Original Article

Pattern of Dyslipidaemia in Patients with Chronic Kidney Disease

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ABSTRACT

Context: Dyslipidaemia is one of the traditional risk factors for cardiovascular disease (CVD) and rapid progression of chronic kidney disease (CKD) to end-stage renal disease.

Aims: The aim of the study was to determine the prevalence and pattern of dyslipidaemia and assess the cardiovascular risk in various stages of CKD.

Setting and Design: A retrospective study of 109 adult pre-dialysis CKD patients who attended nephrology outpatient clinic or admitted at the Obafemi Awolowo University Teaching Hospitals' Complex between 2008 and 2015.

Materials and Methods: Patients' sociodemographic characteristics, aetiology of CKD, anthropometry, blood pressure, fasting lipid profile, electrolytes, urea and creatinine at contact were retrieved from their hospital records. Estimated glomerular filtration rate at presentation was calculated using CKD EPI equation. Dyslipidaemia was defined according to the National Cholesterol Education Program Adult Treatment Panel III's final report. Cardiovascular risk was assessed using atherogenic indices. Atherogenic index of plasma (AIP) >0.24 was regarded as high cardiovascular risk.

Statistical Analysis Used: Data were analysed using the statistical package for the social sciences (SPSS) software version 22.

Results: A total of 109 pre-dialysis CKD patients were studied. Overall prevalence of dyslipidaemia amongst pre-dialysis CKD patient in this study was 90.8%. The mean serum total cholesterol, low-density lipoprotein cholesterol (LDLc), high-density lipoprotein cholesterol (HDLc), very LDLc , non-HDLc and triglycerides were 5.9 (±2.80) mmol/L, 3.6 (±2.50) mmol/L, 1.3 (±0.70) mmol/L, 0.73 (±0.40) mmol/L, 4.5 (±2.60) mmol/L and 1.6 (±1.0) mmol/L, respectively. The median AIP in the pre-dialysis CKD cohort was 0.40 (interquartile range; 0.21–0.72), while 72.5% may be at high risk of developing CVD (AIP >0.24).

Conclusion: Dyslipidaemia is common amongst CKD patients. CKD patients show significant abnormalities of lipid metabolism which may contribute to CVD

Key words: Cardiovascular disease in chronic kidney disease, dyslipidaemia, hyperlipidaemia

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INTRODUCTION

Chronic kidney disease (CKD) is a global public health problem associated with increased morbidity and mortality, cardiovascular disease (CVD) being responsible for majority of them. In fact, about 50% of patients with end-stage renal disease (ESRD) die from a cardiovascular event.[1] Cardiovascular mortality is 30 times higher in dialysis patients and 500 times higher in 25–34-year-old ESRD patients than in individuals from the general population.[1] Those with mild-to-moderate CKD are likely to succumb to CVD before they even progress to ESRD.[2,3]

The association between CKD and CVD is multifactorial.[4] Dyslipidaemia which is one of the traditional risk factors for CVD is a major culprit. At the earlier stages of CKD, patients

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develop both quantitative and qualitative abnormalities in lipoprotein metabolism which usually follows a worsening course as renal function deteriorates regardless of the aetiology of CKD. Early in CKD, there are alterations in apolipoproteins, lipid transfer proteins, lipolytic enzymes and lipoprotein receptors.[1,5,6] As renal function declines, triglyceride (TG) concentrations increase and high-density lipoprotein cholesterol (HDLc) concentrations decline and become dysfunctional, while levels of low-density lipoprotein cholesterol (LDLc) remain within the normal range or become slightly low.[1,5,6] Furthermore, there is progressive accumulation of small less dense and oxidised LDL particles, decreased concentration of apolipoprotein A-containing lipoproteins and increased concentrations of TG-rich apolipoprotein B-containing lipoproteins as glomerular filtration rate (GFR) declines (Stages 4 and 5). Together these, abnormalities contribute to the risk of arteriosclerotic CVD which adversely affects the progression of renal disease and contributes to cardiovascular morbidity and mortality.

In this study, we set out to determine the pattern of dyslipidaemia in various stages of CKD and to assess the cardiovascular risk by correlating the atherogenic indices with the severity of CKD.

