

Original Research Article

A study of lipid profile and ECG changes in chronic kidney disease patients

K. Ganesan¹, N. Ramesh^{2*}

¹Assistant Professor, Department of General Medicine, Government K.A.P. Viswanatham Medical College, Trichy, Tamil Nadu, India

²Assistant Professor, Department of General Medicine, Thanjavur Medical College, Thanjavur, Tamil Nadu, India

*Corresponding author email: rameshnatarajan12@gmail.com

	International Archives of Integrated Medicine, Vol. 6, Issue 3, March, 2019. Copy right © 2019, IAIM, All Rights Reserved. Available online at http://iaimjournal.com/ ISSN: 2394-0026 (P) ISSN: 2394-0034 (O)	
	Received on: 22-02-2019 Source of support: Nil	Accepted on: 26-02-2019 Conflict of interest: None declared.
How to cite this article: K. Ganesan, N. Ramesh. A study of lipid profile and ECG changes in chronic kidney disease patients. IAIM, 2019; 6(3): 60-64.		

Abstract

Introduction: Hyperlipidemia, one of the important risk factor of atherosclerosis, is an abnormality commonly encountered in patients with chronic kidney disease. There are several other important risk factors, such as smoking, proteinuria, oxidative stress, inflammation and dyslipidemia that independently or in combination with elevated blood pressure, can cause deterioration in renal function.

Aim of the study: To analyze lipid alterations that can occur in chronic kidney disease patients.

Materials and methods: This study was conducted among 50 patients with chronic kidney disease. All the patients with established chronic kidney disease ensured with radiological evidence and on conservative treatment were included in the study. Lipid profile parameters are estimated under standard techniques.

Results: Total cholesterol (TC), Triglycerides (TGL), High-Density Lipoprotein (HDL), and Low-Density Lipoprotein (LDL) of all the patients were investigated for the lipid profile study. It was found that the majority (80%) of the study participants were found to have low HDL, 62% of the participants were found to have a high triglyceride level. Overall, seven patients (14%) showed abnormal lipid profile with respect to all the four parameters.

Conclusion: HDL-C levels were lower and triglycerides, total cholesterol, and TGL levels were higher in the study group. There is a statistically significant increase in serum triglycerides level in patients with CKD stage 3, 4 and 5. Predominant lipid abnormalities were reduced HDL-C levels and elevated TGL.

Key words

Renal Failure, Smoking, Atherosclerosis, Obesity, Lipid Profile.

Introduction

Chronic kidney disease is an irreversible deterioration of renal function, which results from diminished effective functioning of renal tissue. Ensuing impairment of excretory, metabolic and endocrine functions of the kidney leads to the development of clinical syndrome of uremia [1]. The severity of the consequences of CKD has however undergone profound changes since the advent of dialysis. Cardiovascular disease is a major cause of morbidity and mortality among patients with chronic kidney disease [2]. The growing recognition that dyslipidemia is a major risk factor for coronary heart disease has prompted interest in the identification and management of abnormalities in plasma lipids and lipoproteins. The majority (58%) of patients with CKD die from cardiovascular causes, making CVD the leading cause of death in patients with CKD [3, 4]. Therefore it is essential to study uremic dyslipidemia since optimal treatment is essential for the prevention or delay of cardiovascular complications in patients with CKD. It is important not only to identify these patients early but also to treat their dyslipidemias intensively before they develop ESRD [5]. An association between lipids and kidney disease was first noted by Virchow who described fatty degeneration of renal epithelium in Bright's disease in 1860. In chronic kidney disease, the most prevalent lipid disorders are hypertriglyceridemia and decreased HDL concentration [6]. LDL levels are usually normal or marginally increased. Also, there are reports available regarding accelerated atherosclerosis in chronic renal failure due to altered lipid metabolism [7].

Materials and methods

A descriptive observational study was conducted during 2017-2018. All the patients in this study group were selected from those who were admitted to the Department of Internal Medicine, Govt. Mahatma Gandhi Memorial Government

Hospital, attached to K.A.P. Viswanatham Govt. Medical College, Trichy. This study was conducted among 50 patients with chronic kidney disease. All the patients with established chronic kidney disease ensured with radiological evidence and on conservative treatment were included in the study. Patients with nephrotic syndrome and patients on drugs like β blockers, statins, and oral contraceptive pills were excluded from the study.

Study tools: History regarding symptoms and duration of the kidney disease, hypertension, diabetes, smoking, alcoholism, drug intake, and treatment were elicited. A detailed clinical examination was performed in all patients including Height and Weight, Blood Pressure, renal function tests, abdominal ultrasonogram, and Electrocardiogram were done for all patients. After 12 hours of overnight fasting blood sample was taken for lipid profile and for TSH levels from patients. The following tests were also performed: Fasting blood glucose and postprandial (mg/dL), Hemoglobin A1C, Serum albumin, 24 hours urine protein, urine P/Cr ratio and Estimated Glomerular filtration rate (eGFR) as assessed by CKD-EPI. Radiological imaging studies by Ultra sonogram showed reduced kidney size (< 9 cm) in one or both kidneys were considered as radiological evidence for chronic kidney disease. LDL > 130 mg/dl, HDL < 40 mg/dl, TGL > 160 mg/dl, TC > 200 mg/dl were considered abnormal.

