BMC Nephrology

RESEARCH Open Access

Association between time-varying serum lipid levels and all-cause mortality in haemodialysis and peritoneal dialysis patients

Zhenjian Xu[1,2], Yuchun Zeng[1,2], Yingyan Liang[1,2], Xuefeng Xie[1,2], Xiaomei Li[1,2], Qiongqiong Yang[1,2*] and Peifen Liang[1,2*]

Abstract

Objective Changes in lipid levels over time and the associated all-cause mortality have not yet been studied in different populations of patients undergoing dialysis. This study aimed to investigate the differences in time-varying serum lipid levels and all-cause mortality among haemodialysis (HD) and peritoneal dialysis (PD) patients over a 5-year follow-up period.

Methods This observational study included Chinese patients with end-stage renal disease (ESRD) who started HD or PD at Sun Yat-sen Memorial Hospital from January 2010 to February 2018. Changes in lipid profiles and trends of change in overall survival rates between the two groups were investigated. Risk factors for the outcome were identified, and the optimal cut-off values were determined using ROC analysis. Additionally, the relationship between the group variable and the outcome measure was assessed using linear regression with a generalized estimating equation (GEE) model.

Results A total of 141 patients (74 HD patients and 67 PD patients) were enrolled in the study. Forty-three (30.71%) patients died during the follow-up period. Compared with the HD group, the PD group had significantly greater triglyceride (TG) (Year 1 and Year 2) and low-density lipoprotein cholesterol (LDL-C) (Year 2) levels and significantly lower high-density lipoprotein cholesterol (HDL-C) (Year 1 and Year 2) levels. There was no significant difference in total cholesterol (TC) levels. The GEE results revealed similar changes in lipid levels between HD patients and PD patients over time. The Kaplan–Meier survival curve revealed that there was no significant difference in overall survival between the two groups (log-rank test, $P=0.119$). Furthermore, the multivariate Cox proportional hazard regression models demonstrated that baseline HDL-C levels (HR: 0, 95% CI: 0 to 0.11; $P=0.004$) and changes in LDL-C levels from baseline to 3 years of follow-up(difference from 0 to 3 years of follow-up) (HR: 0.21, 95% CI: 0.09 to 0.53; $P<0.001$) were associated with a greater risk of death in HD patients. An increase in LDL-C levels (difference from 0 to 3 years of follow-up) ≤ 0.24 mmol/L in HD patients and age ≥ 53 years in all patients initiating dialysis was associated with a significantly increased risk of mortality.

*Correspondence:
Qiongqiong Yang
yangqq@mail.sysu.edu.cn
Peifen Liang
lpeif@mail.sysu.edu.cn

Full list of author information is available at the end of the article

Conclusion The baseline levels of HDL-C and changes in LDL-C levels over a three-year period were significant predictors of all-cause mortality in HD patients, which differed from the lack of significant risk factors observed in the PD group.

Keywords Lipid, Haemodialysis, Peritoneal dialysis, Mortality, Time-varying

Introduction

Chronic kidney disease (CKD) has emerged as a significant public health issue worldwide, affecting over 850 million people globally [1]. In China, it is estimated that 8.2% of the adult population, approximately 82 million individuals, is affected by CKD [2]. For patients with ESRD, renal replacement therapy through peritoneal dialysis (PD) or haemodialysis (HD) remains the primary treatment option. Dyslipidaemia, characterized by elevated triglycerides (TG), low high-density lipoprotein cholesterol (HDL-C), and atherogenic lipoprotein profiles, is a pervasive metabolic disorder in dialysis patients [3]. Cardiovascular disease (CVD) remains a leading cause of both morbidity and mortality among patients undergoing dialysis [4]. Cross-sectional data reveal that 60% of ESRD patients exhibit lipid abnormalities, with distinct patterns between HD and PD modalities [4]. The impact of dyslipidaemia in dialysis patients may extend beyond CVD, influencing dialysis-related outcomes such as residual kidney function, PD and HD technique survival, and overall mortality [5]. Paradoxically, observational studies report a "reverse epidemiology" phenomenon where lower LDL-C levels predict increased mortality in dialysis patients—a finding that challenges conventional lipid management paradigms [6–7]. Despite these insights, critical knowledge gaps persist. Most current studies rely on single-timepoint lipid measurements and modality-specific analyses are scarce, the relationship between dyslipidaemia and mortality in dialysis patients requires further investigation.

Against this backdrop, we analyzed 5-year longitudinal lipid profiles in 141 dialysis patients (HD = 74, PD = 67) from a tertiary care center. Our objectives were threefold: (1) compare lipid trajectory patterns between HD and PD subgroups, (2) identify mortality-associated lipid dynamics, and (3) evaluate the incremental prognostic value of time-varying lipid analysis over traditional risk models.

Methods

Patients

This retrospective, observational cohort study included 141 ESRD patients who started dialysis (74 HD patients and 67 PD patients) from January 2010 to February 2018 at Sun Yat-sen Memorial Hospital. Eligible patients were aged ≥ 18 years. The primary outcome was all-cause mortality.

The study followed the tenets of the Declaration of Helsinki, and informed consent was obtained from all the participants. This study was approved by the institutional review board of Sun Yat-sen Memorial Hospital of Sun Yat-sen University, Guangzhou, China.

Data collection

The clinical characteristics included age, sex, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), smoking history, primary cause of CKD, hypertension history, CVD history, dialysis vintage, Kt/V, and peritonitis history (for PD) and cause of death.

