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EVALUATION OF LIPID PROFILE IN PATIENTS OF CHRONIC KIDNEY DISEASE WITH Lp (a) REFERENCE

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ABSTRACT:

Chronic renal disease combined with abnormal lipid profile causes cardiovascular disease on other side Lipoprotein(a) is risk factor for atherosclerosis. The study includes 50 control group and 50 are cases in both the groups blood urea, Serum creatinine, serum triglyceride, serum total cholesterol, High Density Lipoprotein Cholesterol (HDL-C) were analyzed, while Low Density Lipoprotein Cholesterol (LDL-C) was calculated by using Frieldwald formula.Lp (a) is estimated by using Latex DAIICHI kit method. The serum levels of Triacylglycerols, Total Cholesterol and LDL Cholesterol were raised as compared to corresponding values in Control group. The serum level of HDL cholesterol in the present study was significantly as compared to Control group. Therefore as observed in the present study there is dyslipidaemia associated with chronic renal failure. This dyslipidaemia needs prompt attention and measures for rectification to prevent or delay atherosclerotic complications of chronic renal failure.

Keywords: Chronic Renal disease, Lipid profile, LP(a)

INTRODUCTION

Chronic Renal Disease is a progressive pathology of kidney affecting either the glomerular or the tubulo-interstitial or both leading sooner or later to end stage renal failure in the course of time. It is associated with a progressive reduction of glomerular filtration rate due to irreversible nephron loss. This progressive event often occurs well after the subsidence of acute nephropathy and of its initiating events. This decline in renal function is linear in great majority of patients with chronic renal disease due to glomerulonephritis, tubulo-interstitial nephropathy and diabetic nephropathy [1]. Patients with chronic glomerular diseases tend to have a faster rate of deterioration than those with tubulo-interstitial nephropathies and hypertensive nephropathies. This progression also depends upon degree of renal functional impairment, hypertension at presentation as well as on the severity of proteinuria. This progressive chronic renal disease is characterized histologically by glomerular, tubulo-interstitial and vascular scarring.

Chronic Renal Failure involves initiating mechanisms specific to the underlying etiology as well as a set of progressive mechanisms that are a common consequence following long term reduction of renal mass, irrespective of etiology. Such reduction of renal mass causes structural and functional hypertrophy of surviving nephrons. Clinically the patients of chronic renal diseases are asymptomatic, with the progression of disease process and with the

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increasing amount of nephrons loss, the patient reaches the stage of end stage renal disease (ESRD), which is characterized by prolonged signs and symptoms of uraemia. The manifestations of end stage renal disease remain constant in all the chronic renal diseases irrespective of diverse aetiology. These manifestations underscore the kidney not only as an excretory organ but also its importance in metabolic functions of the body. Chronic renal disease is almost asymptomatic till reduction of glomerular filtration rate up to 35-50% of normal, below which symptoms like hypertension and anaemia begin to appear. The uremic syndrome results from functional derangement of many organ systems, although the prominence of specific symptoms varies among patients. Azotaemia refers to the retention of nitrogenous waste products as renal insufficiency develops. Uraemia refers to the more advanced stages of progressive renal insufficiency when the complex, multi-organ system derangements become clinically manifest. The abnormalities of uremic syndrome as a consequence to the accumulation of products of protein metabolism on the one hand and abnormalities consequent to the loss of other renal functions on the other hand such as fluid and electrolyte homeostasis and synthesis of certain hormones [2].

