JHWCR JOURNAL OF HEALTH, WELLNESS AND COMMUNITY RESEARCH 3007-0570

DOI: https://doi.org/10.61919/yw9apg69

Original Article

A Comparative Analysis of Acute Kidney Injury Severity and Associated Factors in Diabetic and Non-Diabetic Patients Following Myocardial Infarction

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"Cite this Article" Received: 23 March 2026; **Accepted**: 07 July 2026; **Published**: 23 July 2026

Author Contributions: Concept: HSQ, RN; Design: HSQ, AF, RN; Data Collection: AF, HA, MHT; Analysis: MHT, RN; Drafting: HSQ, AF, HA, MHT, RN. **Ethical Approval**: The University of Lahore, Lahore, Pakistan. **Informed Consent**: Written informed consent was obtained from all participants; **Conflict of Interest**: The authors declare no conflict of interest. **Funding**: No external funding; **Data Availability**: Available from the corresponding author on reasonable request; **Acknowledgments**: NA

ABSTRACT

Background: Acute kidney injury is an important complication of myocardial infarction, but comparative evidence describing renal, glycaemic, and cardiac differences between diabetic and non-diabetic patients with established post-MI AKI remains limited. Objective: To compare AKI severity and associated renal, glycaemic, and cardiac parameters between diabetic and non-diabetic patients following myocardial infarction. Methods: This comparative cross-sectional study included 248 post-MI patients with AKI recruited from the Punjab Institute of Cardiology and Mayo Hospital, Lahore, comprising 124 diabetic and 124 non-diabetic participants. AKI was classified using KDIGO serum creatinine criteria. Categorical variables were compared using Chi-square tests, and non-normally distributed continuous variables were analysed using the Mann–Whitney U test. Results: Overall, 50.0% had Stage 2 and 30.6% had Stage 3 AKI. Diabetes status was strongly associated with random blood glucose category ($\chi^2(2) = 91.681$, $p < 0.001$; Cramér's V = 0.608), HbA1c category ($\chi^2(2) = 153.249$, $p < 0.001$; Cramér's V = 0.786), and absolute serum creatinine category ($\chi^2(2) = 25.451$, $p < 0.001$; Cramér's V = 0.320). Significant continuous-variable differences were observed for eGFR ($p = 0.015$), initial troponin ($p = 0.033$), and random blood glucose ($p < 0.001$), whereas serum creatinine, urea, blood urea nitrogen, and LVEF were not significantly different. Conclusion: Diabetic and non-diabetic post-MI patients with AKI demonstrated distinct renal, glycaemic, and cardiac profiles. Comprehensive assessment is warranted irrespective of documented diabetes status. Keywords: Acute kidney injury; myocardial infarction; diabetes mellitus; cardiorenal syndrome; acute hyperglycaemia; estimated glomerular filtration rate; KDIGO.

INTRODUCTION

Acute kidney injury (AKI) is an important complication of myocardial infarction (MI) and is associated with prolonged hospitalization, increased healthcare utilization, and elevated risks of short- and long-term morbidity and mortality (1). Even when renal function appears to recover during the index admission, AKI may independently predict subsequent chronic kidney disease, heart failure, recurrent cardiovascular events, and death (2). Renal impairment following MI is commonly recognized through changes in serum creatinine and estimated glomerular filtration rate (eGFR), both of which have important prognostic implications in patients with acute coronary syndromes (3,4). However, serum creatinine may rise relatively late during acute renal injury, and its interpretation can be affected by age, sex, muscle mass, baseline renal function, and the dynamic physiological changes occurring during critical illness.

