Risk factors associated with treatment failure in peritoneal dialysis-associated peritonitis

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Abstract. Within the present study, the aim was to identify the factors associated with treatment failure in peritoneal dialysis-associated peritonitis (PDAP), thereby enabling earlier clinical identification and timely recognition of patients at an increased risk of worse prognosis. The present retrospective study utilized patients diagnosed with PDAP at the Affiliated Yongchuan Hospital of Chongqing Medical University (Chongqing, China), between May 2016 and December 2024. PDAP episodes were classified as treatment success or failure. Baseline clinical features and relevant laboratory indices were compared across the two groups. Potential risk factors for PDAP were explored using Least Absolute Shrinkage and Selection Operator (LASSO) regression in conjunction with generalized estimating equation (GEE) logistic regression analysis. The discriminative performance of the identified predictors for treatment failure was subsequently assessed through receiver operating characteristic (ROC) curve analysis. A total of 72 patients undergoing peritoneal dialysis experienced 103 PDAP episodes. Among these, 71 episodes (68.93%) resulted in treatment success and 32 (31.07%) resulted in treatment failure. LASSO regression identified four potential predictors. GEE logistic regression analysis showed that D-dimer [odds ratio (OR)=3.57; 95% CI: 1.83-6.95; P<0.001], systemic inflammatory response index (SIRI; OR=1.10; 95% CI: 1.05-1.15; P<0.001) and serum phosphorus (OR=3.16; 95% CI: 1.14-8.76; P=0.027) were independent risk factors for treatment failure in PDAP episodes. ROC analysis indicated that D-dimer exhibited the highest predictive performance [area under the curve (AUC)=0.76; 95% CI: 0.64-0.86], with an optimal cutoff value of 1.39 (sensitivity: 62.5%; specificity: 83.1%), outperforming SIRI (AUC=0.67; 95% CI: 0.54-0.80) and serum phosphorus (AUC=0.64; 95% CI: 0.51-0.76). The present study concluded that elevated D-dimer, SIRI and serum phosphorus were independently associated with treatment failure in PDAP, with D-dimer level demonstrating the strongest predictive value.

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Introduction

Compared with hemodialysis (HD), peritoneal dialysis (PD) provides multiple benefits, including greater treatment flexibility, fewer dietary limitations and improved maintenance of residual kidney function (1). Globally, PD accounts for 10-11% of all dialysis modalities, with marked regional variation and its utilization has increased steadily over the past decade (2). This increase has been largely attributable to health policy measures encouraging home-based dialysis, supported by international clinical guidelines such as those issued by Kidney Disease: Improving Global Outcomes (3) which recognize PD as a key component of patient-centered and sustainable management of end-stage kidney disease. Consistent with these recommendations, national and regional registry data have demonstrated a continued rise in both incident and prevalent PD populations, highlighting the growing role of PD in contemporary renal care (4).

Despite these developments, PD-associated peritonitis (PDAP) remains a major complication of PD and a leading cause of technique failure. Although advances in catheter technology, connection devices, patient training and antimicrobial management have contributed to a reduction in peritonitis incidence, PDAP continues to be a predominant cause of hospitalization, catheter removal and transfer to HD (5). Importantly, treatment failure not only adversely affects individual patient outcomes but also compromises long-term PD technique survival and the sustainability of PD programs (6).

Current management strategies for PDAP primarily rely on standardized empiric and pathogen-directed antibiotic regimens (7). While effective for many patients, these approaches do not fully account for the marked heterogeneity in treatment response observed in routine clinical practice. Existing guidelines, including the International Society for Peritoneal Dialysis peritonitis recommendations (2016 and 2022 updates) (7,8), primarily focus on diagnosis and antimicrobial

management, but provide limited guidance on validated indicators for predicting treatment failure, and no standardized risk prediction tools have been established (5,9,10), and clinicians often lack objective parameters to support timely escalation or individualized management decisions. As the global PD population continues to expand, there is an increasing clinical need to more precisely identify determinants associated with therapeutic failure in PDAP. Therefore, the present study aimed to identify factors associated with unsuccessful treatment outcomes in patients with PDAP, with the goal of enabling earlier intervention and improving clinical prognosis.

Materials and methods

Study population. Patients diagnosed with PDAP and treated at the Peritoneal Dialysis Center within the Department of Nephrology and Rheumatology at Yongchuan Hospital (Chongqing, China) between May 2016 and December 2024 were enrolled in the present study. The inclusion criteria were as follows: Patients aged ≥18 years who were undergoing PD and had a confirmed diagnosis of PDAP. The exclusion criteria comprised the following: i) Presence of malignant tumors, hematologic malignancies, liver cirrhosis or autoimmune diseases; ii) concurrent treatment with PD and HD or a history of kidney transplantation; iii) history of acute or chronic infection within the preceding 3 months; iv) occurrence of thromboembolic events such as cerebral infarction, myocardial infarction, pulmonary embolism or deep vein thrombosis within the past 3 months; v) use of corticosteroids, immunosuppressive agents, anticoagulants or antiplatelet drugs; and vi) incomplete clinical data.

