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# **ORIGINAL RESEARCH**

# Correlation of HbA1c and dyslipidemia in patients of CKD

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## Abstract

**Background**: The frequency of chronic kidney disease (CKD), a serious health issue, is rising globally, especially in those with metabolic diseases such dysglycemia disorders. The glycated hemoglobin concentration (HbA1c) can reveal dysglycemia diseases, such as the prediabetic state, while also serving as a valuable indicator of mean blood glucose levels during the previous three months. Although it is not always present, dyslipidemia (DLP) is a frequent consequence of increasing renal disease. Elevated triglyceride levels, low HDL cholesterol, and small, dense LDL particle size are characteristics of DLP, a renal and cardiovascular risk factor.

Aim: The present study was carried out to investigate the association of CKD with HbA1c, dyslipidemia and electrolytes in pts. of renal failure.

**Methods and materials**: It was a hospital based case-control study. The study was done on 109 patients of CKD as cases and 105 patients as controls who did not have CKD.By using an HPLC (High-Performance Liquid Chromatography) system with affinity columns to separate HbA1c molecules from other hemoglobin molecules, glycated hemoglobin (HbA1c) measurement has been carried out. Based on the proportion of HbAlc peak area to all hemoglobin peak areas, the HbAlc content is determined. Enzymatic color test was used for the quantitative determination of HDL-cholesterol in human serum and plasma on OLYMPUS analyzer.

**Results:** Mean HbA1c of cases is 7.32 (SD 2.82) and control is 5.46 (SD 1:78). Difference is significant p value less than 0.0001.58 CKD patients (53.21%) had HbA1c > 5.6 compared to 36 controls (34.29%) which is significant with p value 0.0060, considered very significant, 95% Confidence Interval 1.256 to 3.783 and odds ratio 2.180.47 (43.12%) CKD patients were diabetic compared to 21(20%) controls as per HbA1c level  $\geq$ 6.5 with p value 0.0004, considered extremely significant, 95% Confidence Interval: 1.647 to 5.583, odds ratio 3.032.In our study, dyslipidemia was present in 43.12% (n=47) among CKD cases and in 26.67% (n=28) among control group.Using Fisher's Extract test, with 95% Confidence Interval (CI): 1.173 to 3.706, Odds Ratio - 2.085, the p value is 0.0147, considered significant. So, dyslipidemia was present in significant number of CKD patients as compared to control groups.

**Conclusion**: In our study high HbA1c were significantly associated with CKD patients compared to non-CKDindividuals. Prevalence of dyslipidemia was significantly higher in CKD group compared to control group in the form of increased TG, LDL and low

HDL.According to our country's trend, we have got relevant results indicating emergent epidemics of diabetes, hypertension.

Keywords: CKD, HbA1c, Dyslipidemia.

#### Introduction

A spectrum of various pathophysiologic processes, impaired kidney function, and a steady fall in glomerular filtration rate (GFR) are all included in chronic kidney disease (CKD). The frequency of CKD, a serious health issue, is rising globally, especially in those with metabolic diseases such dysglycemia disorders. The glycated hemoglobin concentration (HbA1c) can reveal dysglycemia diseases, such as the prediabetic state, while also serving as a valuable indicator of mean blood glucose levels during the previous three months.<sup>1-4</sup>

The most frequent cause of end stage renal disease (ESRD), diabetic nephropathy. Strict glycemic control slows down the onset and progression of diabetic problems, and there is evidence that diabetic patients with advanced CKD benefit from better metabolic control. Dialysis and renal failure might have an impact on the homeostasis of glucose and insulin. The accuracy of HbA1c in the presence of CKD and ESKD is called into doubt, and CKD has an impact on the validity of measures of long-term glycaemia management. Recent studies have concentrated on the reliability of indicators of longer-term glycaemia management. The daily finger stick blood test used to check blood sugar levels is different from the HbA1c test. The target HbA1c value for someone with diabetes is 7%. A normal level for someone who does not have diabetes is 4-5.9%.<sup>5-7</sup>

