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Original Article

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Pre and Post Dialysis Variations in Serum Lipid Profile among End Stage Renal Disease Patients

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ABSTRACT

Introduction: Chronic Kidney Disease (CKD) is a devastating disease and 50% of CKD patients die from Cardiovascular Disease (CVD) rather than End Stage Renal Disease (ESRD). Dyslipidaemia is a major contributor of CVD in these patients. The exact effect of Renal Dialysis (RD) on lipid parameters in ESRD is not clearly elucidated.

Aim: To assess the variations in serum lipid profile and study the pattern of lipoproteins by agarose gel electrophoresis before and after RD.

Materials and Methods: In this case-control study, 30 ESRD patients (15 male and 15 female) receiving maintenance renal dialysis were taken as cases and 30 age and sex matched healthy individuals were recruited as controls. Lipid profile parameters were estimated in Siemens automated analyser. Lipoprotein electrophoresis was done on Sebia Semi automated analyser. The data were analysed using GraphPad prism. Student's t-test for normally distributed data, Mann-Whitney U test for non-normally distributed data and ANOVA for comparison of more than two groups was done. A p-value <0.05 was considered statistically significant.

Results: In ESRD cases there was a significant increase (p <0.001) in Total Cholesterol (TC), Low Density Lipoprotein Cholesterol (LDL-C), High Density Lipoprotein Cholesterol (HDL-C) after RD compared to the values before RD (151.4±38.92 versus 183±57.18 mg/dL; 75.3±24.66 mg/dL versus 84.23±29.74; 31.9±9.74 mg/dL versus 35.57±12.49 mg/dL); respectively. Triglycerides (TG) and Very Low Density Lipoprotein (VLDL) values decreased after RD compared to the values before RD (123.6±50 versus 121.4±49.26; 24.73±10 versus 24.29±9.85); (p>0.05). Fractionation of lipoproteins reveals a significant increase in lipoprotein a {Lp(a)} as compared to controls (21.20±17.46 versus 12.38±10.63, p<0.05). The values of alpha and beta lipoproteins in electrophoresis didn't correlate well with the values obtained from automated enzymatic lipoprotein measurements in ESRD cases after dialysis (r=0.22, p=0.18).

Conclusion: There is a remarkable increase in atherosclerotic risk after RD noted due to significant increase in TC, LDL-C and Lp(a) which might increase the risk of CVD in RD subjects.

Keywords: Cardiovascular disease, Chronic kidney disease, Dyslipidaemia, Lipoprotein agarose gel electrophoresis

INTRODUCTION

Chronic kidney disease is defined as a permanent and significant reduction in Glomerular Filtration Rate (GFR) [1]. The estimated prevalence of CKD in India is up to 785 people per million populations [2].

Several studies have revealed that more than 50% of CKD patients die from CVD rather than develop ESRD [3-5]. The predominant cause of CVD is dyslipidaemia along with increased incidence of both traditional and non-traditional risk factors [6]. The most distinctive lipid abnormality in these patients is increased TG, VLDL and Intermediate Density Lipoprotein (IDL) due to reduced clearance of these triglyceride-rich lipoproteins and low levels of HDL-C [7]. LDL-C may remain normal quantitatively but gets altered qualitatively. Oxidative stress, hyperhomocysteinaemia and alterations in lipid metabolism observed in haemodialysis patients could increase LDL-C oxidation [8]. Lipid abnormalities not only increase the risk of atherosclerosis and CVD but also play a crucial role in the initiation and progression of glomerular and tubulo-interstitial diseases [9].

The renal replacement therapy, the mainstay of the management of CKD is beyond the reach of a large number of CKD patients in many developing countries, including India [10]. Maintenance Renal Dialysis (MRD) is the supportive treatment for such patients [11].

Dialysis is effective for amelioration of uremic symptoms and its toxicity. The effect of haemodialysis on LDL-C and HDL-C inflammatory properties is not known. Dialysis may attenuate LDL-C inflammatory activity and restore HDL-C anti-inflammatory properties by removing the potential pro-oxidant and proinflammatory uremic toxins. Conversely, LDL-C and HDL-C inflammatory activities can be transiently intensified due to exposure to dialyser membrane and tubing, mechanical stress in the roller pump, and influx of impurities from dialysate compartment during haemodialysis [12].

Treatment for hyperlipidaemia in the primary and secondary prevention of CVD is well recognised in the general population while the treatment of hyperlipidaemia in the dialysis population has remained controversial [13].

