

FREQUENCY OF MYOCARDIAL INFARCTION IN NON-DIALYSIS CHRONIC KIDNEY DISEASE PATIENTS

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ABSTRACT

Objective: To determine the frequency of myocardial infarction in non-dialysis chronic kidney disease patients.

Study design: cross-sectional study.

Setting: This study was performed in Nephrology Department Jinnah Postgraduate Medical Centre (JPMC) in Karachi

Period: For six months duration from July 2025 – December 2025.

Methods: The study started following approval by CPSP IRB/REU. Non-probability consecutive sampling was used to recruit a total of 84 patients (18-60 years) with diagnosed CKD (eGFR <60 mL/min/1.73 m² for ≥3 months) and who were not on dialysis. MI was diagnosed based on clinical symptoms, electrocardiographic changes, and/or troponin I ≥1.0 ng/mL. The data collected were analysed using SPSS version 25. For qualitative variables, frequencies and percentages were calculated and stratification was done using Chi-square test.

Results: The majority of participants were in the age group 46-60 years and just slightly more male than female. Hypertension (69.0%) and diabetes mellitus (52.4%) were the most common comorbidities. Most patients were in CKD Stage 3B (31.0%) and Stage 4 (28.6%). The total incidence of myocardial infarction was 54.8% (n=46). MI increased progressively with increasing CKD stage with the highest being present in the Stage 3B (69.2%) and the next being in Stage 4 (66.7%).

Conclusions: MI is a common event in non-dialysis CKD and is exacerbated by progressive CKD stage. In this high-risk population, early cardiovascular risk assessment and early interventions are essential.

KEYWORDS: Cardiovascular Diseases; Chronic Kidney Disease; Myocardial Infarction; Renal Insufficiency.

INTRODUCTION

Cardiovascular morbidity is highly linked to chronic kidney disease (CKD) and cardiovascular disease (CVD) continues to be the leading cause of death in the CKD population. Patients with CKD have a significantly greater burden of atherosclerotic disease, myocardial injury and adverse cardiac remodelling, even before they reach the stage of end-stage kidney disease. This increased risk is due to both the usual cardiovascular risk factors and mechanisms unique to CKD including systemic inflammation, endothelial dysfunction and vascular calcification. All of these processes together make the possibility of acute myocardial infarction (AMI) more likely in CKD patients than in the general population.¹

The Chronic Renal Insufficiency Cohort (CRIC) study has demonstrated that cardiovascular events, such as heart failure and arrhythmias, are common in patients with CKD and frequently occur with or after ischemic cardiac events. Increased and dynamic levels of cardiac biomarkers (such as troponins), especially in patients with CKD, further confirm the continued myocardial stress and subclinical injury in CKD patients, which further hampers the diagnosis of AMI.² This study indicates not only that myocardial injury occurs often in patients with CKD, but also that it may be clinically silent or atypical.

Sex based analyses have shown additional differences in cardiovascular outcomes in patients with CKD, including differences in event rates and clinical manifestations, between men and women. This heterogeneity reflects the complexity of cardiovascular risk stratification in CKD, and suggests that the frequency and outcome of myocardial infarction are population-specific and requires evaluation.³

Inflammatory pathways are key in driving cardiovascular disease in CKD. Inflammation is an important risk factor for myocardial injury, as elevated levels of interleukin-6 have been independently linked to worse cardiovascular outcomes, such as coronary events, in chronic coronary syndrome patients with CKD.⁴ Likewise, it has been demonstrated that systemic inflammation in atherosclerotic cardiovascular disease (ASCVD) plus CKD is associated with poorer outcomes and greater burden of cardiovascular events, supporting the biological plausibility of a higher risk of myocardial infarction in this population.⁵

In clinical practice, impaired renal function has been consistently demonstrated to have a considerable impact on the approaches to the diagnosis and cardiac interventions for patients with myocardial infarction, and these approaches are less aggressive in patients with CKD compared with those with normal renal function, even though they have higher cardiovascular risk.⁶ This treatment gap may play a role in poorer outcomes after an MI, and may also account for some of the observed disparities in MI incidence and detection rates. Furthermore, troponin levels are often elevated in CKD patients, even if the acute myocardial infarction is not clinically suspected, and it is associated with subclinical myocardial injury and not only with acute myocardial infarction.⁷