In the present study, PDAP episodes were considered the unit of analysis. All patients were followed until discontinuation of PD due to transfer to HD, kidney transplantation or mortality, or until December 31, 2024, whichever occurred first.

Data collection. Clinical data, including age and sex distribution, baseline characteristics, PDAP episode information and treatment outcomes were retrospectively extracted from the electronic medical records of all eligible patients. A total of 72 patients who experienced 103 PDAP episodes were included in the present study. At the episode level, the mean age was 51.0 years, with a median of 52 years (range, 20-79 years; interquartile range, 44.5-56.0 years). Among the 103 PDAP episodes, 59 episodes (57.3%) occurred in male patients and 44 episodes (42.7%) occurred in female patients. The collected information included the following: i) General information: Patient age, sex, primary renal disease, comorbidities (hypertension and diabetes), dialysis vintage, precipitating factors for PDAP and whether the episode represented the first occurrence of peritonitis; ii) laboratory parameters: White blood cell count, neutrophil count, lymphocyte count, monocyte count, platelet count, hemoglobin, procalcitonin, serum albumin, serum creatinine, blood urea nitrogen, serum phosphorus, potassium, calcium, D-dimer, activated partial thromboplastin time, total cholesterol, triglycerides, low-density lipoprotein and microbiological culture results; and iii) calculated indices: Systemic inflammatory response index (SIRI) calculated using the following formula: SIRI=(neutrophil count x monocyte count)/lymphocyte count.

Diagnosis and clinical outcomes of PDAP. PDAP diagnosis was made according to the 2022 International Society for Peritoneal Dialysis guidelines for the prevention and management of PDAP (7). PDAP was diagnosed when at least two of the following three diagnostic criteria were met: i) Clinical manifestations consistent with peritonitis, including abdominal pain and/or cloudy dialysis effluent; ii) dialysate leukocyte count >100/μl (or >0.1x10^9/l), with a dwell time of ≥2 h and polymorphonuclear leukocytes accounting for >50% of the total leukocyte count; and iii) positive dialysate culture for pathogenic microorganisms. PDAP treatment failure was defined as the need for catheter removal or mortality within 30 days following the onset of peritonitis (11).

Statistical analysis. Statistical analyses were performed using R software (version 4.1.3; Posit Software, PBC). The normality of continuous variables was assessed using the Shapiro-Wilk test, supplemented by visual inspection of histograms and Q-Q plots. Normally distributed continuous variables are presented as the mean ± SD and compared using independent-sample t-tests, whereas non-normally distributed variables are presented as medians (IQR) and were analyzed using the Mann-Whitney U test. Categorical variables are reported as counts and percentages. All categorical variables were compared using Fisher's exact test with Monte Carlo simulation (10,000 replicates), which provides accurate P-value estimation regardless of the expected counts in contingency table cells. Variable selection was conducted using Least Absolute Shrinkage and Selection Operator (LASSO) regression with the λ.1se criterion and variables with non-zero coefficients were entered into a generalized estimating equation (GEE) logistic regression model with patient identity as the clustering variable to account for repeated peritonitis episodes within the same patient. Receiver operating characteristic (ROC) curve analysis was used to evaluate predictive performance. A two-sided $P<0.05$ was considered to indicate a statistically significant difference.

In the present study, the unit of analysis was the peritonitis episode rather than the individual patient. As a number of patients experienced >1 episode of peritonitis during the present study period, GEE logistic regression with patient identity as the clustering variable was used to account for intra-patient association arising from repeated episodes. GEE logistic regression analysis was performed to identify factors associated with treatment failure. The present study primarily aimed to explore episode-level risk factors.

Results

Baseline characteristics. Overall, 72 patients undergoing PD experienced a total of 103 episodes of PDAP. Among these, 71 episodes (68.93%) were classified as treatment successes and 32 (31.07%) as treatment failures. The treatment success group showed a significantly higher proportion of first-episode peritonitis compared with the treatment failure group ($P<0.05$). By contrast, no significant differences were observed between the groups with respect to sex, age, dialysis duration, underlying renal disease, comorbid conditions or precipitating causes of peritonitis ($P>0.05$). Detailed results are presented in Table I.

Table I. Baseline characteristics of 103 PD-associated peritonitis episodes.

Variable	Treatment success (n=71)	Treatment failure (n=32)	P-value
Age, years (IQR)	52 (43.50-55.00)	53 (46.50-63.00)	0.255
Men, n (%)	40 (56.34)	19 (59.38)	0.773
Primary kidney disease, n (%)			0.180
Hypertensive nephropathy	2 (2.82)	2 (6.25)	
Chronic glomerulonephritis	40 (56.34)	11 (34.38)	
Diabetic nephropathy	5 (7.04)	4 (12.50)	
Other	24 (33.80)	15 (46.88)	
Hypertension, n (%)	69 (97.18)	29 (90.63)	0.348
Diabetes mellitus, n (%)	9 (12.68)	8 (25.00)	0.119
First-onset peritonitis, n (%)	48 (67.61)	12 (37.50)	0.004
Predisposing factors, n (%)			0.812
Diarrhea	13 (18.31)	7 (21.88)	
Improper exchange procedure	4 (5.63)	2 (6.25)	
Constipation	2 (2.82)	2 (6.25)	
Other	52 (73.24)	21 (65.63)	
PD duration, n (%)			0.648
<1 years	23 (32.39)	8 (25.00)	
1-2 years	17 (23.94)	7 (21.88)	
2-3 years	9 (12.68)	2 (6.25)	
3-4 years	12 (16.90)	10 (31.25)	
4-5 years	5 (7.04)	3 (9.38)	
5-6 years	5 (7.04)	2 (6.25)	

PD, peritoneal dialysis.

