G/L)	−0.207	**<0.001****
Hemoglobin (g/L)	0.021	0.596
RDW (%)	0.125	**0.001***
Serum urea nitrogen (mmol/L)	0.031	0.431
Serum creatinine (μmol/L)	0.021	0.588
Corrected calcium (mmol/L)	−0.030	0.949
Phosphorus (mmol/L)	0.002	0.960
Serum uric acid (μmol/L)	0.031	0.434
Triglyceride (mmol/L)	0.038	0.336
Cholesterol (mmol/L)	0.096	**0.015***
Serum albumin (g/L)	0.004	0.917
Total Kt/V	−0.01	0.801

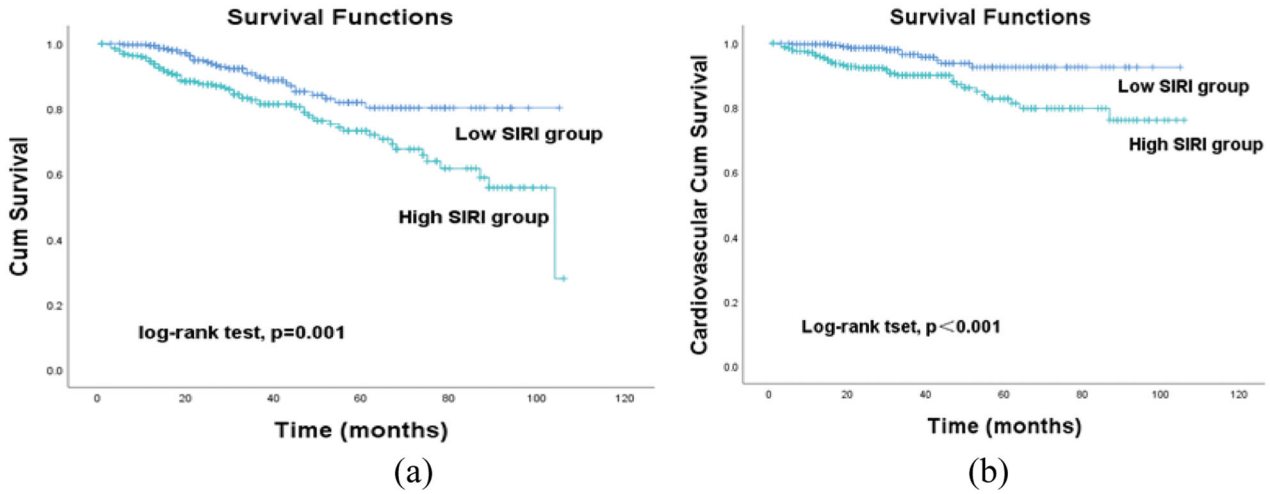
Bold values are when p is less than 0.05 or 0.01.
*$p < 0.05$; **$p < 0.01$.
RDW: red blood cell distribution width.

The ability of SIRI, NLR, and MLR to predict outcomes of PD patients was compared by measuring the AUC (Figure 3). SIRI showed a comparable power compared with the NLR and MLR for predicting all-cause and CVD-related deaths.

Discussion

Microinflammation is universal and uncontrolled in patients undergoing PD. Chronic inflammation contributes to the uremic phenotype (such as CVD, PEW, and osteoporosis) in PD patients, and thus is a major contributor to increased all-cause and cardiovascular-related mortality [22]. SIRI is a novel marker of microinflammation, and to date, no studies have assessed whether SIRI levels could be associated with poor prognosis in PD patients. To the best of our knowledge, this is the first study to identify the relationship between SIRI and risk of mortality in patients on PD. Specifically, our data indicate that higher SIRI levels were predictive of an increased incidence of all-cause and CVD deaths in PD patients, an association that was still evident after proper adjustment of the data. Additionally, SIRI had a prognostic value comparable to the NLR and MLR.

First, by analyzing the data in the low and high SIRI groups, we found that patients in the high SIRI group were older on average than patients in the low SIRI group. The relationship between aging and inflammation has been well described in previous literature.