Laboratory parameters of PD patients and HD patients were measured at the initiation of dialysis (baseline: Year 0), including haemoglobin (HB), serum calcium (Ca), serum phosphorus (P), prealbumin (PALB), serum albumin (ALB), serum uric acid (UA), serum creatinine, free fatty acid (FFA), superoxide dismutase (SOD), serum ferritin, total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), high-sensitivity C-reactive protein (hs-CRP), and intact parathyroid hormone (iPTH).Data were collected at baseline and annually thereafter (Year 1–5). All patients were monitored until death, or the end of 5-year follow-up.

Statistical analysis

Data are expressed as the means ± standard deviations (SDs) or median and interquartile range (IQR) for continuous variables, and as numbers and percentages (%) for categorical variables. Non-normally distributed variables were analyzed using non-parametric tests (Mann-Whitney U for comparisons, Friedman test for trends). Missing data (< 5% for most variables) were handled via multiple imputation. Student's independent t test was used to compare the mean differences between the HD and PD groups. For categorical data, comparisons were made using either the chi-square test or Fisher's exact test when the expected frequency was ≤ 5. We utilized repeated-measures ANOVA to evaluate the temporal changes in variables measured repeatedly. The Kaplan-Meier method, coupled with the log-rank test, was applied to assess differences in survival patterns between the groups. Cox proportional hazards models, both univariate and multivariate, were engaged to identify predictors of survival status. The estimated hazard ratio and its 95% confidence interval were reported as both statistics and forest plots. The continuous associated factors were used in the ROC analysis, and the AUC, sensitivity, specificity, Youden index, and suggested cut-offs

were reported. A generalized estimating equation (GEE) linear regression model was implemented to explore the relationship between group characteristics and outcome measures across six time points: initial assessment and at one, two, three, four, and five-year follow-ups. An AR(1) correlation matrix was adopted for the repeated measures data. $P<0.05$ was considered to indicate that a difference was statistically significant. The statistical software R(version 4.4.0) was utilized for all these analyses.

Results

Characteristics of the enrolled population

A total of 141 patients were included in this study: 74 in the haemodialysis (HD) group and 67 in peritoneal dialysis (PD) group. The average age of the patients was 54.71 ± 13.99 years, and the sex ratio was 1:0.83 (male: female = 77:64). Patients were followed up for a median time of 52 months (3 ~ 60 months), and 43 (30.71%) patients died during the study period. 8.2 deaths per 100 patient-years (43 deaths/525 total patient-years). As indicated in Table 1, significant differences were found in baseline age, DBP, smoking rate, primary cause of CKD, hypertension, CVD, death rate, cause of death, and UA and SOD levels (all $P<0.05$). Patients in the HD group were significantly older and had higher rates of smoking hypertension, CVD, and mortality. They also had significantly lower DBP, UA and SOD levels.

Linear trends and paired comparisons among time points

The results (P values) of the linear trend analysis for each group, as indicated in Table 2, revealed that in both the HD and PD groups, there was a linear trend in the levels of Ca, ALB, UA, ferritin, SOD and iPTH from year 0 to year 5.

However, a linear trend in HB was observed only in the HD group, not in the PD group. Figure 1 shows that ferritin, SOD and iPTH exhibited particularly linear increases, UA showed a particularly linear decrease, whereas ALB and HB remained relatively stable throughout the observation period. The lipid profile, whereas UA showed a particularly linear decrease. The lipid profile, including TG, TC, HDL-C and LDL-C, did not exhibit a linear trend. The GEE results presented in Table 3 indicate significant differences in various laboratory parameters between the HD and PD groups over the entire period. As indicated, the PD group presented significantly lower levels of HB, P, ALB, SOD, and ferritin than did the HD group over time (all $P<0.05$). The GEE results also revealed similar changes in lipid levels between HD patients and PD patients over time.

Survival analysis and factors associated with overall survival

The overall survival time was shown in Table 1. Figure 2 presents the results of the Kaplan–Meier survival analysis comparing the HD and PD groups. There was no significant difference in overall survival between the two groups (log-rank test, $P=0.119$).

Furthermore, univariate and multivariate Cox regression analyses, which investigated the associations between independent variables and overall survival, are shown in Table 4. As indicated, HD and PD patients had different risks, with age and HDL-C levels being significant according to the univariate analysis results. In the multivariate Cox regression analysis, no significantly associated factors were found in the PD group, whereas HDL-C levels (HR: 0.00, 95% CI: 0.00 to 0.11; $P=0.004$) and LDL-C levels (difference from 0 to 3 years) (HR: 0.21, 95% CI: 0.09 to 0.53; $P<0.001$) were associated with a greater risk of death in HD patients. For all patients, age (HR: 1.04, 95% CI: 1.00 to 1.07; $P=0.032$), CVD (HR: 2.78, 95% CI: 1.15 to 6.72; $P=0.023$), Ca (difference from 0 to 3 years) (HR: 6.09, 95% CI: 1.34 to 27.64; $P=0.019$), and PALB (difference from 0 to 3 years) (HR: 1.07, 95% CI: 1.03 to 1.11; $P<0.001$) were significantly associated with the multivariate results. Patients with older age, CVD, and high levels of Ca (difference from 0 to 3 years) or PALB (difference from 0 to 3 years) had a greater risk of death. These results are also shown in Fig. 3 in a forest plot.

ROC analysis

Table 5; Fig. 4 show the results of the ROC analysis of continuous variables that were found to be associated with death via multivariate Cox regression models. As indicated, a medium AUC was found among the results, and significant differences were found in age (all patients) and LDL-C levels (difference from 0 to 3 years; HD group). An increase in LDL-C levels (difference from 0 to 3 years) ≤ 0.24 mmol/L in HD patients and age ≥ 53 years in all patients who starting dialysis was associated with significantly increased mortality.

