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Medicine

CLINICAL STUDY OF ISCHEMIC HEART DISEASE IN CHRONIC KIDNEY DISEASE PATIENTS AT TERTIARY CARE CENTRE

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ABSTRACT **Background:** Chronic Kidney Disease (CKD) is independently associated with increased risk of Ischemic Heart Disease (IHD), contributing significantly to morbidity and mortality. The interplay between renal dysfunction and cardiovascular pathology necessitates comprehensive clinical profiling. **Objectives:** To study the clinical profile, biochemical parameters, and cardiovascular manifestations in CKD patients with coexisting IHD. **Methods:** An observational cross-sectional study was conducted at a tertiary care hospital involving 76 adult CKD patients diagnosed with IHD. Inclusion criteria included age >18 years, eGFR <60 mL/min/1.73m², and evidence of IHD. Data were collected using a structured proforma, including history, physical examination, laboratory investigations (CBC, electrolytes, BUN, creatinine, lipid profile, CRP, troponin-I, CPK-MB), ECG, 2D-ECHO, and USG abdomen. Statistical analysis was performed using SPSS v21.0 with descriptive statistics and ANOVA; $p<0.05$ was considered significant. **Results:** Majority of patients were male (63.2%) with mean age 58.4±12.3 years. Hypertension (94.7%), pallor (81.6%), and lymphadenopathy (96.1%) were common. Elevated BUN (100%) and serum creatinine (93.4%) confirmed renal impairment. Proteinuria (3+, 96.1%) and albuminuria (A3, 78.9%) indicated severe glomerular damage. Mean eGFR declined progressively across stages ($p<0.05$). CRP was elevated in 88.2%, indicating systemic inflammation. ECG showed Q waves (85.5%), ST elevation (59.2%), and LVH (33.3%). Arrhythmias and AMI were more frequent in advanced CKD stages. **Conclusion:** CKD patients with IHD exhibit significant cardiovascular abnormalities, inflammatory markers, and metabolic disturbances, worsening with declining GFR. Early detection and integrated management are crucial to improve outcomes.

KEYWORDS : Chronic Kidney Disease, Ischemic Heart Disease, Cardiovascular Risk, eGFR, Proteinuria, Inflammation, CRP, LVH

INTRODUCTION

Chronic Kidney Disease (CKD) affects over 850 million people globally and is a major contributor to cardiovascular morbidity and mortality [1]. Defined as abnormalities in kidney structure or function persisting for ≥3 months—manifested by eGFR <60 mL/min/1.73m² or markers of kidney damage such as albuminuria, hematuria, or structural abnormalities—CKD is now recognized not only as a progressive renal disorder but also as a powerful risk multiplier for cardiovascular disease (CVD) [2,3].

The Kidney Disease: Improving Global Outcomes (KDIGO) 2012 classification system, reaffirmed in 2024, emphasizes the CGA framework—Cause, GFR category (G1–G5), and Albuminuria category (A1–A3)—to guide prognosis and management [4,5]. This system underscores that even mild reductions in renal function significantly elevate cardiovascular risk, independent of traditional risk factors [6].

Ischemic Heart Disease (IHD) remains the leading cause of death worldwide, accounting for approximately 16% of all global deaths [7]. In CKD patients, the incidence of IHD is 10–30 times higher than in the general population [8]. The pathophysiological link involves multiple mechanisms: endothelial dysfunction, chronic inflammation, vascular calcification, left ventricular hypertrophy (LVH), anemia, and dyslipidemia—all accelerated by uremic milieu [9].

Studies such as the Modification of Diet in Renal Disease (MDRD) and the Atherosclerosis Risk in Communities (ARIC) cohort have demonstrated that reduced creatinine clearance and low eGFR are independently associated with adverse cardiovascular outcomes, including myocardial infarction, heart failure, and sudden cardiac death [10,11]. Manjunath et al. (2003) reported a graded inverse relationship between renal function and cardiovascular risk, reinforcing the need for early cardiovascular risk stratification in CKD [2].

Despite growing evidence, there remains a gap in region-specific data on the clinical and biochemical profile of CKD patients with IHD, especially in low- and middle-income countries where dual disease burden is rising rapidly. In India, IHD occurs 5–10 years earlier than in Western populations, and CKD prevalence is increasing due to rising diabetes and hypertension [12,13].

