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Original Article

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Long-Term Predictors of Mortality in Peritoneal Dialysis Patients

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ABSTRACT

Objective: Peritoneal dialysis (PD) is one of the essential treatments for end-stage renal disease. Cardiovascular disease and peritonitis are the most common causes of death among PD patients. This study aimed to explore the long-term mortality predictors of PD patients.

Methods: One hundred forty-one PD patients were retrospectively included in the study. Patients were selected among cases admitted to the PD outpatient clinic between January 2015 and November 2020. Clinical and laboratory findings obtained at the first visit were recorded. Prevalence of all-cause mortality and associated prognostic factors were analyzed.

Results: The mean age of the patients was 52.5 ± 15.2 years (range 19-86). Thirty-two patients (22.7%) died in the follow-up. Age, hemodialysis (HD) history, diabetes mellitus (DM), ultrafiltration, urine volume, glomerular filtration rate, and normalized protein catabolic rate in modified peritoneal equilibrium test were significantly different between survived and non-survived patients. Non-survived patients had higher ferritin, C-reactive protein (CRP), and CRP to albumin ratio (CAR) (*P*: .003, *P*:<.001; *P*: <.001; respectively). The Cox regression analysis revealed that the presence of DM HR 95%CI: 3.755 (1.703-8.280), *P*: .001, HD history HR 95%CI: 2.843 (1.291-6.263), *P*: .010, and higher CAR ratio HR 95% CI: 4.235 (1.857-9.662), *P*: .001 were independent predictors of all-cause mortality. ROC curve analysis demonstrated that CAR > 1.94 predicted all-cause-mortality with 73% sensitivity and 71% specificity (AUC = 0.789, 95%CI: 0.697-0.881).

Conclusion: The present study demonstrates that DM, HD history, and high CAR are independent predictors of all-cause-mortality among PD patients.

Keywords: Inflammation, mortality, peritoneal dialysis

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INTRODUCTION

Peritoneal dialysis (PD) is one of the best treatments for end-stage renal disease (ESRD) patients. Currently, 11% of ESRD patients are treated with PD.[1] The survival of PD patients is associated with a variety of clinical factors such as demographic findings, comorbidities, nutritional markers, and peritoneal clearance.[2] Cardiovascular disease and peritonitis are the most common causes of death among this population.[3] It is well known that increased age and the presence of diabetes are the major risk factors for mortality in the PD population.[4] In addition, PD patients tend to exhibit persistent inflammation and inflammatory biomarkers such as C-reactive protein (CRP), serum albumin, and ferritin levels are strongly associated with mortality and cardiovascular events in PD patients.[5,6] Inflammation is also negatively associated with residual renal function (RRF), which represents better survival in PD patients. CRP levels are often used to assess systemic inflammation and are associated with all-cause and cardiovascular mortality in patients on PD.[7] Low serum albumin levels can be attributed to inflammation or may also indicate malnutrition and is a negative predictor of survival in patients with ESRD.

In this study, we aimed to investigate the long-term prognostic factors of mortality in PD patients.

METHODS

The investigation conforms to the principles outlined in the Declaration of Helsinki. The ethics committee of Marmara University Medical School approved the study (Protocol code: 09.2020.1151). Patients were selected among cases admitted to the PD outpatient clinic between January 2015 and November 2020.

One hundred forty-one consecutive PD patients were recruited into the study. This was a retrospective, cross-sectional study, and we included patients who had been on PD for at least 3 months. Patients under the age of 18 years and patients with missing data were excluded. Medical history, demographic data, death, cause of death, transfer to hemodialysis (HD), and kidney transplantation were recorded. Duration of PD, PD modality, causes of ESRD, history of HD, peritonitis episodes, baseline residual renal function, and laboratory findings obtained at the first visit were recorded from patient charts.

