



EVALUATION OF RISK FACTORS FOR CARDIOVASCULAR DISEASE IN PATIENTS OF CHRONIC KIDNEY DISEASE.

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Abstract

Background: **Objectives:** To evaluate the prevalence and pattern of major cardiovascular risk factors among patients with chronic kidney disease (CKD) and compare them with age- and sex-matched healthy controls. **Materials and Methods:**

This hospital-based prospective comparative observational study was conducted at Mahatma Gandhi Medical College & Hospital, Jaipur, over a period of 18 months. A total of 600 participants were enrolled, including 300 patients diagnosed with CKD and 300 age- and sex-matched healthy controls. Clinical assessment and laboratory investigations were performed to evaluate cardiovascular risk factors including hypertension, dyslipidaemia, hypertriglyceridaemia, albuminuria, and estimated glomerular filtration rate (eGFR). Statistical analysis was undertaken to compare the prevalence of risk factors between the two groups. **Results:** Hypertension was significantly more prevalent among CKD patients than controls (62% vs. 11%; OR = 13.2, $p < 0.001$). Dyslipidaemia was observed in 50% of cases compared to 32% of controls (OR = 2.13, $p < 0.001$), while hypertriglyceridaemia was present in 29% of cases and 4% of controls (OR = 9.8, $p < 0.001$). Albuminuria was markedly higher among CKD patients, with microalbuminuria present in 55% versus 21.33% of controls and macroalbuminuria in 21.67% versus 0%, respectively. Mean eGFR was significantly lower in the CKD group compared with controls (43.3 vs. 91.08 mL/min/1.73 m²; $p < 0.001$). **Conclusion:** CKD is associated with a substantially increased burden of cardiovascular risk factors, including hypertension, dyslipidaemia, hypertriglyceridaemia, and albuminuria. These findings reinforce the recognition of CKD as a high cardiovascular risk state and emphasize the need for early detection, routine cardiovascular risk assessment, and aggressive risk factor modification to reduce morbidity and mortality.

Keywords: Chronic kidney disease, cardiovascular disease, risk factors, hypertension, dyslipidaemia, albuminuria, glomerular filtration rate.



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INTRODUCTION

Chronic kidney disease (CKD) is a major global public health concern, affecting nearly 15–20% of adults worldwide, typically defined by a reduced glomerular filtration rate (GFR) or the presence of albuminuria.¹ Among its many complications, cardiovascular disease (CVD) is the most clinically significant, accounting for the majority of morbidity and mortality in this population.^{1, 2} While CVD coexists with normal kidney function in approximately 37.5% of the general population, this prevalence rises to nearly 75.3% in patients with CKD Stage 4, underscoring the disproportionate cardiovascular burden in this group.¹

CKD is now recognised as an independent risk factor for CVD beyond traditional determinants such as hypertension, diabetes mellitus, dyslipidaemia, smoking, and obesity.^{2, 3} Disease-specific mechanisms include oxidative stress, endothelial dysfunction, chronic inflammation, accelerated vascular calcification, accumulation of uraemic toxins, anaemia, and persistent activation of the renin–angiotensin–aldosterone system (RAAS) and sympathetic nervous system.^{4, 5} These mechanisms collectively promote uraemic cardiomyopathy, left ventricular hypertrophy, myocardial fibrosis, arrhythmias, heart failure, and sudden cardiac death.⁵

Emerging evidence highlights the prognostic value of serum creatinine-based eGFR and albuminuria in predicting cardiovascular outcomes.³ Both declining eGFR and rising albuminuria independently correlate with adverse cardiovascular events.⁶ Individuals with CKD Stages G3a–G4 (eGFR 15–60 mL/min/1.73 m²) carry a two- to three-fold higher risk of cardiovascular mortality.^{7,8} Early detection, risk stratification, and aggressive cardiovascular risk management are therefore essential in this population.

Aim and objectives

Aim

To study the risk factors for cardiovascular disease in patients with chronic kidney disease.

Objectives

- To assess the prevalence of major cardiovascular risk factors—including hypertension, diabetes mellitus, dyslipidaemia, and obesity—in patients with chronic kidney disease.
- To compare cardiovascular risk factor profiles between CKD patients and healthy controls.

To evaluate the association of CKD stage with cardiovascular risk factors.