The close physiological relationship between the heart and kidneys contributes substantially to the development and severity of AKI following MI. Myocardial ischaemia may reduce cardiac output and renal perfusion, while haemodynamic instability, neurohormonal activation, systemic inflammation, oxidative stress, and exposure to potentially nephrotoxic medications or iodinated contrast media may further compromise renal function (5). This bidirectional interaction is commonly described as acute cardiorenal syndrome and has prompted growing interest in identifying clinical and biochemical markers capable of detecting renal involvement and predicting adverse outcomes earlier than conventional creatinine-based assessment alone (6). Conversely, impaired renal function may aggravate cardiovascular dysfunction through fluid overload, electrolyte imbalance, endothelial injury, and increased inflammatory activity. The association between renal and cardiovascular disease therefore extends beyond an isolated acute event, as pre-existing kidney dysfunction is itself associated with an increased risk of myocardial infarction and poorer cardiovascular outcomes (7).

Diabetes mellitus may further intensify the interaction between myocardial and renal injury. Chronic hyperglycaemia contributes to endothelial dysfunction, microvascular damage, oxidative stress, inflammation, and impaired renal autoregulation, potentially increasing renal susceptibility to acute haemodynamic and metabolic stress following MI (9). Diabetes has also been associated with poorer long-term survival after a first myocardial infarction, although the magnitude of this association may vary according to patient characteristics, including sex and the presence of other cardiovascular risk factors (10,11). Experimental and clinical evidence suggests that diabetes may alter the MI–AKI relationship by increasing the vulnerability of renal tissue to ischaemia and reducing its capacity to compensate for acute reductions in perfusion (9).

Acute renal dysfunction after MI is not limited to patients with pre-existing diabetes. Non-diabetic patients may also develop substantial renal impairment because of acute haemodynamic deterioration, inflammatory responses, sympathetic and hormonal activation, contrast exposure, and metabolic dysregulation during severe illness (12). Acute hyperglycaemia may occur during MI even in individuals without a previous diagnosis of diabetes. This response may reflect increased catecholamine and cortisol concentrations, hepatic glucose production, insulin resistance, and inflammatory activation. Stress-related hyperglycaemia has been associated with adverse cardiovascular and renal outcomes in both diabetic and non-diabetic patients, indicating that glycaemic disturbance during acute MI may have clinical relevance irrespective of a patient's documented diabetic status (13).

Several renal, cardiac, and metabolic parameters may help characterize the clinical severity of AKI following MI. Serum creatinine, blood urea nitrogen, urea, and eGFR provide complementary information regarding renal filtration and nitrogenous waste accumulation. Troponin concentrations reflect myocardial injury, whereas left ventricular ejection fraction provides an estimate of left ventricular systolic function. Random blood glucose and glycated haemoglobin may help differentiate acute glycaemic disturbance from longer-term dysglycaemia, although their interpretation requires consideration of the timing of measurement and the possibility of previously undiagnosed diabetes. Previous studies have identified renal dysfunction, hyperglycaemia, reduced cardiac function, and increased troponin concentrations as clinically relevant factors associated with adverse outcomes after acute MI (14,15). Contemporary MI management therefore increasingly emphasizes individualized assessment of renal, metabolic, and cardiac risk rather than relying on a single clinical or biochemical marker (16).

The clinical presentation and subsequent recognition of MI may also differ according to diabetic status. Patients with diabetes may experience less typical or less intense symptoms, which can delay presentation, diagnosis, reperfusion treatment, and renal assessment (17). Delayed recognition may increase exposure to prolonged haemodynamic and metabolic stress and could influence the severity of renal involvement. Nevertheless, the available literature has more commonly examined AKI within diabetic populations or assessed diabetes as one of several risk factors, rather than directly comparing

renal injury severity and related clinical parameters between diabetic and non-diabetic patients who have already developed AKI following MI.

Comparative evidence from Pakistani tertiary-care settings remains limited. A clearer understanding of the differences in renal filtration, glycaemic status, myocardial injury, cardiac function, and AKI severity between diabetic and non-diabetic patients may support more appropriately targeted clinical assessment during the acute post-MI period. Therefore, this study aimed to compare AKI severity and associated renal, glycaemic, and cardiac parameters between diabetic and non-diabetic patients who developed acute kidney injury following myocardial infarction.