Indian studies on the effect of RD on dyslipidaemia are indeterminate and inconsistent [14,15]. There is insufficient data on the effect of single RD session on lipoproteins in ESRD patients on MRD [16].

Hence, the present study was delineated to assess alterations in the lipoproteins before and after the procedure in a single RD session.

MATERIALS AND METHODS

A case-control study was conducted at Mediciti Hospitals, Hyderabad, Telangana, India, from December 2012-June 2014. A total of 30 ESRD patients (GFR <15 mL/minutes/1.73 m^2) who were maintained on regular maintenance renal dialysis at Mediciti Hospitals after excluding the exclusion criteria {Obesity (Body mass index >24 kg/m^2), nephrotic syndrome, acute renal failure, patients

on ambulatory peritoneal dialysis, patients on lipid lowering drugs, patients on specific diet/specific exercise} were taken as cases and 30 age and sex matched clinically healthy volunteers (GFR >60 mL/minutes/m^2) from the institute staff were screened and included as controls.

Sample size was determined based on Seres DS et al., study [17]. The sample size was 15 in each group with 95% confidence interval and 90% power, to detect the difference in lipid profile values before and after RD using the Polysulphone (PS) dialyser membrane.

Study groups: The study group was divided into six sub groups. They are as follows:

Group I: n=15; Male ESRD cases: Ia: Before RD; Ib: After RD

Group II: n=15; Female ESRD cases: IIa: Before RD; IIb: After RD

Group III: n=15; Age and sex matched healthy male controls.

Group IV: n=15; Age and sex matched healthy female controls.

Institutional Ethical Committee approval was taken before the commencement of the study. A written and informed consent was taken from all the subjects. All procedures performed in the study comply with the ethical standards of the institutional and National/International Research Committee and with the 1964 Helsinki declaration and its later amendments.

Methods

History of all current medications, risk factors for cardiovascular disease, medical and surgical history was taken. All the patients were on low flux polysulphone membrane dialysers from Fresenius FX5/F6. Dialysis blood access was through an arteriovenous fistula. The mean blood flow rate was 300 mL/minute; the mean dialysate flow rate was 500 mL/minute. Heparin was given as a bolus dose of 1500 IU/L; followed by a maintenance dose of 500 IU/L. The patients were dialysed 2-3 times a week and each session on an average lasted for 4 hours. Patients were on dialysis since 1.51±1.49 years (Mean±SD).

Bicarbonate dialysate was used. Dialysis parameters were kept constant for all the patients in the study group. Non-fasting (as it was unethical and impractical to collect fasting samples in the dialysis setting) blood samples were collected from arterial dialysis catheter into serum separator vaccutainer immediately before the RD session and post-dialysis samples were collected 5 minutes after the procedure for equilibration of the graft. The first sample was drawn and discarded (to avoid sample dilution with saline) and second sample was used for analysis. Post 8 hours fasting, serum samples were collected among controls.

Estimation of lipid profile: In the present study TC, HDL-C, TG were estimated in (Dimension Xp and plus) Siemens automated analyser using commercially available Seimens kits. LDL-C was not calculated by Friedewald formula instead, it was estimated by automated enzymatic assays.

Quality control: Internal quality control was done for the measuring parameters by using Biorad lyphochek assayed chemistry control level 1 and level 2.

Fractionation of lipoproteins: Lipoprotein electrophoresis was done on (Hydrasys) Sebia Semi automated analyser using commercially available Sebia Hydragel 7 Lipo+Lp (a). The buffered agarose gels with pH 7.5 were used as electrophoretic support, electrophoretograms were dyed using Sudan Black and scanned by a densitometer (Epson perfection V700) [18].

STATISTICAL ANALYSIS

The data were analysed using GraphPad prism (version 6.0). Descriptive statistics for all parameters were computed. Paired Student's t-test and unpaired t-test for unmatched variables were done for normally distributed data. Mann-Whitney U test was done for non-normally distributed data. ANOVA was done for comparison of more than two groups. A $p<0.05$ was considered statistically significant.

RESULTS

Demographic characteristics in the study group [Table/Fig-1]: There was no significant age difference found between all the four groups ($p>0.05$). The ESRD patients had a lower BMI compared to controls ($p<0.05$). The systolic blood pressure and diastolic blood pressure, ($p<0.05$) urea and creatinine ($p<0.001$) were significantly higher in ESRD patients compared to controls.