Discussion

In this study, we investigated the associations of time-varying serum lipid levels with all-cause mortality in haemodialysis and peritoneal dialysis patients at our centre. Compared with the HD group, the PD group presented significantly greater triglyceride (TG) (Year 1 and Year 2) and low-density lipoprotein cholesterol (LDL-C) (Year 2) levels and significantly lower high-density lipoprotein cholesterol (HDL-C) (Year 1 and Year 2) levels. There was no significant difference in total cholesterol (TC). This pattern aligns with the longitudinal lipid profile trends observed in incident PD patients by Yeoungjee et al. [8],

Table 1 Patient's clinical characteristics and baseline labolatraly data[a]

Parameters	HD (*n* = 74)	PD (*n* = 67)	All (*n* = 141)	*P*
Age, year	59.26 ± 14.34	49.69 ± 11.80	54.71 ± 13.99	< 0.001
Gender, male/female	45/29	32/35	77/64	0.120
BMI, kg/m^2	22.75 ± 3.05	22.68 ± 3.86	22.71 ± 3.45	0.908
SBP, mmHg	148.96 ± 17.08	147.54 ± 26.54	148.28 ± 22.02	0.703
DBP, mmHg	81.70 ± 12.16	90.04 ± 16.41	85.67 ± 14.88	< 0.001
Smoking, *n* (%)	21 (28.38%)	6 (8.96%)	27 (19.15%)	0.003
lipid-lowering therapy, *n* (%)	27(36.49%)	25 (37.31%)	52 (36.88%)	0.919
Etiology of ESRD, *n* (%)				0.009
Chronic glomerulonephritis	24 (32.88%)	27 (40.91%)	51 (36.69%)	
Hypertensive nephrosclerosis	12 (16.44%)	20 (30.30%)	32 (23.02%)	
Polycystic kidney disease	1 (1.37%)	3 (4.55%)	4 (2.88%)	
Obstructive nephropathy	2 (2.74%)	4 (6.06%)	6 (4.32%)	
Lupus nephritis	4 (5.48%)	5 (7.58%)	9 (6.47%)	
Drug-induced injury	1 (1.37%)	0 (0.00%)	1 (0.72%)	
Vasculitis nephritis	1 (1.37%)	0 (0.00%)	1 (0.72%)	
Diabetic nephropathy	27 (36.99%)	7 (10.61%)	34 (24.46%)	
Unknown	1 (1.37%)	0 (0.00%)	1 (0.72%)	
OS, month	51.25 ± 13.67	53.90 ± 14.20	52.39 ± 13.90	0.300
Hypertension, *n*(%)	53 (71.62%)	34 (50.75%)	87 (61.70%)	0.011
Death, *n*(%)	29 (39.73%)	14 (20.90%)	43 (30.71%)	0.016
Cause of death, *n*(%)				0.001
CVD	15 (51.72%)	4 (28.57%)	19 (44.19%)	
Aortic dissection	1 (3.45%)	0 (0.00%)	1 (2.33%)	
Cerebral hemorrhage	5 (17.24%)	1 (7.14%)	6 (13.95%)	
Infection	2 (6.90%)	3 (21.43%)	5 (11.63%)	
Tumor	5 (17.24%)	0 (0.00%)	5 (11.63%)	
Gastrointestinal bleeding	1 (3.45%)	0 (0.00%)	1 (2.33%)	
Unknown	0 (0.00%)	6 (42.86%)	6 (13.95%)	
HB, g/L	74.42 ± 19.46	78.73 ± 22.42	76.47 ± 20.95	0.224
Ca, mmol/L	1.93 ± 0.25	1.92 ± 0.28	1.92 ± 0.26	0.804
P, mmol/L	2.08 ± 0.59	2.05 ± 0.70	2.07 ± 0.64	0.719
PALB, g/L	0.30 ± 0.08	0.31 ± 0.10	0.30 ± 0.09	0.372
TG, mmol/L	1.52 ± 0.75	1.71 ± 0.95	1.61 ± 0.86	0.208
TC, mmol/L	4.64 ± 1.41	4.60 ± 1.78	4.62 ± 1.59	0.887
HDL-C, mmol/L	1.12 ± 0.39	1.11 ± 0.46	1.11 ± 0.42	0.956
LDL-C, mmol/L	2.74 ± 1.07	2.91 ± 1.21	2.82 ± 1.14	0.389
ALB, g/L	32.59 ± 5.38	32.59 ± 5.25	32.59 ± 5.30	0.999
UA, μmol/L	473.0(195.50)	572.0(189.50)	528.0(218.0)	0.024
hsCRP, mg/L	7.26(35.93)	4.92(18.48)	6.55(23.21)	0.300
SOD, U/ml	85.41 ± 19.37	96.84 ± 18.05	90.84 ± 19.55	< 0.001
Ferritin, μg/L	187.4(351.62)	193.0(341.70)	193.0(336.43)	0.646
iPTH, pg/ml	216.5(387.25)	247.0(370.5)	227.0(378.50)	0.681

[a] Mean ± SD, [b]median (IQR), unless noted otherwis; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; ESRD: end-stage renal disease; OS: overall survival; CVD: cardiovascular disease; HB: hemoglobin; Ca: serum calcium; P: serum phosphorus; PALB: prealbumin; TG: triglycerides; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; ALB: serum albumin: UA: serum uric acid; hs-CRP: high-sensitivity C-reactive protein; SOD: superoxide dismutase; iPTH: intact parathyroid hormone

who similarly reported sustained TG elevation despite biocompatible solution use. Notably, our findings extend prior observations by demonstrating these differences persist through longitudinal assessment.