MATERIALS AND METHODS

It is a retrospective study of adult pre-dialysis CKD patients attending nephrology outpatient clinic or admitted at the Obafemi Awolowo University Teaching Hospitals' Complex between 2008 and 2015. Those with documented fasting lipid parameters at their first visit were recruited into the study. Patients with chronic liver disease patients, significant alcohol intake, ESRD patients, transplant patients and pre-dialysis CKD already on corticosteroid therapy and lipid lowering therapy at the time of presentation were excluded from the study. Their sociodemographic characteristics, aetiology of CKD, anthropometry, blood pressure, fasting lipid profile, electrolytes, urea and creatinine at contact were retrieved from their hospital records.

Estimated GFR (eGFR) at presentation was calculated using CKD EPI equation.[7] CKD was staged according to the KDIGO guidelines.[8] Total cholesterol (TC), LDLc, HDLc and TGs at presentation were retrieved from the hospital records, while very LDLc (VLDLc) values were calculated using Friedewald formulae.[9]

VLDLc = TG mmol/L/2.2

Non-HDL cholesterol (non-HDLc) was estimated using this formula:[10]

Non-HDL-cholesterol = (TC − HDLc).

Atherogenic indices were calculated as follows:

Castelli risk index I (CRI I) = TC/HDLc

Castelli risk index II (CRI II) = LDLc/HDLc

Atherogenic coefficient = (TC − HDLc)/HDLc

Atherogenic index of plasma (AIP) = log (TG/HDLc).

Dyslipidaemia was defined according to the National Cholesterol Education Program Adult Treatment Panel III's final report as any or the combination of the following: a serum TC ≥200 mg/dl (5.18 mmol/l), TG ≥150 mg/dl (1.70 mol/l), LDLc of ≥100 mg/dl (2.59 mol/l) and HDLc <40 mg/dl (<1.03 mol/l).[11]

Normal VLDLc is between 2 and 30 mg/dl (abnormal VLDLc defined as values ≥30 mg/dl or 0.7758 mmol/l). Non-HDLc ≥130 mg/dl (3.37 mmol/L) was considered abnormal.

AIP of −0.3–0.1, 0.1–0.24 and >0.24 were regarded as low, intermediate and high cardiovascular risks, respectively.[12]

Diagnostic criteria for the various chronic kidney disease aetiologies

Chronic glomerulonephritis (CGN) was diagnosed in those presenting with any of the following: nephrotic syndrome, nephritic syndrome, persistent asymptomatic proteinuria ≥2+, persistent asymptomatic haematuria of glomerular origin and renal biopsy histology report in keeping with GN with or without a decline in GFR.

Hypertension-attributed CKD was diagnosed in hypertensive patients with a family history of hypertension, whose hypertension predates evidence of renal disease (proteinuria and serum creatinine >1.2 mg/dl), with evidence of Left Ventricular Hypertrophy (LVH) on echocardiography/electrocardiography and/Grade 1 or 2 hypertensive retinopathy and minimal proteinuria of ≤2+ or <500 mg of protein/24 h in the absence of nephrotoxin exposure, congenital, immune complex disease or intrinsic renal disease or other systemic illnesses associated with renal damage.[13]

Diabetic nephropathy was diagnosed in those diabetes mellitus (DM) patients with persistent macroalbuminuria (300 mg/d or >200 µg/min) confirmed on two occasions 3–6 months apart.[14]

Autosomal dominant polycystic kidney disease (ADPKD) was diagnosed using the unified ultrasonographic diagnostic criteria.[15]

Lupus nephritis was diagnosed in patients with systemic lupus erythematosus with renal involvement (urine protein ≥3+ or ≥0.5 g/24 h or cellular cast or renal biopsy report in keeping with lupus nephritis).[6]

Patients with obstructive nephropathy were usually referred by urologists and had either a BPH or prostatic cancer with symptoms of bladder outlet obstruction, ultrasound features of obstructive nephropathy and impaired renal function.

Statistical analysis

Data were analysed using the SPSS Statistics for Windows, version 22.0 (SPSS Inc., Chicago, Ill., USA). Categorical variables were summarised as percentages. Continuous variables were expressed as means ± standard deviation (SD) or median interquartile range. Association between categorical variables was assessed using the Chi-square test, whereas

means were compared using the independent Student's *t*-test or one-way analysis of variance. $P < 0.05$ was considered statistically significant.