This study aims to evaluate the clinical characteristics, laboratory parameters, and cardiovascular manifestations across different stages of CKD in patients with coexisting IHD, providing insights into early markers of cardiovascular risk and informing integrated care strategies.

MATERIALS AND METHODS

Study Design

An observational, cross-sectional study was conducted at a tertiary care teaching hospital over a two-year period after obtaining institutional ethical clearance.

Study Setting

The Department of Medicine, [ACPM MEDICAL COLLEEGE AND HOSPITAL], a tertiary care centre serving urban and rural populations.

Study Population

All adult patients attending outpatient (OPD) and inpatient (IPD) departments diagnosed with both Chronic Kidney Disease and Ischemic Heart Disease.

Sample Size

Seventy-six (76) patients were enrolled based on inclusion and exclusion criteria.

Sampling Technique

Consecutive non-probability sampling was used.

Inclusion Criteria

- Age >18 years
- Confirmed CKD (eGFR <60 mL/min/1.73m² or markers of kidney damage for ≥3 months)
- Evidence of IHD (clinical, ECG, echocardiographic, or biochemical confirmation)
- Willingness to provide informed written consent

Exclusion Criteria

- Pregnancy
- Shock or hemodynamic instability
- Severe sepsis
- Multi-organ failure
- Thyrotoxicosis

Data Collection Procedure

After obtaining informed consent, a detailed history was taken using a pre-designed proforma. This included demographic data, past medical history (diabetes, hypertension, CAD), addiction history (tobacco, alcohol, drugs), and symptoms related to CKD and IHD.

A thorough general and systemic examination was performed.

Table No 1: Variation of Left Ventricular Ejection Fraction (LVEF) Across CKD Stages in Patients with IHD

	stages of CKD	N	Mean	Std. Deviation	P Value
LVEF	1.00	2	39.00	0.00	< 0.05
	2.00	4	46.00	7.11	
	3.00	20	43.20	5.14	
	4.00	41	42.92	4.83	
	5.00	9	44.11	5.81	
	Total	76	43.19	5.08	

In our study, the mean Left Ventricular Ejection Fraction (LVEF) among CKD patients was 43.19 ± 5.08. Across CKD stages, LVEF showed variation, being lowest in Stage 1 (39%) and slightly higher in advanced stages (Stage 5: 44.1%). The difference in LVEF across CKD stages was statistically significant (P < 0.05), suggesting a stage-wise impact of CKD on cardiac function

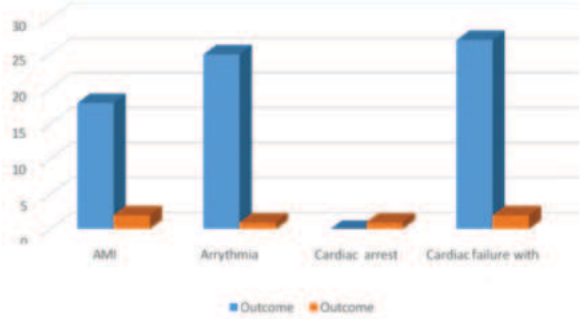
Table No 2: Relation Between Left Ventricular Hypertrohy (LVH) and CKD Staging in Patients with IHD

		Stage of CKD					P Value
		1.00	2.00	3.00	4.00	5.00	
LVH	A	0	1	3	0	0	< 0.05
		0.0%	25.0%	75.0%	0.0%	0.0%	
	P	2	3	17	41	9	
		2.8%	4.2%	23.6%	56.9%	12.5%	

The occurrence of Left Ventricular Hypertrophy (LVH) across various stages of Chronic Kidney Disease (CKD) shows a statistically significant association (P < 0.05): LVH was absent in all patients with Stage 1, 4, and 5 CKD. LVH was most frequently absent in Stage 3 (75%), but rare in other stages. In contrast, LVH was present in the majority of patients with Stage 4 CKD (56.9%), followed by Stage 3 (23.6%) and Stage 5 (12.5%).