The elevated LDL-C in PD patients contrasts with the China Dialysis Collaborative Study [9], which found no modality-specific LDL-C differences in a multicenter cohort. However, our results support the interaction effects between glucose metabolism and lipid profiles in PD patients described by Yiping et al. [10], where dyslipidemia exacerbated mortality risk in those with impaired fasting glucose. The lack of total cholesterol (TC) difference between groups parallels findings from the atherosclerosis progression study by Guiomar et al. [11],

Table 2 The results (*P*-values) of linear trend analysis among years in HD and PD group

		HB	Ca	*P*	PALB	TG	TC	HDL-C	LDL-C	ALB	UA	hsCRP	SOD	Ferritin	iPTH
Linear trend	HD	< 0.001	< 0.001	0.824	0.457	0.390	0.360	0.810	0.294	0.003	0.003	0.996	< 0.001	< 0.001	0.007
	PD	0.306	< 0.001	0.419	0.583	0.270	0.897	0.113	0.959	< 0.001	< 0.001	0.949	0.071	0.033	0.005

suggesting modality-specific effects on lipid subfractions rather than overall cholesterol homeostasis.

The association between LDL-C trajectory (ΔLDL-C ≤ 0.24 mmol/L) and mortality in HD patients warrants particular attention. This contrasts with the Chronic Renal Insufficiency Cohort (CRIC) analysis by Simon et al. [12], where LDL-C variability showed no mortality association in non-dialysis CKD patients. However, our results align with the GOULD registry [13] demonstrating that dynamic lipid changes better predict outcomes than static values in advanced CKD populations. The differential HDL-C mortality association in HD versus PD patients echoes the renal function-stratified results from the ODYSSEY OUTCOMES trial [14], where HDL-C's protective effects diminished with worsening kidney function.

The lack of significant lipid-mortality associations in PD patients contrasts with findings from Yiping et al. [10], who identified interactive effects between lipids and glucose metabolism. This discrepancy may reflect our cohort's smaller PD sample size ($n = 67$ vs. $n = 1{,}452$ in [10]) or differences in glucose-lowering therapy adherence. Our findings reinforce the KDIGO 2024 guidelines [15] emphasizing individualized lipid targets based on dialysis modality and comorbidity profile.

Regarding therapeutic implications, our results partially support the ESC guideline position [16] cautioning against universal statin use in dialysis patients, yet suggest dynamic lipid monitoring (as proposed in JCL recommendations [17]) might identify subgroups benefiting from intensified therapy. The observed LDL-C trajectory effects align with mechanistic insights from PCSK9 studies [18–20], particularly the lipoprotein receptor recycling dynamics described by David et al. [21]. While FOURIER-OLE subanalysis [14] showed PCSK9 inhibitors effectively lower LDL-C in CKD stages 3–4, our data underscore the need for dialysis-specific trials as mandated in KDIGO 2024 [15].

Study limitations should be interpreted through the lens of the CRIC study methodology [11], which similarly faced challenges in distinguishing CKD-related versus treatment-related lipid changes. While residual confounding from unmeasured lipid-lowering therapy (LLT) adherence or temporal effects cannot be excluded, the non-differential LLT use between HD and PD groups suggests minimal impact on modality-specific comparisons. Specifically, we emphasize the critical need for prospective studies to systematically collect detailed time-varying data on LLT and longitudinal dialysis adequacy metrics—including Kt/V, residual renal function (CrCl), and modality-specific complication profiles (e.g., peritonitis frequency in PD, vascular access events in HD)—as these parameters may modulate lipid