Chronic kidney disease patients mostly affected to cardiovascular complication than compared with other abnormalities and end stage renal disease patients also die due to cardiovascular complication than kidney problem. The most common cause for cardiovascular abnormality is due to abnormal lipid profile so we take study the lipid profile pattern in CKD. High plasma levels of Lp(a) are associated with increased risk for atherosclerotic cardiovascular disease. Structurally, Lp(a) closely resembles LDL. Its protein moiety contains apolipoprotein B-100 and apolipoprotein(a)

MATERIALS AND METHODS:

Selection of Controls Fifty healthy individuals from amongst hospital staff and their relatives of the age group of 35-65 years of either sex were taken as controls. The selection was done purely basing on the history, diet and physical examination. Routine blood and urine investigations and biochemical tests were also done for the control group. Patients with the history of diabetes mellitus, myxoedema, hypertension, nephrotic syndrome, obstructive jaundice, congestive cardiac failure, systemic lupus erythematosus, alcohol abuse, steroid therapy and those who were suffering from diseases likely to alter the lipid profile were excluded from this study.

Selection of Cases

The materials of this study included fifty patients with definite evidence of chronic renal disease. The diagnosis of chronic renal disease was done depending upon the criteria by Alfrey [3] as follows: - a) Compromised renal function b) Clinically significant proteinuria c) Urinary sediment alterations

The blood urea was estimated by GLDH– Urease method [4]. Serum creatinine was estimated by Jaffes method [5]. The serum total cholesterol and High Density Lipoprotein Cholesterol (HDL-C) were analysed using cholesterol oxidase method [6, 7], triglyceride assessment was carried out by glycerol kinase method [8], while Low Density Lipoprotein Cholesterol (LDL-C) was calculated by using Fieldward formula [9]. Lp (a) is estimated by using Latex DAIICHI kit method.

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE8, 2023

RESULTS AND DISCUSSION

The objective of this study was to determine the serum lipid profile in chronic renal diseases of diverse etiology and to find out if there was any relation between the degree of renal damage and serum lipid profile in these patients. In addition, an attempt was made in the current study to assess whether there was any alteration in the VLDLc to HDLc ratio in such patients in view of the observations made by kindler et al, [10] who showed that increase in this ratio in CKD patients make them more susceptible to coronary heart disease. The diagnosis of chronic renal disease was made on the basis of history, physical examination, urine analysis and biochemical investigation for renal functions such as blood urea and serum creatinine. Other investigations like plain X-ray of abdomen, ultrasonogram, intravenous pyelogram and histopathological study were considered in certain cases to establish the diagnosis. Only the chronic renal disease primarily affecting the kidneys had been taken into consideration in this study. Renal involvement secondary to some extra-renal diseases had been excluded. All the patients were subjected to determination of their serum lipid profile like total cholesterol, HDL cholesterol, LDL cholesterol, VLDL cholesterol and triacylglycerol. The results so obtained were compared with the lipid profile of healthy persons taken as control. Statistical evaluations were carried out to confirm any deviation from the normal values distribution. The sex distribution among Controls and Cases were 64% and 66% respectively for males and 36% and 34% in females respectively

Table 1. Sex distribution among Controls and Cases

Group	Total number	Male		Female	
		Number	Percentage	Number	Percentage
Control	50	32	64%	18	36%
Cases	50	33	66%	17	34%

Blood urea concentration in control and case groups in the present study the mean blood urea value of case group (94.28 mg/dL ± 15.91) was in higher level as compared to control group (27.04mg/dL ± 5.86). This increase is statistically significant ($p < 0.001$)

Bagdade et al observed in their 13 undialyzed chronic renal disease patients that, all the cases (100%) had a blood urea nitrogen value more than 50mg/dL, which was equivalent to blood urea level of more than 107mg/dL [11]. Jain et al in their study of 6 patientsof chronic renal disease showed blood urea level to be more than 40mg/dL in all cases [12]. Attman and Alaupovic observed in their 50 uremic patients the mean serum urea level to be 178 mg/dL [13]. In the present study the mean value of blood urea was found to be more or less similar with the mean values of all the above authors. High blood urea concentration in CRF patients is due to pre-renal, renal and post-renal causes [14].

