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COMPARATIVE STUDY OF LIPOPROTEIN (a) AND LIPID PROFILE IN CHRONIC KIDNEY DISEASE PATIENTS WITH HEMODIALYSIS AND WITHOUT HEMODIALYSIS

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ABSTRACT: AIM: This study was under taken to compare the lipid profile pattern including Lipoprotein (a) or Lp (a) levels , Total cholesterol , Triglycerides, High density lipoprotein , Low density lipoprotein and other biochemical parameters like Bun, Creatinine, Fasting Plasma Glucose and Post prandial Plasma Glucose in Chronic kidney disease patients with Hemodialysis and without Hemodialysis. **INTRODUCTION:** Chronic kidney disease (CKD) is associated with early development of atherosclerosis and increased risk of cardiovascular morbidity and mortality which is the leading cause of death among these patients. Alterations in lipid metabolism resulting in abnormal lipoprotein composition and concentration (dyslipidemia) have been noticed in chronic renal insufficiency. Dyslipidemia is a primary risk factor in the development of a number of disease multitudes ranging from atherosclerosis to stroke. Dyslipidemia may be worsened by dialysis, especially continuous ambulatory peritoneal dialysis (CAPD). Dyslipidemia among HD patients negatively impacts cardiovascular profiles, which in turn influence the frequency and/or duration of hospitalizations. **METHODS AND MATERIALS:** The study was conducted among subjects attending Nephrology Department, Sri Ramachandra Medical College and Research Institute Chennai, Tamil Nadu as inpatients. The study was conducted over a period of three months. The study includes 40 CKD patients in the age group of 40 to 60 years. They were divided into 2 groups. Group 1 consisted of subjects with chronic kidney disease with hemodialysis. Group 2 consisted of subjects with chronic kidney disease without hemodialysis. Each group had 10 males and 10 females. **RESULTS:** The mean and standard deviation of biochemical parameters of the two groups was calculated. The biochemical parameters include Bun, Creatinine, Fasting Plasma Glucose, Post prandial Plasma Glucose, Lipoprotein (a), Total Cholesterol, Triglycerides, High density lipoprotein and Low density lipoprotein. Data evaluation was done using SPSS programme. The results were expressed as Mean with standard deviation. The P value < 0.05 was considered significant. **CONCLUSION:** In this study, there is no significant difference in all the biochemical parameters between chronic kidney disease patients with Hemodialysis and without Hemodialysis.

KEYWORDS: Lipoprotein (a), Hemodialysis, Chronic kidney disease, Low density lipoprotein, High density lipoprotein.

INTRODUCTION: Chronic kidney disease (CKD) is associated with early development of atherosclerosis and increased risk of cardiovascular morbidity and mortality which is the leading cause of death among these patients. Alterations in lipid metabolism resulting in abnormal lipoprotein composition and concentration (dyslipidemia) have been noticed in chronic renal insufficiency.

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Dyslipidemia is a primary risk factor in the development of a number of disease multitudes ranging from atherosclerosis to stroke.^{1,2} The risk is substantially elevated in patients with end-stage renal disease (ESRD) (such patients show various abnormalities in plasma-lipids and lipoproteins that are called uremic dyslipidemia.^{3,4}

Disturbances of lipid transport and metabolism are common complications of chronic renal failure, regardless of the cause of renal disease, which may persist or deteriorate during renal replacement therapy.^{4,6} Patients with chronic kidney disease (CKD) usually have an elevated ratio of low-density lipoprotein (LDL) cholesterol to high-density lipoprotein (HDL) cholesterol.^{6,10}

Dyslipidemia may be worsened by dialysis, especially continuous ambulatory peritoneal dialysis (CAPD). Dyslipidemia among HD patients negatively impacts cardiovascular profiles, which in turn influence the frequency and/or duration of hospitalizations.¹¹

AIM: This study was under taken to compare the lipid profile pattern including Lipoprotein (a) or Lp (a) levels , Total cholesterol , Triglycerides, High density lipoprotein ,Low density lipoprotein and other biochemical parameters like Bun, Creatinine, Fasting Plasma Glucose and Post prandial Plasma Glucose in Chronic kidney disease patients with Hemodialysis and without Hemodialysis.