MATERIALS AND METHODS

This hospital-based comparative cross-sectional study was conducted at the Punjab Institute of Cardiology and Mayo Hospital, Lahore. The study was designed to compare the severity of acute kidney injury and selected renal, glycaemic, and cardiac parameters between diabetic and non-diabetic patients following myocardial infarction. Reporting was structured in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology guidelines for cross-sectional studies. As the study included only patients who had already developed AKI following MI, the analysis evaluated differences in AKI severity and associated clinical parameters rather than the incidence or risk of developing AKI in the overall post-MI population.

A total of 248 patients with myocardial infarction followed by acute kidney injury were enrolled through purposive non-probability sampling. Participants were divided equally according to documented diabetic status, with 124 diabetic and 124 non-diabetic patients. The sample size was calculated using G*Power version 3.1.9.7 for a two-tailed comparison between two independent groups. The calculation was based on an effect size of 0.4597 derived from data reported by Pakfetrat et al. (18), a type I error probability of 0.05, statistical power of 95%, and an allocation ratio of 1:1. The minimum required sample was 124 participants in each group, producing a total sample of 248 and an estimated actual power of 0.9501.

Patients older than 40 years who had experienced either ST-segment elevation myocardial infarction or non-ST-segment elevation myocardial infarction and subsequently met the diagnostic criteria for AKI were eligible for inclusion. Both patients with and without a documented diagnosis of diabetes mellitus were included. Patients with pre-existing chronic kidney disease, valvular heart disease, or myocardial infarction without subsequent AKI were excluded. Patients who underwent coronary artery bypass grafting or received dialysis during the index clinical episode were retained in the study so that patients with more severe clinical presentations were not systematically excluded. Their inclusion was considered when interpreting the severity distribution because surgical procedures, haemodynamic instability, contrast exposure, and renal replacement therapy may be associated with more severe renal dysfunction.

Myocardial infarction was classified as STEMI or NSTEMI according to the diagnosis recorded in the patients' clinical records. Cases in which the myocardial infarction subtype could not be clearly determined from the available information were retained as an unclear MI-type category. Diabetes status was determined from the documented clinical diagnosis contained in the patients' medical records. Glycated haemoglobin and random blood glucose measurements were analysed as indicators of chronic and acute glycaemic status, respectively, but were not used alone to redefine the recorded diabetic classification.

Acute kidney injury was identified and staged according to the Kidney Disease: Improving Global Outcomes serum creatatinine criteria. Stage 1 AKI was defined as an increase in serum creatinine of at least 0.3 mg/dL within 48 hours or an increase to 1.5–1.9 times the available baseline value within seven days. Stage 2 was defined as an increase to 2.0–2.9 times baseline, whereas Stage 3 was defined as an increase to at least three times baseline, an absolute serum creatinine concentration of at least 4.0 mg/dL, or the requirement for renal replacement therapy. AKI diagnosis and staging were based on the available

serial serum creatinine measurements documented during the index hospitalization. Urine-output measurements were not consistently available for all participants and were therefore not incorporated into the principal staging analysis.

Data were collected using a structured data-collection instrument and information obtained from the participants' clinical records. Demographic and clinical variables included age, sex, diabetic status, type of myocardial infarction, AKI stage, and relevant renal, glycaemic, and cardiac measurements. Renal parameters included serum creatinine, urea, blood urea nitrogen, and eGFR. Glycaemic assessment included random blood glucose and glycated haemoglobin, while cardiac assessment included initial troponin concentration and left ventricular ejection fraction. Laboratory values and cardiac measurements were recorded using the results available during the index hospitalization.

The primary outcome was the severity of AKI according to the KDIGO staging classification. Secondary variables included serum creatinine, urea, blood urea nitrogen, eGFR, random blood glucose, glycated haemoglobin, initial troponin concentration, and left ventricular ejection fraction. Absolute serum creatinine categories were treated as descriptive concentration ranges and were not considered interchangeable with KDIGO AKI stages, which were based on changes in serum creatinine relative to the available baseline measurement.

