International Journal of Current Advanced Research
ISSN: O: 2319-6475, ISSN: P: 2319-6505, Impact Factor: 6.614
Available Online at www.journalijcar.org
Volume 9; Issue 03 (A); March 2020; Page No.21454-21456
DOI: http://dx.doi.org/10.24327/ijcar.2020.21456.4216

Research Article

COMPARATIVE EVALUATION OF SERUM LIPID PROFILE IN CKD PATIENTS AND HEALTHY INDIVIDUALS

Preeti Kumari*[1] and Mithilesh Kumar Singh[2]

[1]B. Pharm, M.Sc Medical Biochemistry Teerthanker Mahaveer Medical College and Research Center, Teerthanker Mahaveer University, Moradabad, Uttar Pradesh
[2]BDS, M.Sc Medical Biochemistry School of Medical Sciences and Research, Sharda University, Greater Noida, Uttar Pradesh

A R T I C L E I N F O

Article History:
Received 06th December, 2019
Received in revised form 14th January, 2020
Accepted 23rd February, 2020
Published online 28th March, 2020

Key words:
CKD, Lipid profile, Atherosclerosis, Renal function test

A B S T R A C T

Background: The associated serum lipid alterations in CKD enhance the risk of atherosclerosis and favor the higher incidence of cardiovascular complications. Dyslipidemia is a most significant risk factor for cardiovascular morbidity and mortality, and it is common along with patients having CKD.
Objective: To evaluate serum lipid profile in CKD patients and healthy individuals.
Materials and methods: 50 patients aged 18-60 years who were diagnosed as Chronic Kidney Disease by determination of serum urea, serum creatinine level and 50 healthy individuals as controls. Overnight fasting venous blood samples were collected from both groups for Lipid Profile estimation. Urea and Creatinine estimation were also done to test for renal function.
Results: Dyslipidemia was observed in CKD patients characterized by a statistically significant increase in serum cholesterol, serum triglycerides, serum LDL &serum VLDL with decreases in serum HDL in CKD patients when compared with the controls.
Conclusion: The accompanying serum lipid alterations in CKD enhance the risk of atherosclerosis and favor the higher incidence of cardiovascular complications. So, the strict monitoring of lipid profile can reduce the morbidity &mortality rate and will also improve the quality of life of CKD patients.

INTRODUCTION

Chronic kidney disease is also known as chronic renal failure. In CKD the kidney Glomerular Filtration Rate (GFR) is decreased <60 ml/min/1.73m^{2}for at least 3months and kidney becomes damaged.[1]Cardiovascular disease is one of major basis of morbidity and mortality with patients of chronic kidney disease.[2]

The upward identification that dyslipidemia has to be a major risk factor for coronary heart disease that encourage in recognition and management of abnormalities in plasma lipids and lipoproteins.[3] Dyslipidemia is highly familiar in patients on maintenance hemodialysis (MHD), with high proportion of the atherogenic triad (hypertriglyceridemia) eminent Very Low Density Lipoprotein (VLDL) and reduced High Density Lipoprotein (HDL).[4]High Density Lipid Cholesterol insufficiency and their abnormal functions in CKD patients have a major cause of atherosclerosis.[5]So, the strict monitoring of lipid profile along the treatment of CKD to avoid various complications and will also improve the quality of life of CKD patients.

*Corresponding author: **Preeti Kumari**
B. Pharm, M.Sc Medical Biochemistry Teerthanker Mahaveer Medical College and Research Center, Teerthanker Mahaveer University, Moradabad, Uttar Pradesh

MATERIALS AND METHODS

Total 50 chronic kidney disease patients and 50 age, sex matched Normal individuals were selected for the study, and Informed consent was taken from each subject before collecting the blood sample.

Inclusion criteria: CKD patients within the age 18-60 years of both Sexes

Exclusion criteria: Acute Renal Failure, Pregnant and lactating women, Malignancy, Cardiac disease

Sample: Overnight fasting venous blood samples was collected aseptically in a plain vial for Serum Lipid Profile, Urea and Creatinine estimation.