Laboratory characteristics. Significant differences (P<0.05) between the two groups were observed for hemoglobin, serum albumin, serum phosphorus, monocyte count, lymphocyte count, platelet count, D-dimer level, SIRI and microbiological culture results. Detailed data are presented in Table II.

Influencing factors of PDAP treatment failure. To identify candidate risk factors for treatment failure, LASSO logistic regression with 10-fold cross-validation was performed. Using the λ.1se rule, the optimal penalization parameter was selected at λ.1se=7.038 (λ.min=4.406). At λ.1se, four predictors retained non-zero coefficients and were therefore selected for subsequent modeling: Serum albumin, serum phosphorus, D-dimer and SIRI (Figs. 1 and 2).

Variables identified by LASSO regression, including serum albumin, serum phosphorus, D-dimer and SIRI, were subsequently incorporated into a GEE logistic regression model to determine independent predictors of PDAP treatment failure. After adjustment, higher serum phosphorus [odds ratio (OR)=3.16; 95% CI: 1.14-8.76; P=0.027], higher D-dimer (OR=3.57; 95% CI: 1.83-6.95; P<0.001) and higher SIRI (OR=1.10; 95% CI: 1.05-1.15; P<0.001) were independently associated with an increased risk of treatment failure (Table III).

Predictive factors for treatment failure in patients with PDAP. ROC curve analysis showed that the D-dimer level achieved an area under the curve (AUC) value of 0.76 (95% CI: 0.64-0.86) for predicting treatment failure in PDAP. At the optimal cutoff value of 1.39, the sensitivity was 62.5% and the specificity was 83.1%, indicating an improved predictive performance compared with SIRI (AUC=0.67; 95% CI: 0.55-0.80) and serum phosphorus (AUC=0.64; 95% CI: 0.51-0.76). The corresponding ROC curves are shown in Fig. 3.

Discussion

Elevated D-dimer, SIRI and serum phosphorus levels were demonstrated to be independent predictors of treatment failure in PDAP, with D-dimer level showing the strongest predictive value in the present study.

Peritonitis is one of the most severe complications of PD. Despite extensive investigation, to the best of our knowledge, no universally accepted predictor of treatment failure in PDAP has been established. A previous investigation indicated that numerous parameters, including dialysis vintage, serum albumin level, dialysate white blood cell count on day 5 and the causative microorganism, may hold prognostic value for PDAP outcomes (11). However, additional studies have shown that these factors do not consistently predict treatment outcomes (5,12). Previously, a number of hematologic inflammation-based markers, including the neutrophil-to-lymphocyte ratio (13), systemic immune-inflammation index (14) and SIRI (15) have been

Table II. Laboratory characteristics of peritoneal dialysis-associated peritonitis episodes in the treatment success and failure groups.

Variable	Treatment success (n=71)	Treatment failure (n=32)	P-value
Hemoglobin, g/l	106.73±22.87	92.94±16.74	0.003
Serum albumin, g/l	32.10±5.22	28.06±6.85	0.001
Serum phosphorus, mmol/l	1.27±0.41	1.51±0.57	0.037
Serum calcium, mmol/l	2.18±0.18	2.12±0.33	0.315
Total cholesterol, mmol/l	4.36±0.82	4.44±1.20	0.696
White blood cell, 10^9/l	8.20 (6.15-10.40)	10.95 (6.57-14.30)	0.119
Neutrophil, 10^9/l	6.44 (4.89-9.09)	9.65 (5.09-12.69)	0.105
Monocyte, 10^9/l	0.38 (0.28-0.52)	0.50 (0.30-0.86)	0.040
Lymphocyte, 10^9/l	0.74 (0.54-0.97)	0.49 (0.39-0.80)	0.005
Platelet, 10^9/l	194.00 (162.00-236.50)	245.50 (182.25-300.25)	0.006
Serum creatinine, μmmol/l	842.00 (654.50-1027.00)	899.50 (668.75-1304.50)	0.312
Serum urea, mmol/l	18.40 (14.24-22.17)	16.90 (14.01-26.36)	0.814
Serum potassium, mmol/l	3.80 (3.35-4.10)	3.65 (3.25-4.73)	0.808
D-dimer, μg/ml	0.70 (0.47-1.15)	1.58 (0.84-2.25)	0.000
APTT, sec	26.20 (24.50-28.80)	27.50 (25.67-30.92)	0.119
Triglycerides, mmol/l	1.19 (0.84-1.82)	1.33 (0.97-1.58)	0.477
LDL, mmol/l	2.15 (1.89-2.71)	2.17 (1.90-2.84)	0.845
Procalcitonin, ng/l	1.20 (0.55-7.83)	3.05 (0.54-12.26)	0.285
SIRI, 10^9/l	3.21 (1.59-5.64)	9.90 (2.56-17.67)	0.005
Infection type, n (%)			0.033
Gram-positive strain	31 (43.66)	11 (34.38)	
Gram-negative strain	11 (15.49)	9 (28.13)	
Culture-negative peritonitis outcome	21 (29.58)	8 (25.00)	
Polymicrobial peritonitis	8 (11.27)	1 (3.13)	
Fungi peritonitis	0 (0.00)	3 (9.38)	

