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Lipid abnormality in chronic kidney disease: Descriptive study

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Abstract
The dyslipoproteinemia seems to result primarily from abnormalities in the transport and metabolism of triglyceride rich lipoprotein. The pathogenic links between the progressive decrease in renal function and the development of lipoprotein abnormalities are still poorly understood. Patients presenting to the hospital and diagnosed with CKD were included in the study after obtaining informed consent until sixty cases were collected. The history of the onset, progression, duration of various symptoms, drug and diet history was noted. Laboratory investigations like basic blood profile, blood urea, serum creatinine, serum cholesterol, serum triglyceride, serum LDL, serum HDL, Lp(a) and ultrasound abdomen was done. The mean value of Serum Triglycerides is 159.76 mg/dl, Serum cholesterol is 184.17 mg/dl, Serum HDL is 41.8 mg/dl, Serum LDL is 111.37 mg/dl, Serum VLDL is 31.1 mg/dl and lipoprotein (a) is 39.26mg/dl.

Keywords: CKD, Lipid Profile, Cholesterol

1. Introduction

The term lipid refers to substances with poor water solubility. These include biologically important materials such as sterols, including cholesterol, that are composed of hydrocarbon rings and glycerides such as triglycerides and phospholipids that are chiefly composed of hydrocarbon chains. The most common fats of diet are the neural fats also known as triglycerides. Each molecule of triglycerides is composed of a glycerol nucleus and three fatty acids. This neutral fat is found in food of both animal and plant origin. The diet also contains phospholipids and cholesterol esters which are considered as fats due to the presence of fatty acids [1].

Hyperlipemia is a more general term, which includes not only lipemias but also any abnormal elevation of the other plasma lipids. Thus familial hypercholesterolemia (IIA) is hyperlipoproteinemia (LDL and Lp (a) increased) or hyperlipidemia (cholesterol increased) but not lipemia (since the plasma is clear) [2].

Disturbance of lipo-protein metabolism in renal diseases have known to clinics since the early 19th century. Bagdae *et al.* [3], reported hypertriglyceridemia in patients with non nephrotic renal failure. Subsequently, a secondary type of hyper lipoproteinemia in uremia was recognized. Hypertriglyceridemia is most common plasma lipid abnormality in patients with chronic kidney disease on conservative management [4].

The elevated TG is due to accumulation of VLDL, IDL & LDL. The total cholesterol is either normal or slightly increased with fraction of cholesterol in VLDL and late pre-beta-lipoprotein being increased. HDL and its cholesterol fraction is decreased.

Shah *et al.* in conservatively managed renal failure patients reported hypertriglyceridemia with no significant change in levels of total cholesterol, HDL and LDL levels[5].

Progressive renal insufficiency is associated with abnormal lipoprotein transport which may be manifested as hyper lipidemia in a substantial number of patients.

The dyslipoproteinemia seems to result primarily from abnormalities in the transport and metabolism of triglyceride rich lipoprotein. The pathogenic links between the progressive decrease in renal function and the development of lipoprotein abnormalities are still poorly understood[4].

Impaired TG removal also contribute to the observed elevation since subnormal post heparin lipolytic activity was observed in all uremic subjects after appropriate dietary preparations, the subnormal lipolytic activity suggests that low tissue levels of lipoproteins lipase are also associated with azotemic state. Further suggestive evidence is that impaired removal also contributes to elevation of triglyceride in uremia is provided by the observation, that, triglyceride levels are higher than expected for any given level of immuno reactive insulin[3].

2. Methodology

Minimum 100 cases of chronic kidney disease, both males and females, admitted in Navodaya Medical College, Hospital and Research Centre, Raichur during period of approximately 1 ½ year will form part of study.

Design of the study

Cross sectional descriptive study.

Procedure

Patients presenting to the hospital and diagnosed with CKD were included in the study after obtaining informed consent until sixty cases were collected. The history of the onset, progression, duration of various symptoms, drug and diet history was noted. Laboratory investigations like basic blood profile, blood urea, serum creatinine, serum cholesterol, serum triglyceride, serum LDL, serum HDL, Lp (a) and ultrasound abdomen was done.

Inclusion criteria

Patients diagnosed as chronic kidney disease on conservative treatment or hemodialysis.

Exclusion criteria

1. Diabetes Mellitus
2. Renal transplant patients

3. Patients on lipid lowering drug.
4. Patients less than 18 years
5. Hypo/Hyperthyroidism

Mean, standard deviation and confidence interval was calculated and the same represented by graphs. Student t test was used to calculate the significance between means.

3. Results

Table 1: Mean Lipid Profile

Lipid profile	Mean(mg/dl)
Total cholesterol	184.17
Triglyceride	159.76
HDL	41.8
LDL	111.37
VLDL	31.1
Lp(a)	39.26

It can be seen that the mean value of Serum Triglycerides is 159.76 mg/dl, Serum cholesterol is 184.17 mg/dl, Serum HDL is 41.8 mg/dl, Serum LDL is 111.37 mg/dl, Serum VLDL is 31.1 mg/dl and lipoprotein (a) is 39.26mg/dl.