Although it is not always present, dyslipidemia (DLP) is a frequent consequence of increasing renal disease. Elevated triglyceride levels, low HDL cholesterol, and small, dense LDL particle size are characteristics of DLP, a renal and cardiovascular risk factor. High cardiovascular morbidity and mortality of CKD are attributed to dyslipidemia. Small lipoproteins are created as a result of the oxidative alteration that LDH and HDL particles go through, and the generation of oxidized LDL is increased.<sup>8-12</sup>

The management of all lipid disorders should start with therapeutic lifestyle changes (TLCs), according to the Executive Summary of the Third Report of the National Cholesterol Education Program [NCEP] Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III) guidelines. The fact that the TLCs may lessen cardiovascular risk through mechanisms other than reducing LDL cholesterol. LDL-C has been identified by ATP III as the main target of cholesterol-lowering treatment. Obtaining lipoprotein values requires 9–12 hours of fasting. There have been very few studies which have collectively analyzed the association of CKD with HbA1c, dyslipidemia and electrolytes in patients of renal failure.<sup>13-17</sup>This study was conducted to evaluate association of CKD with HbA1c, dyslipidemia and electrolytes in patients of renal failure.

#### **Methods and Materials**

It was a hospital-based case-control study. The study was done on 109 patients of CKD as cases and 105 patients as controls who did not have CKD. Consecutive adult patients (>20 years of age) of both sexes. Cases are defined as patients (>20 years of age) with a diagnosis of CKD. The controls are defined as age and gender-matched patients (>20 years) without diagnosis of CKD.

#### Inclusion criteria

#### All case patients as defined -

CKD encompasses a spectrum of different pathophysiological processes associated with abnormal kidney function and a progressive decline in glomerular filtration rate (GFR).

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#### **Exclusion criteria**

A. Patients <20 years

B. Unwilling Patients

## **Estimation of lipid profile**

Serum was analyzed for lipid profile by a semi-autoanalyzer machine.

## Estimation of total cholesterol level (CHOD/PAP method)

It is done by using a cholesterol kit which contains -

- (1) Enzyme Reagent 1(L1).
- (2) Enzyme Reagent 2 (L2)
- (3) Cholesterol Standard (S) 200mg/dl.

## Estimation of triglyceride level

It was estimated using ENZOPAK Triglycerides (DST) in GPO method.

## **Estimation of HDL- Cholesterol level**

Enzymatic color test for the quantitative determination of HDL-cholesterol in human serum and plasma on OLYMPUS analyzer.

### Estimation of LDL and VLDL-Cholesterol levels

Low density lipoprotein cholesterol (LDL-C) and very low- density lipoprotein cholesterol (VLDL-C) was calculated using formulas that is VLDL-C = TG/5, LDL-C = TC- (HDL-C + VLDL-C).

#### **Estimation of HbA1C**

By using an HPLC (High-Performance Liquid Chromatography) system with affinity columns to separate HbA1c molecules from other hemoglobin molecules, glycated hemoglobin (HbA1c) measurement has been carried out. Based on the proportion of HbA1c peak area to all hemoglobin peak areas, the HbA1c content is determined.

## **Estimation of Urea and Creatinine**

GLDH Kinetic method for the determination of urea in serum.

## Estimation of serum creatinine

It was done by Jaffe's Alkaline Picrate Method. Creatinine in alkaline medium reacts with picric acid to form a red tautomer of creatinine picrate the intensity of which is measured at 520nm.

## Estimation of Creatinine Clearance by MDRD formula

The creatinine clearance rate (CCr or CrC1), which is used to calculate the GFR, is the amount of blood plasma cleared of creatinine per unit of time. Any substance that has a constant level in the blood is readily filtered, but neither reabsorbed by the kidneys nor released by them was used to determine glomerular filtration rate (GFR). The amount of the material in the urine that came from a calculable volume of blood is consequently the rate that is being measured. However, in clinical settings, GFR is assessed using estimations of creatinine clearance based on serum creatinine levels or actual creatinine clearance. The most used formula is the "4-variable MDRD," which estimates GFR using four variables: serum creatinine, age, race, and gender.

eGFR = 1.86 serum creatinine  $^{-1.154}$  x Age  $^{-0.203}$  x [ 1.212 if Black] x [0.742 if Female].

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It calculates GFR adjusted to body surface area and is based on the subject's age, race, and gender as well as serum creatinine levels. Transplant recipients are among the populations where the MDRD equation has been independently verified.

