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Original article:

A Study of assessment of Dyslipidemia in undialysed chronic kidney disease patients

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Abstract:

Introduction- Patients with chronic kidney disease (CKD) are at an increased risk for cardiovascular disease and have a higher prevalence of hyperlipidaemia (or dyslipidaemias) than the general population. Therefore, it is important to screen all patients with CKD for dyslipidaemias and treat them appropriately as they are considered "a very high- risk" group for CVD.

Material & method- We studied lipid profile status and Biochemical markers of renal function in undialysed chronic kidney disease patients and healthy controls. 25 cases and similar number of age and sex matched control are included in study.

Conclusion- In our study the lipid abnormality found was Increased Triglyceride, serum cholesterol, VLDL and Decreased HDL which was statistically significant.

Keywords: chronic kidney disease, Hemodialysis, Lipid profile.

INTRODUCTION

Patients with chronic kidney disease (CKD) are at an increased risk for cardiovascular disease and have a higher prevalence of hyperlipidaemia (or dyslipidaemias) than the general population[1-2]. In patients with pre-existing CVD, the presence of CKD is associated with an increased risk of recurrent cardiovascular events[3].The majority (58%) of patients die from cardiovascular causes, making CVD the leading cause of death in patients with CKD[4].Indeed, even mild renal insufficiency has been shown to be associated with increased rates of cardiovascular events[5-6]. Furthermore, patients on dialysis have 10 to 20 times higher cardiovascular mortality rates than the general population[7]. Therefore, it is important to screen all patients with CKD for dyslipidaemias and treat them appropriately as they are considered "a very high- risk" group for CVD.

As reduced GFR is by itself associated with hyperlipidaemia, the strong and independent correlation between proteinuria and reduced GFR also increases the risk of hyperlipidaemia and proteinuria[8]. Conversely, proteinuria has an inverse correlation with the level of HDL cholesterol[9]. Hyperlipidaemia and Progression of Kidney Disease It has long been suggested that hyperlipidaemia could cause renal injury and contribute to the progression of renal disease[10]. Most studies have been small and a meta-analysis of these studies to assess the effect of lipid reduction on the progression of renal disease has shown that lipid reduction may preserve GFR and reduce proteinuria[11]. More

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recent studies have shown that HMG- CoA reductase inhibitors (statins) can reduce proteinuria and slow the decline in renal function[11-14].

MATERIAL AND METHODS

The study was hospital based case control observational study conducted in upgraded department of medicine JLN Medical College Ajmer. We studied lipid profile status and Biochemical markers of renal function in undialysed chronic kidney disease patients and healthy controls. 25 cases and similar number of age and sex matched control are included in study. The chronic kidney disease patients visiting medicine OPD and medicine ward were screened for lipid profile. Subjects were included in study after taking informed consent.

INCLUSION CRITERIA -

Patients of age ≥18 years having established chronic kidney disease and not on dialysis.

EXCLUSION CRITERIA

1. Age <18 years
2. Patients on lipid lowering drug
3. Patients of thyroid disorder
4. Pregnancy
5. Any History of dialysis
6. Diabetes mellitus
7. Obese
8. Alcoholic
9. Smoker
10. Refusal to give consent

GFR calculated by-

Estimated GFR (ml/min per $1.73m^2$) = $1.86 \times (cr/88.4)^{-1.154} \times (Age)^{-0.203}$

(multiply by 0.742 for female) .

Informed consent was obtained from all study participants and ethics committee of our tertiary care hospital approved the study.

Data Analysis: Relationship between Serum Lipid Profile and Biochemical Markers of renal function is determined by using -

1. Chi-square test
2. Coefficient of correlation

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OBSERVATION AND DISCUSSION

Table 1Distribution According to Age

Group	N	Mean	SD	Minimum	Maximum	P Value LS
case	25	43.96	14.273	18	66	
control	25	45.04	13.945	20	70	0.788 NS

All Cases and controls are age matched.

Table 2 Distribution According to Sex

Sex	Cases		Controls		Total
	No	%	No	%	No
F	8	32	8	32	16
M	17	68	17	68	34
Total	**25**	**100**	**25**	**100**	**50**

All cases and controls are sex matched.

. **Table No. 3Distribution of the S. UREA (mg/dl) among the Groups**

S.UREA(mg/dl)						
GROUP	**N**	**Mean**	**SD**	**Minimum**	**Maximum**	**P Value LS**
Case	25	132.37	79.338	45	315	
Control	25	26.04	8.590	14	44	<0.001S

The mean level of S. Urea in cases was significantly higher than controls (p<0.001).