RESULTS

Pre-dialysis chronic kidney disease

A total of 109 pre-dialysis CKD patients were studied. Of the 109 pre-dialysis cohort, 69 (63.3%) were males while females constituted 36.7% (n = 40). The mean age was 47 (±16.2) years (range - 15–85 years). CGN was the most common aetiology of CKD (40.4%), followed by hypertension-attributed CKD (31.2%), DM (13.8%), ADPKD (9.2%), sickle cell nephropathy (1.8%), lupus nephritis (1.8%) and obstructive nephropathy (1.8%). Median eGFR was 18.1 ml/min/1.73 m^2 (7.1–45.81 ml/min/1.73 m^2). Majority of these patients were in Stage 5 CKD; 45 (41.3%), 9 (8.3%) in Stage 1, 11 (10.1%) in Stage 2 and 30 (27.5%) in Stage 3, while 14 (12.8%) were in Stage 4 CKD.

The mean (±SD) body mass index, systolic blood pressure and diastolic blood pressure were 25. 69 (±4.59) kg/m^2, 154 (±31) mmHg and 95 (±19) mmHg, respectively.

Prevalence of elevated TC amongst pre-dialysis CKD was 54.1%, 62.4% had elevated LDLc, 42.2% had low HDLc, whereas 34.9% had elevated TG. Overall prevalence of dyslipidaemia amongst pre-dialysis patient in this study was 90.8% with mean serum TC, LDLc, HDLc, VLDLc, non-HDLc and TG, being 5.9 (±2.80) mmol/L, 3.6 (±2.50) mmol/L, 1.3 (±0.70) mmol/L, 0.73 (±0.40) mmol/L, 4.5 (±2.60) mmol/L and 1.6 (±1.0) mmol/L, respectively.

The prevalence of elevated TC, LDLc, TG, non-HDLc, and low HDLc amongst pre-dialysis CKD was 54.1%, 62.4%, 34.9%, 58.7% and 42.2%, respectively. Approximately, 72.5% of them had AIP >0.24.

The prevalence of the lipid fractions varied across the various stages of CKD [Table I]. The prevalence of elevated TC and non-HDLc fractions was found to be significantly different across the CKD stages (P = 0.022 and 0.017, respectively). There was no significant difference between the prevalence of elevated serum TG, LDLc, low HDLc and high AIP and the overall prevalence of dyslipidaemia across the CKD stages (P = 0.695, 0.380, 0.088, 0.187 and 0.341, respectively).

The mean TC, LDLc, VLDc, TG and non-HDLc were significantly different across the CKD stages with P = 0.001, 0.015, 0.012, 0.012 and 0.001, respectively [Table II]. *Post hoc* analysis revealed that mean TC and non-HDLc were only significantly lower in Stage 5 pre-dialysis CKD when compared with Stage 2 CKD (P = 0.047 and 0.047, respectively).

The eGFR positively correlated with TC (r = 0.426, P = 0.001), LDLc (r = 0.280, P = 0.004), TG (r = 0.256, P = 0.019), VLDLc (r = 0.256, P = 0.019) and non-HDLc (r = 0.402, P = 0.001).

The lipid ratios were found to be elevated in pre-dialysis CKD [Table II]. Castelli risk index I, atherogenic coefficient and AIP did not correlate with the severity of CKD (P = 0.078, 0.078 and 0.300, respectively).

Comparison of lipid parameters across the various aetiologies of chronic kidney disease

The prevalence and pattern of dyslipidaemia across the various aetiologies is as shown in Tables III and IV. All the CGN patients in our cohort had dyslipidaemia. The prevalence of dyslipidaemia were 86.7%, 82.4% and 80% in those with diabetes, hypertension and ADPKD, respectively. The proportion of CKD patients with elevated TC, non-HDLc and overall dyslipidaemia vary significantly across the various aetiologies, $P \leq 0.05$ [Table III]. The mean TC, LDLc and non HDLc were found to be significantly different across the different aetiologies ($P \leq 0.05$).

Median Castelli risk index I, atherogenic coefficient and AIP were similar across the various aetiologies of CKD (P = 0.067, 0.067 and 0.600, respectively).