#### Ultra-sonography (USG) for detection of CKD

The kidneys are tiny and the renal parenchyma is thin in chronic renal failure. Although US can accurately measure renal length, it cannot be utilized to determine the origin of renal scarring because it only displays renal scars without the calyces in connection to them. The most frequent anomaly is a widespread rise in cortical reflectivity above that of the nearby liver. Pyramids look apparent because to enhanced cortico-medullary differentiations.

#### **Statistical Analysis**

Following the collection of all the data, master charts were created using Microsoft Office Excel 2007 and SPSS 21 was used for statistical analysis. For each group, the mean and standard deviation (SD) were computed independently. To determine differences between the two groups' mean values for various parameters, an unpaired (two-tailed) Student's t-test was used. By using univariate analysis, the proportions of the HbA1c and dyslipidemia components were compared between case and control individuals. P-values under 0.05 were considered significant.

#### Results

Majority of male cases were in age group of 36-50 years and 66- 80 years, followed by in 51-65 years age group and most of female cases were of 51-65 years of age group. (Table 1, figure 1) In our study, we found majority of cases are of CKD stage 4 (38.53%), followed by CKD stage 5 ESRD (30.27%). (Table 2, figure 2)Proteinuria was defined as an albumin to creatinine ratio of 30 mg/g or greater (which includes both the categories of microalbuminuria and macroalbuminuria). In our study, among 109 cases of non-edematous CKD proteinuria was found in 52 cases. According to staging criteria of CKD, 4 case of stage 1, 14 cases of stage 2, 9 cases of stage 3, 13 cases of stage 4 and 12 cases of stage 5 had proteinuria. (Figure 3).History of smoking was present in 33.94% (n=37) cases as compared to 19.05% (n=20) controls. (Table 3).

Mean HbA1c of cases is 7.32 (SD 2.82) and control is 5.46 (SD 1:78). Difference is significant p value less than 0.0001.58 CKD patients (53.21%) had HbA1c > 5.6 compared to 36 controls (34.29%) which is significant with p value 0.0060, considered very significant, 95% Confidence Interval 1.256 to 3.783 and odds ratio 2.180.47 (43.12%) CKD patients were diabetic compared to 21(20%) controls as per HbA1c level  $\geq$ 6.5 with p value 0.0004, considered extremely significant, 95% Confidence Interval: 1.647 to 5.583, odds ratio 3.032. (table 4,figure4)

In our study, dyslipidemia was present in 43.12% (n=47) among CKD cases and in 26.67% (n=28) among control group.Using Fisher's extract test, with 95% Confidence Interval (CI): 1.173 to 3.706, odds ratio - 2.085, the p value is 0.0147, considered significant. So, dyslipidemia was present in significant number of CKD patients as compared to control groups.

#### **Total Cholesterol**

In our study, case group had mean total cholesterol (T.Ch.) levelof 178.76 mg% (SD 40.50 mg %) and control group had mean T. Ch. 178.56 mg% (SD 32.40 mg%). The difference of mean is not significant(p value = 0.97). Among the CKD cases, 28.44% (n=31) had T.Ch.>200 mg%

compared to 25.71% (n=27) in controls. Is it not significant value 0.7586 odd ratio 1.148, 95% CI 0.6276 to 2.101.

## Triglyceride

In this study, case group had mean triglyceride (TG) level of 164.03 mg% (SD 54.03 mg%) and control group had mean TG 132.39mg% (SD 51.41 mg%). The difference of mean is significant (p value less than 0.0001). Among the CKD cases, 36.70% (n=40) had TO > 150 mg% compared to 20% (n=21) in controls. Significant, p value 0.0097, odd ratio 2.319, CI 1.251 to 4.297.

## High density Lipoprotein (HDL)

In our study, case group had mean HDL levels of 43.51 mg% (SD 10.02 mg%) and control group had mean 47.45 mg% (SD 9.07 mg%). The difference of mean is significant (p value = 0.0029). Among the CKD cases, 40.37% (n=44) had low HDL according to sex (< 40 in case of male and < 50 in case of female), compared to 23.81% (n=25) in controls. Significant, p value 0.0127 odd ratio 2.166, 95% CI 1.201 to 3.908.