Table No. 4Distribution of the S.Creatinine (mg/dl) Among the Groups

S CREATININE(mg/dl)						
GROUP	**N**	**Mean**	**SD**	**Minimum**	**Maximum**	**P Value LS**
Case	25	3.272	0.9563	1.7	5.2	
Control	25	0.757	0.2144	0.4	1.1	<0.001S

The mean level of S.Creatinine in Case was significantly higher than controls (p<0.001).

Table No. 5Distribution of the Mean TG (mg/dl) among the groups

TG (mg/dl)						
Group	**N**	**Mean**	**SD**	**Minimum**	**Maximum**	**P Value LS**
Case	25	230.92	72.808	120	380	
Control	25	115.92	29.433	66	167	<0.001S

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The mean level of TG in cases was significantly higher than controls (p<0.001).

Table No. 6 Distribution of the S.TG (mg/dl) Among the Groups

TG (mg/dl)[2]	Case		Control		Total
	No	%	No	%	
Abnormal (>150)	20	80	1	4	21
Normal (≤150)	5	20	24	96	29
Total	**25**	**100**	**25**	**100**	**50**

Proportion of the cases of abnormal S.TG level was significantly more in cases as compared to controls (p<0.001).

Table No. 7 Distribution of the HDL (mg/dl) among the groups

HDL(mg/dl)						
Group	**N**	**Mean**	**SD**	**Minimum**	**Maximum**	**P Value LS**
Case	25	30.84	5.444	20	42	0.001S
Control	25	37.36	4.508	30	44	

The mean level of HDL in Cases was significantly lower than controls (p<0.001).

HDL (mg/dl)	Case		Control		Total
	No	%	No	%	
Low (<40mg/dl)	24	96	15	60	39
Normal (≥40mg/dl)	1	4	10	40	11
Total	**25**	**100**	**25**	**100**	**50**

Proportion of the cases low HDL level was significantly more in cases as compared to controls (p<0.01).

Table No. 8 Distribution of the VLDL (mg/dl) Among the Groups

VLDL(mg/dl)						
Groups	**N**	**Mean**	**Std. Deviation**	**Minimum**	**Maximum**	**P Value LS**
Case	25	31.311	18.83	10.0	105.0	
Control	25	22.200	4.96	15.0	31.0	0.024 S

The mean level of VLDL in Case was significantly higher than controls (p=0.024).

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Table No. 9 Distribution of the T. Cholesterol (mg/dl) among the groups

T. CHOLESTEROL (mg/dl)						
Group	**N**	**Mean**	**SD**	**Minimum**	**Maximum**	**P Value LS**
Case	25	295.04	96.29	100	429	0.001 S
Control	25	158.40	38.09	111	250	

The mean level of T. Cholesterol in cases was significantly higher than controls (p=0.001).

T. CHOLESTEROL (mg/dl)2	**Case**		**Control**		**Total**
	No	**%**	**No**	**%**	**No**
Abnormal(>200mg/dl)	19	76	3	12	22
Normal (<200mg/dl)	6	24	22	88	28
Total	**25**	**100**	**25**	**100**	**50**

Proportion of the cases of high T. Cholesterol level was significantly more in cases as compared to controls (p<0.001).

Table No. 10 Distribution of the T. LDL (mg/dl) among the groups

LDL(mg/dl)						
Groups	**N**	**Mean**	**SD**	**Minimum**	**Maximum**	**P Value LS**
Case	25	59.96	31.759	26	129	
Control	25	75.08	25.722	48	168	0.105 NS

There was no significant difference in mean LDL levels between cases and controls
(p=0.105).

LDL(mg/dl)	**Case**		**Control**		**Total**
	No	**%**	**No**	**%**	**No**
Abnormal (>!30mg/dl)	0	0	1	4	1
Normal (<130mg/dl)	25	100	24	96	49

No significant difference was observed according to proportion of the cases in LDL (mg/dl) (p=1.00).

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Table No. 11 Distribution eGFR (ml/min) per 1.73m^2 among the groups

eGFR (ml/min) per 1.73m^2						
Group	**N**	**Mean**	**SD**	**Minimum**	**Maximum**	**P Value LS**
Case	25	27.10	8.80	14.9	44.1	
Control	25	143.72	41.09	90.9	218.0	0.001 S

Mean eGFR (ml/min) per 1.73 m^2 was significantly higher in controls as compared to cases (p=0.001).

CONCLUSION

Patients with CKD are at high risk of developing cardiovascular disease and they have a higher prevalence of dyslipidaemias compared to the general population. Most CKD patients do not develop kidney failure but die as a result of CVD. It is recognised that CVD begins in the early stages of CKD. Therefore, it is important not only to identify these patients early but also to treat their dyslipidaemias intensively before they develop end-stage renal disease. Most patients will require lifestyle modification and lipid-lowering therapy (statins). In our study the lipid abnormality found was Increased Triglyceride, serum cholesterol, VLDL and Decreased HDL which was statistically significant.

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