MATERIALS AND METHODS: The study was conducted among subjects attending Nephrology Department, Sri Ramachandra Medical College and Research Institute Chennai, Tamil Nadu, as inpatients. The study period was from January to March 2005, for a period of three months. The study includes 40 CKD patients in the age group of 40 to 60 years. The CKD patients were classified based on GFR as per the NKF/DOQI guidelines.

They were divided into 2 groups. Group 1 consisted of subjects with chronic kidney disease on hemodialysis. Group 2 consisted of subjects with chronic kidney disease without hemodialysis. Each group had 10 males and 10 females. Group 1 included 20 patients with CKD stage V (GFR < 15ml /min / 1.73 m²) on hemodialysis. Group 2 included 20 patients in CKD stage I to IV (GFR of 15 to 59 ml /min / 1.73 m²).

Patients with Diabetes, obesity, liver disease and systemic illness were excluded from the study.

STUDY DESIGN: As it is a comparative cross-sectional study, all patients underwent a full medical history that included age, family history of diabetes, hypertension, coronary artery disease, Chronic kidney disease, duration of Chronic kidney disease, type of dialysis, smoking and alcohol, Drug history and treatment history for any other disease was collected through a standard questionnaire.

Blood samples were collected after 12 hours of fasting in the vacutainers for estimation of glucose, lipoprotein (a), lipid profile, Bun and creatinine. Blood samples were collected in the morning after 12 hours of overnight fasting. The samples were separated by centrifugation at 2400 rpm.

BIOCHEMICAL METHODS: Lipoprotein (a) levels was determined by Agglutination reaction using Latex daiichi kit in Konelab 60 autoanalyser .Serum total cholesterol and triglycerides and HDL were estimated using Randox kits by enzymatic endpoint analysis.

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Serum LDL-cholesterol levels was calculated using Freidwald's formula: $\text{LDL cholesterol} = \text{Total cholesterol} - \text{HDL cholesterol} - \text{TGL}/5$. GFR was calculated using Modification of Diet in Renal Disease (MDRD) formula.

Glucose was analyzed by enzymatic end point method in konelab 60 automated systems using commercially available kit by Accurex. Bun and creatinine was analyzed by Endpoint method in Kone lab 60 automated systems using commercially available kit by Trace.

STATISTICAL ANALYSIS: Data evaluation was done using SPSS programme. The results were expressed as Mean with standard deviation. The P value < 0.05 was considered significant.

RESULTS: A total number of 40 subjects were recruited for the study .Among them 20 were CKD patients with Hemodialysis and 20 were CKD patients without Hemodialysis. Data evaluation was done using SPSS programme.

The mean and standard deviation of all the biochemical parameters were calculated and their results shown in Table 1. The P value was used to compare the different groups. The P value < 0.05 was considered significant. As per Table 1, there was no significant difference in Bun (44.3 ± 15.3) and creatinine (3.9 ± 1.1) in CKD patients with Hemodialysis compared to Bun (44.5 ± 11.6) and creatinine (4.2 ± 1.1) in CKD patients without hemodialysis.

There was no significant difference in FPG (118.8 ± 59.3) and PPPG (158.4 ± 71.9) in CKD patients with Hemodialysis compared to FPG (115.0 ± 25.9) and PPPG (160.8 ± 48.0) in CKD patients without hemodialysis. There was no significant difference in Total cholesterol (232.0 ± 24.9), Triglycerides (180.1 ± 7.9), HDL(29.7 ± 4.7), LDL(177.8 ± 11.4) and Lipoprotein (a) (74.3 ± 7.0) in CKD patients with Hemodialysis compared to Total cholesterol (224.8 ± 27.7), Triglycerides (180.4 ± 7.3), HDL(30.8 ± 4.1), LDL(178.8 ± 10.2) and Lipoprotein (a) (72.0 ± 6.4) in CKD patients without hemodialysis.

