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Baseline serum triglyceride predicts early-onset peritonitis and prognosis in incident CAPD patients

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Abstract

We aimed to investigate the hypothesis that serum triglyceride (TG) may be an independent predictor of early-onset peritonitis and prognosis in incident continuous ambulatory peritoneal dialysis (CAPD) patients.

In this retrospective, observational study, we screened 291 adults admitted to the PD center of the Wuhan No. 1 hospital from August 1, 2013 to November 31, 2017. All biochemical data were collected at the first 1 to 3 months after the initiation of CAPD. Early-onset peritonitis was defined as peritonitis occurring within 6 months after the initiation of PD. All of PD patients were followed up to July 31, 2018. The primary endpoint was the incidence of early-onset peritonitis while the second endpoints included overall mortality and technical failure.

A total of 38 patients occurred early-onset PD peritonitis and the Lasso logistic regression selected TG and age in the final model for early-onset peritonitis. We divided patients into two groups based on the median baseline TG levels: TG ≥ 1.4mmo/L group (n = 143) and TG < 1.4mmol/L group (n = 148). There were 34 (11.7%) patients died and 33 (11.3%) patients transferred to hemodialysis during the follow-up, Moreover, a level of TG ≥ 1.4mmol/L at the initiation of CAPD was associated with a significantly increased probability of technical failure (hazard ratio, HR, 1.30; 95% confidence interval, 95% CI, 1.09 to 2.19, $P = .043$) and overall mortality (HR, 2.33; 95% CI, 1.16–4.72, $P = .018$).

Serum TG levels measured at the initiation of PD therapy is an independent predictor of early-onset peritonitis and prognosis of CAPD patients.

Abbreviations: ACEI/ARB = angiotensin-converting enzyme inhibitors/angiotensin receptor blocker, AUROC = area under receiver operation curve, BMI = body mass index, CAPD = continuous ambulatory peritoneal dialysis, CCB = calcium channel blockers, CKD = chronic kidney disease, ESRD = end-stage renal disease, GFR = glomerular filtration rate, HDL-C = high density lipoprotein cholesterol, HR = hazard ration, LDL-C = low density lipoprotein cholesterol, MAP = mean arterial pressure, RRF = residual renal function, TG = triglyceride.

Keywords: early-onset peritonitis, early-onset peritonitis, prognosis, triglyceride

1. Introduction

Over the past decades, continuous ambulatory peritoneal dialysis has become a relatively convenient, cheap, and well-established renal replacement therapy method for end-stage renal disease (ESRD) patients, especially those residing in the rural areas.[1,2] Despite great improvements in dialysis systems, antibiotic treatment, and well-developed training protocols for PD patients and staff, peritonitis is still a major cause of catheter loss and technique failure, resulting in patient demoralization and a higher risk of mortality.[3–6] Thus, identifying patients who are at high risk of PD peritonitis may result in a lower rate of switching to hemodialysis therapy and avoidance of prolonged hospitalization and escalating health costs.[7,8]

Hypertriglyceridemia is one of the most common types of dyslipidemia in patients with chronic kidney disease and has been demonstrated to be a predictor of cardiovascular disease mortality in different populations.[9,10] A previous observational study in 1,053 incident PD patients demonstrated that lipid-modifying medication may be associated with improved clinical outcomes.[11] However, the role of elevated triglyceride levels on prognosis in patients with ESRD is limited. Moreover, to the best of our knowledge, no study has investigated the association between serum triglyceride levels and the risk of peritonitis for incident CAPD patients. In the current study, we aimed to explore the hypothesis that TG levels at the beginning of PD therapy may be an independent predictor of early peritonitis and prognosis in CAPD patients.

2. Methods

2.1. Participants

From August 1, 2013 to November 31, 2017, a total of 349 consecutive patients were recruited from a single PD center of

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SW and HT contributed equally to this article.

The authors declare that there are no conflicts of interest.

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Wuhan No. 1 Hospital. All adult patients with end-stage renal disease who initiated continuous ambulatory PD at our hospital and underwent PD for least for 3 months, excluded those who had been treated with hemodialysis or who received kidney transplantation, were included in this study. After exclusion of those who did not have serum triglyceride measurements during the first 6 months of PD initiation, we further excluded those who attended other PD centers during the follow-up. Finally, 291 patients were enrolled in this study (Fig. 1). The most common primary renal disease was chronic glomerulonephritis (41.6%), followed by hypertension (18.2%), diabetic nephropathy (16.5%), and reflux nephropathy (6.5%).

This retrospective study was performed in accordance with the Declaration of Helsinki and was approved by the ethics committee of Wuhan No. 1 Hospital. Due to the nonintrusive nature of this study, the requirement for written consent was waived.