APTT, activated partial thromboplastin time; LDL, low-density lipoprotein; SIRI, systemic inflammatory response index. Values are presented as the median (IQR) unless indicated.

Table III. Generalized estimating equation logistic regression analysis for predicting treatment failure in peritoneal dialysis-associated peritonitis episodes.

Variable	Odds ratio	95% CI	P-value
Serum phosphorus	3.16	1.14-8.76	0.0266
D-dimer	3.57	1.83-6.95	0.0002
SIRI	1.10	1.05-1.15	0.0001
Serum albumin	0.91	0.82-1.01	0.0638

SIRI, systemic inflammatory response index.

explored as potential prognostic indicators of infectious and inflammatory diseases.

D-dimer level is a biomarker reflecting a hypercoagulable state and secondary fibrinolysis (16). Numerous studies have indicated that coagulation parameters are associated with inflammatory responses and may therefore serve as valuable markers of disease severity and prognosis in infectious conditions (17-20). In severe infections, endothelial damage triggers the exposure of tissue factors and activates the extrinsic coagulation cascade, triggering secondary fibrinolysis and generation of D-dimer as a degradation product (21). Concurrently, endothelial anticoagulant activity decreases, while platelet activation and aggregation increase, ultimately leading to microthrombus formation (22). Theoretically, microvascular thrombosis may impair peritoneal tissue perfusion and disturb microcirculatory homeostasis by reducing capillary blood flow, inducing local hypoxia, promoting endothelial dysfunction and triggering inflammatory responses (23,24). Thereby limiting solute transfer and reducing the delivery of antimicrobial agents and immune effector cells to the infection site. Impaired clearance of metabolic byproducts may further aggravate local tissue damage and microbial growth, mechanisms similar to those previously described in microcirculatory dysfunction during intra-abdominal infection-associated sepsis (25). In the present study, PDAP episodes with treatment failure exhibited significantly higher D-dimer levels compared with those with treatment success. GEE logistic regression analysis demonstrated that D-dimer independently predicted treatment failure in PDAP episodes.

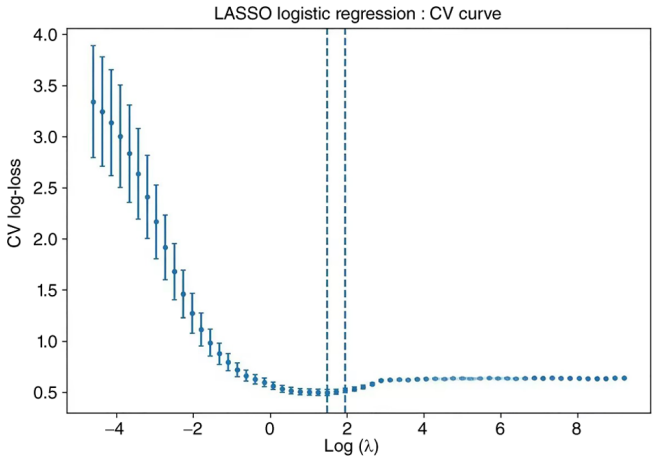

Figure 1. Cross-validation curve for LASSO logistic regression. Vertical dashed lines indicate λ.min and λ.1se. LASSO, Least Absolute Shrinkage and Selection Operator; CV, cross validation.

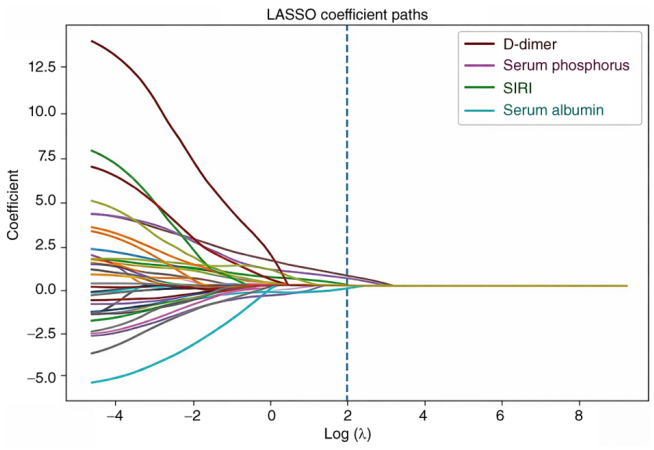

Figure 2. Coefficient paths for predictors across log(λ). Vertical dashed line indicates λ.1se. Only the four variables retained in the final model are labeled; all other variable paths are shown to illustrate the selection process.