## Low Density Lipoprotein (LDL)

In this study, case group had mean LDL level of 125.06 mg% (SD 56.51 mg %) and control group had mean 99.59 mg% (SD 39.69 mg %). The difference of mean is significant (pvalue=0.0002). LDL>100 mg/dl was present in 46 CKD patients (42.20%) compared to 24 (22.86%) in control group. Significant no. of CKD patients had high LDL, p value 0.0034, odd ratio 2.464, 95% CI 1.361 to 4.461.

So, we found statistically significant Dyslipidemia in CKDpatients as compared to non-CKD age and sex-matched control group. (table 5, figure 5,6)

Age group	Male (case)	Female (case)	Male (control)	Female (control)
21-35	4	6	7	9
36-50	23	9	20	11
51-65	20	19	21	12
66-80	22	6	13	12
Total	69	40	61	44

## Table 1: Age and sex-wise distribution of case and control groups

#### Table 2: Percentage Distribution of stages of CKD in case group

Stage	Stage 1	Stage2	Stage 3	Stage 4	Stage 5
	(CFR <u>&gt;</u> 90)	(CFR=60-89)	(CFR=30-59)	(CFR=15-29)	(CFR<15)
Cases (CKD)	2(1.83%)	12 (11.01%)	20(18.34%)	42(38.53%)	33 (30.27%)

#### **Table 3: History of Smoking**

Stage	Male case	Female case	Total case	Control
Smoker	34(31.19%)	3(2.75%)	37(33.94%)	20(19.05%)
Non- smoker	32(29.36%)	40(36.70%)	72(66.06%)	85(80.95%)

#### Table 4: Distribution of HbA1c in Subcategories among case and control

Hb1AC	Normal (≤5.6)	Impaired (5.7-6.4)	Diabetic (≥6.5)
Case	51(46.79%)	11(10.09%)	47(43.12%)

	Control	69(65.71%)	15(14.29%)	21(20%)	
Table 5: I	Distribution	of Dyslipidemia i	n Subcategories amon	g case and in cont	rol

Dyslipidemia	Case	Control	
Present	47 (43.12%)	28 (26.67%)	
Absent	62 (56.88%)	77 (73.33%)	













#### Discussion

Although CKD, diabetes, dyslipidemia is quite common in India, lack of detailed study on their association from our country prompted us to undertake the present study. In this study, we proposed to determine the association between dyslipidemia, dysglycemia & CKD in patients in Indian population. By using statistical analysis, we can now extrapolate the data to the Indian population.

In our study, we included a total of 109 patients as cases (non-edematous CKD) and 105 ageand sex-matched controls. There were no significant differences in gender between the groups. The mean age of the patient group was 54.25 years (standard deviation [SD] 14.73 years, range 22-80 years) and that of the control group was 51.58 years (SD 15.59 years, range 21-80 years). There were no significant differences in the mean age and the distribution of age (p=0.199). Levin et al<sup>18</sup> in astudy described ageing as a non-regulatory factor for the development of CKD. Majority of male cases were in age group of 36-50Years and 66-80 Years and female cases were of 51-65 years.History of alcoholism was present in 27.52% cases as compared to 30.48% in controls which is not significant. But history of smoking was present in 33.94% cases as compared to 19.05% controls and their associations were significant. In the CHOICE study, about 28% of the patients were current smokers on dialysis. Orth SR et al<sup>19</sup> in their study showed the association of smoking with progression of kidney disease and of CVD in patients with reduced GFRsBryson CL et a1<sup>10</sup> in a cross-sectional study showed that the adjusted risk for having a GFR 60 ml/mm per 1.73 m<sup>2</sup> was approximately two-fold higher in current smokers compared withthose who never smoked.

In our study we found significant difference in HbA1c level between cases and controls (p<0.0001).58 CKD patients (53.21%) had HbA1c>5.6 compared to 36 controls (34.9%), which is significant with p value 0.006, odd ratio =2.180, 95% CI= 1.256-3.783.47 CKD

patients (43.12%) were diabetic compared to 21 controls (20%) as per HbA1c level more than 6.5with p value 0.0004, considered extremely significant,odd ratio = 3.032, 95% CI=1.647-5.583.Tonelli et al<sup>20</sup> reported that about 38.5% of CKD patients had diabetes mellitus and the presence of diabetes was associated withCVD in CKD stages 1 through 4. Doyle M. Cummings et al. showed the strongest predictor of change in eGFR was the proportion of HbAlc values >7%.