Glucose, blood urea nitrogen (BUN), creatinine, uric acid, serum albumin, sodium, potassium, serum calcium, phosphorus, and intact parathyroid hormone (iPTH), 25-OH vitamin D, ferritin, HbA1c, complete blood count, and CRP were recorded on the first visit as a part of routine care. Serum levels of uric acid, phosphorus, calcium, total cholesterol, triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein, and ferritin were analyzed using standard laboratory methods and expressed as milligrams per deciliter. CRP levels were measured using the nephelometric method (Date Behring Siemens, Marburg, Germany) and expressed as milligrams per liter. iPTH serum level was determined by enzyme-amplified sensitive immunoassay (Roche Diagnostics, IN, USA) expressed as nanograms per liter. 25-OH vitamin D levels analyzed by chemiluminescent immunoassay. We calculated the CRP to albumin ratio (CAR) (mg/g) by dividing CRP level to albumin level.

We used the modified peritoneal equilibrium test (PET) for assessing water and solute transport across the peritoneal membrane. The standard procedure for PET started with drainage of fluid from the patient's peritoneal cavity, followed by the 3.86% glucose dialysis solution's infusion with a dwell time of 4 h. Dialysate samples (10 mL) were collected at 0 h (immediately after infusion) and 1, 2, and 4 h after infusion. Blood samples (10 mL) were collected at 0 h (immediately after infusion) and at 1, 2, and 4 h after infusion. Dialysis infusion initiation and drainage times were recorded.[8] Residual renal function (24-h urine volume), dialysate volume, PET ultrafiltration (UF), eGFR, nPCR, KT/V, D/P creatinine, PET permeability types were obtained. The first PET was performed 6 months after starting PD and was recorded. The primary endpoint of the study was all-cause of mortality during follow-up. PD patients were divided into 2 groups as survived and non-survived. Clinical demographic and laboratory data of the groups were analyzed and compared.

Statistical Analysis

All statistical tests were performed with Statistical Package for the Social Sciences (SPSS) version 22.0 (IBM SPSS Corp.; Armonk, NY, USA. Categorical variables were presented as numbers and percentages and compared with the chi-square test. Continuous variables were presented as mean ± standard deviation. Continuous variables with parametric distribution were compared with independent samples *t*-test. The distribution of data was assessed by using a one-sample Kolmogorov–Smirnov test. Mann–Whitney *U* test was used to compare continuous variables, while the chi-square test was used to compare categorical variables. The Cox proportional hazards models were used to assess independent predictors of all-cause mortality in patients who receiving PD. A receiver operating characteristic (ROC) curve was generated to determine the predictive power of CAR for mortality. Sensitivity and specificity values were also computed for the optimum cut-off value of the CAR. *P*-values < .05 were interpreted as statistically significant.

RESULTS

One hundred forty-one PD patients were consecutively included in the study. The mean age of patients was 52.5 ± 15.2 years (range 19-86 years), and 71 patients (50.4%) were men. The mean duration of PD was 65.8 ± 54.7 months, and 103 (73%) of patients were on continuous ambulatory PD, 38 (27%) of those were on automated PD. Twenty-seven (19.1%) of the patients had a history of HD before starting PD, and none of the patients had a history of transplantation before starting PD.

Thirty-two patients (22.7%) died; 39 (27.7%) patients were transferred to HD; 18 (12.8%) patients received a kidney transplant, and 52 (36.8%) patients continued on PD therapy during

Main Points

- Peritoneal dialysis (PD) is one of the essential treatments for end-stage renal disease. Cardiovascular disease and peritonitis are the most common causes of death among PD patients. This study aimed to explore the long-term mortality predictors of patients who are receiving PD.
- Thirty-two patients (22.7%) died in the follow-up. Non-survived patients had higher ferritin, C-reactive protein (CRP) and CRP to albumin ratio (CAR).
- The present study demonstrates that the presence of DM, HD history, and high CAR are independent predictors of all-cause mortality among PD patients.
- Chronic exposure of inflammation could explain accelerated atherosclerosis in PD patients. Early diagnosis and controlling inflammation may reduce the mortality of peritoneal dialysis patients.