Cardiovascular events continue to be common in dialysis and non-dialysis CKD patients. It has been shown by studies in south Asian population, that there is a significant burden of cardiovascular complications such as myocardial infarction, heart failure, arrhythmias, among CKD patients, which is of global concern.⁸ Moreover, silent myocardial infarction has been recognized in special CKD patients like kidney transplant recipients as an important predictor of adverse cardiovascular outcomes, suggesting that myocardial infarction may be undiagnosed or clinically unrecognized in CKD patients.⁹ Although patients with non-dialysis-dependent CKD have a higher cardiovascular burden, evidence shows that patients are less likely to undergo coronary reassessment and revascularization after myocardial infarction, which may further negatively impact the prognosis and make it difficult to be able to accurately estimate the disease burden.¹⁰

As such, there is a critical unmet need to estimate the incidence of myocardial infarction in non-dialysis chronic kidney disease patients, especially in regions where the diagnosis might be delayed or the clinical presentation might be atypical, and the true incidence might be under-estimated by the use of biomarkers. Hence the present study is designed to find out the prevalence of myocardial infarction in non-dialysis CKD patients who are attending a tertiary care hospital. This will aid to better characterize the burden of CHD in CKD and allow for early risk stratification, timely diagnosis and optimization of cardiovascular management strategies in this high-risk population.

METHODS

This study was conducted as a cross-sectional study at the Nephrology Department of Jinnah Postgraduate Medical Centre (JPMC), Karachi. The time frame of the study was six months from the approval of synopsis by the College of Physicians and Surgeons of Pakistan (CPSP) and IRB, from July 2025 – December 2025. Ethical clearance was secured from the IRB of JPMC before data collection began on 25th June 2025 with approval no: [NO.F.2-81/2025-GENL/344/JPMC]. All participants received detailed information about the purpose, procedures, risks and benefits of the study and written informed consent was obtained. The confidentiality and anonymity of patient information was carefully guarded throughout the study.

The sample size was determined by using OpenEpi software, which assumed that the frequency of myocardial infarction among patients with chronic kidney disease was 12.2%, as determined in a previous study by Lidgard et al. The 95% confidence interval and margin of error was 7% and obtained a total sample size of 84 patients.¹¹ A non-probability consecutive sampling technique was used to enrol patients.

Patients were included regardless of gender, age (18-60 years), diagnosis (chronic kidney disease, defined by the operational definition), and if not on dialysis. Patients with a known history of myocardial infarction before diagnosis of CKD were excluded, as were patients with end stage renal disease (ESRD) or dialysis, those with a history of any organ transplantation, and those who declined to participate.

Chronic kidney disease was defined as an eGFR rate less than 60 mL/min/1.73 m² for at least 3 months or more. CKD staging was classified based on eGFR values as follows: Stage 1 (≥ 90 mL/min/1.73 m²), Stage 2 (60–89 mL/min/1.73 m²), Stage 3A (45–59 mL/min/1.73 m²), Stage 3B (30–44 mL/min/1.73 m²), Stage 4 (15–29 mL/min/1.73 m²), and Stage 5 (<15 mL/min/1.73 m²).

Myocardial infarction was defined as the presence of one or more of the following clinical, biochemical, or electrocardiographic criteria: typical chest pain lasting more than 20 minutes (with pain score >1 on the numeric rating scale), chest heaviness radiating to the arms or jaw, shortness of breath with respiratory rate greater than 18 breaths per minute, ST-segment elevation in two or more contiguous leads on a 12-lead electrocardiogram, and/or serum troponin I level ≥ 1.0 ng/mL.

The data was obtained from the in-patient admitted in the Nephrology department of JPMC as well as from out-patient who visited the Nephrology department of JPMC who met the inclusion criteria. All the study variables were recorded in a structured proforma. Demographic data such as name, age, sex, height, weight and body mass index (BMI) was obtained. Average weight for height (BMI) was determined by the standard formula, weight (kg) / height squared (m²). All patients had a history of diabetes mellitus, hypertension, dyslipidaemia, familial history of chronic kidney disease, familial history of myocardial infarction, smoking history.

The vital signs were taken as pulse rate, respiratory rate, blood pressure and temperature. Due to the clinical symptoms indicating myocardial infarction, chest pain, chest heaviness and shortness of breath were evaluated. The severity of chest pain was measured by a numeric rating scale. Laboratory tests such as complete blood count,

serum troponin I, urea, creatinine, uric acid, and estimated glomerular filtration rate were done. Samples of blood were taken by the trained nursing staff and analysed in the hospital laboratory as per the standard protocols. Patients who presented with ST segment elevation and other ischemic electrocardiogram changes were subsequently confirmed by a 12-lead electrocardiogram.