A previous study showed long-term glycemic variability expressed by SD of HbA1c predicted development of renal and cardiovascular complications.

In our study, we found statistically significant difference in lipid profile of CKD patients compared to control group. Dyslipidemia was present in 43.12% (n=47) among CKD cases and in 26.67% (n=28) among control group (p = 0.0147, odd ratio 2.085, 95% CI-1.173-3.706). We found significant higher values of triglyceride (p < 0.0001), HDL (p=0.0029) and LDL (p=0.0002) among CKD patients. There was no statistically significant difference in Total Cholesterol values of CKD and control population (p=0.97).

Sowers JR<sup>25</sup>et al observed that in chronic kidney disease total cholesterol and low-density lipoprotein (LDL) cholesterol, the routine lipid parameters, are usually in the high, normal range or even low. Main dyslipidemic disturbance is hypertriglyceridemia. It is already seen in early stages of CKD and present in up to 70% of ESRD patients, but hemodialysis tends to improve triglyceridemia at least in nondiabeticpatients. Low HDL cholesterol has been ascribed to low activity of lecithin-cholesterol acyltransferase which normally increases the uptake of esterified cholesterol by HDL. The change in HDL concentration is favored by the common presence of microinflammation in uremic patients.

In the MMKD study (Mild and Moderate Kidney Disease) 227 patients with primary kidney disease were followed for 7 years, measuring baseline, and assessing doubling of serum creatinineand/or ESRD as the endpoint. The study documented a progressive decrease of HDL cholesterol in the earliest stages of primary kidney disease accompanied by a continuous increase in triglycerides and a significant decrease of average Lipoprotein (A).

Indian studies on lipid profile in CRF have not beenconsistent. Sharma et al<sup>21</sup> and Kunde et al<sup>22</sup> observed no hyperlipidemia in patients of CRF. On the other hand, Gupta R<sup>23</sup> et al and Das DS et al<sup>24</sup>observed lipid abnormalities similar to those reported in Western studies i.e., hypertriglyceridemia and reduced high density lipoprotein (HDL). A previous study demonstrated that CRF is commonly accompanied by lipid abnormality in the form of hypertriglyceridemia. This is like the observations made in Western studies and recent Indian studies. This lipid abnormality could be due to increased hepatic synthesis of VLDL triglycerides and/or defective triglyceride removal.

Thus, considering the more atherogenic lipid profile in Asian ethnic population, the significant link of dyslipidemia with CKD revealed in the present study is well validated and comparable to the data found in several studies in other ethnic population.

## Conclusion

In our study high HbA1c were significantly associated with CKD patients compared to non-CKDindividuals. Prevalence of dyslipidemia was significantly higher in CKD group compared to control group in the form of increased TG, LDL and low HDL.According to our country's trend, we have got relevant results indicating emergent epidemics of diabetes, hypertension

## References

1. Fortu no, O. Beloqui, G. San Jos'e, M. U. Moreno, G. Zalba, and J. D'ez, "Increased phagocytic nicotinamide adenine dinucleotide phosphate oxidase-dependent superoxide production in patients with early chronic kidney disease," *Kidney International*. *Supplement*, no. 99, pp. S71-S75, 2005.