	Months of follow-up								
	0	12	24	36	48	60	72	84	96
Low SIRI	320	293	194	128	82	50	29	13	2
High SIRI	326	284	188	134	87	61	38	24	8

Figure 2. Kaplan–Meier curves were used to analyze study endpoints that occurred during follow-up in patients grouped by the systemic inflammation response index (SIRI): overall survival (a) and cardiovascular event-free survival (b).

Table 3. Univariate and multivariate Cox regression analyses of prognostic factors for all-cause mortality.

	Univariate analyses			Multivariate analyses		
Variable	HR	95% CI	*p*	HR	95% CI	*p*
SIRI (<1.28 *vs.* ≥1.28)	2.065	1.347–3.166	0.001	2.007	1.304–3.088	0.002
Sex (male = 1; female = 2)	0.850	0.561–1.288	0.443			
Age (years)	1.047	1.030–1.063	<0.001	1.037	1.020–1.054	<0.001
BMI (kg/m^2)	1.038	0.955–1.129	0.376	–	–	–
Cardiovascular disease (no = 0; yes = 1)	1.644	1.094–2.471	0.017	–	–	–
Diabetes mellitus (no = 0; yes = 1)	2.217	1.468–3.348	<0.001	–	–	–
Hemoglobin (g/L)	0.996	0.986–1.007	0.498	–	–	–
RDW (%)	1.093	1.004–1.189	0.039	–	–	–
Serum creatinine (μmol/L)	0.999	0.998–1.000	0.004	–	–	–
Serum albumin (g/L)	0.908	0.871–0.948	<0.001	0.931	0.890–0.974	0.002
Uric acid (μmol/L)	0.999	0.997–1.001	0.494	–	–	–
Cholesterol (mmol/L)	1.051	0.888–1.244	0.565	–	–	–
Triglyceride (mmol/L)	1.079	0.925–1.259	0.334	–	–	–
Total Kt/V	0.748	0.501–1.117	0.156	–	–	–
NLR (<2.59 *vs.* ≥2.59)	1.546	1.028–2.325	0.037	–	–	–
MLR (<0.31 *vs.* ≥0.31)	2.314	1.484–3.068	<0.001	–	–	–

SIRI: systemic inflammation response index; NLR: neutrophil count/monocyte count; MLR: monocyte count/lymphocyte count; RDW: red blood cell distribution width.
In univariate Cox regression analysis, variables with $p < 0.1$ will be included in multivariate Cox regression.

Dysregulation and overactivation of inflammatory processes in the elderly results in thez persistence of chronic inflammatory conditions [23]. Furthermore, patients in the high SIRI group had a higher proportion of patients with diabetes, which is consistent with previous research. Inflammation contributes to the development of diabetes by causing insulin resistance, at the same time, the presence of hyperglycemia exacerbates inflammation [24]. Age and diabetes mellitus are traditional risk factors affecting the prognosis of PD patients. In 2014, a study in China determined that age is an independent risk factor for the prognosis of PD patients, which is consistent with our results [25]. In addition, previous research has shown that the survival rate of non-diabetic nephropathy patients with diabetes mellitus was decreased [26,27]. Diabetes mellitus as a complication of non-diabetic nephropathy has an important role in the prognosis of PD patients. However, in our study, there was not a statistically significant difference in diabetes incidence after multivariate Cox regression analysis, which could be due to the fact that this is a retrospective study with information

Table 4. Univariate and multivariate Cox regression analyses of prognostic factors for CVD mortality.