Table 1. Causes of Death and Hemodialysis Transfer in Peritoneal Dialysis (PD) Patients

Causes of Death	*n* (%)	Causes of Transvers to HD	*n* (%)
Cardiovascular	13 (40.6)	Peritonitis	20 (51.3%)
Pneumonia	5 (15.6%)	PD inadequacy	7 (17.9%)
Peritonitis	5 (15.6%)	Technical failure	4 (10.3%)
Cerebrovascular accident	5 (15.6%)	Patient choice	3 (7.7%)
Peritoneal sclerosis	1 (3.1%)	Peritoneal sclerosis	3 (7.7%)
Malignancy	1 (3.1%)	Tunnel infection	2 (5.1%)
Cholangitis	1 (3.1%)		
Unknown	1 (3.1%)		

HD, hemodialysis; PD, peritoneal dialysis.

follow-up. The causes of death and transfer to HD are shown in Table 1. The leading cause of death was cardiovascular disease (40.6%). The most common causes of transfer to HD were peritonitis (51.3%), inadequate PD (17.9%), and technical failure (10.3%).

The demographic features and baseline laboratory findings of the study groups are summarized in Tables 2and 3. Older age, HD history, and diabetes mellitus (DM) were significantly more prevalent in the non-survived group. Non-survived patients had also lower albumin levels and higher ferritin, CRP, and CAR. Non-survived patients had significantly lower UF, urine volume, Egfr, and nPCR in modified PET. PET measurements of the groups are summarized in Table 4.

Since inflammation is a continuous process during dialysis vintage, we performed a subgroup analysis in which we excluded patients with a history of HD. One hundred fourteen patients were analyzed, and 21(18.4%) patients died during follow-up. Diabetes mellitus (DM) was significantly more prevalent in the non-survived group (38.1% vs. 11.8%; *P*: .007). Non-survived patients had also lower albumin levels 3.6 (0.66) vs. 3.8 (0.68) g/dL; *P*: .017 and higher ferritin 425.0 (468.3) vs. 241.0 (298.5) µg/L; *P*: .001, CRP 14.0 (14.6) vs. 4.0 (6.0) mg/L; $P < .001$, and CAR 3.9 (4.1) vs. 1.0 (1.5) mg/L; $P < .001$. Non-survived patients had significantly lower urine volume 415.0 (895.0) vs. 1000.0 (1275.0) mL/day; *P*: .023 and nPCR 0.89 (0.36) vs. 0.97 (0.30) g/kg/day; *P*: .029 in modified PET.

The Cox regression analyses showed that DM, HD history, and CAR were independent predictors of all-cause mortality (Table 5). ROC curve analysis demonstrated that CAR > 1.94 predicted all-cause mortality with 73% sensitivity and 71% specificity (area under curve = 0.789, 95% CI 0.697-0.881) (Figure 1). However, peritonitis and glucose exposure were similar between non-survived and survived groups.

Table 2. Demographic Data of Non-survived and Survived Peritoneal Dialysis (PD) Patients

Variables	Non-survived (*n* = 32)	Survived (*n* = 109)	*P*
Age, years	59.5 (13.4)	50.4 (15.1)	**.002**
Sex, male *n* (%)	14 (19.7%)	57 (80.3%)	.427
Body mass index, kg/m^2	25.9 (5.8)	25.7 (5.3)	.845
Duration of PD, months	69.9 ±58.3	64.6 (53.8)	.649
Type of PD, CAPD, *n* (%)	24 (23.3%)	79 (76.7%)	1
Glucose exposure, mg/day	131.8 (101.8)	121.4 (73.8)	.521
Urine volume, mL	559.7 (561.7)	1015.4 (884.1)	**.007**
Presence of HD history, *n* (%)	11 (40.7%)	16 (%59.3)	**.020**
Peritonitis, *n* (%)	21 (25.6%)	61 (74.4%)	.416
Cause of ESRD			
Diabetes mellitus, *n* (%)	11 (45.8)	13 (54.2%)	**.006**
Hypertension, *n* (%)	18 (29.5%)	43 (70.5%)	.107
Glomerulonephritis, *n* (%)	3 (8.3%)	33 (91.7%)	**.020**
Polycystic kidney, *n* (%)	2 (22.2%)	7 (77.8%)	1
Vesico-ureteral reflux, *n* (%)	1 (12.5%)	7 (87.5%)	.683
Amyloidosis, *n* (%)	1 (20%)	4 (80%)	1
Nephrolithiasis, *n* (%)	1 (33.3%)	2 (66.7%)	.541
Others, *n* (%)	1 (25%)	3 (75%)	1
Unknown, *n* (%)	4 (22.2%)	14 (77.8%)	1

CAPD, continuous ambulatory peritoneal dialysis; ESRD, end-stage renal disease; HD, hemodialysis; PD, peritoneal dialysis.
Data presented as median (interquartile range).