2.2. Data collection

All data were obtained from the electronic medical records of dialysis facilities. Both demographic and clinical data, including demographic data (age, sex, body mass index, primary cause of ESRD, medication use and comorbid diseases), were collected at the initiation of CAPD. Laboratory measurements, including hemoglobin, serum lipid profiles, albumin, ferritin, C-reactive protein, alkaline phosphatase, serum calcium and serum phosphorus levels, were collected during the first 1 to 3 months after the initiation of CAPD and were collected quarterly to biannually throughout the entire study period. The dialysis dose, estimated by weekly total and peritoneal Kt/V_{urea} using the urea kinetic model, and residual renal function (RRF), calculated from mean values of creatinine clearance and urea clearance and adjusted for body surface area, were measured at least biannually.

Medication use was recorded according to prescriptions and adherence of the patients. All the patients were asked to return to our PD center at least quarterly for the assessment of general conditions and concomitant medications.

Peritonitis was diagnosed when at least two of the following conditions were present within 6 months after PD therapy: clinical symptoms, effluent cell count greater than 100 cells/μL, and a positive effluent culture. Early-onset peritonitis was defined as peritonitis occurring within 6 months after the initiation of PD therapy. Moreover, peritonitis was treated using the recommended standard antibiotic protocols. Appropriate antibiotic therapy was continued for 14 to 21 days depending on the organism.

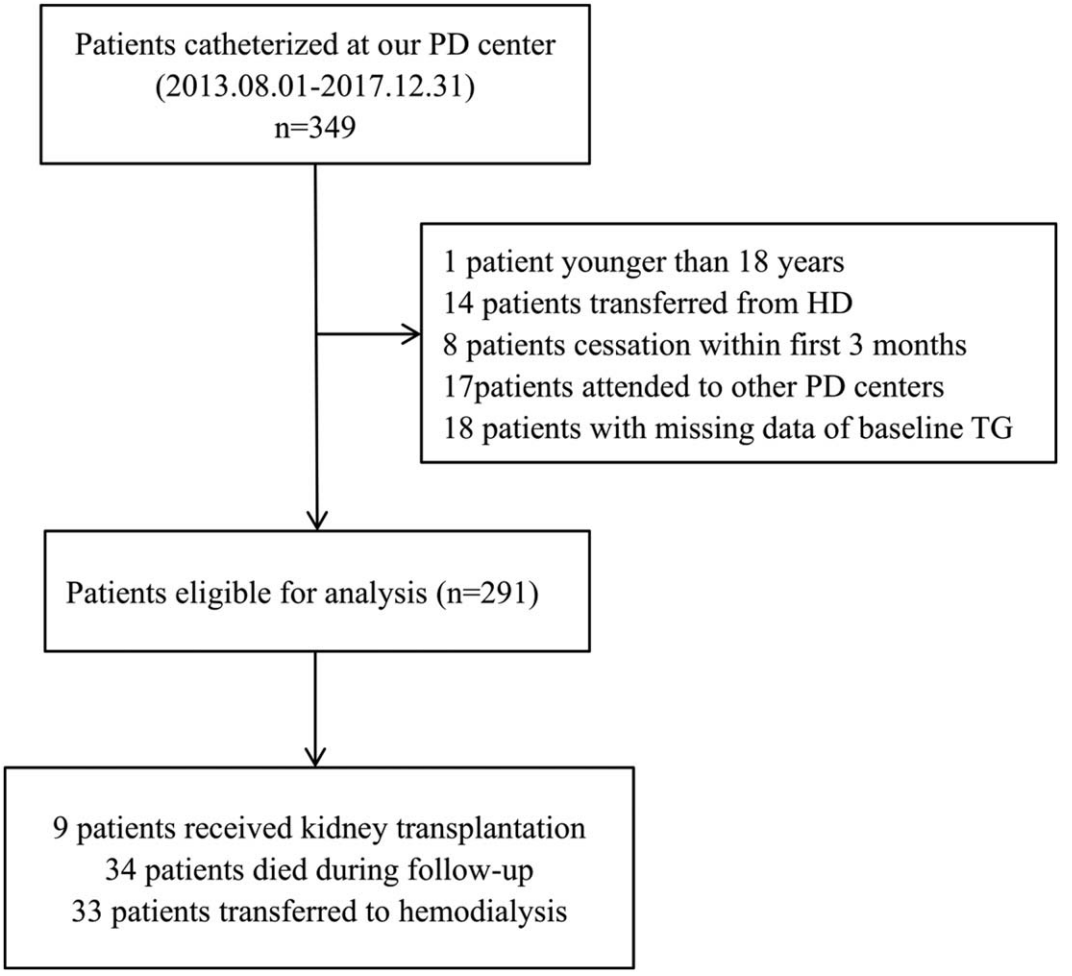


Figure 1. Study flow, including patient enrollment and outcomes.

The primary outcome was the development of early peritonitis, while the secondary primary endpoints were all-cause mortality and technical failure. Patients were censored at the time of kidney transplantation, when they were switched to hemodialysis or lost to follow-up or at the end of the study period (July 31, 2018). Moreover, when analyzing technique failure, switching to hemodialysis and drop-out due to death were regarded as final events; functional dialysis at the end of the study, kidney transplantation and loss to follow-up resulted in censored data.

