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Original Research Article

Study of Lipid Profile and Lipoprotein (A) in Type 2 Diabetic CKD Patients with or without Dialysis

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Abstract

Introduction: This study was conducted to evaluate the lipid profile and lipoprotein (a) level in type 2 diabetic CKD patients with or without dialysis. **Methodology:** A comparative study conducted on 150 chronic kidney patients of age group 30 to 80 years. Group A were healthy control patients (n=50), group B were CKD patients without hemodialysis (n=50) and group C were CKD patients on hemodialysis (n=50). **Results:** The mean age of patients were 52.2±15.4 years, 49.2±14.1 years and 54.8±5.9 years in group- A, B, C, respectively. There was statistically significant difference in lipid profile of patients having CKD with healthy controls, but we do not find any statistically significant difference in lipid profile of CKD patients with and without dialysis. **Conclusion:** Lipid profile was not significantly affected by dialysis or presence of diabetes among CKD patients.

Keywords: Chronic Kidney Disease, Diabetes, Dialysis, Lipid Profile.

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Introduction

Chronic kidney disease (CKD) is one of today's leading public health problems, with increasing frequency and prevalence[1]. Patients with CKD, particularly those with end-stage renal disease (ESRD) who are treated with hemodialysis (HD) or peritoneal dialysis (PD) or those treated with renal transplantation, are at increased risk of developing cardio vascular disease (CVD). Dyslipidemia is a common complication of CKD and lipoprotein metabolism alteration and is associated

with the decline in GFR; hence, lipid profile depends on the level of kidney function and the degree of proteinuria[2].

CKD is associated with dyslipidemia associating hypertriglyceridemia, elevated LDL cholesterol, an accumulation of Apolipoprotein b (Apo b) containing lipoproteins, increased concentrations of lipoprotein (a) particles, and low HDL levels[3]. In CKD, HDL metabolism is impaired[4].

Kidney disease is associated with both an increased risk of vascular disease and an

acquired elevation in LP (a) levels[5]. Lipoprotein (a) is synthesized in the liver and is composed of a single LDL particle linked to a highly polymorphic Apo (a) protein (1). Plasma LP (a) levels vary widely between individuals, from 0 to >200 mg/dl[6]. Present study was conducted to study the lipid profile and lipoprotein (a) level in type 2 diabetic CKD patients with or without dialysis.

Methodology:

This was a comparative study conducted in chronic kidney patients of age group 30 to 80 years. informed consent taken from all candidates. Total 150 candidates were divided into three groups. Group A was a healthy control patient (n=50), group B was CKD patients without hemodialysis (n=50) and group C was CKD patients on hemodialysis (n=50).

After overnight fasting of 12 hours, venous blood is collected for lipid profile and renal function tests. Along with them complete blood count, Liver function tests, urine examination, USG were collected. The serum total cholesterol (TC), high density lipoprotein Cholesterol (HDL-C), triglycerides (TGs) and very

low - density lipoprotein (VLDL) are measured using commercially available Randox auto analyzer and low-density Cholesterol lipoprotein. (LDL-C) calculated from Friedewald's Formula $(LDL=TC-HDL-TG\setminus 5)[5].$ Mean difference between two independent groups was analyzed using the student ttest. This was analyzed using Epi Infotm 7.1.5 and SPSS for windows version 16.0. Calculation of eGFR6 is done by equation from the modification of diet in renal disease study (MDRD). -1.1540.203 Estimated GFR (ml/min per 1.73 m2) = $1.86 \times (SCr) \times (age)$.

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Results:

Total 150 cases were taken and divided into three groups. The mean age of patients was 52.2±15.4 years, 49.2±14.1 years and 54.8±5.9 years in group - A, B, C, respectively. There were 21 males and 29 females in group - A, 39 males and 11 females in group - B and 33 males and 17 females in group - C. The mean BMI of all patients in all the three groups are 22.9±2.4. 27.4 ± 3.4 and 20.7 ± 2.9 respectively. There were17 diabetic patients in group – B and 19 patients were diabetic in group-C (Table:1).

Table:1 Base line characteristics

| Parameter | Group-A (50) | Group-B (50) | Group-C (50) |
|--------------------------|--------------|--------------|--------------|
| Age | 52.2±15.4 | 49.2±14.1 | 54.8±5.9 |
| Sex (M/F) | 21/29 | 39/11 | 33/17 |
| BMI | 22.9±2.4 | 27.4±3.4 | 20.7±2.9 |
| No. of diabetic patients | 0 | 17 | 19 |

Table: 2 Lipid Profile in Healthy controls and CKD Patients

| Lipid Profile | Healthy controls (50) | CKD patients (100) | P-value |
|-------------------|-----------------------|--------------------|---------|
| Total-cholesterol | 189.1±44.43 | 215.7±46.2 | 0.0001 |
| LDL | 122.9±45.79 | 141.7±44.3 | 0.0001 |
| HDL | 36.5±9.2 | 32.6±8.8 | 0.0001 |
| VLDL | 29.9±11.13 | 8.9±13.0 | 0.0001 |
| TG | 149.3±57.92 | 193.6±65.2 | 0.0001 |

The mean cholesterol, LDL, VLDL and TG were high in CKD patients as compared to healthy controls. The results

are statistically significant with P-value <0.05 (Table: 2). similarly, mean lipid profile was not statistically significant for

CKD patients who were on dialysis or not on dialysis with p-value > 0.05 (Table: 3, 4). There was also no statistically

significant difference in lipid profile among diabetics and non-diabetics (P-value >0.05).