ROC curve analysis yielded an AUC of 0.76, with an optimal cutoff value of 1.39, corresponding to 62.5% sensitivity and 83.1% specificity, underscoring its potential clinical value for the early identification of patients at an increased risk of treatment failure.

SIRI is an integrated biomarker derived from peripheral neutrophil, monocyte and lymphocyte counts (26). It reflects the imbalance between pro-inflammatory mediators (neutrophils and monocytes) and immune regulatory elements (lymphocytes) (27). During severe inflammatory responses, neutrophil and monocyte counts typically increase, whereas lymphocyte levels decrease, resulting in an overall increase in SIRI values (28). Such elevation reflects intensified systemic inflammation, a higher infectious burden and impaired host immune defense (29). Consequently, a high SIRI may indicate a pathophysiological state associated with greater disease severity and a higher probability of therapeutic failure. Previous evidence has demonstrated that SIRI is associated with all-cause mortality among patients receiving PD (30). In addition, increased SIRI has been identified as an independent predictor of both all-cause mortality and cardiovascular mortality in patients with chronic kidney disease (31). In the present study, SIRI remained an independent determinant of treatment failure in PDAP. Specifically, each one-unit increment in SIRI corresponded to an ~10% higher risk of treatment failure. ROC analysis indicated that SIRI exhibited a moderate discriminatory performance in predicting treatment outcomes (AUC=0.67). Therefore, SIRI may be more appropriately incorporated as part of a composite risk stratification model, rather than used as a solitary prognostic marker.

A previous study demonstrated that elevated serum phosphorus concentrations are independently associated with an increased risk of all-cause mortality among patients receiving PD (32). However, limited evidence exists regarding the association between serum phosphorus concentrations and PDAP. In the present study, GEE logistic regression analysis indicated that elevated serum phosphorus was associated with PDAP treatment failure (OR=3.16; 95% CI: 1.14-8.76; P=0.027). Fibroblast growth factor-23 (FGF-23), a hormone

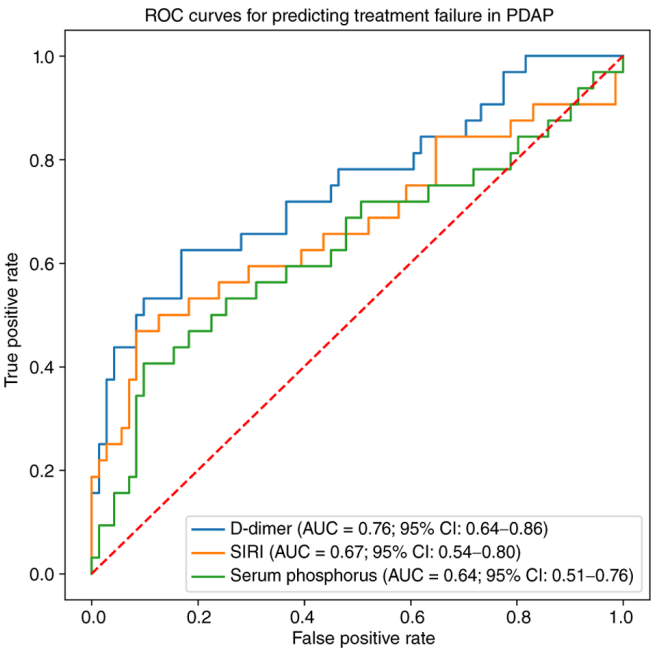

Figure 3. ROC curve of the risk prediction model for PDAP episodes. ROC, receiver operating characteristic; AUC, area under the curve; PDAP, peritoneal dialysis-associated peritonitis; SIRI, systemic inflammatory response index.

primarily secreted by osteocytes that regulates phosphate and vitamin D metabolism (33-35), has been shown to increase in response to hyperphosphatemia and act to reduce serum phosphorus levels (36). However, FGF-23 may also impair immune function. Specifically, it can inhibit the chemotaxis, phagocytosis and bactericidal activity of neutrophils (37) and macrophages (38) which serve key roles in the early immune response to peritoneal infections. Consequently, impairment of these immune functions may hinder effective pathogen elimination and reduce the effectiveness of infection control. In addition, FGF-23 promotes the production of pro-inflammatory cytokines, including IL-6, TNF-α and C-reactive protein (39), potentially exacerbating inflammatory responses and further impairing infection control by promoting immune cell dysfunction and suppressing vitamin D-mediated

antimicrobial pathways (39). Persistent hyperphosphatemia has also been recognized as an important contributor to micro-inflammation in patients with chronic kidney disease (40). It can stimulate the production of pro-inflammatory cytokines, such as IL6, IL-1β and TNF-α in vascular smooth muscle cells, endothelial cells and various other cell types (39,41). Notably, the relatively wide 95% CI observed for serum phosphorus suggests limited precision in the effect estimate, likely associated with the small sample size and limited number of treatment-failure events. Variability in phosphorus levels and residual confounding inherent to the retrospective design may also have contributed to this finding. Therefore, this result should be interpreted cautiously and validated in future larger multicenter studies.