Table No 3: Association Between Type of Ischemic Heart Disease (IHD) and Outcome in CKD Patients

IHD	Outcome		P Value
	ALIVE	DIED	
AMI	18	2	< 0.05
	90.0%	10.0%	
Arrythmia	25	1	
	96.2%	3.8%	
Cardiac arrest	0	1	
	0.0%	100.0%	
Cardiac failure with angina	27	2	
	93.1%	6.9%	



Graph: Association Between Type of Ischemic Heart Disease (IHD) and Outcome in CKD Patients

In our study, survival outcomes varied significantly according to the type of IHD in CKD patients (P < 0.05). Mortality was highest in patients with cardiac arrest (100%), followed by acute myocardial infarction (10%) and cardiac failure with angina (6.9%), while arrhythmia had the lowest mortality (3.8%). This highlights that certain IHD subtypes, particularly cardiac arrest and AMI, are associated with poorer prognosis in CKD patients.

Investigations Included

- Complete blood count (CBC)
- Fasting and postprandial blood glucose, HbA1c
- Serum electrolytes (Na^+, K^+, Ca^{2+}, PO_4^{3-})
- Blood urea nitrogen (BUN), serum creatinine
- Liver function tests (SGPT, SGOT)
- Lipid profile (total cholesterol, HDL, LDL, triglycerides, VLDL, ratios)
- Urine routine and microscopy
- Serum albumin, uric acid, C-reactive protein (CRP)
- Cardiac markers: Troponin-I, CPK-MB
- Imaging: 2D Echocardiography, ECG, ultrasonography (abdomen + pelvis), chest X-ray

Definition of Variables

CKD Staging: Based on KDOQI 2002 guidelines (used in study):

- Stage 1: eGFR ≥90 + kidney damage
- Stage 2: eGFR 60–89
- Stage 3: eGFR
- 30–59
- Stage 4: eGFR 15–29
- Stage 5: eGFR <15 or dialysis-dependent
- IHD Diagnosis: Based on clinical features (chest pain, dyspnea), ECG changes (ST-T changes, Q waves), elevated cardiac enzymes, or prior revascularization.
- Hypertension: SBP ≥140 mmHg or DBP ≥90 mmHg or on antihypertensive therapy.
- Diabetes Mellitus: FBS ≥126 mg/dL, PPBS ≥200 mg/dL, or HbA1c ≥6.5% or on treatment.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 21.0. Quantitative variables were expressed as mean ± standard deviation. Normality was assessed using Shapiro-Wilk test. One-way ANOVA was used for comparison across CKD stages; p-value <0.05 was considered statistically significant.

RESULTS

A total of 76 patients were included in the study. The mean age was 58.4 ± 12.3 years, with males constituting 63.2% (n=48). The majority belonged to stage 4 CKD (41 patients, 53.9%), followed by stage 3 (20, 26.3%), stage 5 (9, 11.8%), stage 2 (4, 5.3%), and stage 1 (2, 2.6%).

Clinical Features

General examination revealed that 82.9% were poorly built, 50% malnourished, and 27.6% febrile. Hypertension was present in 94.7%, pallor in 81.6%, cyanosis in 25.0%, and lymphadenopathy in 96.1%. Clubbing and pedal edema were each seen in 19.7%. Raised JVP was noted in 10.5%, asterixis in 69.7%, and koilonychia in 25.0%.

Common symptoms included fatigue (77.6%), shortness of breath (72.4%), chest pain (68.4%), and joint pain (43.4%). Weight loss (42.1%) and hematuria (42.1%) were also prevalent.

All parameters except HbA1c showed statistically significant variation across stages, reflecting progressive metabolic derangement.

Cardiovascular Manifestations by CKD Stage

AMI was most common in stage 4 (26.8%), while arrhythmias increased progressively, peaking in stage 5 (55.6%). Cardiac failure with angina was highest in stage 4 (39.0%). One cardiac arrest occurred in stage 4.

ECG Findings

Q wave: 85.5% (indicative of prior MI)
ST elevation: 59.2%
ST depression: 17.1%
T-wave inversion: 52.4%
LVH: 33.3%
Irregular rhythm: 18.4%
LVH and T-wave changes were significantly associated with advanced CKD (p<0.05).