DISCUSSION

The present study demonstrates that advanced age, history of HD, presence of diabetes, and systemic inflammation with higher ferritin, CRP, and CAR, lower albumin, and lower urine volume has an impact on PD patient survival. Our results show that diabetes, HD history, and CAR are independent risk factors for all-cause mortality among PD patients.

While our patient's 5-year mortality rate was 22.7%, Utas et al.[9] found 3- and 5-year patient survival rates to be 84.5 and 68.9%, respectively, in 2001 in Turkey. In another study in Turkey, Unsal A et al.[10] found a survival rate of 85.2, 66.5, and 45.3% at 1, 3, and 5 years, respectively. In 2006, United States Renal Data System data showed that the 5-year survival rate was 32% in the United States of America.[11] Our 5-year survival rate of 77.3% and

Table 3. Laboratory Data of Non-survived and Survived Peritoneal Dialysis (PD) Patients

Variables	Non-survived (*n* = 32)	Survived (*n* = 109)	*P*
Glucose, mg/dL	124.8 (61.1)	104.7 (48.8)	.056
BUN, mg/dL	59.8 (24.5)	61.4 (19.3)	.696
Creatinine, mg/dL	7.8 (2.9)	7.8 (2.3)	.971
Albumin, g/dL	3.5 (0.5)	3.8 (0.5)	**.002**
Uric acid, mg/dL	5.8 (1.3)	6.2 (1.4)	.156
Calcium, mg/dL	9.0 (0.7)	8.9 (1.0)	.645
Phosphorus, mg/dL	5.0 (1.6)	5.0 (1.3)	.756
Sodium, mEq/L	137.6 (3.2)	137.2 (3.7)	.615
Potassium, mEq/L	4.4 (0.8)	4.4 (0.7)	.968
Hemoglobin, g/dL	10.4 (1.7)	10.6 (1.5)	.531
Leucocyte, $\times 10^3/\mu L$	7.7 (2.3)	7.5 (2.2)	.601
Platelet, $\times 10^3/\mu L$	251.2 (83.4)	250.4 (79.2)	.963
CRP, mg/L	17.0 (14.4)	6.5 (5.0)	**<.001**
CRP to albumin ratio	5.3 (5.0)	1.7 (5.0)	**<.001**
HbA1c, %	5.8 (1.3)	5.4 (1.0)	.093
25-OH vitamin D, µg/L	7.6 (5.9)	8.8 (5.9)	.344
Parathyroid hormone, ng/L	367.0 (308.3)	456.0 (327.2)	.172
Ferritin, µg/L	515.0 (414.3)	317.7 (292.0)	**.003**

BUN, blood urea nitrogen; CRP, C-reactive protein.
Data presented as median (interquartile range).

Table 4. PET Results of the Non-survived and Survived Patients

	Non-survived	Survived	*P*
PET dialysate volume, mL	7350.0 ± 2770.9	7319.7 (2941.7)	.957
PET UF, mL	584.0 ± 256.0	762.8 (568.7)	**.013**
PET eGFR, mL/min/1.73 m^2	2.7 ± 3.2	4.3 (3.8)	**.023**
PET nPCR, g/kg/day	0.9 ± 0.3	1.1 (0.5)	**.031**
PET KT/V	2.1 ± 0.4	2.2 (0.6)	.198
PET D/P creatinine	0.7 ± 0.1	0.7 (0.1)	.562
PET permeability, *n* (%)			.723
High	8 (28.6%)	20 (71.4%)	
High average	15 (21.7%)	54 (78.3%)	
Low	2 (33.3%)	4 (66.7%)	
Low average	7 (18.4%)	31 (81.6%)	

D/P, dialysate/plasma; eGFR, estimated glomerular filtration rate; nPCR, normalized protein catabolic rate; PET, peritoneal equilibrium test; UF, ultrafiltration.
Data presented as mean + standard deviation.

