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Comparison of Lipid Profile in Chronic Kidney Disease Patients on Conservative Management and Dialysis

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Abstract
CKD leads to a variety of biochemical abnormalities. Important among them are disturbances in water and electrolyte balance. It also causes disturbances in calcium, phosphorous, magnesium, fluoride and sulphate levels. Lipid abnormalities are seen in renal failure. It is an observational cross-sectional study of alterations in lipid profiles in patients with kidney disease of duration more than 6 months. An estimation of total cholesterol, triglycerides, serum HDL, cholesterol and VLDL cholesterol was done by enzymatic method by using an autoanalyser in District Hospital Hi-Tech Laboratory. Overall, lipid profile parameters were better in the cases on conservative management as compared to those on dialysis. There was significant difference in Triglycerides, HDL and VLDL among the two groups, while there was no statistically significant difference in Total cholesterol and LDL. The HDL/Total Cholesterol ratio also was significantly lower in the cases on conservative management as compared to those on dialysis ($p = 0.023$).

INTRODUCTION

The progression of CKD is associated with changes in kidney structure characterized by scarring associated with glomerulosclerosis, tubulointerstitial fibrosis vascular sclerosis. Whereas injury to the glomeruli, tubules, interstitium, or vessels may predominate initially. CKD progression is often associated with damage and scarring affecting all structural components of the kidney. The kidney responds to injury by adaptive changes that lead to remodeling evolving toward either healing and functional recovery or scarring with loss of kidney function and progressive CKD. During the last 25 years, considerable progress has been made in the understanding of the pathways leading to healing and recovery and those favoring the progression to scarring and fibrosis. However, little is known about what determines the kidney's predilection for one or the other pathway. Most forms of chronic kidney damage, such as those induced by diabetes, hypertension, chronic glomerulonephritis, or chronic exposure to infections or nephrotoxins, evolve to progressive scarring with loss of function and CKD. Scarring is characterized by the progressive loss of intrinsic renal cells and their replacement by fibrous tissue made of collagenous extracellular matrix. This affects the glomeruli (glomerulosclerosis) tubules and interstitium (tubule interstitial fibrosis) vessels (vascular sclerosis)[1,2].

CKD leads to a variety of biochemical abnormalities. Important among them are disturbances in water and electrolyte balance. It also causes disturbances in calcium, phosphorous, magnesium, fluoride and sulphate levels. Lipid abnormalities are seen in renal failure. Uric acid levels are increased and late in renal failure, protein deficiency ensues. Blood urea and creatinine are raised in CKD and are commonly used to diagnose renal failure[3].

The presenting symptoms and signs of chronic kidney disease are less overt and specific. The kidney disease must be suspected in cases of unexplained fever, lassitude, anorexia, nausea, weakness anemia. Hypertension, heart failure or oedema may dominate the clinical picture. Neurological disturbances like headache, tremor, coma or convulsions often monopolize the attention when the patient is first seen. Stunted growth may be the chief concern of the child or adolescent with chronic renal failure[4].

Polyuria and nocturia may be the only findings at first. Later the patient complains of feeling weak, easy fatigability, insomnia and slight breathlessness. Poor appetite and bad taste in the mouth set in early. Intractable nausea especially in the morning hours brings the patient to the physician, when acidosis and azotemia become more severe, the patients becomes lethargic and may be troubled by hiccough and uncontrolled twitching's of muscles. If renal failure remains untreated, heart failure, progressive anemia and bleeding from various sites, particularly from the gastrointestinal tract, herald the onset of illness. The skin becomes pale, the breath is uriniferous and fibrinous pericarditis may appear. Within a few weeks patient develop disorientation and coma, followed by death.

MATERIALS AND METHODS

Sample Size: 100.

Method:

- Detailed history was taken from patients and meticulous examination was done according to prepared proforma
- Thorough physical examination of all the systems were done
- Previous hospital records and investigations were recorded.
- Patients were subjected for investigations wherever required.

Inclusion Criteria:

- All stages of stable CKD patients.

Exclusion Criteria:

- Critically ill patients.
- Recent worsening of CKD.
- Acute on chronic renal failure.
- Patients on lipid lowering drugs.
- Associated co-morbid conditions such as febrile illness.

It is an observational cross-sectional study of alterations in lipid profiles in patients with kidney disease of duration more than 6 months. An estimation of total cholesterol, triglycerides, serum HDL, cholesterol and VLDL cholesterol was done by enzymatic method by using an autoanalyser in District Hospital Hi-Tech Laboratory.

Normal healthy individuals without any significant systemic medical illness, individuals with normal biochemical profile and normal USG abdomen were taken for study, however individuals with hypertension, diabetes and significant systemic medical illness were excluded from this.

For all patients urine: Albumin, Sugar, Microscopy, Specific gravity, 24 hours urinary protein was done.

RESULTS AND DISCUSSIONS

Total 100 cases of chronic kidney disease patients were taken.