Data were analysed using IBM SPSS Statistics version 27.0. Categorical variables were summarized as frequencies and percentages. Continuous variables were assessed for distributional characteristics using the Shapiro–Wilk and Kolmogorov–Smirnov tests. As the continuous variables demonstrated non-normal distributions, they were summarized using medians and interquartile ranges and compared between diabetic and non-diabetic groups using the Mann–Whitney U test. The corresponding standardized Z-statistic was used to estimate the effect size as (r=Z/\sqrt{N}), with values of approximately 0.10, 0.30, and 0.50 interpreted as small, moderate, and large effects, respectively.

Associations between diabetes status and categorical variables, including myocardial infarction type and AKI stage, were evaluated using the Pearson Chi-square test. Group-specific frequencies, percentages, correct degrees of freedom, and effect-size estimates were considered when interpreting these comparisons. Statistical significance was established at a two-sided p-value of less than 0.05. Because the analysis was based primarily on unadjusted comparisons, statistically significant variables were interpreted as associated factors rather than independent predictors of AKI severity.

Ethical approval was obtained from the research committee of the University of Lahore. Informed consent was obtained from the participating patients before data collection. Participant information was handled confidentially and used exclusively for research purposes. The study procedures were conducted in accordance with applicable institutional ethical requirements and the principles governing research involving human participants.

RESULTS

A total of 248 patients with acute kidney injury following myocardial infarction were included, comprising 124 patients with diabetes and 124 without diabetes. Of the total sample, 169 (68.1%) were male and 79 (31.9%) were female. Most participants were aged 61–80 years (n = 135, 54.4%), followed by 40–60 years (n = 101, 40.7%) and older than 80 years (n = 12, 4.8%). NSTEMI was the most frequently documented myocardial infarction subtype, occurring in 125 (50.4%) participants, while 99 (39.9%) had STEMI and 24 (9.7%) had an unclear MI subtype.

Table 1. Demographic and Myocardial Infarction Characteristics of the Study Population

Characteristic	Category	n	%
Diabetes status	Diabetic	124	50.0
	Non-diabetic	124	50.0
Sex	Male	169	68.1

Characteristic	Category	n	%
	Female	79	31.9
Age group, years	40–60	101	40.7
	61–80	135	54.4
	>80	12	4.8
Myocardial infarction type	NSTEMI	125	50.4
	STEMI	99	39.9
	Unclear	24	9.7

Abbreviations: NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction.

AKI severity was predominantly classified as Stage 2 or Stage 3. Stage 2 AKI was present in 124 (50.0%) participants, Stage 3 in 76 (30.6%), and Stage 1 in 48 (19.4%). Thus, 200 of the 248 enrolled patients (80.6%) had Stage 2 or Stage 3 AKI. These percentages describe the selected cohort of post-MI patients with established AKI and do not represent the incidence of AKI among all patients with myocardial infarction.

Random blood glucose concentrations were below 140 mg/dL in 86 (34.7%) participants, between 140 and 199 mg/dL in 99 (39.9%), and above 200 mg/dL in 63 (25.4%). The most frequently reported eGFR category was 60–89 mL/min/1.73 m², observed in 92 (37.1%) participants; 79 (31.9%) had an eGFR below 60 mL/min/1.73 m² and 77 (31.0%) had an eGFR of at least 90 mL/min/1.73 m².

Table 2. Overall Distribution of AKI Stage, Random Blood Glucose, and Estimated Glomerular Filtration Rate

Variable	Category	n	%
AKI stage	Stage 1	48	19.4
	Stage 2	124	50.0
	Stage 3	76	30.6
Random blood glucose, mg/dL	<140	86	34.7
	140–199	99	39.9
	>200	63	25.4
eGFR, mL/min/1.73 m²	<60	79	31.9
	60–89	92	37.1
	≥90	77	31.0

Abbreviations: AKI, acute kidney injury; eGFR, estimated glomerular filtration rate. AKI stages were reported according to the manuscript's KDIGO classification. Group-specific AKI-stage counts were not available in the supplied results.