Parameters	Hemodialysis	Without Hemodialysis	P Value	Significant / Not-Significant
Bun	44.3 ± 15.3	44.5 ± 11.6	0.972	Not Significant
Creatinine	3.9 ± 1.1	4.2 ± 1.1	0.392	Not Significant
FPG	118.8 ± 59.3	115.0 ± 25.9	0.798	Not Significant
PPPG	158.4 ± 71.9	160.8 ± 48.0	0.905	Not Significant
Cholesterol	232.0 ± 24.9	224.8 ± 27.7	0.391	Not Significant
Triglycerides	180.1 ± 7.9	180.4 ± 7.3	0.903	Not Significant
HDL	29.7 ± 4.7	30.8 ± 4.1	0.432	Not Significant
LDL	177.8 ± 11.4	178.8 ± 10.2	0.781	Not Significant
Lipoprotein (a)	74.3 ± 7.0	72.0 ± 6.4	0.292	Not Significant

Table 1: Comparison of biochemical parameters between Chronic Kidney Disease patients with and without Hemodialysis

Biochemical Parameters of CKD patients with Hemodialysis and without hemodialysis.

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DISCUSSIONS: As per table 1, there is no significant difference in all the biochemical parameters between chronic kidney disease patients with Hemodialysis compared to Chronic kidney disease patients without Hemodialysis.

In this study, only a small number of Chronic kidney disease patients were included, out of which 20 were with Hemodialysis and the rest without Hemodialysis.

According to Gruber et al, the type of therapy for renal failure does not seem to influence the elevation in Lipoprotein (a) concentration.¹²

Lp (a) is an LDL-like particle having an apolipoprotein (a) which is attached to apolipoprotein B-100 by a disulfide linkage. It is synthesized in the liver, but its sites of catabolism are not clear. The increase in Lp (a) levels in CKD patients could be due to its increased synthesis by the liver or due to its decreased catabolism in kidneys.¹³

A significant decrease in Lp (a) concentrations between the ascending aorta and renal vein¹⁴ and the identification of apo (a) fragments in urine¹⁵ indicate kidneys' active participation in the degradation of Lp (a). Recent studies have also shown a strong genetic basis for the increase in serum Lp(a) levels in chronic kidney disease. In chronic kidney disease, individuals with low molecular weight (LMW) apo(a) isoforms have been shown to have high serum Lp (a) levels and those with high molecular weight HMW apo (a) isoforms have low levels.

Sechi and coworkers studied 160 patients with early impairment of renal function. They found an increase in plasma Lp (a) levels in comparison with healthy controls. In another study, Sechi and colleagues evaluated Lp (a) concentrations and apo (a) isoforms in a group of patients with moderate renal failure. They found an increased plasma Lp (a) concentrations in patients and a similar apo (a) isoform distribution between patients with renal disease and controls.¹⁵

A consistent moderate elevation in plasma lipoprotein (a) concentrations has been observed in large case control studies in hemodialysis patients. Increased plasma free apolipoprotein (a) fragments have been observed in hemodialysis patients, but appear to account for only a small proportion of increased lipoprotein (a) observed in such patients. The frequency of apolipoprotein (a) isoforms in hemodialysis patients is comparable to that in healthy controls.

An increase in lipoprotein (a) plasma levels has been identified specifically in hemodialysis patients exhibiting high molecular weight apolipoprotein (a) isoforms, but this has not been

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confirmed by others in different ethnic groups. It was found that due to unknown reasons, inflammation affected only high molecular weight apolipoprotein (a) isoforms in hemodialysis patients.¹⁶

In hemodialysis patients, by in vivo turnover studies using stable isotope techniques, Frischmann KE et al¹⁷ have elucidated that the fractional catabolic rate of the Apolipoprotein (a) was significantly reduced resulting in its longer residence time in plasma (9 days) compared to the controls (4.4 days). This decreased clearance could be the result of loss in kidney function, in hemodialysis patients. Malnutrition and inflammation have also been associated with high plasma Lp (a) levels in hemodialysis patients.¹⁸

However, it still remains to be clarified through which pathophysiological mechanisms Lp (a) might contribute to the progression of glomerular disease. The underlying mechanisms responsible for the elevation of Lp (a) plasma concentrations in patients with renal insufficiency are not known. Although plasma levels of Lp (a) in healthy individuals are predominantly genetically determined, the alterations seen in conjunction with renal disorders such as advanced renal insufficiency, i.e. ESRD, and nephrotic syndrome are not primarily due to genetic factors.¹²

Plasma triglycerides are predominantly present in 2 types of lipoproteins namely the chylomicrons and VLDL. Hypertriglyceridemia may be due to high production rate of these lipoproteins and a low catabolic rate¹⁹. Renal insufficiency can cause insulin resistance which in turn promotes hepatic VLDL production and hence elevated triglyceride levels.²⁰ But the predominant mechanism for increased triglyceride levels in pre dialysis patients is that of delayed catabolism and hence impaired clearance.