Patients who were on conservative treatment are 60 among them 36 showed normal lipid profile, 24

Table 1: List of chronic kidney disease patients who are managed conservatively and on maintenance hemodialysis

Line of Management	Frequency	Percent
Conservative management	60	60.0
Dialysis	40	40.0
Total	100	100.0

Table 2: Comparison of Lipid profile among chronic kidney disease patients on Conservative management and Dialysis

		Lipid profile		
		Abnormal	Normal	Total
Management	Conservative	24 (40.0%)	36 (60.%)	60 (100.0%)
	Dialysis	30 (75.0%)	10 (25.0%)	40 (100.0%)
Total	54	46	100	

Chi-square = 11.836; p=0.001

Table 3: Comparison of lipid profile in chronic kidney disease patients on Conservative management and dialysis

Parameter	Management	N	Mean	Std. Deviation	Mean difference	p-value
TCH	Conservative	60	199.80	50.200	-7.64	0.408
	Dialysis	40	207.45	35.875		
TG	Conservative	60	146.22	67.662	-50.92	<0.001
	Dialysis	40	197.14	66.114		
HDL	Conservative	60	38.84	6.326	2.74	0.017
	Dialysis	40	36.10	3.933		
LDL	Conservative	60	132.97	48.792	-4.31	0.649
	Dialysis	40	137.28	41.975		
VLDL	Conservative	60	28.29	10.494	-11.42	<0.001
	Dialysis	40	39.71	11.265		
HDL/TCH	Conservative	60	0.208	0.064	0.027	0.023
	Dialysis	40	0.181	0.044		

patients showed variation in lipid profile. Total 40 patients were on dialysis, among them 30 showed abnormal lipid profile fraction and 10 showed normal lipid profile.

Overall, lipid profile parameters were better in the cases on conservative management as compared to those on dialysis. There was significant difference in Triglycerides, HDL and VLDL among the two groups, while there was no statistically significant difference in Total cholesterol and LDL. The HDL/Total Cholesterol ratio also was significantly lower in the cases on conservative management as compared to those on dialysis. (p = 0.023)

Total 60 chronic kidney disease patients were on continuous hemodialysis and 40 chronic kidney disease patients were on conservative management are taken. The patients who were on hemodialysis were suffering from chronic kidney disease from long time compared to patients on conservative management. Probably that might have contributed to increase number of lipid abnormalities in those patients.

The dialysate glucose, acetate buffer, heparinization in hemodialysis contributed to aggravation of hyperlipidemia.

Glucose and acetate primarily contribute to aggravation of the hyperlipidemia.

Heparinization can deplete lipoprotein lipase (LPL) stores, hepatic triglyceride lipase (HTGL) and also inhibit LCAT.

In this study triglycerides were markedly elevated in hemodialysis patients as compared to patients on conservative treatment and this was statistically significant (p<0.05).

Monzani[5], in their study showed hemodialysis patients had general worsening of the lipoprotein profile with elevated APO-E levels and indirect evidence of remnant accumulation. While PTH did not have any significant influence on lipoprotein pattern. Increased insulin levels during HD might partly account for high triglyceride of these patients.

The results point to elevated Apo CIII, reduced Apo CII/Apo CIII and Apo E/ APO CIII ratios as typical features of uremic hyper lipidemia and show that a defective triglyceride removal is the major pathogenic mechanism of uremic high triglyceride.

Hemodialysis treatment generally seems to worsen the lipid and apo lipoprotein pattern observed in predialytic stage of CKD[5].

Mariano Senti *et al.,* in their study on patients with CKD on HD had high triglyceride levels[6].

Increased serum triglyceride levels have been well documented in patients on chronic maintenance hemodialysis.

The total cholesterol is marginally raised in hemodialysis patients as compared to patients on conservative management and it is not statistically significant (P = 0.408).

The LDL cholesterol was not raised significantly in dialysis patient as compared to patient on conservative management (P = 0.649). It is statistically not significant.

Shah BV, *et al.,* showed low values of LDL cholesterol and total cholesterol in dialysis patients[7]. Wheeler DC. stated, increased LDL cholesterol and hyper cholestremia seen in hemodialysis patients[8].

HDL levels were marginally low in dialysis patients as compared to patients on conservative treatment and this was statistically significant (P<0.017).

Mariano Senti, *et al.,* in their study of CKD patients on HD showed low HDL levels[6].

Marion Morena[9], in their study on hemodialysis patients states that hemodialysis patients are exposed to several atherogenic factors resulting from qualitative and functional lipid abnormalities, including triglyceride rich particles, increased susceptibility to LDL oxidation and finally impairment of HDL protective effects.

The results suggest that qualitative abnormalities such as an impairment of HDL associated enzymes are associated with a decrease of HDL levels during hemodialysis.

Hence in addition to the known impairment of reverse cholesterol transport, the reduction of HDL protective capacity against oxidative stress could be involved in the development of HD induced atherosclerosis[9].

The VLDL levels in chronic kidney disease on hemodialysis were increased significantly compared to patients who were on conservative treatment. This increase was statistically significant (P<0.001).

Pedro Botet J. in his study showed increased levels of VLDL fractions in hemodialysis patients. The possible rise of hypertriglyceridemia and changes in VLDL composition as risk factor for coronary heart disease remain a matter of dispute[10].

The HDL/TC ratio is marginally low in hemodialysis patients as compared to patients on conservative treatment (P = 0.023) which was statistically significant.

Alam SM[11], in their study on patients with CKD had elevated TGL in conservatively managed group and markedly decreased HDL cholesterol in both conservative and hemodialysis group.

Bagade JD, *et al.,* in their study on patient with CKD had elevated triglyceride and decreased HDL cholesterol in both conservative and haemodialysis group[12].

CONCLUSION

The HDL/TC ratio was significantly reduced in CKD patients. On comparison of CKD patients on hemodialysis and patients on conservative treatment, there is significant increase in triglyceride and VLDL levels in HD groups.

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