Random blood glucose distributions differed substantially between the diabetic and non-diabetic groups. Among diabetic participants, 76 (61.3%) had random blood glucose below 140 mg/dL, 41 (33.1%) had values of 140–199 mg/dL, and 7 (5.6%) had values above 200 mg/dL. Among participants classified as non-diabetic, the corresponding frequencies were 10 (8.1%), 58 (46.8%), and 56 (45.2%), respectively. The association between documented diabetes status and random blood glucose category was statistically significant and large in magnitude ($\chi^2(2) = 91.681$, $p < 0.001$; Cramér's V = 0.608).

HbA1c distributions also differed markedly between groups. Among diabetic participants, 68 (54.8%) had HbA1c above 6.5%, 55 (44.4%) had HbA1c of 5.7–6.4%, and one (0.8%) had HbA1c below 5.7%. Among participants classified as non-diabetic, 90 (72.6%) had HbA1c below 5.7%, 31 (25.0%) had values of 5.7–6.4%, and three (2.4%) had HbA1c above 6.5%. The association between documented diabetes status and HbA1c category was statistically significant, with a large effect size ($\chi^2(2) = 153.249$, $p < 0.001$; Cramér's V = 0.786).

Table 3. Glycaemic Categories According to Documented Diabetes Status

Variable	Category	Diabetic, n (%)	Non-diabetic, n (%)	χ^2 (df)	p-value	Cramér's V
Random blood glucose, mg/dL	<140	(61.3)	(8.1)	(2)	<.001	0.608
	140–199	(33.1)	(46.8)			
	>200	(5.6)	(45.2)			

Variable	Category	Diabetic, n (%)	Non-diabetic, n (%)	χ^2 (df)	p-value	Cramér's V
HbA1c, %	<5.7	(0.8)	(72.6)	(2)	<0.001	0.786
	5.7–6.4	(44.4)	(25.0)			
	>6.5	(54.8)	(2.4)			

Data are presented as n (% within diabetes-status group). Pearson Chi-square tests were calculated from the complete reported category counts. Cramér's V values of approximately 0.10, 0.30, and 0.50 indicate small, moderate, and large associations, respectively. The three participants classified as non-diabetic who had HbA1c >6.5% should be verified against the original clinical records because this finding may reflect previously unrecognized diabetes, diagnostic misclassification, or another clinical explanation.

Absolute serum creatinine distributions also differed between the groups. Among diabetic participants, 25 (20.2%) had serum creatinine concentrations of 0.3–1.9 mg/dL, 59 (47.6%) had concentrations of 2.0–2.9 mg/dL, and 40 (32.3%) had concentrations above 3.0 mg/dL. Among non-diabetic participants, the corresponding frequencies were 63 (50.8%), 37 (29.8%), and 24 (19.4%). The association between diabetes status and serum creatinine category was statistically significant, with a moderate effect size ($\chi^2(2) = 25.451$, $p < 0.001$; Cramér's V = 0.320).

Table 4. Absolute Serum Creatinine Categories According to Diabetes Status

Serum creatinine, mg/dL	Diabetic, n (%)	Non-diabetic, n (%)	χ^2 (df)	p-value	Cramér's V
0.3–1.9	(20.2)	(50.8)	(2)	<0.001	0.320
2.0–2.9	(47.6)	(29.8)			
>3.0	(32.3)	(19.4)			

Data are presented as n (% within diabetes-status group). The concentration ranges are descriptive serum creatinine categories and should not be interpreted as KDIGO AKI stages, which depend principally on changes relative to baseline creatinine and the requirement for renal replacement therapy.

