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Original Research

Assessment of serum lipid profile in CKD patients

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ABSTRACT:

Background: CKD leads to deterioration of renal function, which results from diminished effective functioning of renal tissues. The present study was conducted to assess the serum lipid profile in CKD patients. **Materials & Methods:** 60 CKD patients of both genders were kept in group I and equal age and gender matched healthy subjects in group II. Assessment of blood urea, serum creatinine, lipid profile: total cholesterol, triglycerides, HDLc, LDLc, VLDLc were performed. **Results:** There were 38 males and 22 female CKD patients. The mean total cholesterol was 130.2 mg/dl in group I and 145.2 mg/dl in group II, TG was 152.4 mg/dl in group I and 98.4 mg/dl in group II, HDLc was 32.5 mg/dl in group I and 46.3 mg/dl in group II, LDLcwas 62.1 mg/dl in group I and 78.4 mg/dl in group II, VLDLc was 31.4 mg/dl in group I and 19.4 mg/dl in group II. The mean blood urea was 124.1 mg/dl in group I and 28.4 mg/dl in group II. The mean serum creatinine was 6.5 mg/dl in group I and 0.92 mg/dl in group II. The difference was significant ($P< 0.05$). **Conclusion:** Increased triglycerides, increased VLDL and reduced HDL are responsible for increased cardiovascular complications in patients with CKD.

Key words: Chronic kidney disease, triglycerides,high density lipoprotein cholesterol

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INTRODUCTION

Chronic kidney disease (CKD) is a condition characterized by progressive loss of renal function over a period of time.[1] CKD leads to deterioration of renal function, which results from diminished effective functioning of renal tissues. Cardiovascular disease is a major cause of morbidity and mortality among patients with CKDs.[2] Most of the patients with CKD die from cardiovascular system complications before ever reaching Stage 5 CKD. Dyslipidemia is a major risk factor for coronary heart disease and it has prompted interest in the identification and management of abnormalities in plasma lipids and lipoproteins.[3]

Dyslipidaemia in CKD patients is characterized by elevated triglyceride (TG), elevated total cholesterol (TC), high density lipoprotein cholesterol (LDL-C) and reduced high density lipoprotein cholesterol (HDL-C). However, total cholesterol may be normal or reduced especially in the presence of malnutrition.[4] The pattern of dyslipidaemia seen in CKD patients is highly atherogenic and is associated with development of atherosclerotic cardiovascular disease and all cause mortality.[5]Dyslipidaemia is a modifiable cardiovascular risk factor, hence early diagnosis and management with both lifestyle modification and lipid lowering medicationswill reduce cardiovascular disease risk and progression to end stage renal disease (ESRD). Treatment of dyslipidaemia using statins has been reported to reduce the rate of decline in glomerular filtration rate (GFR) in CKD patients.[6]The present study was conducted to assess the serum lipidprofile in CKD patients.

MATERIALS & METHODS

The present study comprised of 60CKD patients of both genders. The consent was obtained from all enrolled patients.

Data such as name, age, gender etc. was recorded. CKD patients were kept in group I and equal age and gender matched healthy subjects in group II. The presence of CKD was established based on presence of kidney damage and level of kidney function Glomerular filtration rate (GFR). Markers of kidney damage included abnormalities in the composition of

blood (elevated blood urea and serumcreatinine, abnormalities in serum electrolytes, serum total protein and fraction) or imaging tests (ultrasonogram). Assessment of blood urea, serum creatinine, fasting blood sugar, post prandial blood sugar, serum total protein, serum albumin, serum globulin, lipid profile: total cholesterol, triglycerides, HDLc, LDLc, VLDLc were done by using semiautoanalyzer. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 60		
Gender	**Males**	**Females**
Number	38	22

Table I shows thatthere were 38 males and 22 female CKD patients.

Table II Assessment of lipid profile

Lipid profile	Group I	Group II	P value
Total cholesterol	130.2	145.2	0.04
TG	152.4	98.4	0.01
HDLc	32.5	46.3	0.05
LDLc	62.1	78.4	0.05
VLDLc	31.4	19.4	0.02

Table II, graph I shows that mean total cholesterol was 130.2 mg/dl in group I and 145.2mg/dl in group II, TG was 152.4mg/dl in group I and 98.4mg/dl in group II, HDLc was 32.5mg/dl in group I and 46.3mg/dl in group II, LDLc was 62.1mg/dl in group I and 78.4mg/dl in group II, VLDLc was 31.4mg/dl in group I and 19.4mg/dl in group II. The difference was significant (P< 0.05).