Variables	Group I (n=15)	Group II (n=15)	Group III (n=15)	Group IV (n=15)	ANOVA p-value
Age (years)	50.8±11.59	45.73±8.32	51.4±11.3	45.73±9.01	0.2505#
BMI (kg/m^2)	21.84±2.35	20.37±2.84	23.25±0.82	22.31±1.59	0.0032*
Mean SBP (mmHg)	142.7±15.34	136.7±18.4	128.7±7.43	124.7±6.40	0.0015*
Mean DBP (mmHg)	86±5.07	79.33±8.84	82.67±5.94	80±3.78	0.019*
Urea (mg/dL)	92.13±34.9	82.27±23.6	32±5.38	30.93±6.09	<0.000**
Creatinine (mg/dL)	7.77±3.21	6.86±1.74	0.75±0.18	0.69±0.15	<0.000**

[Table/Fig-1]: Demographic characteristics of the study groups (Mean±SD).
Group I: Male cases; Group II: Female cases; Group III: Male controls; Group IV: Female controls
*: p<0.05-significant; #:p>0.05-not significant; **:p<0.001- highly significant.
BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure

Chronic glomerulonephritis was the major cause of CKD in the study group constituting 30% of the cases. Hypertensive nephrosclerosis and contracted kidneys (as seen on an Ultrasonogram) due to unknown aetiology constituted 17% in the study group.

Lipid profile: The close scrutiny of lipid profile in the ESRD cases proves that after renal dialysis the number of subjects in the desirable group (as per NCEP ATP III guidelines) [19] has decreased compared to the number of subjects in the desirable group before dialysis [Table/Fig-2].

Parameter	Grading	Normal values (mg/dL)	Ia+IIa (n)	Ib+IIb (n)	III+IV (n)
TC	Desirable	<200	27	18	26
	Borderline	200-239	2	7	4
	High	>240	1	4	0
HDL-C	Low	<40	24	21	11
	High	>60	0	1	0
LDL-C	Optimum	<100	25	21	22
	Near Optimum	100-129	4	8	7
	Borderline High	130-159	1	0	0
	High	160-189	0	1	0
	Very High	>190	0	0	1
TG	Normal	150	23	26	26
	High	150-199	4	0	4

[Table/Fig-2]: Grading of Lipid Profile in cases versus controls according to NCEP ATP III guidelines [19].
Groups: Ia: Males before RD; Ib: Males after RD; II a: Females before RD; IIb: Females after RD; III: Male controls; IV: Female controls.
Ia+IIa: All cases before dialysis, Ib+IIb: All cases after dialysis, III+IV: All controls.
TC: Total cholesterol; HDL-C: High density lipoprotein cholesterol; LDL-C: Low density lipoprotein cholesterol; TG: Triglycerides

There was a significant increase ($p<0.0001$) in TC, LDL-C, HDL-C values after RD compared to the values before RD. There was a decreasing trend noted ($p>0.05$) in TG and VLDL after RD compared to the values before RD [Table/Fig-3].

The mean values of blood urea and serum creatinine decreased significantly post-dialysis indicating effective attenuation of uremic toxicity in these patients.

Parameter (mg/dL)	Total cases			Male cases			Female cases		
	Ia+IIa (n=30)	Ib+IIb (n=30)	ANOVA p-value	Ia (n=15)	Ib (n=15)	ANOVA p-value	IIa (n=15)	IIb (n=15)	ANOVA p-value
TC	151.4±38.92	183±57.18	<0.000**	140.1±40.08	160.6±53.05	0.001*	162.8±35.43	205.5±53.68	0.001*
HDL-C	31.9±9.74	35.57±12.49	0.001*	31.53±10.98	34.2±13.99	0.035#	32.27±8.69	36.93±11.11	0.018*
LDL-C[a]	75.3±24.66	84.23±29.74	0.000**	65.93±21.89	72.47±26.61	0.029#	84.67±24.33	96±28.77	0.003*
TG	123.6±50.04	121.4±49.26	0.708#	117.1±46.31	114.5±47.38	0.753#	130.1±54.34	128.3±51.76	0.838#
VLDL	24.73±10.01	24.29±9.85	0.708#	23.43±9.26	22.91±9.48	0.753#	26.03±10.87	25.67±10.35	0.838#
LDL-C[c]	94.81±33.05	123.2±46.08	<0.000**	85.11±34.21	103.35±43.4	0.001*	104.5±29.84	142.9±41.08	0.001*