	Univariate analyses			Multivariate analyses		
Variable	HR	95% CI	*p*	HR	95% CI	*p*
SIRI (<1.28 *vs.* ≥1.28)	3.201	1.628–6.292	0.001	2.847	1.445–5.608	0.002
Sex (male = 1; female = 2)	0.629	0.337–1.176	0.147	–	–	–
Age (years)	1.048	1.025–1.072	<0.001	1.044	1.021–1.068	<0.001
BMI (kg/m^2)	0.927	0.818–1.052	0.240	–	–	–
Cardiovascular disease (no = 0; yes = 1)	1.814	1.019–3.230	0.043	–	–	–
Diabetes mellitus (no = 0; yes = 1)	1.784	0.974–3.266	0.061	–	–	–
Hemoglobin (g/L)	1.002	0.987–1.018	0.776	–	–	–
RDW (%)	1.133	1.011–1.270	0.031	–	–	–
Serum creatinine (μmol/L)	0.999	0.998–1.000	0.076	–	–	–
Serum albumin (g/L)	0.920	0.865–0.978	0.007	–	–	–
Uric acid (μmol/L)	1.000	0.997–1.003	0.908	–	–	–
Cholesterol (mmol/L)	1.066	0.842–1.350	0.596	–	–	–
Triglyceride (mmol/L)	0.968	0.736–1.272	0.813	–	–	–
Total Kt/V	0.503	0.266–0.952	0.035	–	–	–
NLR (<2.59 *vs.* ≥2.59)	2.353	1.259–4.399	0.007	–	–	–
MLR (<0.31 *vs.* ≥0.31)	2.935	1.494–5.768	0.002	–	–	–

SIRI: systemic inflammation response index; NLR: neutrophil count/ monocyte count; MLR: monocyte count/lymphocyte count; RDW: red blood cell distribution width.
In univariate Cox regression analysis, variables with $p < 0.1$ will be included in multivariate Cox regression.

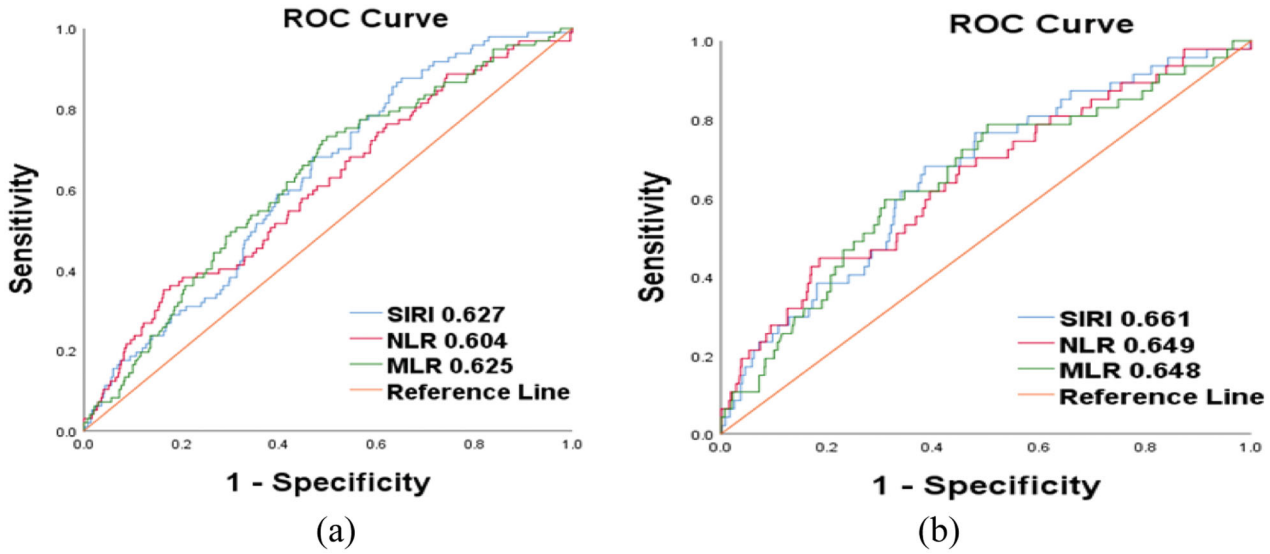


Figure 3. ROC curves were drawn and the areas under the curves (AUC) were calculated to compare the predictive value of SIRI, NLR, and MLR in all-cause death and CVD death in PD patients.

bias. Finally, malnutrition is one of many long-term complications associated with PD, occurring in approximately 30–50% of patients [28]. Serum albumin, one of the nutritive indexes we assessed, was generally recognized as a protective factor in PD patients, with higher serum albumin levels correlated with a lower mortality rate, which we also observed in our study [29,30]. After adjusting for potential confounding factors, Cox regression analysis showed that serum albumin was negatively associated with mortality in PD patients.