Graph I Assessment of lipid profile

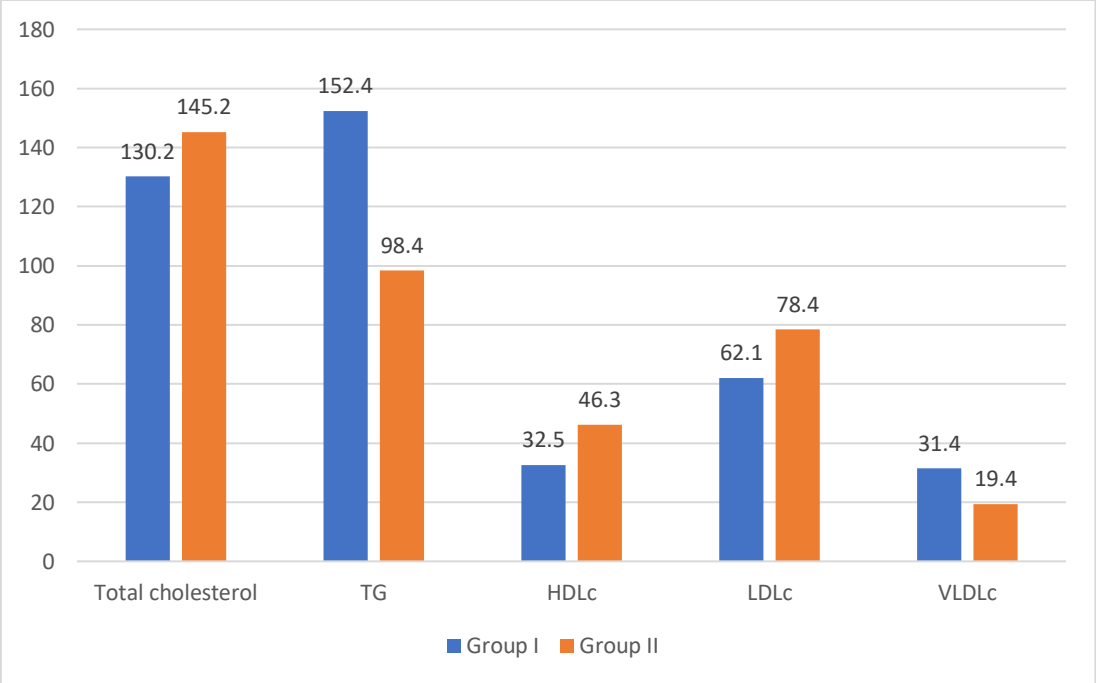
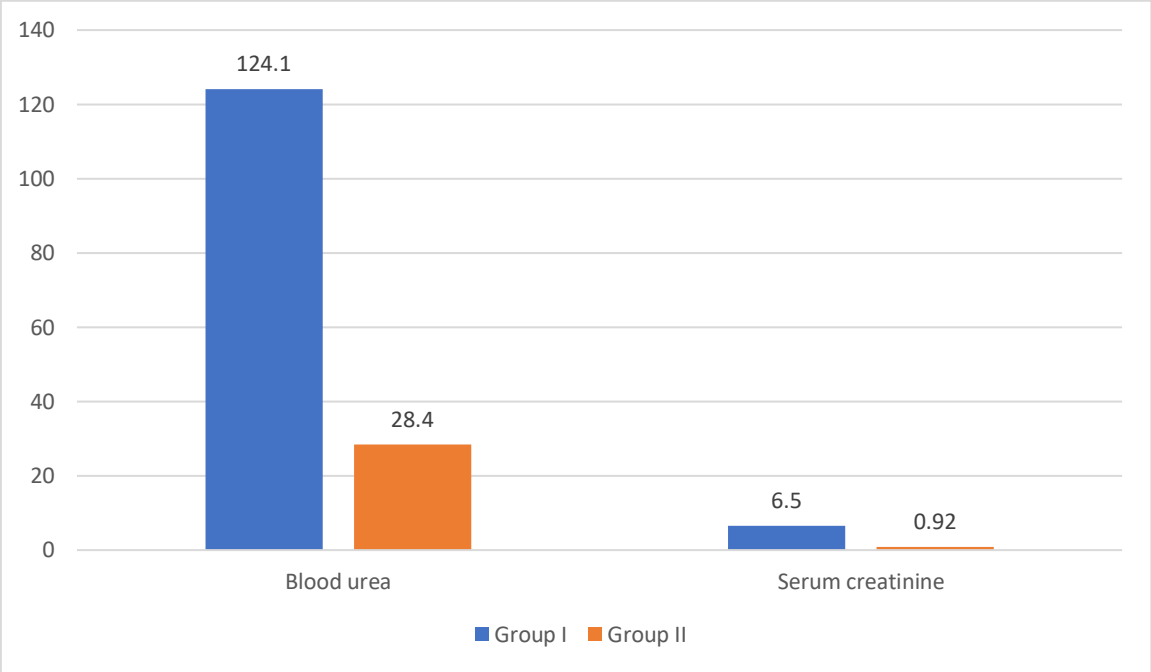


Table III Assessment of renal function test

RFT	Group I	Group II	P value
Blood urea	124.1	28.4	0.01
Serum creatinine	6.5	0.92	0.01

Table III, graph II shows that mean blood urea was 124.1 mg/dl in group I and 28.4 mg/dl in group II. The mean serum creatinine was 6.5 mg/dl in group I and 0.92 mg/dl in group II. The difference was significant (P< 0.05).