DISCUSSION

Majority of our patients in this study were young which is in keeping with reports from previous studies on CKD in Nigeria which usually affect the economically productive age group when compared with developed countries.[16,17] In addition,

Table I: Distribution of abnormal of lipid fractions across the various stages of chronic kidney disease

	All CKD (n=109), n (%)	Stage 1 CKD (n=9), n (%)	Stage 2 CKD (n=11), n (%)	Stage 3 CKD (n=30), n (%)	Stage 4 CKD (n=14), n (%)	Pre-dialysis Stage 5 CKD (n=45), n (%)	P
High TC	59 (54.1)	8 (88.9)	8 (72.2)	17 (56.7)	9 (64.3)	17 (37.8)	0.022
High LDL	68 (62.4)	6 (66.7)	10 (90.9)	20 (66.7)	10 (71.4)	22 (48.9)	0.380
Low HDL	46 (42.2)	3 (33.3)	3 (27.3)	11 (36.7)	5 (35.7)	24 (53.3)	0.088
High TG	38 (34.9)	2 (22.2)	6 (54.5)	12 (40)	6 (42.9)	13 (28.9)	0.695
High-non-HDL cholesterol	64 (58.7)	7 (77.8)	9 (81.8)	20 (66.7)	10 (71.4)	18 (40.0)	0.017
AIP >0.24 high risk CVD	79 (72.5)	4 (44.4)	8 (72.7)	27 (90)	10 (71.4)	31 (68.9)	0.187
Dyslipidemia	99 (90.9)	9 (100)	11 (100)	28 (93.3)	13 (92.9)	38 (84.4)	0.341

CKD: Chronic kidney disease, TC: Total cholesterol, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TG: Triglycerides, AIP: Atherogenic index of plasma, CVD: Cardiovascular disease

Table II: Comparison of lipid parameters across the chronic kidney disease stages

	Mean±SD						*P*
	All CKD (*n*=109) Mean±SD	Stage 1 CKD (*n*=9) Mean±SD	Stage 2 CKD (*n*=11) Mean±SD	Stage 3 CKD (*n*=30) Mean±SD	Stage 4 CKD (*n*=14) Mean±SD	Pre-dialysis Stage 5 CKD (*n*=45) Mean±SD	
TC (mmol/L)	5.9±2.8	6.7±2.4	7.6±3.0	6.6±3.9	6.6±2.5	4.5±1.2	0.001
LDLc (mmol/L)	3.6±2.5	3.8±2.2	4.7±2.6	4.3±3.5	3.9±2.4	2.6±1.1	0.015
VLDLc (mmol/L)	0.73±0.47	0.72±0.61	1.02±0.70	0.72±0.30	1.13±0.77	0.62±0.33	0.012
HDLc (mmol/L)	1.3±0.7	1.8±1.2	1.3±0.46	1.3±0.7	1.4±1.1	1.2±0.5	0.195
TG (mmol/L)	1.6±1.0	1.6±1.4	2.3±1.5	1.6±0.7	2.5±1.7	1.4±0.7	0.012
Non-HDL cholesterol (mmol/L)	4.5±2.6	4.9±2.0	6.2±2.8	5.3±3.6	5.2±2.5	3.4±1.2	0.001
Castelli risk Index I**	4.4 (3.2-5.8)	3.7 (2.5-6.5)	5.2 (4.8-6.4)	4.6 (3.5-6.4)	5.1 (3.1-6.6)	3.9 (2.9-5.4)	0.278
Castelli risk Index II**	2.5 (1.7-3.5)	2.0 (1.2-3.5)	3.2 (2.6-4.7)	2.5 (1.9-4.7)	2.3 (1.8-4.4)	2.4 (1.4-3.1)	0.344
Atherogenic coefficient**	3.4 (2.2-4.8)	2.7 (1.5-5.5)	4.2 (3.8-5.4)	3.6 (2.5-5.4)	4.5 (2.1-5.6)	2.9 (1.9-4.4)	0.278
AIP**	0.40 (0.21-0.72)	0.14 (−0.12-0.97)	0.46 (0.13-0.91)	0.46 (0.31-0.66)	0.66 (0.19-0.86)	0.32 (0.19-0.65)	0.439

**Skewed data expressed in median (IQ range). CKD: Chronic kidney disease, TC: Total cholesterol, LDLc: Low-density lipoprotein cholesterol, VLDLc: Very low-density lipoprotein cholesterol, HDL: High-density lipoprotein, HDLc: HDL cholesterol, TG: Triglycerides, AIP: Atherogenic index of plasma, SD: Standard deviation, IQ: Interquartile