The reduced catabolism is due to the decreased activity of 2 endothelium- associated lipoprotein lipases -hepatic lipase and lipoprotein lipase (LPL). There may be down regulation of the LPL and hepatic lipase enzyme gene expressions, contributed in part by secondary hyperparathyroidism.²¹ The decrease in lipoprotein lipase activity may be due to the increase in plasma apo C-III levels resulting in a decrease in apo C-II/ apo C-III ratio.

Apo C-II is an activator of lipoprotein lipase while apo C-III is a potent inhibitor of LPL and so the increase in apo C-III levels results in inactivation of lipoprotein lipase resulting in reduced triglyceride lipolysis and hence increased triglyceride levels. Another inhibitor of lipoprotein lipase has been identified as pre β -HDL, whose concentration is found to be elevated in CKD patients.²²

Though many studies have shown hypertriglyceridemia in hemodialysis patients, we did not observe any significant change in this group of patients. This may either be due to the patients receiving carnitine injections, multivitamin supplementations or HMG-CoA inhibitors.^{23,24} These factors could have marginally prevented the rise of serum triglyceride levels in hemodialysis patients.

Many authors have noticed that in hemodialysis patients low serum cholesterol is associated with increased mortality.^{25,26} It appears that many dialysis patients have a condition identified as malnutrition inflammation complex syndrome (MICS), which is a combination of protein-energy malnutrition and inflammation and is related to poor dialysis outcomes²⁷. This MICS leads to a low body mass index, hypocholesterolemia, hypocreatinemia, and hypohomocysteinemia, increasing the risk of death.²⁹

The hypocholesterolemia is a strong mortality risk factor in dialysis patients and a marker of poor nutritional status.²⁸ The mechanism by which systemic inflammation and malnutrition may explain this hypocholesterolemia is unclear.

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A cytokine mediated acute-phase reaction to acute or chronic inflammation may partially account for the hypocholesterolemia (cholesterol- negative acute phase reactant), in dialysis patients by increasing catabolism and decreasing appetite.³⁰

The lowering of LDL in CKD may be due to the same above said reason inflammation/ malnutrition or due to reduced production of LDL resulting in its near normal levels.³¹ The inflammation may change the lipoprotein structure and function by oxidatively modifying low density lipoprotein.

In CKD patients there is a relative increase in IDL (intermediate density lipoproteins) and small dense LDL (sdLDL) particles which undergo further modifications like glycation, oxidation and carbamylation, making them highly atherogenic.³²

These modified lipoproteins are in turn taken up by the scavenger receptors on macrophages and vascular smooth muscle cells, which are increased in uremia, favoring the development of atherosclerotic plaques. Though many studies have shown a reduction in HDL level with progression of renal disease, we could not find any changes.³³

STRENGTH AND LIMITATION OF THE STUDY: The main strength of this study is to reduce the early development of atherosclerosis and increased risk of cardiovascular morbidity and mortality which is the leading cause of death among CKD patients with and without hemodialysis. The number of patients recruited for the study was small which the only limitation of the study.

CONCLUSION: Cardiovascular disease (CVD) is a major cause of mortality in patients with mild to moderate chronic kidney disease and end stage renal disease (ESRD). In our study there was no significant difference in Lipoprotein (a) and Lipid profile between Chronic kidney disease patients on Hemodialysis compared to chronic kidney disease patients without hemodialysis.

As a smaller group was included in this study, this study can be further extended to a larger group to confirm whether early detection and treatment (diet /drug therapy) of this dyslipidemia is quite promising, in the prevention of adverse clinical outcomes in CKD patients with hemodialysis and CKD patients without hemodialysis. Further research can also be done to see the correlation of Lipoprotein (a) and the rate of progression of renal disease and the use of apolipoprotein (a) isoforms as a predictor of cardiovascular disease in hemodialysis patients.

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