2.3. Laboratory measurements

All of the patients were asked to have fasting blood drawn and the blood samples were measured in the center laboratory of our hospital. Evaluation of triglycerides was performed by determination of total values (glycerol-3-phosphate oxidase-phenol + aminophenazone high performance method) using a BM/Hitachi 717/911 analyzer. And the reference range for serum triglyceride in our laboratory is 0.53 to 2.06 mmol/L.

2.4. Statistical analyses

R software (3.6.0.0) was used for all analyses. Descriptive analysis results are reported as the mean ± SD or as medians (interquartile ranges) for continuous variables and as proportions for categorical variables. Participants were divided into two groups based on the median of baseline serum triglyceride levels: <1.4mmol/L and ≥ 1.4mmol/L. We used two-sample *t* tests or the Mann–Whitney U test to compare continuous variables across groups and Pearson chi-squared test (χ^2) to compare categorical variables across groups. We used the Lasso logistic regression model to identify independent risk factors for PD peritonitis. Compared with the traditional stepwise logistic regression analysis, Lasso logistic regression can reduce the estimation variance while providing an interpretable final model, which may more accurate than stepwise selection.[12] We estimated the association between baseline serum triglyceride levels categorized into median and all-cause mortality or technical failure using Cox proportional hazard regression models with three incremental levels of adjustment: Model 1: demographic and clinical characteristics of age, sex, major comorbid conditions (diabetes, hypertension, cardiovascular disease) and medication use (angiotensin-converting enzyme inhibitors/ angiotensin receptor blocker (ACEI/ARB), β-blockers, calcium channel blockers (CCB) and Statin/fibrate); Model 2: Model 1 plus malnutrition and inflammation indices that included body mass index (BMI), mean arterial pressure (MAP), serum total cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), albumin, ferritin, CRP levels and serum electrolyte; Model 3 (fully adjusted model): Model 2 plus residual GFR and dialysis dose (total and peritoneal Kt/V_{urea}). Cumulative survival curves as a function of time were generated by Kaplan-Meier analysis and were compared by log-rank tests. To measure the sensitivity and specificity of serum triglyceride at different cutoff values, we generated a conventional ROC curve. We also calculated the area under receiver operation curve (AUROC) to ascertain the quality of TG as a predictor of outcomes. In these analyses, TG was modeled both as a categorical variable and as a continuous variable. All statistical tests were two-tailed, and $P<.05$ was considered statistically significant.

3. Results

3.1. Study participants

In total, 291 incident PD patients were enrolled in this study, and the baseline demographic and clinical characteristics of the cohort are shown in Table 1, categorized according to serum

Table 1

Baseline characteristics of the CAPD patients.

Characteristics	TG≧1.4mmol/L (n=143)	TG<1.4mmol/L (n=148)	P
Age, year	54.5±13.9	51.5±14.3	.078
Gender, male, n (%)	85 (59.4)	66 (44.6)	.054
BMI, kg/m^2	22.8±4.2	22.2±3.6	.182
Primary cause of ESRD			.326
Glomerulonephritis, n (%)	58 (40.[illegible])	63 (42.6)	
Diabetic nephropathy, n (%)	25 (17.5)	23 (15.5)	
Hypertension, n (%)	28 (19.6)	25 (16.9)	
Reflux nephropathy, n (%)	10 (7.0)	9 (6.1)	
Other or unknown, n (%)	22 (15.4)	28 (18.9)	
Comorbidities			
Coronary artery disease, n (%)	57 (39.9)	59 (39.9)	.634
Hypertension, n (%)	117 (81.8)	122 (82.4)	.164
Diabetes, n (%)	29 (20.3)	40 (27.0)	.094
Treatments			
Statin/fibrate, n (%)	17 (11.9)	19 (12.8)	.762
ACEI/ARB, n (%)	47 (32.9)	42 (28.4)	.660
β-blockers, n (%)	73 (51.0)	77 (52.0)	.442
CCB, n (%)	105 (70.9)	111 (77.6)	.194
Diuretic, n (%)	38 (25.7)	31 (21.7)	.425
SBP, mmHg	141.5±14.4	144.9±16.1	.064
DBP, mmHg	82.9±10.7	84.0±11.2	.390
MAP, mmHg	102.4±10.2	104.3±11.1	.140
Dialysis dose			
Weekly total Ccr	71.8±11.7	72.8±15.5	.349
Weekly kidney Ccr	36.7±10.7	34.6±10.5	.609
Weekly total K_t/V_{urea}	2.5±0.6	2.1±0.6	.354
Weekly peritoneal K_t/V_{urea}	0.6±0.1	0.7±0.2	.855
Residual GFR (ml/min/1.73m^2)	5.3±1.0	4.8±0.9	.386
PET at baseline	0.7±0.1	0.7±0.2	.542
Laboratory variables			
Leukocyte, $\times 10^9$/L	5.9±1.8	6.7±2.2	<.001
Erythrocyte, $\times 10^{12}$/L	3.3±0.6	3.5±0.7	.012
Hemoglobin, g/L	95.5±16.8	101.4±19.3	.006
Serum albumin, g/L	34.7±5.3	36.9±5.2	<.001
Total cholesterol, mmol/L	5.2±1.8	4.3±1.1	<.001
Serum triglyceride, mmol/L	2.4±0.6	1.0±0.3	<.001
HDL-C, mmol/L	1.1±0.2	1.2±0.3	.390
LDL-C, mmol/L	3.5±0.6	2.6±0.5	.023
Calcium, mmol/L	2.2±0.3	3.4±0.5	.363
Phosphorus, mmol/L	1.5±0.4	1.5±0.5	.706
Potassium, mmol/L	4.1±0.7	4.1±0.6	.856
Sodium, mmol/L	151.1±29.1	142.3±23.1	.335
Alkaline phosphatase, U/L	97.3±17.1	79.0±10.1	.150
Blood urea nitrogen, mmol/L	17.7±6.7	20.7±12.0	.397
Serum creatinine, umol/L	759.9±212.6	718.0±215.7	.137
Uric acid, umol/L	408.8±99.0	422.3±85.9	.214
Outcomes			
Early peritonitis	28 (18.9)	10 (7.0)	.015
Overall mortality	23 (16.1)	11 (7.4)	.022
Technical failure	47 (31.8)	20 (14.0)	.018