Table 5. COX Regression Analysis Showing Independent Predictors of All-Cause Mortality

	Hazard Ratio (95% CI)	*P*
Hemodialysis history	2.843 (1.291-6.263)	.010
Diabetes mellitus	3.755 (1.703-8.280)	.001
CAR ≤ 1.94	4.235 (1.857-9.662)	.001

CAR, C-reactive protein to albumin ratio.

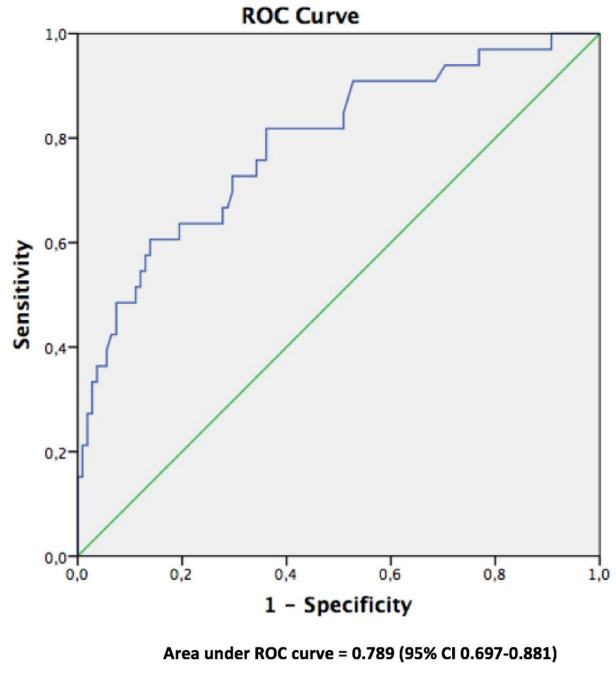

Figure 1 : Receiver operating characteristic analysis demonstrated that C-reactive protein (CRP) to albumin ratio (CAR) > 1.94 predicts all-cause mortality with 73% sensitivity and 71% specificity.

was better than previously published studies. Cardiovascular disease was the most common cause of death, followed by infection in our cohort, which was similar to other studies.[3,12]

In our study, non-survived patients had older age than the survived group, similar to findings in Sakaci et al.'s[13] study. They showed that mortality was higher in elderly patients and advanced age, comorbidities, peritonitis attacks, hypoalbuminemia and decreased daily urine volumes (<100 mL) were factors affecting mortality. Elderly dialysis patients tend to have more comorbidities, such as cardiovascular diseases, malnutrition, and cerebrovascular accidents. On the other hand, the prevalence of elderly patients requiring dialysis treatment has also increased in the recent years. These factors may contribute to mortality in advanced ages.

Similar to our results, the presence of HD history, diabetes, and low serum albumin were found to be poor predictors of patient survival by Unsal et al.[10] Fluid status and volume homeostasis

are cornerstones of therapy in PD patients. It is well known that RRF decreases in PD patients switched to HD. Inadequate fluid removal contributes to hypertension and also cardiovascular diseases and may play a potential role in the increased mortality of these patients. Park et al.[14] showed that absence of RRF and hypoalbuminemia affects the survival of PD patients. Besides serum albumin levels, urine volume was also significantly lower in non-survived patients in our cohort.

Co-existing cardiovascular diseases and PD membrane alterations due to advanced glycation end products in PD patients with diabetes were the major reasons for increased mortality in these patients. In our study, 34.4% of non-survived patients had DM, and it was significantly higher than the survived group (12%). Similarly, Sakaci et al.[13] showed that diabetes and comorbidities affect mortality rates in elderly PD patients. Unsal et al.[10] retrospectively evaluated 322 PD patients and showed that patient survival was significantly lower in diabetic patients than non-diabetics. Patients with a history of HD had a higher mortality rate than those without a history of HD.

Elevated serum ferritin levels have been observed in patients with renal impairment, and increasing evidence has shown that hyperferritinemia is significantly associated with poor prognosis and higher mortality in PD patients.[15] While the mechanisms underlying the relationship between higher ferritin levels and mortality are not fully understood, possible explanations have been proposed. Serum ferritin leaks from damaged cells, and circulating ferritin can cause oxidative stress; moreover, released iron can stimulate further cell damage.[16,17] Fu et al.[12] showed that hyperferritinemia was associated with increased mortality in PD patients. These results are in accord with our observation that increased ferritin levels are associated with higher mortality in PD patients.