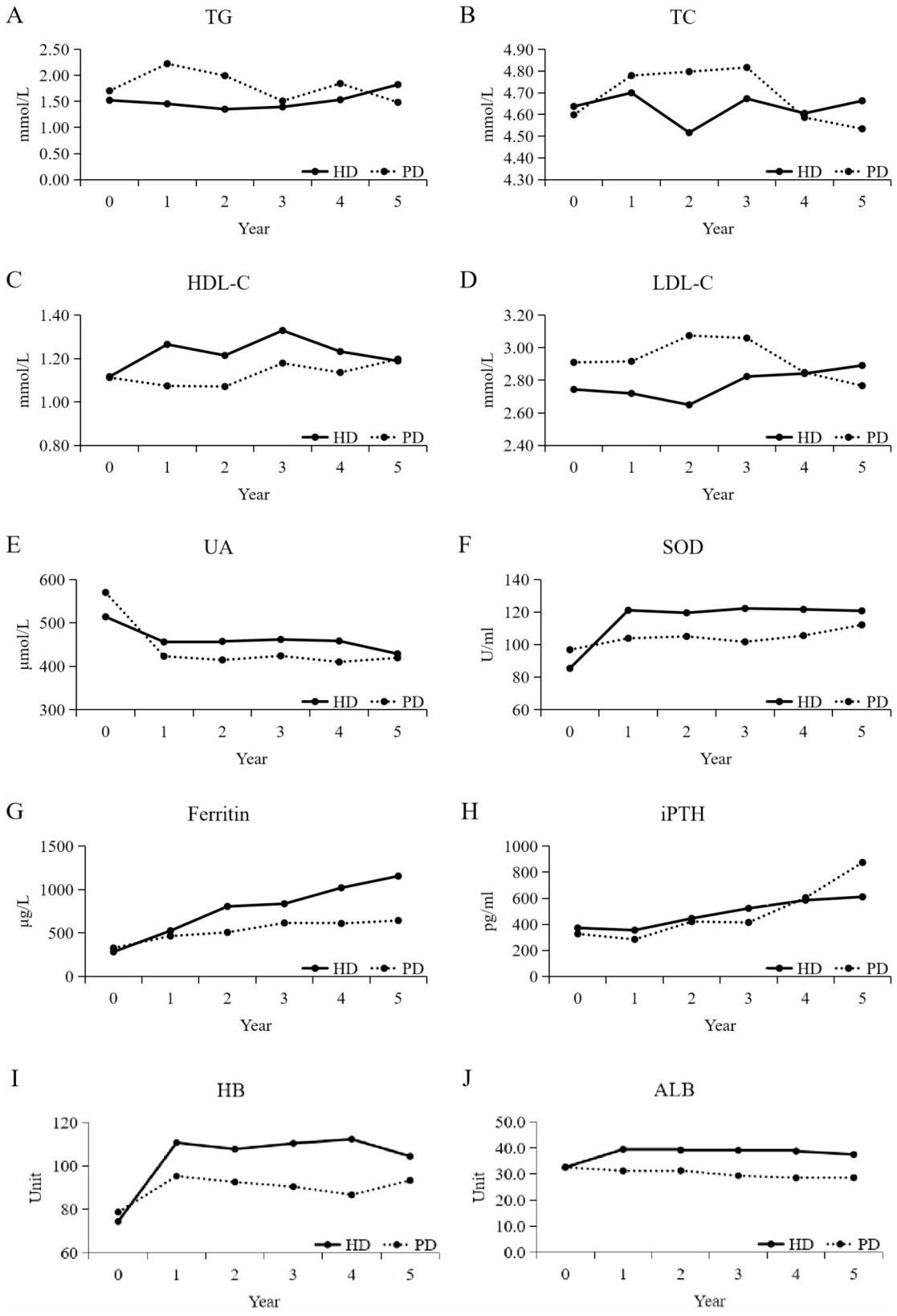

Fig. 1 The line chart of index results from year 0 to year 5, including TG (**A**), TC (**B**), HDL-C (**C**), LDL-C (**D**), UA (**E**), SOD (**F**), Ferritin (**G**), iPTH (**H**), HB (**I**) and ALB (**J**)

Table 3 The linear regression results of group variable to each index under GEE models

Parameter	B (95% CI)	*P*
HB	-12.27 (-17.85 to -6.69)	< 0.001
Ca	0.63 (-0.71 to 1.98)	0.357
P	-0.16 (-0.30 to -0.03)	0.019
PALB	0.22 (-0.17 to 0.61)	0.270
TG	0.25 (-0.07 to 0.58)	0.127
TC	0.08 (-0.28 to 0.44)	0.680
HDL-C	-0.07 (-0.17 to 0.03)	0.173
LDL-C	0.17 (-0.08 to 0.42)	0.176
ALB	-6.62 (-7.70 to -5.54)	< 0.001
UA	-6.90 (-33.13 to 19.33)	0.606
hsCRP	-3.18 (-12.26 to 5.90)	0.493
SOD	-9.09 (-13.50 to -4.68)	< 0.001
Ferritin	-263.67 (-409.54 to -117.79)	< 0.001
iPTH	-13.23 (-140.77 to 114.30)	0.839

Linear regression coefficient B is the estimation of group variable where PD comparing to HD, i.e., HD as reference group

trajectory-outcome relationships. Future investigations should prioritize standardized documentation of these parameters alongside advanced lipoprotein subfraction analysis to enable precision phenotyping in dialysis populations.

Conclusion

Our study reveals comparable five-year survival rates between HD and PD patients, yet identifies distinct lipid-mortality associations: baseline HDL-C and three-year LDL-C trajectory are critical prognostic markers in HD patients, while no significant associations emerged in PD cohorts. These findings underscore the necessity of modality-specific lipid management strategies—prioritizing dynamic LDL-C monitoring in HD and HDL functional restoration in PD. Time-averaged lipid targets rather than static thresholds, and precision interventions targeting HD-specific oxidative