ACEI/ARB = angiotensin-converting enzyme inhibitors/ angiotensin receptor blocker, BMI = body mass index, CCB = calcium channel blockers, CRP = c-reactive protein, DBP = diastolic blood pressure, GFR = glomerular filtration rate, HDL-C = high-density lipoprotein cholesterol, iPTH = intact parathyroid hormone, LDL-C = low-density lipoprotein cholesterol, MAP = mean arterial pressure, PET = peritoneal equilibration test, SBP = systolic blood pressure.

triglyceride levels. The mean age was 53.0 ± 14.1 years. The mean and median baseline values for observed serum triglyceride levels were 1.7 and 1.4 mmol/L, respectively. Patients with elevated serum triglycerides had higher levels of total cholesterol and LDL-C, whereas they had lower hemoglobin levels and serum albumin concentrations. However, there was no difference in demographic data, comorbidities or drug usage between the two groups. Nevertheless, the achieved dialysis adequacy and residual glomerular filtration rate (GFR) at baseline were not different according to triglyceride categorization.

3.2. TG as a predictor of the primary end point

In this study, PD peritonitis occurred in 38 patients within six months after the initiation of CAPD. In addition, 21 peritonitis episodes involved gram-positive organisms, 12 episodes involved gram-negative organisms, 3 episodes were culture-negative, and 2 episodes involved fungal organisms. A much higher incidence of early-onset PD peritonitis was found in the TG ≥ 1.4 mmol/L group than in the TG < 1.4 mmol/L group (18.9% vs 7.0%). The independent risk factors for PD peritonitis were determined by Lasso logistic regression analysis. Each colored line represents a variable in the model. Among the 37 variables, TGs and age (coefficients = 0.139 and 0.011, respectively, Fig. 2) had nonzero coefficients and were selected in the final model for PD peritonitis. Moreover, a higher TG value was associated with early PD peritonitis (OR = 2.10; 95% CI, 1.01 to 4.35; $P = .048$) in the fully adjusted model when the TG level was analyzed as a categorical variable. For predicting PD peritonitis, the AUC of TGs for the initiation of CAPD was 0.728 (Fig. 3A). A cutoff of 1.6 mmol/L yielded good specificity (73.5%) and sensitivity (58.6%).

3.3. The association between serum TG and secondary outcomes

After a median follow-up of 24.4 months, 34 all-cause deaths occurred, 33 patients switched to hemodialysis therapy for any reason, and 9 patients underwent kidney transplantation. Among the 34 patients who died, the causes of death were as follows: 15 cardiovascular disease, 5 peritonitis and 14 unknown causes or other causes. The reasons for switching to hemodialysis included 18 cases of peritonitis, 5 cases of inadequate dialysis, 4 cases of mechanical malfunction and 6 cases for other reasons.

Moreover, a level of TG ≥ 1.4 mmol/L at the initiation of CAPD was associated with a significantly increased probability of technical failure (HR, 1.30; 95% CI, 1.09 to 2.19, P = 0.043) and overall mortality (HR, 2.33; 95% CI, 1.16–4.72, $P = .018$) (Table 2 and Fig. 4). In the prespecified subgroup analysis, patients with TG ≥ 1.4 mmol/L had a higher risk of overall mortality and technical failure than patients with TG < 1.4 mmol/L in some subgroups. Moreover, the AUC of TG for technical