[Table/Fig-3]: Lipid profile (Mean±SD) before and after haemodialysis in the ESRD cases.
Groups: Ia: Males before RD; Ib: Males after RD; II a: Females before RD; IIb: Females after RD; III: Male controls; IV: Female controls. Ia+IIa: All cases before dialysis, Ib+IIb: All cases after dialysis
[c]: Calculated LDL-C; [a]: Automated LDL-C; *: p<0.05-significant; #: p>0.05-not significant; **: p<0.001- highly significant
TC: Total cholesterol; HDL-C: High density lipoprotein cholesterol; LDL-C: Low density lipoprotein cholesterol; TG: Triglycerides

Parameter (mg/dL)	Total cases versus Total controls			Male cases versus Male controls			Female cases versus Female controls		
	Ia+IIa (n=30)	III+IV (n=30)	ANOVA p-value	Ia (n=15)	III (n=15)	ANOVA p-value	IIa (n=15)	IV (n=15)	ANOVA p-value
TC	151.4±38.92	181.3±24.01	0.111#	140.1±40.08	178.9±30.57	0.006*	162.8±35.43	183.6±15.6	0.046*
HDL-C	31.9±9.74	41.53±3.60	0.841#	31.53±10.98	41.87±3.23	0.002*	32.27±8.69	41.2±4.02	0.001*
LDL-C[a]	75.3±24.66	95.3±11.15	0.035*	65.93±21.89	97.53±7.15	<0.000**	84.67±24.33	93.07±14.00	0.256#
TG	123.6±50.04	129.9±25.52	0.486#	117.1±46.31	126.3±23.51	0.498#	130.1±54.34	133.4±27.74	0.837#
VLDL	24.73±10.01	25.97±5.10	0.486#	23.43±9.26	25.27±4.70	0.498#	26.03±10.87	26.67±5.55	0.837#
LDL-C[c]	94.81±33.05	113.8±22.67	0.109#	85.11±34.21	111.9±28.2	0.027*	104.5±29.84	115.7±16.17	0.211#

[Table/Fig-4]: Comparison of Lipid profile (Mean±SD) among the subgroups.
Groups: Ia: Males before RD; Ib: Males after RD; II a: Females before RD; IIb: Females after RD; III: Male controls; IV: Female controls. Ia+IIa: All cases before dialysis, III+IV: All controls
[c]: Calculated LDL-C; [a]: Automated LDL-C; *: p<0.05-significant; #: p>0.05-not significant; **: p<0.001-highly significant
TC: Total cholesterol; HDL-C: High density lipoprotein cholesterol; LDL-C: Low density lipoprotein cholesterol; TG: Triglycerides

There were significantly higher LDL-C values in female ESRD cases compared to male ESRD cases both before and after RD (p<0.05).

In male ESRD cases the values of TC, LDL-C, HDL-C were significantly (p<0.01) lower compared to male healthy controls. There was no significant difference in TG, VLDL between them [Table/Fig-4].

In female ESRD cases the values of TC (p<0.05), HDL-C (p<0.01) were significantly lower compared to healthy controls. There was no significant difference in LDL-C, TG, and VLDL between them [Table/Fig-4].

Fractionation of lipoproteins [Table/Fig-5]: There was a significant difference in cases compared to controls in the mean values of the percentage of Chylomicrons (CM) (8.97±5.91 versus 2.03±1.61) (p<0.001); LDL-C (37.74±5.80 versus 27.64±20.79) (p<0.001); HDL-C (15.02±10.34 versus 23.68±5.74) (p<0.01). Compared to the pre-dialysis values the post-dialysis values of HDL-C did not differ significantly (p>0.05). There was no significant difference in the mean values of VLDL percentage in cases compared to controls (p>0.05) (27.18±22.5 versus 24.18±10.47). There was significant difference in the mean values of Lp (a) percentage in cases compared to controls. Lp (a) was significantly higher (p<0.05) in all stages of CKD compared to controls (21.20±17.46 versus 12.38±10.63). The α-lipoproteins were significantly lower in cases compared to controls.

Variable	Cases pre RD	Cases post RD	Controls
α	15.02±10.34	13.11±10.09#	23.68±5.74*
Pre-β	27.18±22.51	24.96±21.65#	24.18±10.47
β	32.98±19.92	27.64±20.79#	37.74±5.80
Lp (a)	21.20±17.46	22.20±12.28#	12.38±10.63*

[Table/Fig-5]: Lipoprotein electrophoresis.
α: Alpha lipoprotein- HDL-C; Pre-β: Pre-beta lipoprotein-VLDL; β: beta lipoprotein-LDL-C; RD: Renal dialysis; *: p<0.05; #: p>0.05 (in cases pre RD compared to post RD and controls)

DISCUSSION

In the present study, the age group of ESRD patients on MRD was 48.27±10.24 years; this was in accordance with other studies in developing countries [2,20]. The reason for the disparity compared to studies in western population where the mean age is (>65 years) might be due to increased incidence of hypertension and diabetes mellitus (which are the common risk factors for CKD) along with other factors like smoking, genetic, socio-cultural factors, therapeutic modalities and the pattern of diseases causing CKD [21].