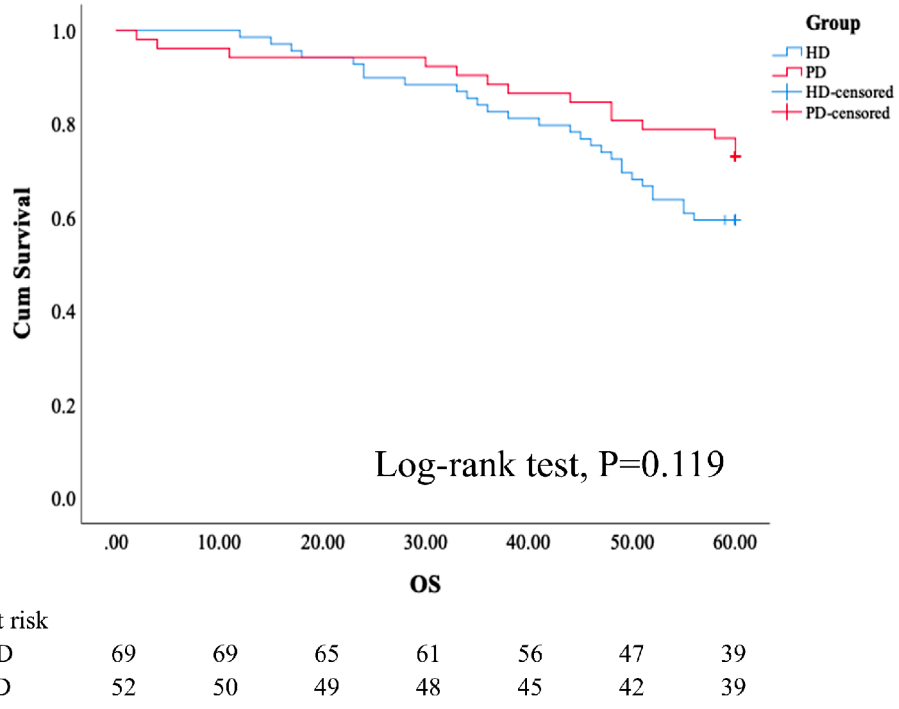

Fig. 2 The Kaplan-Meier survival functions of HD and PD groups to overall survival

Table 4 Univariate and multivariate Cox regression results of independent variables to overall survival among groups

Variables	Univariate						Multivariate			
	ALL		HD		PD		All		HD	
	HR[a] (95% CI)[b]	*P*	HR (95% CI)	*P*	HR (95% CI)	*P*	HR (95% CI)	*P*	HR (95% CI)	*P*
Age	1.05 (1.02 to 1.07)	< 0.001	1.04 (1.01 to 1.07)	0.008	1.05 (1.01 to 1.10)	0.024	1.04 (1.00 to 1.07)	0.032		
CVD	2.42 (1.27 to 4.60)	0.007	2.27 (1.08 to 4.79)	0.031			2.78 (1.15 to 6.72)	0.023		
TG	0.59 (0.35 to 0.98)	0.040								
HDL–C			0.26 (0.08 to 0.88)	0.030	2.19 (1.00 to 4.80)	0.050			0.00 (0.00 to 0.11)	0.004
hsCRP					1.01 (1.00 to 1.01)	0.017				
Ferritin	1.00 (1.00 to 1.00)	0.140	1.00 (1.00 to 1.00)	0.013						
Difference 0 to 1										
ALB					0.93 (0.88 to 0.98)	0.013				
SOD	0.99 (0.98 to 1.00)	0.036	0.98 (0.96 to 0.99)	< 0.001						
Ca	4.38 (1.13 to 17.02)	0.033					6.09 (1.34 to 27.64)	0.019		
hsCRP					0.99 (0.98 to 1.00)	0.017				
PALB	1.05 (1.01 to 1.09)	0.014					1.07 (1.03 to 1.11)	< 0.001		
iPTH					1.00 (1.00 to 1.01)	0.021				
Defference 0 to 3										
LDL-C			0.70 (0.49 to 0.99)	0.043					0.21 (0.09 to 0.53)	< 0.001
ALB			0.89 (0.81 to 0.98)	0.017						
SOD			0.98 (0.96 to 1.00)	0.043						
PALB					1.06 (1.01 to 1.11)	0.014				
hsCRP					1.02 (1.00 to 1.03)	0.010				
Ferritin					1.00 (1.00 to 1.00)	0.028				

a HR: Hazard ratios; b 95% CI: 95% confifidence interval

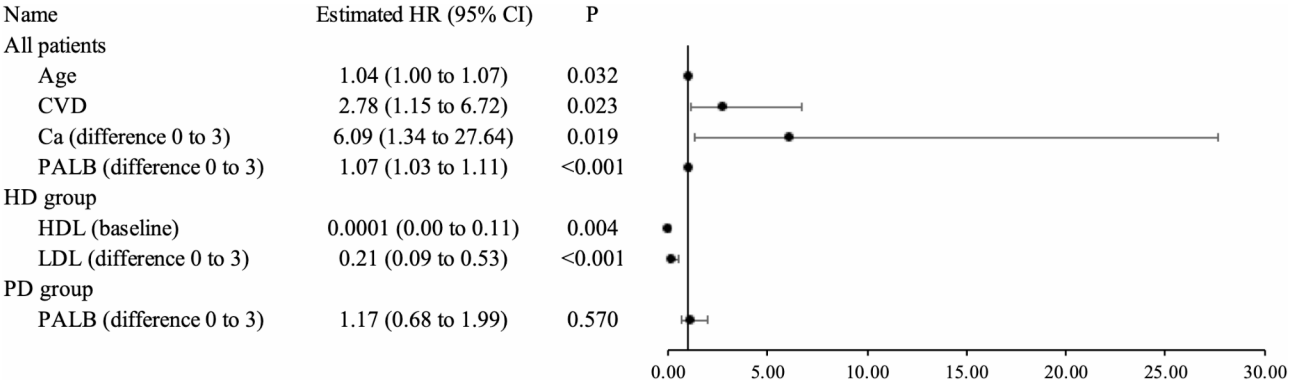

Fig. 3 The forest plot of all estimated hazard ratio (HR) in multivariate models

Table 5 ROC analysis results

Parameters	AUC (95% CI)	*P*	Sensitivity	Specificity	Youden index	Suggested cut-off
All patients						
Age	0.75 (0.66 to 0.83)	< 0.001	0.84	0.58	0.42	52.5
Ca (diff. 0 to 3)	0.63 (0.50 to 0.75)	0.055	0.46	0.84	0.30	0.575
PALB (diff. 0 to 3)	0.59 (0.45 to 0.73)	0.165	0.36	0.89	0.25	-0.08
HD group						
HDL-C (baseline)	0.62 (0.49 to 0.75)	0.094	0.52	0.70	0.22	0.965
LDL-C(diff. 0 to 3)	0.66 (0.50 to 0.82)	0.049	0.56	0.77	0.32	-0.24
PD group						
PALB (diff. 0 to 3)	0.60 (0.29 to 0.90)	0.461	0.50	0.89	0.39	-0.085

stress and PD-related HDL glycation maybe important in clinical practice. Future research must address: the paradoxical dissociation between LDL-C levels and mortality in PD patients through advanced lipoprotein subfraction analysis; long-term effects of lipid trajectory modulation on dialysis technique survival in multicenter cohorts. By bridging these gaps, we may ultimately achieve personalized lipid stewardship in dialysis care—a paradigm shift from reactive correction to proactive risk stratification.