Furthermore, numerous studies have reported that hypoalbuminemia is associated with adverse outcomes in patients undergoing PD, including an increased incidence of peritonitis (42), a higher risk of technique failure (17) and reduced long-term survival (43). In the present study, serum albumin demonstrated a borderline inverse association with treatment failure (OR=0.91; P=0.064). Despite this finding having not reached statistical significance in the GEE logistic regression model, the observed trend suggests that serum albumin may represent an important clinical factor. Possible explanations for the present result include serum albumin levels being influenced by both nutritional and inflammatory conditions and indirectly affecting infection outcomes by modulating immune responses through mechanisms including maintenance of oncotic pressure, antioxidant activity, binding and transport of inflammatory mediators, and support of immune cell function (44,45). When stronger inflammatory markers such as D-dimer and SIRI were included in the GEE regression analysis, the independent predictive value of albumin may have been partially attenuated, thereby diminishing its statistical significance. Nevertheless, the consistent protective trend observed in the present study suggests that nutritional status may remain an important contributor to treatment outcomes in PDAP.

The present study exhibits a number of limitations. First, it was a single-center retrospective study with a relatively limited sample size, including 103 PDAP episodes from 72 patients. The limited sample size may have reduced statistical power of the analysis and contributed to the relatively wide CIs observed for certain variables, such as serum phosphorus. Second, as a number of patients experienced multiple episodes of peritonitis during the present study period, the analysis was performed at the episode level. Although this approach allowed the evaluation of episode-specific risk factors, repeated episodes occurring within the same patient may have introduced intra-patient association. In the present study, GEE logistic regression was applied to account for within-patient association. Despite this, residual association among recurrent episodes may still have existed, and future studies with larger sample sizes should aim to further explore more advanced statistical approaches, such as mixed-effects models, to better account for associations among recurrent episodes. Third, although the number of variables included in the multivariable model was limited and selected using LASSO regression, the relatively small number of treatment failure events (n=32) may still raise concerns regarding model stability. Although only four predictors were included in the final model, the event-per-variable (EPV) ratio was 8, slightly below the commonly recommended threshold of ≥10 EPV for logistic regression models (46), thus the findings should be interpreted cautiously and validated in larger studies.

In summary, the present retrospective study examined elevated D-dimer, SIRI and serum phosphorus levels as independent predictors of treatment failure in PDAP, with D-dimer demonstrating the strongest predictive performance. These findings suggest that readily available laboratory indicators reflecting coagulation and systemic inflammation may provide useful information for the early identification of PDAP episodes at increased risk of treatment failure. Further large-scale multicenter prospective studies are warranted to validate these findings and to develop integrated predictive models incorporating inflammatory, coagulation, and microbiological indicators to improve risk stratification and clinical management of PDAP.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

YJ and YL conceived and designed the present study. YJ, XL and TP collected, analyzed and interpreted the data. YJ wrote the manuscript and YL provided critical revision. YJ and YL confirm the authenticity of all the raw data. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate

The present study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Yongchuan Hospital affiliated to Chongqing Medical University (Chongqing, China; approval no. 2024EC0066).