Statistical methods: Frequencies of normal and abnormal values of LDL, HDL, TGL and Total cholesterol presented. Chi-square test was performed to test the significance of the lipid profile with the ECG changes. A p value of <0.05 was considered significant.

Results

Among the 50 patients studied, the majority were males 39 (78%) and 11 (22%) were females. The

mean age of the subjects was 53.82 ± 10.07 years and the majority (39.58%) of subjects was in the age group of 40-49 years. Age of the patients varied from 40 to 71 years. It was found that there were ten smokers and six alcoholics among the male study participants. A majority (76%) of the study participants was found to have diabetes, hypertension or both (Hypertension-58%, Diabetes-2% and both -16%). There were twenty patients (40%) with urine albumin positive. The majority (92%) of the patients BMI was found to be within normal limit (18.5 to 25 kg/m²).

Lipid profile: Total cholesterol (TC), Triglycerides (TGL), High-Density Lipoprotein (HDL), and Low-Density Lipoprotein (LDL) of all the patients were investigated for the lipid profile study. It was found that the majority (80%) of the study participants were found to have low HDL, 62% of the participants were found to have a high triglyceride level. Overall, seven patients (14%) showed abnormal lipid profile with respect to all the four parameters.

HDL pattern Serum HDL values ranged between 23 mg/dl to 46 mg/dl. LDL pattern Patients showed abnormal LDL levels (130 mg/dl) were found in 12 patients. Their mean value was 188.78 mg/dl and the standard deviation was 5.680. **TGL pattern:** TGL value in our study group ranged between the minimum of 73 mg/dl and the maximum of 313 mg/dl. TGL levels were abnormal in 31 patients >160 mg/dl). Mean and standard deviation of the study group were 165.04 and 50.8. **Total Cholesterol:** Range of TC levels in the study group was 120 mg/dl to 280 mg/dl. Total cholesterol was more than 200 mg/dl in 20 patients. The mean values and standard deviations of the study group were 188.78 and 40.16 (**Table – 1**).

In 50 patients, 11 had GFR <15 ml, 28 had 15-29 ml, 11 had 30-50 ml (**Table – 2**).

Out of 50 patients, 17 (34%) of patients showed changes suggestive of LVH and 16 (32%) of patients showed ischemic changes. The risk

factors which are responsible for increased morbidity and mortality were hypertension, DM, high LDL, low HDL, and smoking (**Table – 3**).

Table - 1: CKD patients with abnormal lipid profile.

Type of Lipid Disorders	No. of Patients (%)
Elevated Cholesterol	20 (40%)
Decreased HDL	40 (80%)
Elevated Triglycerides	31 (62%)
Increased LDL cholesterol	12 (24%)

Table - 2: GFR values.

GFR	Frequency (%)
<15 ml	11 (22%)
15-29 ml	28 (56%)
30-50 ml	11 (22%)

Discussion

CRF is a worldwide health problem and is the leading cause of morbidity and mortality in the developed world. Patients with CRF are at high risk for CVD and cerebrovascular disease (CBVD), and they are more likely to die of CVD than to develop ESRD [8]. CRF is associated with premature atherosclerosis and increased incidence of cardiovascular morbidity and mortality. The low HDL levels in patients with CKD in our study were consistent with Lee et al. who studied the lipid profile in CRF patients. These low HDLC levels were also an independent risk factor for the development of CKD in the Framingham offspring study [9]. Several mechanisms may underlie these reductions in HDLC levels, which is usually an indication of impaired reverse cholesterol transport. Thus, uremic patients usually exhibit decreased levels of apolipoprotein AI and AII (the main protein constituent of HDL) [10]. Diminished activity of LCAT (the enzyme responsible for the esterification of free cholesterol in HDL particles), as well as increased activity of cholesterol ester, transfers protein that facilitates the transfer of cholesterol esters from HDL to TGL-rich lipoproteins that reduce serum concentrations of HDL cholesterol. Hypertriglyceridemia represents an early feature

of renal failure [11]. Indeed, previous studies have shown that patients with impaired renal function exhibit increased concentrations of TGLs even though serum creatinine levels were within normal limits. In addition, individuals with renal insufficiency usually display an abnormal increase in serum TGLs after a fat meal (postprandial lipemia). Experimental studies revealed that accumulation of TGL-rich lipoprotein (very LDL [VLDL], chylomicrons, and their remnants) in individuals with predialysis CKD is mainly due to their decreased catabolism [12]. The down regulation of the expression of several genes along with the changes in the composition of lipoprotein particles and the direct inhibitory effect of various uremic toxins on the enzymes involved in lipid metabolism represents the most

important pathophysiological mechanism underlying the development of hypertriglyceridemia in renal failure. LDL was significantly elevated than that of controls in our study [13]. We found that 44% of patients showed elevated LDL levels. It was found that 60.5% of patients have elevated LDL-C than non-CKD patients (P = 0.06).TC levels were significantly elevated in our study group. However, most of the studies did not observe hypercholesterolemia [14]. The possible reason for the hypercholesterolemia in our study is the significant elevation of cholesterol-containing lipid fractions (IDL and LDL.ECG changes: Out of 50 patients, 17 (34%) of patients showed changes suggestive of LVH and 16 (32%) of patients showed ischemic changes [15].