High plasma CRP levels are frequently observed in CKD patients, and prospective studies have shown that CRP predicts mortality in dialysis patients.[18] Wang et al.[19] showed that increased CRP levels have independent prognostic value in PD patients. Chen et al.[20] found that CRP was an independent predictor for cardiovascular and all-cause mortality in PD patients. Our findings are similar to those reported in these previous studies.

Albumin is a negative acute-phase reactant and hypoalbuminemia is associated with mortality. In our study, serum albumin levels were significantly lower in the non-survived group. There are various studies showing low serum albumin levels to be associated with increased mortality in PD patients.[21,22] Alves et al.[21] showed that inflammation has a catalytic effect on serum albumin, an independent risk predictor for poor outcome only in the presence of inflammation, whereas hypoalbuminemia without inflammation carries a negligible mortality risk. Thus, it may be reasonable to evaluate serum albumin levels with regard to other inflammatory markers in PD patients.

Indeed, CAR, a composite indicator of inflammation and nutritional status, has recently been recognized as an independent prognostic marker for use in several cancer types, Crohn's disease, vasculitides and critically ill patients.[22-25] Data on usefulness of CAR in PD patients are scarce.[8] Also, there are currently limited data from which to determine the safe cut-off values for serum CAR levels to predict mortality in PD patients. In our cohort, a cut-off value for CAR of 1.94 is a significant risk factor for mortality. Liu S et al.[8] showed that elevated serum CAR in PD patients is independently associated with increased risk for all-cause mortality. Further studies are needed to investigate the impact of CAR on mortality in PD patients.

The major limitation of our study was the small sample size and its retrospective nature. We didn't investigate changes in the laboratory values, glucose exposure and residual renal function of the patients within 5 years of follow-up, which could have had an impact on mortality. In view of decreasing global PD rates, we believe our study will help to understand risk factors associated with mortality despite the small sample size.

CONCLUSION

Inflammation markers are essential for predicting mortality in PD patients. In this study, we found that CAR, which is strong marker of inflammation, was an independent predictor of mortality in patients on PD therapy. Chronic inflammation may be responsible for accelerated atherosclerosis in PD patients. Therefore, all treatments, which decrease inflammation, may reduce mortality in PD patients.

Ethics Committee Approval: Ethics committee approval was received from the Ethics Committee of Marmara University Medical School approved the study (Protocol code: 09.2020.1151).

Informed Consent: Informed consent was not obtained due to the retrospective design of this study.

Peer-review: Externally peer-reviewed.

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REFERENCES

1. Li PK, Chow KM, Van de Luijtgaarden MWM, et al. Changes in the worldwide epidemiology of peritoneal dialysis. *Nat Rev Nephrol*. 2017;13(2):90-103. [CrossRef]
2. Genestier S, Hedelin G, Schaffer P, Faller B. Prognostic factors in CAPD patients: a retrospective study of a 10-year period. *Nephrol Dial Transplant*. 1995;10(10):1905-1911.