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

References

1. Andreoli MCC and Totoli C: Peritoneal dialysis. Rev Assoc Med Bras (1992) 66 (Suppl 1): S37-S44, 2020.
2. Thongprayoon C, Wathanavasin W, Suppadungsuk S, Davis PW, Miao J, Mao MA, Craici IM, Qureshi F and Cheungpasitporn W: Assessing global and regional public interest in home dialysis modalities from 2004 to 2024. Front Nephrol 4: 1489180, 2024.
3. Perl J, Brown EA, Chan CT, Couchoud C, Davies SJ, Kazancioğlu R, Klarenbach S, Liew A, Weiner DE, Cheung M, *et al*: Home dialysis: Conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. Kidney Int 103: 842-858, 2023.
4. Cho Y, Cullis B, Ethier I, Htay H, Jha V, Arruebo S, Caskey FJ, Damster S, Donner JA, Levin A, *et al*: Global structures, practices, and tools for provision of chronic peritoneal dialysis. Nephrol Dial Transplant 39 (Suppl 1): ii18-ii25, 2024.
5. Liu X, Qin A, Zhou H, He X, Cader S, Wang S, Tang Y and Qin W: Novel predictors and risk score of treatment failure in peritoneal Dialysis-related peritonitis. Front Med (Lausanne) 8: 639744, 2021.
6. Perl J, Davies SJ, Lambie M, Pisoni RL, McCullough K, Johnson DW, Sloand JA, Prichard S, Kawanishi H, Tentori F and Robinson BM: The peritoneal dialysis outcomes and practice patterns study (PDOPPS): Unifying efforts to inform practice and improve global outcomes in peritoneal dialysis. Perit Dial Int 36: 297-307, 2016.
7. Li PK, Chow KM, Cho Y, Fan S, Figueiredo AE, Harris T, Kanjanabuch T, Kim YL, Madero M, Malyszko J, *et al*: ISPD peritonitis guideline recommendations: 2022 update on prevention and treatment. Perit Dial Int 42: 110-153, 2022.
8. Li PKT, Szeto CC, Piraino B, de Arteaga J, Fan S, Figueiredo AE, Fish DN, Goffin E, Kim YL, Salzer W, *et al*: ISPD peritonitis recommendations: 2016 update on prevention and treatment. Perit Dial Int 36: 481-508, 2016.
9. Mao Y, Xiao D, Deng S and Xue S: Development of a clinical risk score system for peritoneal dialysis-associated peritonitis treatment failure. BMC Nephrol 24: 229, 2023.
10. Li J, Liu Y, Lu Y, Fu C, Ye Z and Zhang Z: Dynamic changes in serum albumin and ferritin indicate higher risk of early-onset peritonitis in peritoneal dialysis patients. Ther Apher Dial: Jan 2, 2025 (Epub ahead of print). doi: 10.1111/1744-9987.14244.
11. Meng L, Zhu X, Yang L, Li X, Cheng S, Guo S, Zhuang X, Zou H and Cui W: Development and validation of a prediction model for treatment failure in peritoneal dialysis-associated peritonitis patients: A multicenter study. Nan Fang Yi Ke Da Xue Xue Bao 42: 546-553, 2022 (In Chinese).
12. Htay H, Cho Y, Pascoe EM, Darssan D, Nadeau-Fredette AC, Hawley C, Clayton PA, Borlace M, Badve SV, Sud K, *et al*: Center effects and peritoneal dialysis peritonitis outcomes: Analysis of a national registry. Am J Kidney Dis 71: 814-821, 2018.
13. Su N, Zheng Y, Zhang X, Tang X, Tang LW, Wang Q, Chen X, Wang X, Wen Y, Feng X, *et al*: Platelet-to-lymphocyte ratio and the first occurrence of peritonitis in peritoneal dialysis patients. BMC Nephrol 23: 415, 2022.
14. Li G, Yu J, Jiang S, Wu K, Xu Y, Lu X, Wang Y, Lin J, Yang X, Li Z, *et al*: Systemic immune-inflammation index was significantly associated with all-cause and cardiovascular-specific mortalities in patients receiving peritoneal dialysis. J Inflamm Res 16: 3871-3878, 2023.
15. Yang Y, Xu Y, Liu S, Lu P, Zhou H and Yang M: The systemic inflammation indexes predict all-cause mortality in peritoneal dialysis patients. Renal failure 45: 2160348, 2023.
16. Tripodi A and Di Micco P: How we manage a high D-dimer. Haematologica 109: 933-943, 2024.
17. Cerda-Mancillas MC, Santiago-Germán D, Andrade-Bravo B, Pedraza-Olivares F, Valenzo-Hernández F, Leaños-Miranda A and Isordia-Salas I: D-dimer as a biomarker of severity and adverse outcomes in patients with community acquired pneumonia. Arch Med Res 51: 429-435, 2020.
18. Schupp T, Weidner K, Rusnak J, Jawhar S, Forner J, Dulatahu F, Brück LM, Hoffmann U, Kittel M, Bertsch T, *et al*: D-Dimer levels and the disseminated intravascular coagulation score to predict severity and outcomes in sepsis or septic shock. Clin Lab: May 1, 2023 (Epub ahead of print). doi: 10.7754/Clin.Lab.2022.221015.
19. Dumache R, Muresan CO, Laitin SMD, Ivanovic N, Chisalita A, Herlo A, Marinescu A, Lazureanu EV and Cut TG: COVID-19 organ injury pathology and D-Dimer expression patterns: A retrospective analysis. Diagnostics (Basel) 15: 1860, 2025.
20. Balaceanu LA and Dina I: D-dimers in advanced liver cirrhosis: Useful biomarker or not? Am J Med Sci 368: 415-423, 2024.
21. Fiusa MM, Carvalho-Filho MA, Annichino-Bizzacchi JM and De Paula EV: Causes and consequences of coagulation activation in sepsis: An evolutionary medicine perspective. BMC Medicine 13: 105, 2015.
22. Doganyigit Z, Eroglu E and Akyuz E: Inflammatory mediators of cytokines and chemokines in sepsis: From bench to bedside. Hum Exp Toxicol 41: 9603271221078871, 2022.
23. Levi M and van der Poll T: Inflammation and coagulation. Crit Care Med 38 (Suppl 2): S26-S34, 2010.
24. De Backer D, Orbegozo Cortés D, Donadello K and Vincent JL: Pathophysiology of microcirculatory dysfunction and the pathogenesis of septic shock. Virulence 5: 73-79, 2014.
25. Iba T, Helms J and Levy JH: Sepsis-induced coagulopathy (SIC) in the management of sepsis. Ann Intensive Care 14: 148, 2024.
26. Qi Q, Zhuang L, Shen Y, Geng Y, Yu S, Chen H, Liu L, Meng Z, Wang P and Chen Z: A novel systemic inflammation response index (SIRI) for predicting the survival of patients with pancreatic cancer after chemotherapy. Cancer 122: 2158-2167, 2016.
27. Wei Y, Wang T, Li G, Feng J, Deng L, Xu H, Yin L, Ma J, Chen D and Chen J: Investigation of systemic immune-inflammation index, neutrophil/high-density lipoprotein ratio, lymphocyte/high-density lipoprotein ratio, and monocyte/high-density lipoprotein ratio as indicators of inflammation in patients with schizophrenia and bipolar disorder. Front Psychiatry 13: 941728, 2022.
28. Agnello L, Giglio RV, Bivona G, Scazzone C, Gambino CM, Iacona A, Ciaccio AM, Lo Sasso B and Ciaccio M: The value of a complete blood count (CBC) for sepsis diagnosis and prognosis. Diagnostics (Basel) 11: 1881, 2021.
29. Wang Z, Zhang W, Chen L, Lu X and Tu Y: Lymphopenia in sepsis: A narrative review. Crit Care 28: 315, 2024.
30. Li J, Li Y, Zou Y, Chen Y, He L, Wang Y, Zhou J, Xiao F, Niu H and Lu L: Use of the systemic inflammation response index (SIRI) as a novel prognostic marker for patients on peritoneal dialysis. Ren Fail 44: 1227-1235, 2024.
31. Gu L, Xia Z, Qing B, Wang W, Chen H, Wang J, Chen Y, Gai Z, Hu R and Yuan Y: Systemic inflammatory response index (SIRI) is associated with all-cause mortality and cardiovascular mortality in population with chronic kidney disease: Evidence from NHANES (2001-2018). Front Immunol 15: 1338025, 2024.
32. Lopes MB, Karaboyas A, Zhao J, Johnson DW, Kanjanabuch T, Wilkie M, Nitta K, Kawanishi H, Perl J and Pisoni RL; PDOPPS Steering Committee: Association of single and serial measures of serum phosphorus with adverse outcomes in patients on peritoneal dialysis: Results from the international PDOPPS. Nephrol Dial Transplant 38: 193-202, 2023.
33. Liu CT, Lin YC, Lin YC, Kao CC, Chen HH, Hsu CC and Wu MS: Roles of serum calcium, phosphorus, PTH and ALP on mortality in peritoneal dialysis patients: A nationwide, population-based longitudinal study. Sci Rep 7: 33, 2017.
34. Huo Z, Liu D, Ye P, Zhang Y, Cao L, Gong N, Dou X, Ren C, Zhu Q, Li D, *et al*: Longer serum phosphorus time in range is associated with lower mortality risk among peritoneal dialysis patients: A multicenter cohort study. BMC Nephrol 25: 117, 2024.
35. White KE and Ix JH: Regulation of FGF23 production and phosphate metabolism. Nat Rev Nephrol 19: 185-193, 2023.
36. Fitzpatrick EA, Han X, Xiao Z and Quarles LD: Role of fibroblast growth Factor-23 in innate immune responses. Front Endocrinol (Lausanne) 9: 320, 2018.
37. Rossaint J, Oehmichen J, Van Aken H, Reuter S, Pavenstädt HJ, Meersch M, Unruh M and Zarbock A: FGF23 signaling impairs neutrophil recruitment and host defense during CKD. J Clin Invest 126: 962-974, 2016.
38. Han X, Li L, Yang J, King G, Xiao Z and Quarles LD: Counter-regulatory paracrine actions of FGF-23 and 1,25(OH)2 D in macrophages. FEBS Lett 590: 53-67, 2016.
39. Raju S and Saxena R: Hyperphosphatemia in kidney failure: Pathophysiology, challenges, and critical role of phosphorus management. Nutrients 17: 1587, 2025.
40. Yamada S, Tokumoto M, Tatsumoto N, Taniguchi M, Noguchi H, Nakano T, Masutani K, Ooboshi H, Tsuruya K and Kitazono T: Phosphate overload directly induces systemic inflammation and malnutrition as well as vascular calcification in uremia. Am J Physiol Renal Physiol 306: F1418-F1428, 2014.