Mann–Whitney U testing showed statistically significant differences between diabetic and non-diabetic participants in eGFR, initial troponin, and random blood glucose. The eGFR comparison yielded U = 6409.000, Z = −2.430, and p = 0.015, corresponding to a small effect size of r = 0.154. Initial troponin also differed significantly between groups (U = 6561.500, Z = −2.133, p = 0.033), with a small effect size of r = 0.135. The strongest continuous-variable group difference was observed for random blood glucose (U = 4611.500, Z = −5.808, $p < 0.001$), with a moderate effect size of r = 0.369.

No statistically significant continuous-variable differences were identified for serum creatinine (p = 0.077), urea (p = 0.506), blood urea nitrogen (p = 0.643), or left ventricular ejection fraction (p = 0.468). The corresponding effect sizes were small or negligible. Although the categorical serum creatinine distribution differed significantly between groups, the Mann–Whitney comparison of the underlying continuous serum creatinine values did not reach statistical significance. These analyses address different characteristics of the data and should be interpreted together only after verification of the original continuous measurements, category coding, and group assignments.

Table 5. Mann–Whitney U Comparisons Between Diabetic and Non-Diabetic Participants

Variable	Mann–Whitney U	Wilcoxon W	Z	p-value	Effect size, r	Magnitude
Serum creatinine, mg/dL	6761.000	14387.000	−1.766	0.077	0.112	Small
Urea, mg/dL	7345.500	15220.500	−0.665	0.506	0.042	Negligible
Blood urea nitrogen, mg/dL	7444.500	15319.500	−0.463	0.643	0.029	Negligible
eGFR, mL/min/1.73 m²	6409.000	14035.000	−2.430	0.015	0.154	Small
Initial troponin, ng/mL	6561.500	14187.500	−2.133	0.033	0.135	Small
Left ventricular ejection fraction, %	7301.000	14927.000	−0.725	0.468	0.046	Negligible
Random blood glucose, mg/dL	4611.500	12486.500	−5.808	<0.001	0.369	Moderate

Effect size was calculated as (r=|Z|/\sqrt{248}). Values of approximately 0.10, 0.30, and 0.50 were interpreted as small, moderate, and large effects, respectively. Group-specific medians and interquartile ranges were not available in the supplied manuscript and should be inserted from the original SPSS output before publication. The sign of Z was not used to infer direction because the group-coding and rank order were not reported.

Overall, the findings demonstrated substantial differences in glycaemic profiles according to documented diabetes status. Participants classified as non-diabetic more frequently had random blood glucose above 200 mg/dL, whereas participants with diabetes more frequently had HbA1c above 6.5%. However, the presence of HbA1c above 6.5% in three participants classified as non-diabetic requires verification before the raised random glucose values in this group can be attributed specifically to stress-related hyperglycaemia.

Renal comparisons showed a higher proportion of diabetic participants in the upper absolute serum creatinine categories, and eGFR differed significantly between groups. Nevertheless, the continuous serum creatinine comparison was not statistically significant, and the absence of group-specific medians and IQRs prevents precise quantification of the direction and clinical size of these differences. Initial troponin differed significantly between groups, while left ventricular ejection fraction did not. Because these were unadjusted comparisons, the observed differences should be interpreted as associations rather than independent effects of diabetes.