Methods: Serum cholesterol was estimated by the Cholesterol oxidase method (ERBA diagnostics Mannheim Gmbh).[6] Serum triglyceride was estimated by the Glycerol kinase method (ERBA diagnostics Mannheim Gmbh).[7] Serum HDL was estimated by the End point Trinder reaction (ERBA diagnostics Mannheim Gmbh).[8] Serum VLDL was estimated by the calculation formula TG/5.[6]

Serum LDL was estimated by the Friedwald's Equation(LDL = Total Cholesterol – HDL –TG/5).[9] Serum urea was estimated by Urease Method (ERBA diagnostics Mannheim Gmbh). Serum creatinine was estimated by Jaffe's Method (ERBA diagnostics Mannheim Gmbh).

RESULT

SPSS 16 versions were selected for the calculation of various biochemical parameters in the study.The analyzed data was correlated by calculating p-values considered significant,

{P <0.05 – Significant, P <0.001 – Highly Significant}.

Table 1 Age and sex distribution among CKD patients (n=50)

Age group (years)	No. Of males	No. Of females	Total no. Of cases
18-30	5	7	12
31-40	8	0	8
41-50	4	9	13
51-60	11	6	17
TOTAL	28	22	50

Table 2 Shows that mean value of urea and creatinine in CKD patients and controls.

Groups	Blood Urea	Serum creatinine
Controls (mean±SD)	26.62±8.659	0.83±0.187
Patients (mean±SD)	148.96±61.126	6.25±2.828
P value	<0.001	<0.001

Table 3 Shows that the comparison of lipid profile (serum cholesterol, TG, HDL, LDL &VLDL) between controls and CKD patients, which was statistically significant (p< 0.001&0.005)

Parameter's	Controls (mean±SD)	CKD patients (mean±SD)	P value
Cholesterol	161.04±19.065	195.23±65.109	0.001
TG	128.14±41.922	253.41±111.980	0.000
HDL	43.00±9.570	24.86±10.585	0.000
LDL	90.97±22.447	119.68±53.098	0.001
VLDL	25.40±9.469	50.68±22.396	0.000

DISCUSSION

In our study the level of the blood urea and creatinine were increased in CKD patients as compared with control. The serum level of these analytes was increased because of the decreased Glomerular filtration rate.[1]

The result shows that there aresignificant alterations in the lipid profiles of the CKD patients, the serum cholesterol, triglycerides, LDL and VLDL were significantly higher in CKD patients group than healthy controls. Whereas the levels of serum HDL where lower in CKD patients. The pathophysiological basis for dyslipidemia in CKD is not only increased rate of atherosclerosis in the renal microcirculation, but also increased lipoprotein in glomerular apparatus and stimulates inflammation mediators and contributes for fibrogenesis.[10,11]

According to Tsumura *et al*. Hypercholesterolemia is one of the abnormalities in patients with CKD, which attributed to hypercholesterolemia due to heavy proteinuria in CKD involves altered gene expression of HMG-CoA reductase, 7 alpha hydroxylase and hepatic LDL receptor.[12]

The accumulation of triglycerides leading to triglyceridemia in CKD is the outcome of both a high production, and a low catabolism of triglycerides.[13]The significant decrease in a HDL in CKD can be attributed to Decrease levels of apolipoprotein, Diminished activity of lecithin-cholesterol acyltransferase (LCAT), increased activity of Cholesteryl Ester Transfer Protein (CETP).[14,15]

Elevated plasma LDL cholesterol is common in nephrotic syndrome, but it is not a typical feature of patients with advanced CKD.[16]The factor which elucidate the increase in serum VLDL are increased activity of CETP which increases transfer of cholesterol ester to VLDL and promotes more VLDL formation, Increased apo C-III, which is an LPL inhibitor inhibiting the degradation of VLDL.[17]

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How to cite this article:

Preeti Kumari and Mithilesh Kumar Singh (2020) 'Comparative Evaluation of Serum Lipid Profile in Ckd Patients and Healthy Individuals', *International Journal of Current Advanced Research,* 09(03), pp. 21454-21456.
DOI: http://dx.doi.org/10.24327/ijcar.2020. 21456.4216

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