41. Voelkl J, Lang F, Eckardt KU, Amann K, Kuro OM, Pasch A, Pieske B and Alesutan I: Signaling pathways involved in vascular smooth muscle cell calcification during hyperphosphatemia. Cell Mol Life Sci 76: 2077-2091, 2019.
42. Zha D, Yang X and Xi H: Association of hypoalbuminemia with the risk of peritoneal dialysis-associated peritonitis in peritoneal dialysis patients: A meta-analysis. Blood Purif: Feb 10, 2025 (Epub ahead of print). doi: 10.1159/000543693.
43. Zhang L, Cao T, Li Z, Wen Q, Lin J, Zhang X, Guo Q, Yang X, Yu X and Mao H: Clinical outcomes of peritoneal dialysis patients transferred from hemodialysis: A matched case-control study. Perit Dial Int 33: 259-266, 2013.
44. Arques S: Human serum albumin in cardiovascular diseases. Eur J Intern Med 52: 8-12, 2018.
45. Soeters PB, Wolfe RR and Shenkin A: Hypoalbuminemia: Pathogenesis and clinical significance. JPEN J Parenter Enteral Nutr 43: 181-193, 2019.
46. van Smeden M, Moons KGM, de Groot JAH, Collins GS, Altman DG, Eijkemans MJ and Reitsma JB: Sample size for binary logistic prediction models: Beyond events per variable criteria. Stat Methods Med Res 28: 2455-2474, 2018.