MATERIALS AND METHODS

Study Setting and Design

This hospital-based prospective comparative observational study was conducted in the Department of General Medicine at Mahatma Gandhi Medical College & Hospital, Jaipur, Rajasthan, over 18 months from the date of Institutional Ethics Committee (IEC) approval. Written informed consent was obtained from all participants, and confidentiality of patient information was strictly maintained throughout.

Study Population and Sample Size

A total of 600 participants were enrolled: 300 CKD patients (cases) and 300 age- and sex-matched healthy individuals (controls). The sample size was calculated at a 90% confidence level with a 5% margin of error. Eligible participants were consecutively enrolled until the required sample size was achieved.

CKD Definition and Staging

CKD was defined per KDIGO guidelines as abnormalities of kidney structure or function for more than 3 months with health implications, including eGFR < 60 mL/min/1.73 m² or evidence of kidney damage.⁹ Staging was based on eGFR values:

- Stage 1: eGFR ≥90 mL/min/1.73 m² (with kidney damage)
- Stage 2: eGFR 60–89 mL/min/1.73 m²
- Stage 3: eGFR 30–59 mL/min/1.73 m²
- Stage 4: eGFR 15–29 mL/min/1.73 m²
- Stage 5: eGFR <15 mL/min/1.73 m²

Inclusion and Exclusion Criteria

Cases included patients aged 18–60 years with diagnosed CKD not yet on haemodialysis who provided written consent. Controls were age- and sex-matched healthy individuals without renal disease. Patients were excluded if they had established cardiovascular disease, liver disease, hypothyroidism, inflammatory or autoimmune disorders, neoplasms, pregnancy, psychiatric illness, or were receiving lipid-altering drugs.

Methodology

All participants underwent detailed clinical assessment including history, anthropometric measurements (BMI, waist circumference), and blood pressure recording. Laboratory investigations included haemoglobin, blood urea, serum creatinine, electrolytes, eGFR, urine albumin-to-creatinine ratio (UACR), serum albumin, fasting blood glucose, HbA1c, lipid profile, C-reactive protein, and homocysteine. Electrocardiography was performed in all participants.

Statistical Analysis

Data were analysed using SPSS version 29.0. Quantitative variables were expressed as mean ± SD and qualitative variables as frequency and percentage. Student's t-test, Mann–Whitney U test, Chi-square test, ANOVA, and logistic regression were applied as appropriate. A p-value < 0.05 was considered statistically significant.

RESULTS

Demographic Profile

The age distribution differed significantly between cases and controls ($p < 0.001$). The majority of CKD patients were older: 41.0% in the 51–60 years category and 32.0% in the 41–50 years group. The mean age of cases (47.35 ± 8.73 years)

was significantly higher than that of controls (38.49 ± 9.42 years). This is consistent with published literature showing CKD prevalence increases progressively after the age of 40 years.

Table 1. Age distribution of study participants

Age Group (Years)	Cases n (%)	Controls n (%)
< 30	6 (2.00)	61 (20.33)
31–40	75 (25.00)	141 (47.00)
41–50	96 (32.00)	60 (20.00)
51–60	123 (41.00)	38 (12.67)
Total	300	300
Mean $\pm$ SD	47.35 ± 8.73 years	38.49 ± 9.42 years
p-value	< 0.0001	

Regarding gender, females were predominant among cases (57.0%) while males were predominant among controls (59.0%; $p < 0.001$). This pattern may reflect differences in healthcare-seeking behaviour, hormonal influences, and survival advantages of women with chronic disease.

Anthropometric and Haemodynamic Parameters

Mean BMI (24.28 ± 2.61 vs 24.8 ± 3.05 kg/m²; $p = 0.07$) and waist circumference (88.67 ± 6.89 vs 87.67 ± 7.7 cm; $p = 0.07$) were comparable between groups. This paradox may reflect CKD-associated body composition changes, fluid retention, sarcopenia, and protein-energy wasting that mask true adiposity when assessed by BMI alone. In contrast, both systolic (130.56 ± 12.85 vs 116.66 ± 6.69 mmHg; $p < 0.001$) and diastolic blood pressure (81.4 ± 8.82 vs 73.63 ± 4.26 mmHg; $p < 0.001$) were significantly elevated in CKD patients.