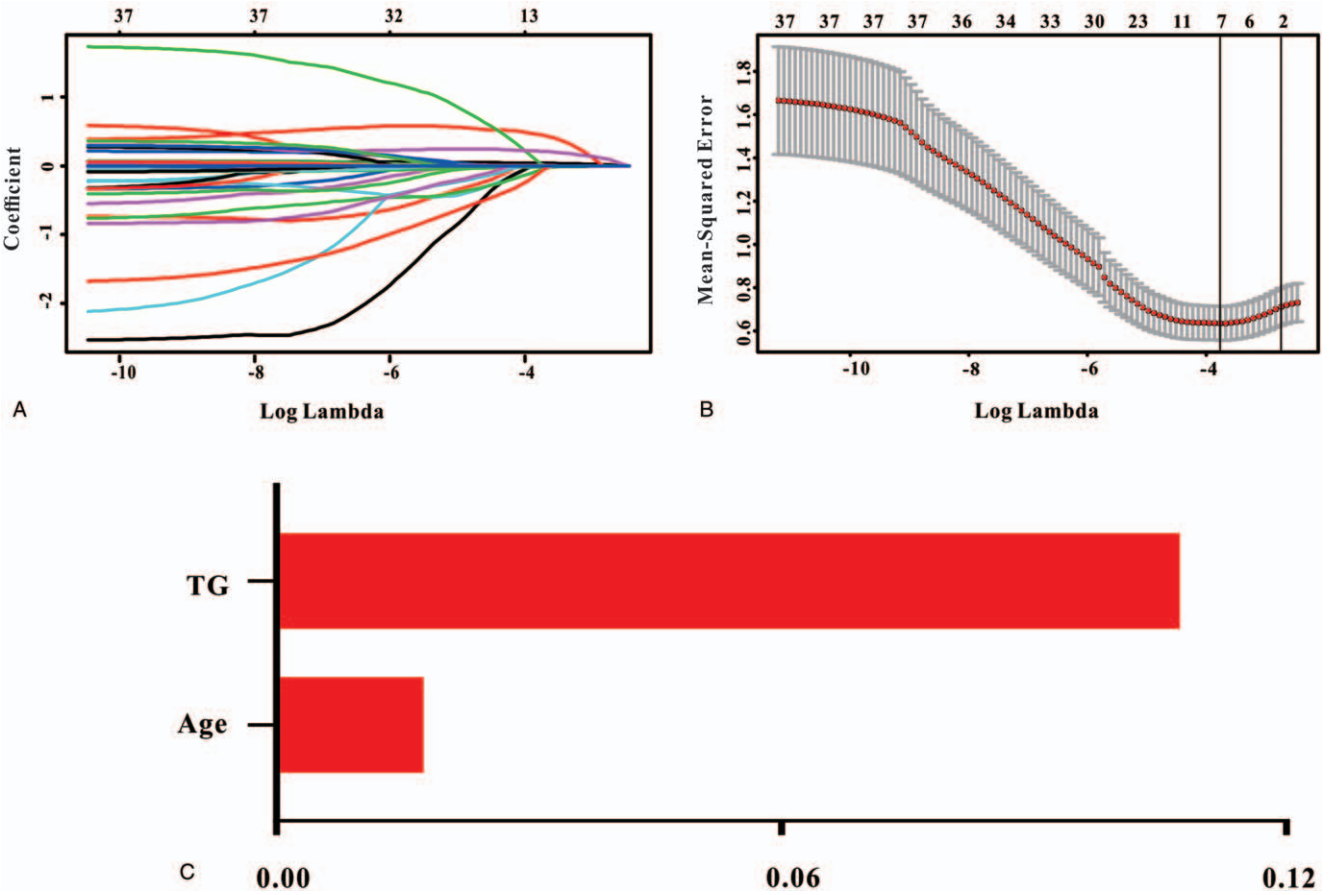
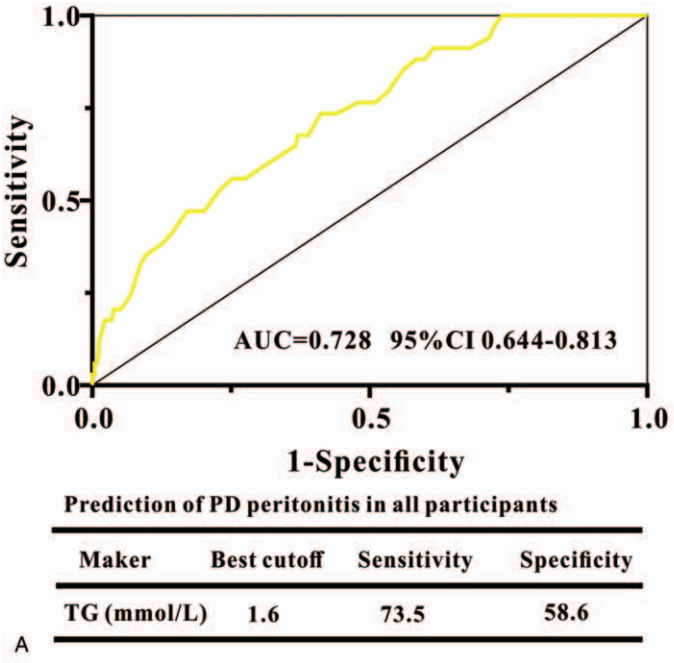