Table 2.Blood urea Level

Group	Urea mg/dl		
	Range	Mean	± SD
Control (n=50)	15 – 37	27.04	5.86
Cases (n=50)	54- 128	94.28	15.91

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE8, 2023

Z value: 28.04 p value < 0.001

Serum creatinine concentration in control and case groups In the present study the mean serum creatinine value of case group (2.29 mg/dL ± 0.57) was in higher level as compared to control group (0.80mg/dL ± 0.22). This increase is statistically significant (p <0.001)

Mordasini et al in their study of 13 cases of chronic uremia found the mean value of serum creatinine to be 8.30mg/dL [15]. Grutzmacher et al observed that the mean creatinine concentration in group-II, group-III and group-IV of chronic renal failure with advancing deterioration were 0.98 mg/dL ± 0.16, 2.08 mg/dL ± 0.61 and 5.64 mg/dL ± 2.93 [16]. There was gradual increase in the serum creatinine concentration with the progression of renal damageHigh serum creatinine concentration in CRF patients is due to any cause of a reduced GFR [17].

Table 3.Serum Creatinine Levels

Group	Creatinine mg/dl		
	Range	Mean	± SD
Control (n=50)	0.7 – 1.3	0.99	0.20
Cases (n=50)	4.8 – 8.1	6.5	1.01

Z value: 17.32 p value < 0.001

Mean fasting serum triacyl glycerol levels in control and case groups In the present study the mean fasting serum triacylglycerol value of case group (211.92mg/dL ± 15.72) was higher as compared to control group (101.4mg/dL ± 14.58). This increase is statistically significant (p <0.001).

Bagdade et al observed increased triacyl glycerol levels in patients of chronic renal failure (209.30mg/dL ± 91.00) [18]. Rappaport observed increased triacyl glycerol levels in patients of chronic renal failure (191.00mg/dL ± 84.00) [19]. Basha et al observed increased triacyl glycerol levels in patients of chronic renal failure (171.08mg/dL ± 99.25) [20]. Sharma et al observed increased triacyl glycerol levels in patients of chronic renal failure (96.40mg/dL ± 10.80) [21] Mean serum triacyl glycerol level in our present study was in agreement with the studies of Bagdade et al and Rapaport.

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE8, 2023

Table 4.Mean Fasting Serum Triacylglycerols Levels

Group	Serum Triacylglycerols(mg/dl)		
	Range	Mean	± SD
Control (n=50)	73 – 140	101.4	14.58
Cases (n=50)	186 - 239	211.92	15.72

Z value: 36.43 p value< **0.001**

The possible factors for this increase in triacylglycerol level may be due to increased triacylglycerol release or decreased triacylglycerol removal into the circulation or a combination of both. The CRF-induced hypertriacyl glycerolmia, abnormal composition and impaired clearance of triacyl glycerolrich lipoprotein and their remnants are primarily due to down regulation of lipoprotein lipase, hepatic lipase, and the very-low-density lipoprotein receptor, as well as, up regulation of hepatic acyl-CoA cholesterol acyltransferase (ACAT) [22].

Increased triacylglycerol synthesis is a minor cause of hypertriacylglycerolemia in CRF It may result due to excess carbohydrate intake, improper utilization of carbohydrates, impaired beta-oxidation of fattyacids due to carnitine deficiency and hyperinsulinism resulting from insulin resistance. Hyper insulinaemia resulting from peripheral insulin resistance may be responsible for part of the hypertriacylglycerolemia [23].