- 2. ADVANCE Collaborative Group; Patel A, MacMahon S, Chalmers J, et al. Intensive blood glucose control and vascular outcomes in patients with type 2 diabetes. *N Engl J Med*. 2008; 358:2560-2572.
- 3. Aneja A, El-Atat F, McFarlane SI, et al. Hypertension and obesity. *Recent Prog Horm Res* 2004;59: 169-205. [PubMed:14749502].
- 4. Anoop Mishra, Lokesh Khurana. The Metabolic Syndrome in South Asians: Epidemiology, Determinants, and Prevention. *Metabolic Syndrome and Related Disorders* 2009; 7:497-514.
- 5. Bagdade J, Casaretto A. Effect of chronic uremia, hemodialysis and renal transplantation on plasma lipids and lipoproteins. J Clin Invest 1976; 87:37 41.
- 6. Becker B, Kronenberg F, Kielstein JT, et al. Renal insulin resistance syndrome, adiponectin, and cardiovascular events in patients with kidney disease: the mild and moderate kidney disease study. *J Am Soc Nephrol*. 2005; 16: 1091-1098.
- Beddhu S, Pappas LM, Ramkumar N, Samore M. Effects of body size and body composition on survival in hemodialysis patients. *J Am Soc Nephrol* 2003; 14:2366-2372. (PubMed: 129373151.
- 8. Bojestig M, Arnqvist HJ, Hermansson G, et al: Decliningincidence of nephropathy in insulin-dependent diabetes mellitus.*N EnglJMed* 1994; 330(1):15-18.
- 9. Borch-Johnsen K: The prognosis of insulin-dependent diabetes mellitus. An epidemiological approach. *Dan Med Bull* 1989; 39:336-349.
- 10. Bryson CL, Ross HJ, Boyko EJ, Young BA: Racial and ethnicvariations in albuminuria in the US Third National Health andNutrition. Examination Survey (NHANES III) population:Associations with diabetes and level of CKD. *Am J Kidney Dis*48:720-726, 2006).
- 11. Buller CE, Nogareda JG, Ramanathan K, et al: The profileof cardiac patients with renal artery stenosis. *J Am Coil Cardiol*2004; 43(9):1606-1613.
- 12. Zoccali, "Overweight, obesity and metabolic alterationsin chronic kidney disease," *Prilozi*, vol. 30, no. 2, pp. 17-31,2009.
- 13. Chan MK, Varghese Z, Moorhead JF. Lipid abnormalities in uremia. *Kidney Int*1981; 19:625 637.
- 14. Chen J, Muntner P, Hamm LL et al. The metabolic syndromeand chronic kidney disease in U.S. adults. *Ann Intern Med*2004; 140: 167-174.
- 15. Chen J, Muntner P, Hamm LL, et al: The metabolic syndromeand chronic kidney disease in U.S. adults. *Ann Intern Med* 2004;140(3):167-174.
- 16. Chmielewski M, Carrero JJ, Nordfors L, et al. Lipiddisorders in chronic kidney disease: reverse epidemiology andtherapeutic approach. *J Nephrol.* 2008; 21: 635-644.
- 17. Chmielewski M, Carrero JJ, Nordfors L, et al. Lipiddisorders in chronic kidney disease: reverse epidemiology and therapeutic approach. *J Nephrol.* 2008; 21: 635-644.
- 18. Levin A. Identification of patients and risk factors in chronic kidney disease-evaluating risk factors and therapeutic strategies. *Nephrol Dial Transplant* 2001; 16(Suppl 7): 57-60.
- 19. Orth SR, Stockmann A, Conradt C, et al: Smoking as a risk factor for end-stage renal failure in men with primary renal disease. *Kidney Int* 1998; 54(3):926-931.
- 20. Tonelli M, Bohm C, Pandeya S, Gill J,Gill J, Levin A, Kiberd BA. Cardiac risk factors and the use of cardioprotective medications in patients with chronic renal insufficiency. *Am J Kidney Dis*2001; 37:484-489.
- 21. Sharma BK, Jindal SK, Rana DS. Absence of hyperlipidemia in patients of chronic renal failure in Chandigarh. *Indian J Med Res* 1980; 72:461 464.
- 22. Kunde Mani MK, Kuruvilla KC. Lipid abnormality in chronic renal failure and hemodialysis. *J Assoc Physicians India* [abstract] 1977; 25:1013

- 23. Gupta R, Kaul V, Bhagat N; et al. Trends in prevalence of coronary risk factors in an urban Indian population: Jaipur Heart Watch-4.Indian Heai4 J 2007: 346-53.
- 24. Das BS, Mishra SK, Rao DVP. Serum lipids in chronic renalfailure. J Assoc Physicians India 1984; 32: 1019 1021
- 25. Sowers JR. Metabolic risk factors and renal disease. KidneyInt. 2007; 71:719-20.