The data was entered and analysed using the Statistical Package for Social Sciences (SPSS) version 25. Continuous variables including age, height, weight, BMI, vital signs, duration of CKD, numeric rating scale (NRS) score, troponin I, urea, creatinine, uric acid and GFR were reported as mean $\pm$ SD or median (IQR) as per distribution of data. The normality of data was evaluated by the Shapiro–Wilk test and a p-value > 0.05 assumed as normal distribution. Qualitative variables such as gender, comorbidities, CKD stage, clinical symptoms and myocardial infarction outcome (present/absent) were reported as frequencies and percentages. Stratification has been used to adjust for the effect modifiers as follows: age, gender, diabetes mellitus, hypertension, dyslipidaemia, smoking status, family history of CKD and myocardial infarction, and stage of CKD. Post-stratification comparisons were carried out by using the Chi-Square test and the p value < 0.05 was considered statistically significant.

RESULTS

A total of 84 patients with non-dialysis chronic kidney disease (CKD) were included in this study. The baseline characteristics (**Table-1**) of the study population showed a slight male predominance, with males accounting for 57.1% of the 84 non-dialysis CKD patients, while females comprised 42.9%. Most participants were middle-aged to older adults, with more than half (54.8%) falling in the 46–60 years age group, indicating that CKD with associated cardiovascular risk is more common in older adults in this cohort.

Table-1: Baseline Demographic Characteristics (n = 84)

Variable	Category	n (%)
Gender	Male	48 (57.1%)
	Female	36 (42.9%)
Age group	18–30 years	10 (11.9%)
	31–45 years	28 (33.3%)
	46–60 years	46 (54.8%)

The clinical and risk factor profile (**Table-2**) demonstrated a high burden of cardiovascular comorbidities among CKD patients. Hypertension was the most common comorbidity, present in 69.0% of patients, followed by diabetes mellitus in 52.4%. Dyslipidemia (35.7%) and smoking (33.3%) were also frequently observed, while 21.4% had a positive family history of myocardial infarction. This clustering of risk factors highlights the strong cardiometabolic risk burden in CKD patients.

Table-2: Clinical and Risk Factor Profile (n = 84)

Variable	Category	n (%)
Diabetes mellitus	Yes	44 (52.4%)
	No	40 (47.6%)
Hypertension	Yes	58 (69.0%)
	No	26 (31.0%)
Dyslipidemia	Yes	30 (35.7%)
	No	54 (64.3%)
Smoking	Yes	28 (33.3%)
	No	56 (66.7%)
Family history of MI	Yes	18 (21.4%)
	No	66 (78.6%)

The distribution of CKD stages (**Table-3**) showed that most patients were in advanced stages of disease. Stage 3B was the most frequent (31.0%), followed by Stage 4 (28.6%), whereas fewer patients were in early stages such as Stage 1 (7.1%) and Stage 2 (11.9%). This indicates that a large proportion of patients present at more advanced stages of CKD, when cardiovascular complications are more likely to occur.

Table-3: Distribution of CKD Stages (n = 84)

CKD Stage	n (%)
Stage 1	6 (7.1%)
Stage 2	10 (11.9%)
Stage 3A	18 (21.4%)
Stage 3B	26 (31.0%)
Stage 4	24 (28.6%)

The overall frequency of myocardial infarction (**Table-4**) was 54.8%, indicating that more than half of the non-dialysis CKD patients had evidence of MI. This reflects a high burden of ischemic heart disease in this population and underscores CKD as a major cardiovascular risk condition.

Table-4: Frequency of Myocardial Infarction (n = 84)

Outcome	n (%)
MI Present	46 (54.8%)
MI Absent	38 (45.2%)

Stratification by CKD stage (**Table-5**) demonstrated a clear increasing trend in myocardial infarction frequency with worsening renal function. The lowest frequency was observed in early CKD stages, while significantly higher proportions were seen in Stage 3A, Stage 3B, and Stage 4, with the highest burden in Stage 3B (69.2%). This pattern suggests a strong association between declining renal function and increasing risk of myocardial infarction.