In the present study, the ESRD patients had a significant lower BMI compared to the controls (21.11±11.67 versus 22.77±1.30 kg/m^2); (p<0.05). These results were in accordance with other studies where significant lower (BMI) in RD patients compared to controls was identified [22,23].

The lower BMI noted in the present study can be attributed to a condition identified as Malnutrition-Inflammation Complex Syndrome (MICS) seen in most of the dialysis patients, which is a combination of protein energy malnutrition and inflammation. This syndrome leads to low BMI, hypocholesterolemia, hypocreatininaemia and hyperhomocystinemia, increasing the risk of death [22].

The mean values of blood urea (87.20±29.68 versus 31±5.38 mg/dL) and serum creatinine (7.32±2.58 versus 0.72±0.17 mg/dL) in the cases were significantly higher (p<0.0001) when compared to controls.. The mean GFR in this study group was 10.78±4.63 mL/minutes/1.73 m^2 which was significantly low compared to controls (p<0.001).

Triglycerides: The mean value of triglycerides in cases before RD was 123.63±50.05 mg/dL and decreased after dialysis 121.43±49 mg/dL and the TG in controls was 129.87±25.52 mg/dL. The mean differences were not significantly different in cases compared to controls (p>0.05). Hypertriglyceridaemia was observed in only 8 out of 30 RD cases (26%).

Though these results were different from the classical picture of higher degree of hypertriglyceridaemia noted in the western CKD population, they were in concordance with other Indian studies [11,17,20,24].

RD patients have lower concentrations of TG compared to the non-dialysis CKD patients possibly representing an attenuation of the dyslipidaemia following haemodialysis which causes improved triglyceride removal by increasing post heparin lipolytic activity and decreasing peripheral resistance to insulin after initiation of dialysis.

Diet plays a significant role in the genesis of hypertriglyceridaemia. Indian CKD populations follow a diet which is low in calories and high in polyunsaturated fatty acids [17,22].

Total cholesterol: The mean values of TC increased significantly after dialysis compared to the values before dialysis (151.43±38.92 mg/dl versus 183.03±57.18 mg/dL); (p<0.0001). The mean of TC in controls was 129.87±25.52 mg/dL. The mean differences were significantly different between cases and controls (p<0.001). These results are in accordance to other studies [11,16,17].

Seres DS et al., reported an increase in TC post RD and that was attributed to an increase mainly due to HDL-C (27%) and 6% increase is due to LDL-C [17]. Blankestijn PJ et al., reported an increase in TC post RD which was mainly due to a significant increase in LDL-C [16].

In the present study, the TC values increased significantly and it attributed in large to the increase in non-HDL-C (obtained by subtracting HDL-C from TC) which was a significant and independent marker of CV mortality that integrated atherogenic potentials of VLDL, Intermediate Density Lipoprotein (IDL) and LDL-C [25].

HDL-cholesterol: In agreement with Seres DS et al., the HDL-C values in the present study increased significantly after RD (31.9±9.73 versus 35.56±12.48 mg/dL) (p<0.001) compared to the values before RD [17]. The mean HDL-C value among controls is 41.53±3.60 mg/dL. The mean value of HDL-C among cases was found to be significantly lower compared to controls (p<0.0001).

Renal dialysis procedure significantly lowers, but does not normalize the pro-inflammatory activity of LDL-C, and enhances the ability of HDL-C to suppress LDL-C inflammatory activity [12].

LDL cholesterol: The mean values of LDL-C increased significantly after RD (75.3±24.65 versus 84.23±29.74 mg/dL); (p<0.001). LDL-C in patients was significantly lower than the LDL-C in controls (75.3±24.65 versus 95.3±11.15 mg/dL) (p<0.05). These results are consistent with Seres DS et al., [17]. In this study, a highly significant difference (p<0.0001) was noted between the LDL-C values obtained by Friedewald formula versus the automated LDL-C values (values pre RD: 94.81±33.05 mg/dL versus 75.30±24.66), (values post RD: 123.2±46.08 mg/dL versus 84.23±29.74), (controls: 113.67±22.67 versus 95.30±11.15 mg/dL).