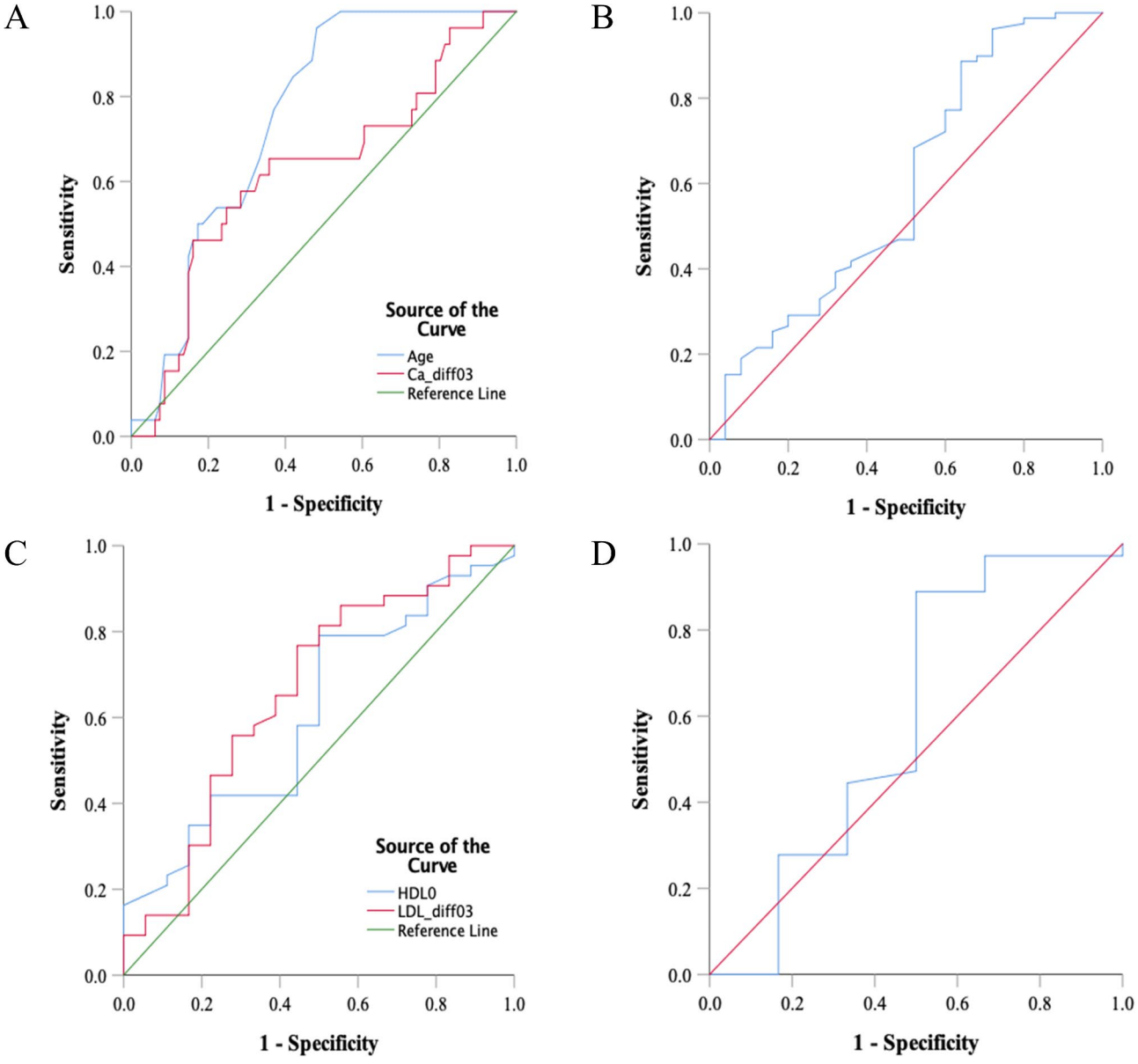

Fig. 4 The ROC of independent variables to dead outcome, including all patient's age and Ca (difference 0 to 3) (**A**), all patient's PALB (negative related to dead outcome) (**B**), HD group's HDL-C (baseline) and LDL-C (difference 0 to 3) (both negative related to dead outcome) (**C**), PD group's PALB (negative related to dead outcome) (**D**)

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12882-025-04119-x.

Supplementary Material 1

Acknowledgements
We would like to thank all the medical staff for their assistance and advice with the treatment and nursing care in this study.

Author contributions
Peifen Liang, Zhenjian Xu and Qiongqiong Yang wrote the main manuscript text and Yingyan Liang, Xuefeng Xie and Xiaomei Li prepared Figs. 1 and 2, Peifen Liang, Zhenjian Xu and Qiongqiong Yang prepared Figs. 3 and 4. All authors reviewed the manuscript.

Funding
This work was supported by grants from the Guangdong Natural Science Foundation (2023A1515011036, 2020A1515010429 and 2022A1515012308).