3. Mehrotra R, Devuyst O, Davies SJ, Johnson DW. The current state of peritoneal dialysis. *J Am Soc Nephrol*. 2016;27(11):3238-3252. [CrossRef]
4. Kendrick J, Teitelbaum I. Strategies for improving long-term survival in peritoneal dialysis patients. *Clin J Am Soc Nephrol*. 2010;5(6):1123-1131. [CrossRef]
5. Ishii J, Takahashi H, Kitagawa F, et al. Multimarker approach to risk stratification for long-term mortality in patients on chronic hemodialysis. *Circ J*. 2015;79(3):656-663. [CrossRef]
6. Park SH, Stenvinkel P, Lindholm B. Cardiovascular biomarkers in chronic kidney disease. *J Ren Nutr*. 2012;22(1):120-127. [CrossRef]
7. Li W, Xiong L, Fan L, et al. Association of baseline, longitudinal serum high-sensitive C-reactive protein and its change with mortality in peritoneal dialysis patients. *BMC Nephrol*. 2017;18(1):211. [CrossRef]
8. Liu S, Qiu P, Luo L, et al. Serum C-reactive protein to albumin ratio and mortality associated with peritoneal dialysis. *Ren Fail*. 2020;42(1):600-606. [CrossRef]
9. Utaş C., Turkish Multicenter Peritoneal Dialysis Study Group. Patient and technique survival on CAPD in Turkey. *Perit Dial Int*. 2001;21(6):602-606. [CrossRef]
10. Unsal A, Koc Y, Basturk T, et al. Clinical outcomes and mortality in peritoneal dialysis patients: a 10-year retrospective analysis in a single center. *Clin Nephrol*. 2013;80(4):270-279. [CrossRef]
11. Collins AJ, Kasiske B, Herzog C, et al. Excerpts from the United States renal data System 2006 annual data report. *Am J Kidney Dis*. 2007;49(1)(suppl 1):A6-7, S1. [CrossRef]
12. Fu S, Chen J, Liu B, et al. Systemic inflammation modulates the ability of serum ferritin to predict all-cause and cardiovascular mortality in peritoneal dialysis patients. *BMC Nephrol*. 2020;21(1):237. [CrossRef]
13. Sakacı T, Ahbap E, Koc Y, et al. Clinical outcomes and mortality in elderly peritoneal dialysis patients. *Clinics*. 2015;70(5):363-368. [CrossRef]
14. Park JY, Cho JH, Jang HM, et al. Survival predictors in anuric patients on peritoneal dialysis: A prospective, multicenter, propensity score-matched cohort study. *PLoS One*. 2018;13(4):e0196294. [CrossRef]
15. Hur SM, Ju HY, Park MY, et al. Ferritin as a predictor of decline in residual renal function in peritoneal dialysis patients. *Korean J Intern Med*. 2014;29(4):489-497. [CrossRef]
16. Kell DB, Pretorius E. Serum ferritin is an important inflammatory disease marker, as it is mainly a leakage product from damaged cells. *Metallomics*. 2014;6(4):748-773. [CrossRef]
17. Hasuike Y, Nonoguchi H, Tokuyama M, et al. Pathological role of aminolevulinate in uremic patients. *Ther Apher Dial*. 2011;15(1):28-33. [CrossRef]
18. Wanner C, Zimmermann J, Schwedler S, Metzger T. Inflammation and cardiovascular risk in dialysis patients. *Kidney Int Suppl*. 2002;61(80):99-102. [CrossRef]
19. Wang AY, Woo J, Lam CW, et al. Is a single time point C-reactive protein predictive of outcome in peritoneal dialysis patients? *J Am Soc Nephrol*. 2003;14(7):1871-1879. [CrossRef]
20. Chen T, Hassan HC, Qian P, Vu M, Makris A. High-sensitivity troponin T and C-reactive protein have different prognostic values in hemo- and peritoneal dialysis populations: a cohort study. *J Am Heart Assoc*. 2018;7(5):e007876. [CrossRef]
21. Alves FC, Sun J, Qureshi AR, et al. The higher mortality associated with low serum albumin is dependent on systemic inflammation in end-stage kidney disease. *PLoS One*. 2018;13(1):e0190410. [CrossRef]
22. Oh TK, Song IA, Lee JH. Clinical usefulness of C-reactive protein to albumin ratio in predicting 30-day mortality in critically ill patients: a retrospective analysis. *Sci Rep*. 2018;8(1):14977. [CrossRef]
23. Mao M, Wei X, Sheng H, et al. C-reactive protein/albumin and neutrophil/lymphocyte ratios and their combination predict overall survival in patients with gastric cancer. *Oncol Lett*. 2017;14(6):7417-7424. [CrossRef]
24. Qin G, Tu J, Liu L, et al. Serum albumin and C-reactive protein/albumin ratio are useful biomarkers of Crohn's disease activity. *Med Sci Monit*. 2016;22:4393-4400. [CrossRef]
25. Moon JS, Ahn SS, Park YB, Lee SK, Lee SW. C-reactive protein to serum albumin ratio is an independent predictor of all-cause mortality in patients with ANCA-associated vasculitis. *Yonsei Med J*. 2018;59(7):865-871. [CrossRef]
26. Liu S, Qiu P, Luo L, et al. Serum C-reactive protein to albumin ratio and mortality associated with peritoneal dialysis. *Ren Fail*. 2020;42(1):600-606. [CrossRef]