Graph II Assessment of renal function test



DISCUSSION

Cardiovascular disease is the leading cause of hospitalization and mortality in patients with chronic kidney disease.[7] The process of cardiovascular disease most likely started in early stages of CKD considering its severity at commencement of renal replacement therapy (RRT). Dyslipidaemia is one of the recognized traditional cardiovascular risk factors in the general population as well as CKD patients.[8] This cardiovascular risk factor occurs commonly in patients with CKD. Dyslipidaemia is associated with rapid decline in renal function and commencement of RRT in CKD patients.[9] The precise mechanism is unknown, but it has been postulated that mesangial cells bind and take up oxidized LDL which then causes injury to mesangial, epithelial and endothelial cells by favouring recruitment of inflammatory cells such as macrophages which release cytokines, chemokines and growth factors.[10] This subsequently leads to glomerulosclerosis. Hypercholesterolaemia and hypertriglyceridaemia also cause podocyte injury and mesangial sclerosis, subsequently leading to glomerulosclerosis.[11,12]The present study was conducted to assess the serum lipidprofile in CKD patients.

We found that there were 38 males and 22 female CKD patients. The mean total cholesterol was 130.2 mg/dl in group I and 145.2 mg/dl in group II, TG was 152.4 mg/dl in group I and 98.4 mg/dl in group II, HDLc was 32.5 mg/dl in group I and 46.3 mg/dl in group II, LDLcwas 62.1 mg/dl in group I and 78.4 mg/dl in group II, VLDLc was 31.4 mg/dl in group I and 19.4 mg/dl in group II.Adejumo et al[13]determined the prevalence and pattern of dyslipidaemia in pre-dialysis CKD patients. 105 consecutive pre-dialysis CKD patients recruited over twoyears and 105 age and sex matched control subjects were included. Data obtained from participants included demographics, bodymass index, and aetiology of CKD. Blood sampling was done for the determination of creatinine and fasting serumlipids. The mean age of the CKD and control subjects were 46.98±16.81 and 47.57±15.97 years respectively witha male:female ratio of 1.7:1. The median atherogenic index of plasma (AIP), low density lipoprotein-cholesterol andtriglyceride (TG) were significantly higher in the CKD patients while mean high density lipoprotein-cholesterol(HDL-C) was significantly lower in the CKD patients (p=<0.001). The overall prevalence of dyslipidaemia in theCKD patients was 60% which was significantly higher than 39% in the control (p=0.002). The prevalence of highAIP, elevated TG and reduced HDL-C increased with worsening renal function. Dyslipidaemia was commoner infemale CKD patients (p=0.02) and those who were ≥ 45years (p=0.94).

We observed that mean blood urea was 124.1 mg/dl in group I and 28.4 mg/dl in group II. The mean serum creatinine was 6.5 mg/dl in group I and 0.92 mg/dl in group II.Phukan et al[14]evaluated the serum lipidprofile in CKD patients and to find the pattern of its alteration inboth haemodialyzed and conservatively treated CKD patients.Seventy- one randomly selected CKDpatients attending a tertiary care hospital of Assam during oneyear of time frame (40 haemodialyzed and 31 conservativelytreated) along with 50 apparently healthy controls were includedin the study. Test for serum lipid profile, urea creatinine, FBS,PPBS, total protein and albumin were carried out in all the casesand controls. Triglyceride level (TGL) of CKD group

157.88±61.82,controls 96.98±37.52, very low-density lipoprotein (VLDL) ofCKD group 31.58±12.36, controls 19.39±7.50 was marginallyelevated and high-density lipoprotein (HDL) of CKD group33.40±9.06, controls 45.95±10.35 was significantly reduced inthe patient group as compared to the controls and the resultswere statistically highly significant with p-value<0.001. Totalcholesterol (CKD group 128.2±53.57, controls 142.53±31.44)and LDL (CKD group 63.23±46.47, controls 77.35±26.81)were lower in the patient group as compared to the controls,however the difference was statistically not significant (pvalue 0.09 and 0.059 respectively). There was no statisticallysignificant difference of lipid profile between hemodialyzed andconservatively treated CKD groups and there was no genderrelated variation of lipid profile too.

CONCLUSION

Authors found that increased triglycerides, increased VLDL and reduced HDL are responsible for increased cardiovascular complications in patients with CKD.

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