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
The study followed the tenets of the Declaration of Helsinki, and informed consent was obtained from all the participants. The study was approved by

the institutional review board of Sun Yat-sen Memorial Hospital of Sun Yat-sen University, Guangzhou, China (SYSKY-2024-860-01).

Clinical trial number
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Nephrology, Sun Yat-Sen Memorial Hospital of Sun Yat-Sen University, Guangzhou 510120, China
[2]Guangdong Provincial Key Laboratory of Malignant Tumor Epigenetics and Gene Regulation, Sun Yat-Sen Memorial Hospital of Sun Yat-Sen University, Guangzhou 510120, China

Received: 1 January 2025 / Accepted: 11 April 2025
Published online: 19 April 2025

References

1. Qishu L, Wei L. L-shaped association between dietary niacin intake and chronic kidney disease among adults in the USA: a cross-sectional study. Ren Fail. 2024;46(2).
2. Wang L, Xu X, Zhang M, Hu C, Zhang X, Li C, et al. Prevalence of chronic kidney disease in China: results from the sixth China chronic disease and risk factor surveillance. JAMA Intern Med. 2023;183(4):298–310.
3. Qunibi WY. Dyslipidemia in dialysis patients. Semin Dial. 2015 Jul-Aug;28(4):345– 53.
4. Tingting G, Wenqi X, Huanqing G, Jijuan Z, Jiaojiao Z, Kaiyue W, et al. Relationship between control of cardiovascular risk factors and chronic kidney disease progression, cardiovascular disease events, and mortality in Chinese adults. J Am Coll Cardiol. 2024;84(14).
5. Natalia S. Dyslipidemia in peritoneal dialysis: implications for peritoneal membrane function and patient outcomes. biomedicines. 2024;12(10).
6. Chieh-Li Y, Pei-Chun F, Cheng-Chia L, Jia-Jin C, Chao-Yu C, Yi-Ran T, et al. The role of maintaining lower LDL-C level during Statin treatment for advanced CKD patients. Atherosclerosis. 2024;399(0).
7. Kalantar-Zadeh K, Block G, Humphreys MH, Kopple JD. Reverse epidemiology of cardiovascular risk factors in maintenance dialysis patients. Kidney Int. 2003;63(3):793–808.
8. Yeoungjee C, Janine B, Sonja S, Jutta P-D, Carmel MH, Goce D, et al. Longitudinal trend in lipid profile of incident peritoneal dialysis patients is not influenced by the use of biocompatible solutions. Perit Dial Int. 2015;36(2).
9. Fanfan H, Jianping J, Jianghua C, Xueqing Y, Qiugen Z, Pingyan C, et al. China collaborative study on dialysis: a multi-centers cohort study on cardiovascular diseases in patients on maintenance dialysis. BMC Nephrol. 2012;13:0.
10. Yiping X, Zhong Z, Yi L, Zhijian L, Yi Z, Zhibin L, et al. Interaction effect between fasting plasma glucose and lipid profiles on mortality of peritoneal dialysis patients. Clin Kidney J. 2023;16(4).
11. Guiomar M, Stuart P, Virginia M, Andrea M, Ruth O, Inés G-L, et al. Determinants of progression and regression of subclinical atherosclerosis over 6 years. J Am Coll Cardiol. 2023;82(22).
12. Simon C, Jessy Korina P-E, Katherine MS, Sushrut SW, Finnian RMC. Myeloperoxidase and the risk of CKD progression, cardiovascular disease, and death in the chronic renal insufficiency cohort (CRIC) study. Am J Kidney Dis. 2019;76(1).
13. Aleesha S, Mikhail K, James A, dL, Qi G, Katherine EM, Shushama A, et al. Use of lipid-lowering therapies in patients with chronic kidney disease and atherosclerotic cardiovascular disease: 2-year results from getting to an improved understanding of low-density lipoprotein cholesterol and dyslipidemia management (GOULD). Clin Cardiol. 2022;45(12).
14. José T, Philippe Gabriel S, Deepak LB, Vera AB, Rafael D, Shaun GG, et al. Effect of alirocumab on major adverse cardiovascular events according to renal function in patients with a recent acute coronary syndrome: prespecified analysis from the Odyssey outcomes randomized clinical trial. Eur Heart J. 2020;41(42).
15. KDIGO. clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int. 2024;105(0).
16. Jean-Philippe C, Holger T, Emanuele B, Olivier B, Johann B, Deepak LB, et al. 2020 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J. 2020;42(14).
17. Gloria Lena V, Jijia W, Scott MG. Chronic kidney disease and statin eligibility. J Clin Lipidol. 2020;15(1).
18. Kevin F, Maria F-K, Svetlana S-M, Abigail L, Brian RB, Jessica ES, et al. Effect of an RNA interference drug on the synthesis of proprotein convertase subtilisin/kexin type 9 (PCSK9) and the concentration of serum LDL cholesterol in healthy volunteers: a randomised, single-blind, placebo-controlled, phase 1 trial. Lancet. 2013;383:9911.
19. Julia B, Kausik KR. Novel and future lipid-modulating therapies for the prevention of cardiovascular disease. Nat Rev Cardiol. 2023;20(9).
20. Konrad Z, Wiktoria G, Jacek M, Jolanta M. Kidney and lipids: novel potential therapeutic targets for dyslipidemia in kidney disease? expert opin ther targets. 2022;26(11).
21. David S, Danilo F, Thimoteus S. Proprotein convertase subtilisin/kexin type 9 in kidney disease. Nephrol Dial Transpl. 2019;34(8).

Publisher's note