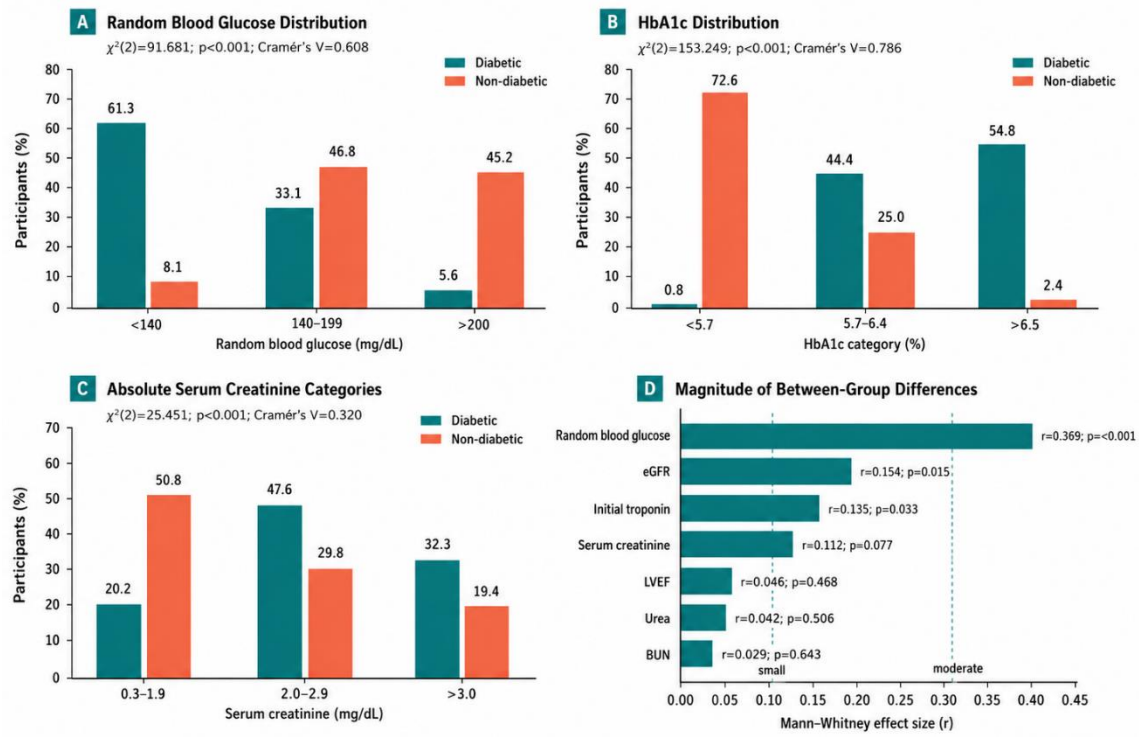

Figure 1. Comparative glycaemic, renal, and cardiac profiles in diabetic and non-diabetic patients with acute kidney injury following myocardial infarction. Panel A presents random blood glucose categories according to documented diabetes status. Random blood glucose above 200 mg/dL was observed in 45.2% of non-diabetic participants compared with 5.6% of diabetic participants, whereas values below 140 mg/dL were recorded in 61.3% and 8.1%, respectively. The association was statistically significant and large in magnitude ($\chi^2(2) = 91.681$, $p < 0.001$; Cramér's $V = 0.608$). Panel B shows HbA1c distributions; 54.8% of diabetic participants had HbA1c >6.5%, compared with 2.4% of those classified as non-diabetic, while HbA1c <5.7% occurred in 0.8% and 72.6%, respectively ($\chi^2(2) = 153.249$, $p < 0.001$; Cramér's $V = 0.786$). Panel C demonstrates absolute serum creatinine categories. Concentrations above 3.0 mg/dL were present in 32.3% of diabetic and 19.4% of non-diabetic participants, while concentrations of 0.3–1.9 mg/dL occurred in 20.2% and 50.8%, respectively ($\chi^2(2) = 25.451$, $p < 0.001$; Cramér's $V = 0.320$). Panel D summarizes Mann–Whitney effect sizes. The largest between-group difference was observed for random blood glucose ($r = 0.369$, $p < 0.001$), followed by eGFR ($r = 0.154$, $p = 0.015$), initial troponin ($r = 0.135$, $p = 0.033$), and serum creatinine ($r = 0.112$, $p = 0.077$). Differences in LVEF, urea, and blood urea nitrogen were negligible. Absolute serum creatinine categories are descriptive and should not be interpreted as equivalent to KDIGO AKI stages.