Table III: Distribution of abnormal of lipid fractions across the various chronic kidney disease etiologies

	All CKD (*n*=109), *n* (%)	CGN (*n*=44), *n* (%)	Hypertension (*n*=34), *n* (%)	Diabetes (*n*=15), *n* (%)	ADPKD (*n*=10), *n* (%)	Other etiologies (*n*=6), *n* (%)	*P*
High TC	59 (54.1)	30 (68.2)	13 (38.2)	9 (60.0)	3 (30)	4 (66.7)	0.042
High LDL	68 (62.4)	33 (75.0)	18 (52.9)	7 (46.7)	6 (60)	4 (66.7)	0.205
Low HDL	46 (42.2)	22 (50.0)	12 (35.3)	7 (46.7)	3 (30)	2 (33.3)	0.611
High TG	38 (34.9)	19 (43.2)	10 (29.4)	5 (33.3)	0 (0)	4 (66.7)	0/227
High-non-HDL Cholesterol	64 (58.7)	32 (72.7)	16 (47.1)	10 (66.7)	2 (20)	4 (66.7)	0.016
AIP >0.24 high risk CVD	79 (72.5)	33 (75)	24 (70.6)	10 (66.7)	8 (80)	5 (83.3)	0.931
Dyslipidemia	99 (90.9)	44 (100)	28 (82.4)	13 (86.7)	8 (80)	6 (100)	0.046

CKD: Chronic kidney disease, TC: Total cholesterol, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TG: Triglycerides, AIP: Atherogenic index of plasma, CVD: Cardiovascular disease, CGN: Chronic glomerulonephritis, ADPKD: Autosomal dominant polycystic kidney disease. Other etiologies include 2 sickle cell nephropathies, 2 lupus nephritis and 2 obstructive nephropathies

Table IV: Comparison of lipid parameters across the different etiologies of chronic kidney disease

Lipid parameters	Mean±SD						*P*
	All CKD (*n*=109) Mean±SD	CGN (*n*=44) Mean±SD	Hypertension (*n*=34) Mean±SD	Diabetes (*n*=15) Mean±SD	ADPKD (*n*=10) Mean±SD	Other etiologies (*n*=6) Mean±SD	
TC (mmol/L)	5.9±2.8	7.1±3.6	4.7±1.3	5.6±2.1	4.3±1.2	6.6±2.3	0.001
LDLc (mmol/L)	3.6±2.5	4.6±3.3	2.7±1.1	3.0±1.5	2.5±0.8	4.2±2.2	0.004
VLDLc (mmol/L)	0.73±0.47	0.83±0.57	0.62±0.28	0.74±0.50	0.5±0.1	0.99±0.7	0.220
HDLc (mmol/L)	1.3±0.7	1.3±0.9	1.2±0.4	1.4±0.9	1.3±0.6	1.5±0.8	0.959
TG (mmol/L)	1.6±1.0	1.8±1.3	1.4±0.6	1.6±1.0	1.2±0.3	2.2±1.5	0.220
Non-HDL cholesterol (mmol/L)	4.5±2.6	5.7±3.4	3.5±1.2	4.3±1.8	3±0.9	5.1±2.5	0.001
Castelli risk Index I**	4.4 (3.2-5.8)	5.2 (3.7-7.3)	3.8 (2.7-5.2)	4.6 (3.1-5.6)	3.5 (2.7-4.4)	4.9 (2.9-8.1)	0.067
Castelli risk Index II**	2.5 (1.7-3.5)	2.8 (2.1-4.6)	2.3 (1.4-3.2)	1.9 (1.7-3.9)	2.2 (1.4-3.1)	3.4 (1.3-5.9)	0.094
Atherogenic coefficient**	3.4 (2.2-4.8)	4.2 (2.7-6.3)	2.8 (1.7-4.2)	3.6 (2.1-4.6)	2.5 (1.7-3.4)	3.9 (1.9-7.1)	0.067
AIP**	0.40 (0.20-0.72)	0.47 (0.14-0.84)	0.32 (0.21-0.62)	0.44 (0.13-0.69)	0.33 (0.18-0.44)	0.59 (0.09-0.96)	0.600