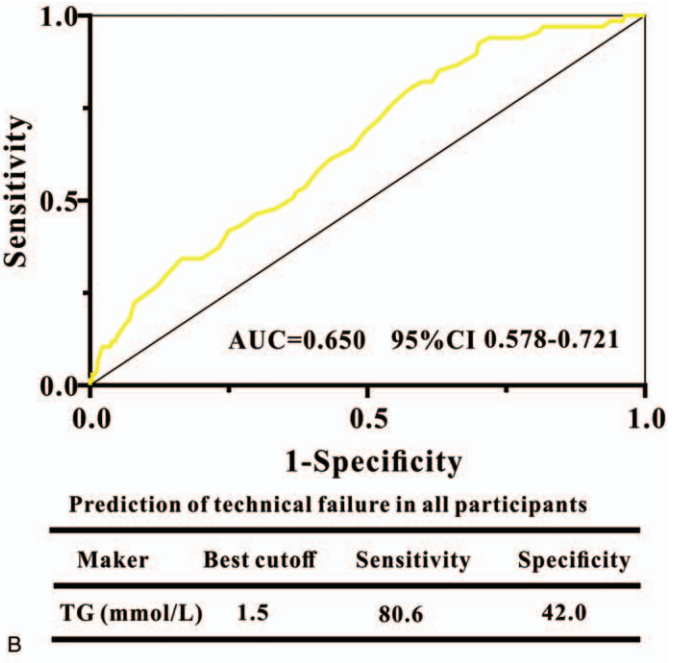
Figure 2. Selection of informative factors associated with early-onset peritonitis using the LASSO logistic regression model. (A) LASSO coefficient profiles of the 37 clinical features. (B) Selection of the tuning parameter (λ). (C) Histogram shows the coefficients of individual features that contribute to the final logistic model.



Prediction of PD peritonitis in all participants

Maker	Best cutoff	Sensitivity	Specificity
TG (mmol/L)	1.6	73.5	58.6

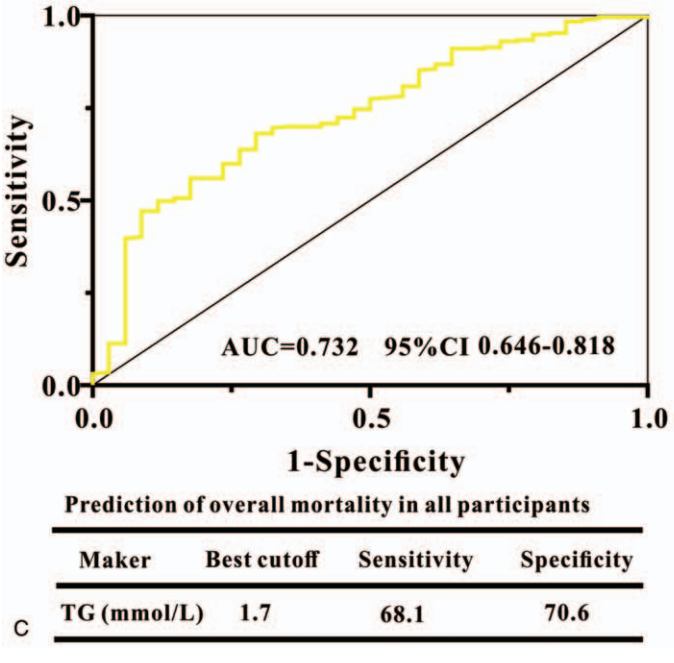
A



Prediction of technical failure in all participants

Maker	Best cutoff	Sensitivity	Specificity
TG (mmol/L)	1.5	80.6	42.0

B



Prediction of overall mortality in all participants

Maker	Best cutoff	Sensitivity	Specificity
TG (mmol/L)	1.7	68.1	70.6

C

Figure 3. ROC analyses for predicting early-onset peritonitis (A), technical failure (B) and overall mortality (C).

failure and overall mortality were 0.650 and 0.732, respectively (Fig. 3A-B).

4. Discussion

In the current study, the results revealed an association between serum TG levels and the incidence of PD peritonitis and mortality in CAPD patients. When the patients were divided into two groups based on TG levels, the incidence of PD peritonitis, the rate of technical failure and mortality were much higher in the elevated TGs group than in the lower TGs group after adjustment for a variety of other clinical and laboratory variables. In summary, our data suggest that serum TGs measured at the initiation of CAPD may be a good predictor for identifying patients at high risk of PD peritonitis and mortality.

PD-related peritonitis, as one of the most common complications during PD therapy, has been reported to contribute directly or indirectly to ~ 20% of PD technique failures and 2% to 6% of deaths.[13,14] Several risk factors have been identified in previous studies, including BMI, albumin, obesity, educational status, smoking and so on.[15–17] However, to the best of our knowledge, no study has investigated the relationship between lipids and peritonitis. In the current study, since Lasso logistic regression can reduce the estimation variance while providing a more

Table 2

Cox proportional hazards analysis for overall mortality and technical failure.