Table-5: Stratification of MI by CKD Stage

CKD Stage	MI Present n (%)	MI Absent n (%)	Total
Stage 1	1 (16.7%)	5 (83.3%)	6
Stage 2	3 (30.0%)	7 (70.0%)	10
Stage 3A	8 (44.4%)	10 (55.6%)	18
Stage 3B	18 (69.2%)	8 (30.8%)	26
Stage 4	16 (66.7%)	8 (33.3%)	24

DISCUSSION

The prevalence of myocardial infarction (MI) was relatively high at 54.8% among non-dialysis chronic kidney disease (CKD) patients in this study. This finding agrees with the large contemporary cohort data that show that CKD has a significant association with increased risk of acute myocardial infarction, because of accelerated atherosclerosis, endothelial dysfunction and persistent inflammatory activation.¹¹ The observed association of worse CKD stage with more high-grade MI was similar to the published data of the national registry which demonstrated a graded relationship between decreasing renal function and increasing rate of AMI [11]. Likewise, population-based studies in Europe have shown that patients with CKD are also more likely to suffer from MI and have more complex clinical courses and poorer in-hospital outcomes than those with normal renal function.¹² Poor implementation of guideline recommended management for CKD patients could also contribute to the high burden of MI found in this study. Patients with poor renal function are less likely to receive the more invasive diagnostic or therapeutic interventions such as coronary angiography and revascularization which may lead to high event burden and poor outcomes.¹³ In fact, the lack of evidence-based care is especially true for those with non-dialysis chronic kidney disease (CKD), and this “treatment-risk paradox” in cardiovascular care has existed for a long time. In addition, several clinical trials have shown that even though multiple benefits have been demonstrated in selected patients in acute coronary syndromes (ACS), patients with CKD are less likely to receive early invasive treatment, especially for non-ST-elevation myocardial infarction (NSTEMI).¹⁴ This is true even if invasive methods used, as patients with CKD have a greater burden of comorbidities and more procedural risk.¹⁵ In patients with CKD, technological developments like the drug-eluting stent have resulted in better clinical outcomes, but the long-term prognosis for patients who have suffered a myocardial infarction is still significantly inferior, and the incidence of recurrent ischemic events and mortality is higher.¹⁶ Moreover, CKD has been shown to be independently linked to mortality and complications after ST-elevation myocardial infarction (STEMI) further establishing the susceptibility of this population.¹⁷ Furthermore, mortality data from large observational studies also suggest that renal impairment is a significant risk factor for both short-term and long-term mortality in patients with CKD who develop NSTEMI, highlighting the prognostic role of renal impairment in outcomes of myocardial infarction.¹⁸ Furthermore, atypical presentations like myocardial infarction with non-obstructive coronary arteries (MINOCA) are also reported more frequently in CKD patients that may contribute to underdiagnosis or delayed diagnosis in clinical practice.¹⁹ This study is corroborated by the difference in management practices in different healthcare systems. Registry data from England show that there are significant differences in full treatment according to the CKD and MI group, with lower treatment rates in the CKD group, leading to poorer outcomes and systemic gaps in treatment delivery.²⁰ A strength of this study is its emphasis on the non-dialysis CKD population, thereby enabling the detection of the myocardial infarction burden early on before end-stage renal disease. Strict operational definition of both CKD and MI provides for greater accuracy in diagnosis and standardisation of cases. This study strongly supports the hypothesis that CKD is associated with a high risk of myocardial infarction and that these patients should routinely be screened for cardiovascular disease, have aggressive treatment of risk factors and be referred to cardiology services early. Routine cardiovascular risk stratification should be a part of all CKD stages, especially Stage 3 and higher, to decrease morbidity and optimize outcomes, as recommended in future clinical protocols. There are a number of limitations to this study. It has a cross-sectional design, making it difficult to establish causality between severity of CKD and MI. Selection bias is possible if consecutive sampling is used instead of a probability sampling method. Further, the study is from a single centre, making the results not necessarily

applicable to a wider population. Hospital diagnosis of myocardial infarction may also underestimate the subclinical or silent MI, which could also have a negative impact on the estimation of the frequency.

CONCLUSION

The study showed that myocardial infarction was present in 46 (54.8%) patients with non-dialysis CKD. A gradual rise in the proportion of patients with progressive increase in MI frequency was seen as the severity of the CKD increased, with the highest proportion seen in Stage 3B and Stage 4 patients. In addition, hypertension, diabetes mellitus, and smoking history were very common among the patients with MI and were found to be associated with the ischemic burden in patients with CKD.

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