Lipoprotein lipase and hepatic triacylglycerol lipase (HTGL) are the two key enzymes of plasma triacylglycerol catabolism, are decreased in plasma of CRF patients. Heparin is responsible for release of these enzymes into circulation. Post heparin lipolytic activity is consistently decreased in uremic patients. Reduced hepatic triacylglycerol lipase activity has been demonstrated in patients with even moderate and terminal renal failure and correlated with reduced G.F.R. Furthermore, apo C-II is the cofactor for lipoprotein lipase catalyzed lipolysis, whereas apo C-III inhibits this reaction. In addition, the decreased ratio of apo C-II to apo C-III in plasma VLDL and IDL in chronic renal failure patients could affect the lipoprotein lipase activity although there is no absolute deficiency of apo C-II in uremia, Reduced lecithin cholesterol acyl transferase (LCAT) activity has been reported in uremic patients. This is particularly noteworthy, since the activity of this enzyme is usually increased in other hypertriacylglycerolemic states. Low lecithin cholesterol acyl transferase activity may reflect low levels of endogenous substrate and activator apolipoprotein A in these studies. It has been suggested that circulating inhibitors account for reduced lipolytic activity. Uremic plasma appears to contain a nondialysable inhibitor of lipoprotein lipase, which is detectable in both acute renal failure and chronic renal failure [24, 25].

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE8, 2023

Insulin is also a known stimulator of lipoprotein lipase. Insulin resistance occurs in uremia as far as its glucose uptake is concerned. It may also be possible that a resistance to insulin's stimulatory activity on lipoprotein lipase exists. One of the possible links between decreased lipoprotein lipase activity, insulin resistance and hyperinsulinemia may be due to increased level of parathyroid hormone. Elevated growth hormone and parathyroid hormone had been demonstrated and these may be responsible for insulin resistance. Reduced concentrations of 1,25 dihydroxy cholecalciferol may impede the insulin release and in some patients results in a relative insulin deficiency. Glucagon, which stimulates lipolysis are also found to be elevated [26, 27].

Finally reactions distal to lipoprotein lipase may be defective and become rate limiting in triacylglycerol assimilation. This is suggested by the finding that fatty acid incorporation into adipocytes is reduced in uremic subjects [28].

Mean fasting serum total cholesterol levels in control and case groups In the present study the mean fasting serum total cholesterol value of case group (221.3mg/dL ± 14.88) was higher as compared to control group (169.18mg/dL ± 10.64). This increase is statistically significant ($p<0.001$)

Mordasini et al observed increased total cholesterol levels in patients of chronic renal failure (254.00mg/dL ± 36.00) [15]. Rappaport. observed increased total cholesterol levels in patients of chronic renal failure (208.70mg/dL ± 88.20) [19]. Attman et al observed increased total cholesterol levels in patients of chronic renal failure (221.00mg/dL ± 56.00) [13]. Mean serum total cholesterol level in our present study was in agreement with the studies of Rapaport , Attman et al. Increased serum total cholesterol resulted from Increased synthesis of cholesterol. Increasing amount of proteinuria occurring during chronic renal disease causes compensatory increase in hepatic protein synthesis of 3- hydroxy 3-methylglutaryl CoA (HMG CoA) reductase. That causes increased cholesterol synthesis. Insulin resistance that occurs during chronic renal disease results in excess insulin secretion. This insulin is a contributing factor for increased cholesterol synthesis. The apo-B compenent of LDL receptor is also deformed in uremia. In the uremic atmosphere there occurs carbamylation and oxidation of lipoproteins. These factors interfere with the recognition and receptor mediated uptake of lipoproteins. So decreased cholesterol entry through LDL mechanism results in increased HMG-CoA activity and cholesterol synthesis [29]

Table 5.Fasting Serum Total Cholesterol Levels

Group	Serum Total Cholesterol (mg/dl)		
	Range	Mean	± SD
Control (n=50)	152 – 192	169.18	10.64
Cases (n=50)	188 – 242	221.3	14.88

Z value: 20.14; p value <0.001

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE8, 2023

Mean fasting serum HDL - cholesterol levels in control and case groups In the present study the mean fasting serum HDL cholesterol value of case group (42.58mg/dL ± 6.07) was in lower level as compared to control group (47.48mg/dL ± 4.87). This decrease is statistically significant (p <0.001).