This ascertains the fact that use of Friedewald formula to calculate LDL-C is inappropriate in the setting of CKD.

VLDL cholesterol: In the present study, the mean values of VLDL were substantially higher (24.73±10.01 vs. 4.86±1.97 mg/dL) compared to controls (p<0.0001). The VLDL values before and after RD (24.73±10.01 vs. 24.29±9.85 mg/dL) did not differ significantly (p>0.05). This is in accordance with studies of Zulbeari L et al., [22]. VLDL decreases following dialysis with high-flux PS (polysulphone) membranes. The contrast findings in the present study might be due to use of low flux PS membranes. Hence, choice of dialyser membrane has a substantial effect in improving the quality of life in RD patients by reducing the risk of atherosclerosis, cardiovascular morbidity and mortality [23].

Gender variation: There was no significant gender variation noted among controls. In agreement with Aravind KR et al., the levels of non-fasting TC, LDL after RD were significantly higher in females compared to male patients (205.5±53.69 versus 160.6±53.05 mg/dL; 96±28.77 versus 72.47±26.6 mg/dL respectively) [26]. The reason behind this difference could be due to higher BMI and oestrogen imbalance noted with advancing age in females which results in increased stress which in turn increases the deposition of lipid molecules in the adipose tissue [26].

Fractionation of lipoproteins: Conventional lipoprotein measurements fail to capture relevant changes in lipoproteins as these lipoproteins get altered qualitatively as well as quantitatively. The qualitative changes in the form of LDL-C particle size and density can be picked up by performing a fractionation of lipoproteins on agarose gel electrophoresis. The qualitative changes that are expected to occur are presence of small dense LDL-C (sd-LDL), acute phase HDL-C, presence of oxidised LDL-C, IDL and Increased Lp(a).

Chylomicron: These results are comparable and correlate well to the values obtained by direct enzymatic measurements performed. The findings in the present study are in good agreement with Ravichandran R et al., [20].

LDL-C: A disparity (decreasing trend of LDL-C following RD was noted) has been identified in the post dialysis LDL-C values obtained by electrophoresis and the post dialysis automated LDL-C values obtained by direct enzymatic essays.

These contradictory findings can be attributed to the fact that the LDL-C changes in size and density ascertained by Vaziri ND et al., who proved that LDL-C changes qualitatively; by demonstrating an enhanced susceptibility of LDL-C from RD patients to ex-vivo oxidation [12] and transforms into sd-LDL which might also have a different charge compared to LDL-C. It is a well known fact that the main principle for separation of molecules by agarose gel electrophoresis is the difference in size, density and charge. Hence, the migration of molecules differs based on these characteristics.

VLDL: These results are in good correlation with the results of Blankestijn PJ et al., Seres DS et al., and Ravichandran R et al., [16,17,20].

Lp (a): In RD patients this abnormality is even more predominant. The results in the present study are in accordance with that of Ponnudhali D and Nagarajan P [24].

HDL-C: Vaziri ND et al., have elucidated the fact that the quality of HDL-C in RD patients is impaired resulting in a pro-inflammatory acute phase HDL-C [12]. This HDL-C is altered qualitatively and compositionally and hence might have a different charge and migration from that of the normal HDL-C which explains the disparity noted in electrophoresis compared to the enzymatic assays in present study.

Strength

LDL-C was estimated using direct automated enzymatic studies unlike all other studies that relied on Friedewald formula. The lipoproteins were estimated both before and after renal dialysis to assess the effect of the RD procedure. Lipoprotein electrophoresis was done to assess the qualitative changes in the lipoproteins.

LIMITATION

The lipoprotein fractions obtained by electrophoresis could not be eluted or separated for further down-stream analysis by specialised techniques.

CONCLUSION

The major outcome of this study was the elucidation of an elevation in atherogenic lipoproteins post renal dialysis which might significantly contribute to the high rate of cardiovascular morbidity and mortality in these patients. Except for the significant attenuation of TG, VLDL post dialysis there was no noted beneficial effect on dyslipidaemia. There was a significant increase in TC, LDL-C and Lp (a) suggesting the heightened exposure of these patients to atherogenic particles. There was a significant gender variation in LDL-C and TC with higher values noted among females. Further randomized controlled studies have to be done to confirm whether early detection and treatment of this dyslipidaemia is quite promising in the prevention of CVD and adverse clinical outcomes in patients on MRD.

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