We further explored the interaction between SIRI and other factors in our study using Spearman correlation analysis. The RDW has been observed to be a meaningful indicator of anemia and potential metabolic abnormalities including shortening of telomere length, inflammation, and oxidative stress [31]. In our study, we also observed a positive correlation between RDW and SIRI levels, with severe microinflammation correlating with increased severity of anemia in PD patients. Soohoo et al. found that RDW was linked to outcomes and hospitalizations in PD patients, with a higher RDW correlating with a higher risk of mortality and hospitalizations [32]. In our study, SIRI levels also were positively correlated with cholesterol. Increased cholesterol in PD patients could be due to both a loss of protein in the dialysate and stimulation of glucose in the PD solutions that thereby contribute to dyslipidemia in PD patients [33,34]. Previous work in the literature found

that dyslipidemia was a major contributor to chronic intraperitoneal microinflammation [35], which is consistent with our findings that patients in the high SIRI group had higher total cholesterol levels (Table 2).

SIRI values were calculated by use of the following formula: neutrophil count × monocyte count/lymphocyte count. An increase in neutrophil and monocyte count and a decrease in lymphocyte count led to elevated SIRI levels. In previous studies, higher neutrophil and monocyte counts and lower lymphocyte counts have proven to be independent predictors of mortality risk in kidney failure patients [36–38]. In 2007, a 10-year prospective clinical study was conducted in the general population. The results showed that higher SIRI levels were linked to an increased risk of stroke and acute coronary syndrome, as well as all-cause mortality [39]. In addition, SIRI is associated with the prognosis of a variety of malignancies, such as cervical cancer [17]. In our study, we demonstrated that PD patients with a high SIRI level had a significantly increased risk for poor outcomes. Use of Cox proportional hazards models showed that high SIRI had significant predictive values for all-cause and CVD deaths. The risk of all-cause death and CVD death was 2.0 and 2.8 times higher in the high SIRI group than in the low SIRI group, respectively. The underlying mechanism by which SIRI affects the risk of all-cause death and CVD death in PD patients is unclear. SIRI is an indicator of inflammation. It integrates three immune pathways including neutrophils and monocytes that account for the persistent inflammatory response, and lymphocytes that account for immune regulation [40,41]. The higher the ratio, the greater the imbalance, and the more severe the inflammatory response. Based on our findings in this study, SIRI can be used as a risk stratification indicator for PD patients, as the early identification of microinflammation in PD patients along with a timely and powerful clinical intervention can effectively improve quality of life and prolong PD patient survival. Compared with a single indicator or a composite indicator of two factors, the composite indicator of three factors may be more stable and less susceptible to other factors, thus increasing the application value in predicting PD patients' prognoses. Therefore, SIRI may be widely promoted and useful in clinical applications to monitor and assess mortality risk of patients on PD.

Although our data presented here is novel, there are several limitations of our study. First, our study is a retrospective, single-center study and may have potential bias. Second, we only measured patient's baseline parameters; therefore, understanding how SIRI levels can change over time is extremely necessary and deserves further exploration. Finally, the study lacked a complete assessment of inflammatory markers, such as cytokines and high-sensitivity CRP. These markers should be included in further studies to further our understanding of the role inflammation and SIRI play in predicting PD patient prognosis.

Conclusion

In summary, our research presented here demonstrates that SIRI may be a useful index to indicate the prognosis of PD patients, with comparable power to the NLR and MLR.

Ethical approval

The Ethics Committee of Zhujiang Hospital approved this research (Ethics: 2022-KY-072-01), and this research was in adherence with the Declaration of Helsinki.

Author contributions

Jiaqi Li[#]: performed study, analyzed data, wrote article.
Yingxue Li[#]: performed study, wrote article.
Yaowei Zou: performed study, revised the manuscript.
Yaode Chen: performed study, collected data.
Lizhen He, Ying Wang, Jingxuan Zhou, Fangqi Xiao: collected data.
Hongxin Niu: designed study.
Lingli Lu: designed study, revised the manuscript.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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Data availability statement

The data set used in the study can be obtained from the corresponding author upon reasonable request.

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