	Unadjusted		Model 1		Model 2		Model 3	
TG levels	**HR (95% CI)**	***P***	**HR (95% CI)**	***P***	**HR (95% CI)**	***P***	**HR (95% CI)**	***P***
For overall mortality								
Continuous	1.54 (1.19–1.99)	<.001	1.35 (1.17–1.56)	<.001	1.26 (1.13–1.40)	<.001	1.22 (1.10–1.35)	<.001
hypertriglyceridemia	2.22 (1.10–4.50)	.027	1.81 (0.85–3.82)	.123	1.38 (0.26–5.22)	.633	1.26 (0.32–4.89)	.739
TG ≥ 1.4mmol/L	2.16 (1.05–4.43)	.001	1.82 (1.61–2.07)	.029	1.48 (1.20–1.84)	.035	1.30 (1.09–2.19)	.043
For technical failure								
Continuous	1.28 (1.08–1.51)	.004	1.18 (1.04–1.33)	.012	1.13 (1.02–1.25)	.022	1.04 (1.01–1.06)	.031
hypertriglyceridemia	1.44 (0.84–2.47)	.189	1.38 (0.79–2. 42)	.263	1.26 (0.45–3.52)	.670	1.11 (0.37–3.32)	.846
TG ≥ 1.4mmol/L	2.37 (1.40–4.02)	<.001	2.49 (1.46–4.24)	.001	2.30 (1.21–4.39)	.012	2.33 (1.16–4.72)	.018

95%CI = 95% confidence index, HR = hazard ratio, TG = triglyceride.

Adjustment in Model 1: age, sex, major comorbid conditions (diabetes, hypertension, cardiovascular disease) and medication use (ACEI/ARB, β-blockers, CCB and Statin/fibrate); Model 2: Model 1 plus malnutrition and inflammation indices that included BMI, MAP, serum total cholesterol, HDL-C, LDL-C, serum albumin, ferritin, CRP levels and serum electrolyte; Model 3 (fully adjusted model): Model 2 plus residual GFR and dialysis dose (total and peritoneal Kt/V_{urea}).

accurate and interpretable final model than the traditional stepwise logistic regression analysis,[12] we used the Lasso logistic regression model to identify independent risk factors for early-onset peritonitis, and TGs were finally selected. Although the precise mechanisms responsible for this significant association have not been well established, it could be proposed that the presence of inflammation and endothelial dysfunction might underlie this relationship.[18] In addition, a recent study conducted with an untreated hyperlipidemic rat model found a positive correlation between TG levels and serum adenosine deaminase activity, which in fact is described as an unspecific marker of cell-mediated immunity, immune cell activation and inflammation.[19,20] Moreover, this correlation between TG levels and adenosine deaminase activity has also been corroborated in humans.[21]

A bidirectional relationship between serum TG levels and CKD has been suggested by recent studies. Dyslipidemia is common in patients with CKD, especially in patients with ESRD, and is