Table - 3: Lipid profile and its relation with the ECG changes.

Lipid profile		ECG change		χ^2 value	P value
		Abnormal (LVH & Ischemic changes)	Normal		
HDL	Abnormal	25 (62.5%)	15 (37.5%)	0.047	0.827
	Normal	6 (60%)	4 (40%)		
TGL	Abnormal	23 (74.19%)	8 (25.81%)	3.876	0.048
	Normal	8 (42.11%)	11 (57.89%)		
LDL	Abnormal	10 (83.33%)	2 (16.67%)	1.975	0.159
	Normal	21 (55.26%)	17 (44.74%)		
TC	Abnormal	15 (75%)	5 (25%)	1.559	0.211
	Normal	16 (53.33%)	14 (46.67%)		

Conclusion

HDL-C levels were lower and triglycerides, total cholesterol, and TGL levels were higher in the study group. There is a statistically significant increase in serum triglycerides level in patients with CKD stage 3, 4 and 5. Predominant lipid abnormalities were reduced HDL-C levels and elevated TGL. A significant number of patients showing ECG changes of left ventricular hypertrophy 34% and ischemic changes 32%.

Acknowledgments

The authors would like to thank the Professors, Associate Professor, and Postgraduate students,

Department of General Medicine, K.A.P. Viswanatham Govt. Medical College, Trichy for helping with data collection and laboratory analyses.

References

1. Foley RN, Parfrey PS, Sarnak MJ. Clinical epidemiology of cardiovascular disease in chronic renal disease. *American Journal of Kidney Diseases*, 1998 Nov 1; 32(5): S112-9.
2. Chauhan V, Vaid M. Dyslipidemia in chronic kidney disease: managing a high-risk combination. *Postgraduate*

- Medicine, 2009 Nov 1; 121(6): 54-61.
3. Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. *New England Journal of Medicine*, 2004 Sep 23; 351(13): 1296-305.
 4. Shah B, Nair S, Sirsat RA, Ashavaid TF, Nair K. Dyslipidemia in patients with chronic renal failure and in renal transplant patients. *Journal of postgraduate medicine*, 1994 Apr 1; 40(2): 57.
 5. Lee DM, Knight-Gibson C, Samuelsson O, Attman PO, Wang CS, Alaupovic P. Lipoprotein particle abnormalities and the impaired lipolysis in renal insufficiency. *Kidney international*, 2002 Jan 1; 61(1): 209-18.
 6. Vaziri ND, Liang K, Parks JS. Down-regulation of hepatic lecithin: cholesterol acyltransferase gene expression in chronic renal failure. *Kidney international*, 2001 Jun 1; 59(6): 2192-6.
 7. Hunsicker LG, Adler S, Caggiula A, England BK, Greene T, Kusek JW, Rogers NL, Teschan PE, Beck G. Modification of Diet in Renal Disease Study Group. Predictors of the progression of renal disease in the Modification of Diet in Renal Disease Study. *Kidney international*, 1997 Jun 1; 51(6): 1908-19.
 8. Gupta DK. Hyperlipidemia in patients of chronic renal failure. *Bombay Hospital J.*, 1991; 33: 45-50.
 9. Das BS, Misra SK, Rao DV, Satpathy SR, Bose TK. Serum lipid in chronic renal failure. *The Journal of the Association of Physicians of India*, 1984 Dec; 32(12): 1019.
 10. Bagdade JD, Casaretto A, Albers J. Effects of chronic uremia, hemodialysis, and renal transplantation on plasma lipids and lipoproteins in man. *The Journal of laboratory and clinical medicine*, 1976 Jan 1; 87(1): 37-48.
 11. Chan MK, Varghese Z, Moorhead JF. Lipid abnormalities in uremia, dialysis, and transplantation. *Kidney Int.*, 1981; 19(5): 625-37.
 12. Kes P. Lipid abnormalities in chronic renal failure, nephrotic syndrome, and dialysis. *Actamedica Croatica: casopisHravatskeakademijemedicinskih nanosti.*, 2001; 55(4-5): 177-86.
 13. Cheung AK, Parker CJ, Ren K, Iverius PH. Increased lipase inhibition in uremia: Identification of pre- β -HDL as a major inhibitor in normal and uremic plasma. *Kidney international*, 1996 May 1; 49(5): 1360-71.
 14. Ramjan I, Harper L, Mcpake D, Kendall MJ, Wheeler DC. Low-density lipoprotein subfraction profiles in chronic renal failure. *Nephrology, dialysis, transplantation: official publication of the European Dialysis and Transplant Association-European Renal Association*, 1998 Sep 1; 13(9): 2281-7.
 15. Vaziri ND, Liang KH. Down-regulation of hepatic LDL receptor expression in experimental nephrosis. *Kidney international*, 1996 Sep 1; 50(3): 887-93.