Bagdade et al observed decreased HDL cholesterol levels in patients of chronic renal failure (34.50mg/dL ± 23.00) [24]. Mordasini et al observed decreased HDL cholesterol levels in patients of chronic renal failure (38.00mg/dL ± 17.42) [15]. Jain et al observed decreased HDL cholesterol level in patients of chronic renal failure (45.55mg/dL ± 19.80) [30]. Mean HDL cholesterol level in our present study was in between the observations of Mordasini and Jain

The lowered mean HDL cholesterol concentration in chronic renal disease is due to lowered concentration of both HDL2 and HDL3. The decrease of HDL2 is more marked than that of HDL3. Finally, HDL2 and HDL3 of uremic patients are enriched in triacylglycerol. HDL3 is more specifically deficient in cholesteryl esters. The decline in HDL is due to consequence of hypertriacylglycerolemia. There is correlation between LPL activity and HDL cholesterol. Major part of HDL is formed during the catabolism of triacylglycerol rich lipoproteins. HDL2 in CRF is diminished because of decreased lipolysis and HDL3 also registers a decline. Decreased apo A-I concentration in CRF results in reduced LCAT activity and decreased cholesterol esterification. So free cholesterol remains as such inside HDL3. Unesterified cholesterol does not come from tissue or other lipoproteins like chylomicron, LDL, VLDL etc to be esterified and incorporated into HDL2. So HDL cholesterol concentration becomes low [31, 32}

Table 6. Fasting Serum HDL Cholesterol Levels

Group	Serum HDL Cholesterol (mg/dl)		
	Range	Mean	± SD
Control (n=50)	41 – 62	47.48	4.87
Cases (n=50)	30 – 55	42.58	6.07

Z value: 4.44 p value<0.001

Mean fasting serum LDL - cholesterol levels in control and case groups In the present study the mean fasting serum LDL cholesterol value of case group (136.33mg/dL ± 16.15) was higher as compared to control group (101.41mg/dL ± 12.04). Bagdade et al observe d slightly increased LDL cholesterol levels in patients of chronic renal failure (126.20mg/dL ± 47.00) [24]. Mordasini et al observed increased LDL cholesterol levels in patients of chronic renal failure (170.00mg/dL ± 54.42) [15].

Serum LDL cholesterol concentration although with in normal range was slightly increased as compared to control in the observation of Bagdade et al. Mordasini et al revealed in their study a significant increase in serum LDL cholesterol concentration [15]. Jain et al noticed no

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE8, 2023

change in LDL cholesterol (93.33mg/dL ± 17.20) level in their study when compared with that of controls [30]. Mean LDL cholesterol level in our present study was in agreement with Bagdade et al.

Table 7.Fasting Serum LDL Cholesterol Levels

Group	Serum LDL Cholesterol (mg/dl)		
	Range	Mean	± SD
Control (n=50)	74 – 128	101.41	12.04
Cases (n=50)	100 – 174	136.33	16.15

Z value: 12.25; p value<0.001

Mean fasting serum VLDL - cholesterol levels in control and case groups In the present study the mean fasting serum VLDL cholesterol value of case group (42.38mg/dL ± 3.14) was higher as compared to control group (20.28mg/dL ± 2.9). This increase is statistically significant ((p <0.001)

Bagdade et al observed slightly increased VLDL cholesterol levels in patients of chronic renal failure (47.80mg/dL ± 20.00) as compared to control [24]. Mordasini et al observed no significant increase in VLDL cholesterol levels in patients of chronic renal failure (24.00mg/dL ± 3.42) as compared to control [15]. Jain et al observed no significant increase in VLDL cholesterol levels in patients of chronic renafailure (28.33mg/dL ± 2.72) as compared to control [30]. Mean VLDL cholesterol level in our present study was in agreement with Bagdade et al.