The mean values of the different fractions were obtained with respect to the co-morbid conditions present in the study sample. The comparative mean values of the different fractions of lipid in CKD patients with HTN and without HTN is depicted in bar diagram below.

Table 2: Lipid profile in hypertensive and non-hypertensive patients

Lipid Profile of CKD patients	Hypertension	
	Yes	No
Total cholesterol	195.04	174.53
Triglyceride	175.4	145.88
HDL	38.28	44.93
LDL	122.93	101.12
VLDL	34.05	28.48
Lp(a)	41.28	37.47

The p value of S. Triglyceride is 0.030, S. Cholesterol is 0.011, S.HDL is 0.019, S.LDL is 0.011, S. VLDL is 0.044 and Lp(a) is 0.045 which are significant.

Table 3: Tests of equality of Means

Parameters	Tests of equality of Means	
	t	P
Total cholesterol	2.105	0.021
Triglyceride	2.548	0.020
HDL	-2.260	0.027
LDL	1.987	0.026
VLDL	2.548	0.016
Lp(a)	11.983	<0.001

4. Discussion

In our study, the mean value of lipid profile is as follows. Serum cholesterol is 184.17 mg/dl, Serum Triglycerides is 159.76 mg/dl, Serum HDL is 41.8 mg/dl, Serum LDL is 111.37 mg/dl, Serum VLDL is 31.1 mg/dl and lipoprotein (a) is 39.26mg/dl.

One of the study conducted by Mannangi *et al.* [6] also showed similar results. In their study, Mean values in mg/dl were as follows; total cholesterol (196.80±54.58), Triglycerides (187.93±60.61), HDL (38.60±11.54), LDL (125.33±14.38), VLDL (34.86±11.87), Lp(a) (61.98±36.38).

Attman P.O., Alaupovic P., stated, hypertriglyceridemia is the most common plasma lipid abnormality in adult patients and children with renal failure [4].

The cause for hyper triglyceridemia in chronic kidney disease patients, has not been delineated. Available data derived from kinetic studies with intra lipid administration have demonstrated that reduced catabolism of triglyceride is the predominant defect due to deficiency of lipoprotein lipase [6, 8] or hepatic triglyceride lipase or both. These enzymes are the primary mediation of the process, reason for decrease in activity of these enzymes are not clear. Possibly due to;

- Presence of circulatory inhibitor of lipolytic enzymes in the serum.
- Changes in apoprotein concentrations which can effect lipoprotein lipase activity [7].
- Insulin resistance seen in renal insufficiency [4].
- Alteration of lipoprotein substrate [4].

Fuh MMT *et al.* [8] found decrease in plasma HDL cholesterol concentration seen in patients with chronic kidney disease is associated with decrease in both the fractional catabolic rate and the total synthetic rate of apoAI/HDL. The worse the renal function is the slower the fractional catabolic rate and the lower the apoAI/HDL. The changes in lipoprotein lipase activity the enzyme that are responsible for VLDL-triglyceride hydrolysis may play a major role in this regard. For example lipoprotein lipase activity is decreased in patients with chronic renal failure. The lower the lipoprotein lipase activity is the lower the plasma HDL concentration. There is also evidence that patients with chronic kidney disease have a factor in the plasma which inhibits lipoprotein lipase. Thus there are several possible mechanisms involving an abnormality in VLDL - triglyceride hydrolysis which could result in abnormal HDL metabolism and HDL concentration in patients with chronic kidney disease.

Anderson *et al.*, showed increase in LDL levels[9]. The concentration of the most atherogenic lipoprotein LDL is usually normal or only marginally increased. In Uremia LDL lipoproteins qualitatively altered. *In-vitro* studies have shown that LDL isolated from uremic patients is a poor ligand for the LDL apo B/E receptors.

LDL clearance is often abnormally low in patients with chronic renal failure. The delayed clearance of LDL, increases in the time of residence of LDL in the circulation. This leads to number of disturbances in the LDL metabolism modification of LDL such as carbamylation, oxidation. Internalization with glycosaminoglycans suggested to cause accelerated atherosclerosis [10]. Cheung showed increase in very low density lipoproteins (VLDL) [11].

Koch *et al.*, showed association between history of coronary artery stenosis and dyslipidemia in patients with CKD and observed Lp (a) levels were increased along with decreased HDL-Cholesterol, but no change in total cholesterol, LDL-Cholesterol and triglyceride levels [12].

5. Conclusion

In this study, mean values of lipid profile in 100 CKD patients showed increase in triglycerides, LDL, VLDL and Lp(a), decrease in mean value of HDL

6. References

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