Lipid Profile

Mean total cholesterol: 154.21 ± 41.05 mg/dL
HDL: 37.8 ± 8.2 mg/dL
LDL: 86.4 ± 21.3 mg/dL
TG: 154.2 ± 41.0 mg/dL
LDL/HDL ratio: 4.16 ± 1.69
HDL/TG ratio: 0.23 ± 0.08

Dyslipidemia was common, though levels did not show significant variation across stages.

Outcome Analysis

Among 76 patients, 14 deaths occurred, all in stages 4 and 5. No deaths

were recorded in stages 1–3. Mortality was highest in those with AMI (100% mortality in stage 5) and cardiac arrest (100%).

DISCUSSION

This study highlights the high burden of cardiovascular disease in CKD patients, with nearly all exhibiting signs of IHD, inflammation, and metabolic disturbances. The mean age of 58.4 years aligns with earlier onset of CVD in Indian populations [13]. The predominance of males (63.2%) may reflect higher rates of smoking, alcohol use, and occupational stress.

The presence of hypertension in 94.7% supports its role as a key driver of both CKD and IHD. Persistent hypertension leads to glomerular hyperfiltration, vascular stiffness, and LVH—central features in uremic cardiomyopathy [187]. Similarly, diabetes mellitus, though not directly quantified here, was implied by elevated HbA1c (mean 7.72%) and glycosuria (48.7%), consistent with diabetic nephropathy as a major etiology.

Progressive decline in creatinine clearance and rising serum creatinine across stages confirms worsening renal function. Notably, CRP was elevated in 88.2%, indicating a chronic inflammatory state. This is consistent with studies showing that inflammation promotes atherosclerosis, insulin resistance, and muscle wasting in CKD [18,32]. Ridker et al. (2000) established CRP as an independent predictor of cardiovascular events, reinforcing its utility in risk stratification [31].

The high prevalence of proteinuria (96.1%) and A3 albuminuria (78.9%) reflects significant glomerular damage. Ritz et al. (2006) showed that proteinuria independently predicts cardiovascular mortality, possibly via endothelial dysfunction and pro-inflammatory cytokine release [30].

ECG findings reveal a high burden of structural heart disease: Q waves in 85.5% suggest prior silent MI, common in diabetics and uremic patients due to autonomic neuropathy. ST elevation (59.2%) indicates active ischemia, while LVH (33.3%) reflects pressure overload and volume expansion—hallmarks of CKD-related cardiomyopathy [187].

Arrhythmias and sudden cardiac death are major concerns. In our cohort, arrhythmias increased with CKD severity, reaching 55.6% in stage 5. This correlates with electrolyte imbalances (hyperkalemia, hypocalcemia), autonomic dysfunction, and fibrosis [110]. The single cardiac arrest in stage 4 underscores the fragility of these patients.

The absence of mortality in stages 1–3, versus 14 deaths in stages 4–5, emphasizes the importance of early intervention. Timely initiation of RRT, control of hypertension, correction of anemia, and lipid management could delay progression and reduce cardiovascular events [2,3].

Our findings support KDIGO recommendations for integrated care, including ACE inhibitors/ARBs to reduce proteinuria [19], phosphate binders to prevent vascular calcification [20], and sodium bicarbonate to correct metabolic acidosis [21]. Lifestyle modifications—smoking cessation, exercise, dietary changes—are also critical [22].

Limitations

Single-center design limits generalizability
Small sample size (n=76)
Cross-sectional nature prevents causal inference
Lack of long-term follow-up
Scope for Future Research
Longitudinal studies assessing impact of early cardiovascular screening and multidisciplinary care on hard endpoints (mortality, hospitalization) in CKD patients are needed.

CONCLUSION

Patients with CKD and IHD exhibit a complex interplay of metabolic, inflammatory, and structural cardiovascular abnormalities that worsen with advancing renal dysfunction. Key findings include high prevalence of LVH, prior MI, elevated CRP, proteinuria, and arrhythmias—especially in late-stage CKD. These markers should prompt aggressive cardiovascular risk assessment and early intervention. Integrated nephrology-cardiology care models are essential to improve survival and quality of life.

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