Prevalence of Cardiovascular Risk Factors

Table 2 summarises the prevalence of major cardiovascular risk factors and their odds ratios. Hypertension was the most prevalent risk factor among cases (62.0% vs 11.0%; OR = 13.2; $p < 0.001$). Dyslipidaemia was present in 50.0% of cases vs 32.0% of controls (OR = 2.13; $p < 0.001$), with hypertriglyceridaemia (OR = 9.8) and low HDL (OR = 2.52) as the dominant lipid abnormalities. Macroalbuminuria and hypoalbuminaemia were observed exclusively among cases, demonstrating the strongest associations (OR = 167.16 and 151.19 respectively).

Table 2. Prevalence and odds ratios of cardiovascular risk factors (cases vs controls)

Risk Factor	Cases n (%)	Controls n (%)	OR	p-Value
Hypertension	186 (62.00)	33 (11.00)	13.20	< 0.001
Diabetes mellitus	40 (13.33)	27 (9.00)	2.06	0.0115
Obesity	45 (15.00)	63 (21.00)	0.66	0.07
Dyslipidaemia	150 (50.00)	96 (32.00)	2.13	< 0.001
Hypercholesterolaemia	69 (23.00)	39 (13.00)	2.00	0.002
Hypertriglyceridaemia	87 (29.00)	12 (4.00)	9.80	< 0.001
Low HDL	141 (47.00)	78 (26.00)	2.52	< 0.001
High LDL	96 (32.00)	81 (27.00)	1.27	0.21
Hypoalbuminaemia	60 (20.00)	0 (0.00)	151.19	< 0.001
Microalbuminuria	165 (55.00)	64 (21.33)	4.51	< 0.001
Macroalbuminuria	65 (21.67)	0 (0.00)	167.16	< 0.001

Renal Function and Metabolic Parameters

Mean eGFR was markedly lower in cases (43.3 ± 20.99 vs 91.08 ± 20.24 mL/min/1.73 m²; $p < 0.001$) and mean serum creatinine substantially higher (449.94 ± 29.66 vs 99.03 ± 14.37 μ mol/L; $p < 0.001$). UACR was dramatically elevated in cases (328.08 ± 170.62 vs 18.23 ± 2.57 mg/g; $p < 0.001$). Serum albumin was modestly but significantly lower in cases (40.38 ± 6.46 vs 42.01 ± 1.11 g/L; $p = 0.041$).

Table 3. Comparison of renal function and metabolic parameters (cases vs controls)

Parameter	Cases (Mean ± SD)	Controls (Mean ± SD)	p-Value
Serum Creatinine (μmol/L)	449.94 ± 29.66	99.03 ± 14.37	< 0.001
eGFR (mL/min/1.73 m ²)	43.3 ± 20.99	91.08 ± 20.24	< 0.001
Serum Albumin (g/L)	40.38 ± 6.46	42.01 ± 1.11	0.041
UACR (mg/g)	328.08 ± 170.62	18.23 ± 2.57	< 0.001
Fasting Plasma Glucose (mmol/L)	5.39 ± 2.07	4.47 ± 0.30	0.058
Triglycerides (mmol/L)	1.50 ± 0.95	0.91 ± 0.59	< 0.001
HDL (mmol/L)	0.84 ± 0.72	0.98 ± 0.25	0.210
LDL (mmol/L)	3.31 ± 1.25	3.50 ± 0.78	0.290
Total Cholesterol (mmol/L)	4.70 ± 1.52	4.60 ± 1.32	0.680

DISCUSSION

The significantly older mean age of CKD cases (47.35 ± 8.73 years) compared with controls (38.49 ± 9.42 years) is consistent with the established understanding that renal function declines progressively with advancing age due to nephron loss, vascular changes, and cumulative comorbidity exposure. Birhan et al. reported a comparable mean age of approximately 49 years among CKD patients.¹⁰ Go et al. similarly demonstrated that reduced eGFR and CKD prevalence increase markedly after the age of 40 years.¹¹ The predominance of females among cases (57.0%) may reflect differences in healthcare-seeking behaviour, hormonal influences, and survival advantages with chronic disease, though global patterns are mixed. Hill et al. reported that CKD prevalence is frequently higher in women, whereas men are overrepresented in dialysis populations.¹²