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Table: 3 Lipid Profile in CKD Patients with and without Dialysis

| Lipid Profile | CKD patients without dialysis | CKD patients with dialysis | P-value |
|--------------------------|-------------------------------|----------------------------|---------|
| Total Cholesterol | 220.2±46.2 | 211.2±46.1 | 0.0987 |
| LDL | 140.8±45.1 | 142.5±44.1 | 0.992 |
| HDL | 31.8±9.5 | 33.5±8.1 | 0.0601 |
| VLDL | 39.3± 13.6 | 38.5±12.5 | 0.807 |
| TG | 196.7±68.2 | 190.7±62.7 | 0.0601 |

Table: 4 Lipid profile in CKD patients with or without diabetics

| Lipid Profile | CKD with diabetics | CKD without diabetics | P-value |
|-------------------|--------------------|-----------------------|---------|
| Total-cholesterol | 217.6±48.9 | 213.1±57.3 | 0.093 |
| LDL | 129.6±50.8 | 141.9±49.6 | 0.059 |
| HDL | 34.6±8.6 | 31.8±12.1 | 0.88 |
| VLDL | 36.9±13.6 | 39.3±12.8 | 0.708 |
| TG | 184.5±68.4 | 190.5±64.5 | 0.509 |

Here, we also calculated lipoprotein-a level and found that it is elevated in CKD patients as compared to healthy controls (Table:5) and also found that lipoprotein

(a) level was not significantly elevated in CKD patients with or without dialysis (Table: 6).

Table: 5 Lipoprotein (a) level in Healthy control and CKD Patients

| | Healthy controls (50) | CKD patients (100) | P-value |
|----------------|-----------------------|--------------------|---------|
| Lipoprotein(a) | 35.1±16.9 | 46.53±16.72 | 0.0001 |

Table: 6 Lipoprotein (a) level in CKD Patients with and without dialysis

| | CKD patients without dialysis | CKD patients with dialysis | P-value |
|----------------|-------------------------------|----------------------------|---------|
| Lipoprotein(a) | 45.5±15.9 | 47.6±17.6 | 0.6701 |

Discussion:

In this study of CKD individuals with or without diabetics, we demonstrate that GFR impairment is indeed associated with an atherogenic lipid profile, primarily through its strong association with elevated plasma LP (a) levels[7].

Here, there is dyslipidaemia and the mean cholesterol, LDL, VLDL and TG was high in CKD Patients as compared to healthy controls. Mikolasevic et. al. also states that CKD patients who were on HD usually have similar lipid profile to those with non-dialysis-dependent CKD. TC and LDL

levels were generally relatively normal, triglyceride levels were elevated, and HDL was low. In these patients, LDL is rarely markedly elevated[2]. We also calculated lipoprotein (a) level and found that it is elevated in CKD patients as compared to healthy controls. T simihodimos et.al. found that HD patients also have increased plasma LP (a), which is Isoform specific. Malnutrition and inflammation are usually present in this group of patients and together with the impaired clearance of Apo (a) may be responsible for these alterations[8]. Patients with CKD usually have hypertriglyceridemia due to an

increased concentration of triglyceride-rich lipoproteins (VLDL, chylomicrons, and their remnants). Hypertriglyceridemia occurs because of both the delayed catabolism and the increased hepatic production of triglyceride-rich lipoproteins. Delayed catabolism is the most prevalent mechanism responsible for an elevated triglyceride-rich lipoprotein concentration in CKD patients and occurs probably because of a decreased activity of hepatic triglyceride lipase and peripheral lipoprotein lipase. Patients with nondialysis-dependent CKD and without nephrotic syndrome have low HDL and high triglycerides and normal or even low TC and LDL cholesterol, but a more atherogenic profile is hidden behind this spectrum. This profile includes increased Apo lipoprotein b (Apo b), lipoprotein (a) intermediate-and very-lowdensity lipoprotein (IDL cholesterol, VLDL cholesterol; "remnant particles"), and small dense LDL particles. Also, in patients with more severe CKD, LDL, and HDL particles are often modified by the oxidative process, which leads to the formation of small lipoproteins and increased formation of oxidized LDL[9-11].

Thus, cardiovascular disease is a major cause of morbidity and mortality in patients with impaired renal function. Dyslipidemia has been established as a well-known traditional risk factor for cardiovascular disease (CVD) in the general population and it is well known that patients with chronic kidney disease (CKD) exhibit significant alterations in lipoprotein review, metabolism. In this pathogenesis and treatment of CKDinduced dyslipidemia discussed. are Studies on lipid abnormalities in predialysis and hemodialysis patients were analyzed. In addition, the results of the studies that tested the effects of the hypolipidemic drugs on cardiovascular morbidity and mortality in patients with CKD are reported[2].

Conclusion:

Cardiovascular disease (CVD) is the leading cause of death in chronic kidney disease (CKD). One of the most important pathophysiological mechanisms for CVD in patients with CKD is the wide spread and possibly accelerated formation atherosclerotic plaques due to hyperlipidemia. Studies showed that the level of oxidized low-density lipoprotein cholesterol increases, and that high-density lipoprotein cholesterol dysfunction occurs kidney function declines inflammation becomes more prevalent. In this study, we aimed to discuss that there is dyslipidemia in CKD patients irrespective of mode of management, but the derangement is much more common and significant in CKD with hemodialysis group and they are at risk of cardiovascular disease. It is better to start lipid lowering drugs which decrease disease progression and dyslipidemia.

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