**Skewed data expressed in median (IQ range). Other etiologies include 2 sickle cell nephropathies, 2 lupus nephritis and 2 obstructive nephropathies. CKD: Chronic kidney disease, TC: Total cholesterol, LDLc: Low-density lipoprotein cholesterol, VLDLc: Very low-density lipoprotein cholesterol, HDL: High-density lipoprotein, HDLc: HDL cholesterol, TG: Triglycerides, AIP: Atherogenic index of plasma, SD: Standard deviation, CGN: Chronic glomerulonephritis, ADPKD: Autosomal dominant polycystic kidney disease, IQ: Interquartile

we found that the leading cause of CKD in this study was CGN followed by hypertension and DM has been reported by others. [16,17]

Majority of our patients were in CKD Stage 3, 4 and 5 with very few cases in Stages 1 and 2. This finding may be explained by the poor health-seeking behaviours in our environment.

CKD is largely asymptomatic at early stages and so may not be recognised by patients who do not routinely have medical checks.

This study revealed that dyslipidaemia is prevalent amongst our pre-dialysis CKD cohort and it affects almost all the lipid fractions. Over 90.9% of our study population had at least one lipid abnormality, all the patients in Stages 1 and 2 CKD had dyslipidaemia, 93.3% of Stage 3, 92.9% of Stage 4 CKD and 84.4% of Stage 5 pre-dialysis CKD had at least one abnormal lipid fraction. Our findings are similar to that of Akpan *et al*. who studied 100 dialysis-naïve CKD, reported an overall prevalence of 84% amongst their study participants, they are however higher than other previously reported studies in West Africa. [16-19]

One would also expect patients with non-dialysis-dependent CKD to have predominantly low HDLc and high TG and normal or even lower TC and LDLc which is contrary to our findings [Tables I and II]. The possible explanation for this is that lipid profile pattern in dialysis-naïve CKD depends not only on the level of kidney function but also on the degree of proteinuria and nutritional status. Patients with nephrotic syndrome and preserved GFR show a higher atherogenic profile, with markedly elevated plasma cholesterol, TG concentrations, and increased VLDLc and LDLc, intermediate density lipoprotein (IDL), lipoprotein(a) levels and normal, increased or depressed plasma HDLc. [1,6] In this study, the leading aetiology of CKD in our dialysis-naïve cohort is CGN which is a proteinuric CKD. Tables III and IV further corroborates this presumption, elevated TC and LDLc were more prevalent (68.2% and 75%, respectively), and the mean TC and LDLc were higher amongst the CKD patients whose aetiology is CGN when compared to other CKD aetiologies.

Non-HDLc which is a measure of atherogenic lipids such as apolipoprotein(a), LDLc, IDL and VLDLc was prevalent amongst our study population. The prevalence was found to be higher in earlier stages of CKD than in the pre-dialysis Stage 5 CKD [Table I]. Several prospective studies in the Western world and Asia have demonstrated that elevated non-HDLc is a risk factor for CVD in CKD patients. [20-23]

Atherogenic indices have been reported to be higher in CKD population compared to healthy controls. [16,24,25] These parameters are used for assessing the risk of CVD beyond the routine lipid profile parameters. AIP is a marker of atherogenicity which has been reported to have higher predictive value for atherosclerosis and cardiovascular events compared to other atherogenic indices. [24,26] Studies have shown that AIP may be an alternative diagnostic tool for cardiovascular risk assessment when other atherogenic risk parameters such as LDL, TG and HDL are within normal range. AIP is an indirect indicator of small dense LDL levels and other atherogenic lipoprotein phenotype. [24,26,27] The prevalence of pre-dialysis CKD patients with high AIP in our study was in agreement with a previous finding in this environment. [16]

This study is limited by its retrospective nature, lack of non-CKD cohort as control and lack of information on the urinary protein excretion rate of studied patients. A prospective multicentre study with larger sample size that will include dialysis (haemodialysis and peritoneal dialysis patients) and transplant patient cohorts will be required to further strengthen the observations found in this study.

CONCLUSION

Dyslipidaemia is common amongst CKD patients. CKD patients show significant abnormalities of lipid metabolism such as hyper-triglyceridaemia, hypercholesterolaemia (i.e. elevated TC, elevated LDLc, elevated non-HDLc), low HDLc and high atherogenic indices which may contribute to atherosclerosis and CVDs. We advocate early evaluation and management of dyslipidaemia in CKD patients.

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Conflicts of interest

There are no conflicts of interest.

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