Hypertension was the most prevalent cardiovascular risk factor, present in 62.0% of cases versus 11.0% of controls (OR = 13.2; $p < 0.001$). Both systolic and diastolic blood pressure were significantly elevated in CKD patients. These findings align with Birhan et al. who reported hypertension in over 91% of their CKD cohort¹⁰, and with Babua et al. who documented a 90% prevalence.²¹ Samal et al. identified hypertension as an independent predictor of adverse cardiovascular outcomes (regression coefficient 1.45; $p = 0.01$).¹³

Diabetes mellitus was significantly more prevalent among cases (13.33% vs 9.0%; OR = 2.06; $p = 0.0115$), though mean fasting plasma glucose did not reach statistical significance ($p = 0.058$). The prevalence was lower than the 48.1% reported by Birhan et al.¹⁰, which may reflect regional, dietary, and genetic differences. Nabalawi et al. reported an adjusted OR of 0.674 for diabetes in CKD ($p = 0.029$).¹⁴ Persistent hyperglycaemia contributes to glomerular hyperfiltration, mesangial expansion, endothelial dysfunction, and progressive renal fibrosis, making glycaemic control central to CKD management.¹

Dyslipidaemia was present in 50.0% of cases versus 32.0% of controls (OR = 2.13; $p < 0.001$). Hypertriglyceridaemia was the most striking abnormality (29.0% vs 4.0%; OR = 9.8), followed by low HDL (47.0% vs 26.0%; OR = 2.52). Mean LDL and total cholesterol did not differ significantly, consistent with the uraemic dyslipidaemia pattern characterised by hypertriglyceridaemia and reduced HDL rather than elevated LDL. Samal et al. identified dyslipidaemia as an independent predictor of cardiovascular outcomes (regression coefficient 1.30; $p = 0.03$).¹⁵ Current ESC 2023 guidelines recommend LDL-C < 70 mg/dL for CKD Stage 3 and < 55 mg/dL for Stages 4–5.³⁶ Statins remain the cornerstone of lipid-lowering therapy, with the SHARP trial demonstrating significant cardiovascular benefit in CKD patients.¹⁶

CK patients had markedly lower eGFR and higher serum creatinine, both highly significant ($p < 0.001$). These findings are supported by Matsushita et al., who demonstrated that rising serum creatinine and declining eGFR closely correlate with CKD progression and adverse clinical outcomes.¹⁷ Albuminuria was dramatically elevated in cases (mean UACR 328.08 vs 18.23 mg/g). Macroalbuminuria and hypoalbuminaemia were observed exclusively among cases, with extraordinarily high odds ratios (OR = 167.16 and 151.19 respectively). The Chronic Kidney Disease Prognosis Consortium demonstrated that both lower eGFR and higher albuminuria independently predict cardiovascular disease and mortality. Gerstein et al. (HOPE study) showed microalbuminuria to be a powerful predictor of cardiovascular morbidity in both diabetic and non-diabetic individuals.¹⁸ Kaysen et al. reported that hypoalbuminaemia in CKD is a strong predictor of hospitalisation, cardiovascular events, and mortality.¹⁹

Obesity prevalence was comparable between groups (15.0% in cases vs 21.0% in controls; $p = 0.07$), and BMI and waist circumference were not significantly different. Kramer et al. observed that BMI may become a less reliable marker in established CKD due to changes in body composition.²⁰ Kalantar-Zadeh et al. described an “obesity paradox” in dialysis patients, where better nutritional reserves among patients with higher BMI may confer short-term survival advantages.²¹ Despite this finding, obesity remains clinically relevant as a contributor to insulin resistance, hypertension, dyslipidaemia, and glomerular hyperfiltration.^{1,2}

CONCLUSION

This study confirms that patients with chronic kidney disease constitute a high-risk group for cardiovascular disease. Hypertension, dyslipidaemia, hypertriglyceridaemia, low HDL, and albuminuria were significantly more prevalent among CKD patients, with hypertension and albuminuria-related parameters demonstrating the strongest associations. Early, systematic cardiovascular screening and aggressive management of modifiable risk factors—particularly blood pressure, lipids, glycaemia, and urinary albumin excretion—3 are essential to reduce cardiovascular morbidity, delay CKD progression, and improve overall clinical outcomes.

Limitations

- Single-centre design limits generalisability to other regions and healthcare settings.
- The observational design precludes establishment of causal relationships between CKD and cardiovascular risk factors.
- Hospital-based recruitment may introduce selection bias.
- Lifestyle data (dietary habits, physical activity, smoking) relied on patient self-reporting and may be subject to recall bias.

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