The increased VLDL cholesterol concentration in chronic renal disease is due to elevated VLDL levels because of delayed catabolism of VLDL. In uremia the cholesterol content of HDL is low and the apo C-II concentration is also low. Normally this apo C-II is transferred from HDL in plasma to VLDL. In VLDL molecule this apo C-II acts as an activator of lipoprotein lipase (LPL) in extrahepatic tissue. The decreased in apo C-II leads to decreased triacylglycerol catabolism and VLDL metabolism. So VLDL concentration rises [33].

Table 8.Fasting Serum VLDL Cholesterol Levels

Group	Serum VLDL Cholesterol (mg/dl)		
	Range	Mean	± SD
Control (n=50)	14 – 28	20.28	2.9
Cases (n=50)	37 - 48	42.38	3.14

Z value: 36.43 p value <0.001

Ratio of VLDL cholesterol to HDL cholesterol in control and case groups In the present study the ratio of mean fasting serum VLDL cholesterol to HDL cholesterol level of case

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE8, 2023

group (1.02 ± 0.16) was higher as compared to control group (0.43 ± 0.06). This increase is statistically significant (p <0.001)

Kindler et al studied in European sample of patients with chronic renal disease argued that increased VLDL cholesterol to HDL cholesterol ratio was associated with increased incidence of coronary heart disease [10].

Table 9.Ratio of Mean Fasting serum VLDL cholesterol to HDL cholesterol

Group	Serum VLDLc /HDLc		
	Range	Mean	± SD
Control (n=50)	0.30 – 0.49	0.43	0.06
Cases (n=50)	0.79 – 1.21	1.02	0.16

Z value: 23.5 p value<0.001

Mean Serum Lp(a) levels in control and case groups In the present study the ratio of mean Lp(a) level of case group (73.6 ± 3.85) was higher as compared to control group (21.2 ± 3.37). This increase is statistically significant (p <0.001).

Several studies reported that there is continuous raise of Lp(a) in renal failure patients. Lp(a) contributed to increases incidence of cardio vascular complications [34, 35]. Besides decreased catabolism there is increased synthesis of Lp(a) [36]. The increased Lp(a) concentration in renal failure patient due to malnutrition and inflammation and mainly due to decreased clearance

Table 10.Serum Lp(a) Level

Group	Serum Lp(a) (mg/dl)		
	Range	Mean	± SD
Control (n=50)	15 – 26	21.2	3.37
Cases (n=50)	62 – 82	73.6	3.85

Z value: 72.32; p value<0.001

From above data there is an alteration of lipid profile in chronic kidney disease patients which can causes cardiovascular complications. The underlying mechanism for the development of atherosclerosis and coronary heart disease is Hypertyriglyceridemia. Certainly triacylglycerols contribute to the lipid component of atherosclerotic plaque. On the other hand, high serum triacylglycerol raises the concentrations of other lipoproteins that apparently promote atherogenesis like VLDL remnants, chylomicron remants and small dense LDL. Thus the rise is reflected in a raised serum VLDL cholesterol concentration. Hypertyriglyceridemia also reduces protective HDL concentration. Reduced HDL concentration, along with abnormal HDL composition would result in an increase VLDL

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE8, 2023

cholesterol to HDL cholesterol ratio in the serum. This altered ratio is responsible for defective cholesterol removal from tissues notably the arterial wall, contributing to the increased arteriosclerosis in chronic renal disease and predisposing to coronary heart disease. On other side Lp(a) is a strong atherogenic

CONCLUSION

Morbidity and mortality due to cardiovascular complications are increased in Chronic renal failure even in patients with mild renal dysfunction. The mechanisms of increasing in cardiovascular events are unknown, both traditional and novel Cardiovascular risk factors common to both disease processes have been implicated. In our present study Renal dyslipidemia is characterized by raised triglyceride, low HDL. However, altered regulation of lipoprotein metabolism can develop early in renal disease with alteration in apolipoprotein concentrations in spite of normal plasma lipid levels and there is a alteration of Lp(a)which a potent atherogenic. Preventing the altered lipid profile prevent atherogenic risk and mortality.

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