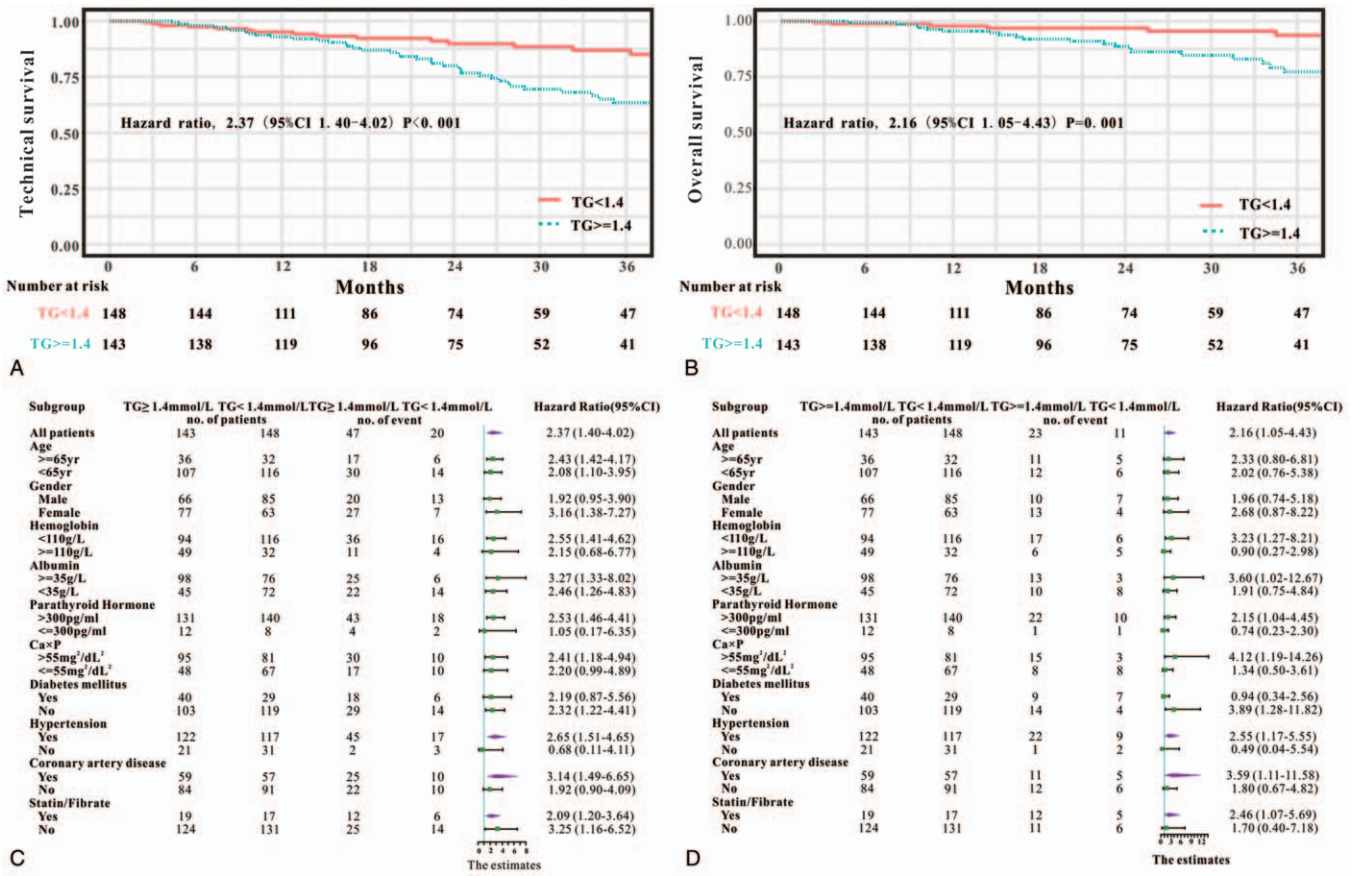


Figure 4. Kaplan-Meier estimates of technical survival, overall survival, and subgroup analyses at the second interim analysis in all participants in this study. Shown are hazard ratios and number of event among TG ≥ 1.4mmol/L group and TG < 1.4mmol/L group.

characterized by high TG and reduced HDL-C levels.[10] On the other hand, there was a significant trend towards deteriorating renal function with increases in triglyceride level categories in a retrospective study of 3748 hospital-based type 2 diabetes mellitus patients, and the researchers further concluded that plasma TGs were an independent risk factor for CKD according to multiple logistic regression after adjustment for other confounders.[22] A similar conclusion has also been drawn in another study. Kazuhiko et al conducted a prospective longitudinal cohort study of 117,279 Japanese people and found a robust and consistent association between serum TG levels and the reduction in eGFR and the incidence and progression of CKD.[23] In contrast, a recent study demonstrated that lipid-lowering therapy prior to hospital admission may improve clinical outcomes and be associated with shorter hospital stays, lower renal replacement therapy requirements and mortality in critically ill patients.[24] However, the role of TGs in the pathogenesis of cardiovascular disease mortality and the role of lipid-modifying medications in dialysis patients remain uncertain. In patients with hemodialysis, there have been limited studies investigating the association between serum TG levels and all-cause and cardiovascular mortality. In the first study on maintenance hemodialysis, serum TGs exhibited a U-shaped relationship,[25] whereas in two later studies, the opposite conclusion was reached.[26,27] Moreover, Arsalan et al demonstrated that serum TGs may be a direct predictor of death in a retrospective study of 1053 chronic PD patients, and they further concluded that treatment of hypertriglyceridemia may be warranted individuals with triglyceride levels >200 mg/dl.[11] A similar conclusion was also made in another study.[28] Our data added evidence that serum TG levels were a significant determinant of mortality and technical failure, which means that each 1 mmol/L increase in TG leads to a 22% increase in the risk of overall mortality and a 4% increase in the risk of technical failure. However, a recent cohort study detected serum TG levels regularly over a 10-year follow-up and demonstrated an association between lower TG levels and higher all-cause mortality in 749 incident PD patients.[29] These paradoxical conclusions may be the reason guidelines regarding the management of lipids in patients with dialysis are still obscure.

This study should be considered in the context of a few inevitable limitations. First, this was a single-center study of 291 patients, and the relatively small sample size and short duration of follow-up may weaken the conclusion. Second, we did not account for changes in TG levels during the follow-up since patients may have undergone lipid-lowering therapy. Furthermore, we did not detect other inflammatory or endothelial markers, which may be used to explore the potential mechanisms.

In summary, the present study demonstrated that serum TG levels measured at the initiation of PD therapy may be an independent predictor of early-onset PD peritonitis and mortality in CAPD patients. Further investigations are needed to identify their utility for dialysis patients and explore the potential mechanisms.

Author contributions

Conceptualization: Hongdan Tian, Yanmin Zhang.
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Validation: Sheng Wan, Yanmin Zhang.
Visualization: Hongdan Tian, Yanmin Zhang.
Writing – original draft: Sheng Wan, Hongdan Tian, Qing Luo, Yanmin Zhang.
Writing – review & editing: Sheng Wan, Li Cheng, Yanmin Zhang.

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