DISCUSSION

This study compared renal, glycaemic, and cardiac characteristics between diabetic and non-diabetic patients who developed acute kidney injury following myocardial infarction. The cohort was predominantly male and older, and most participants had Stage 2 or Stage 3 AKI. The principal comparative findings were significant differences in eGFR, initial troponin, random blood glucose,

HbA1c category, and absolute serum creatinine category according to documented diabetes status. The between-group effect was moderate for random blood glucose but small for eGFR and troponin. Serum creatinine, urea, blood urea nitrogen, and left ventricular ejection fraction did not differ significantly when analysed as continuous variables. Because all participants had already developed AKI, these findings describe clinical differences within an AKI-positive post-MI cohort and do not estimate the incidence or relative risk of AKI following MI.

The predominance of moderate-to-severe AKI in the overall cohort indicates a substantial burden of renal dysfunction among the enrolled patients. Stage 2 AKI accounted for 50.0% of participants and Stage 3 for 30.6%, meaning that 80.6% of the selected cohort had Stage 2 or Stage 3 disease. This distribution should be interpreted within the context of recruitment from tertiary cardiac-care hospitals, where patients may have more complex or severe presentations. A retrospective analysis of patients with acute myocardial infarction similarly demonstrated that severe AKI was associated with higher mortality and longer intensive-care stays than milder renal injury, supporting the clinical importance of identifying renal deterioration early during acute cardiac illness (19). Nevertheless, the present study did not include post-MI patients without AKI and therefore could not establish how frequently AKI developed in the wider MI population.

Several interacting mechanisms may contribute to renal dysfunction after myocardial infarction. Reduced cardiac output, renal hypoperfusion, systemic inflammation, sympathetic activation, oxidative stress, haemodynamic instability, and exposure to iodinated contrast media or nephrotoxic agents may all impair renal function. These factors can operate in both diabetic and non-diabetic patients and form part of the acute cardiorenal interaction. However, contrast exposure, cardiogenic shock, medication use, and detailed haemodynamic variables were not systematically recorded in the present study. Their contribution to the observed renal differences could therefore not be quantified.

A notable finding was the marked difference in random blood glucose distributions. Random blood glucose above 200 mg/dL was recorded in 45.2% of participants classified as non-diabetic but in only 5.6% of diabetic participants. The categorical association was large, and the Mann–Whitney analysis also demonstrated a moderate between-group effect for random blood glucose. Acute hyperglycaemia during critical illness may result from catecholamine and cortisol release, increased hepatic glucose production, inflammatory activation, and transient insulin resistance. A matched case-control study of non-diabetic critically ill patients found that stress hyperglycaemia was associated with AKI across several glucose-based definitions, independently of sepsis and shock, supporting the potential relevance of acute metabolic dysregulation to renal injury (20).

The random glucose pattern nevertheless requires cautious interpretation. The timing of glucose measurement relative to admission, insulin administration, oral hypoglycaemic treatment, nutritional intake, and reperfusion treatment was not reported. Diabetic patients may have received glucose-lowering therapy before sampling, whereas acutely ill patients without documented diabetes may have been sampled before metabolic stabilization. Furthermore, three participants classified as non-diabetic had HbA1c values above 6.5%, raising the possibility of previously unrecognized diabetes or diagnostic misclassification. The term acute hyperglycaemia is therefore more defensible than confirmed stress hyperglycaemia unless the original records establish the absence of pre-existing diabetes and a validated stress-hyperglycaemia definition is applied.

HbA1c distributions were strongly associated with documented diabetes status. More than half of the diabetic group had HbA1c above 6.5%, whereas most non-diabetic participants had values below 5.7%. This pattern was broadly consistent with chronic dysglycaemia among patients with known diabetes, although an HbA1c threshold of 6.5% should not automatically be interpreted as inadequate glycaemic control in every patient. Long-standing hyperglycaemia may increase renal susceptibility through endothelial dysfunction, microvascular injury, oxidative stress, inflammation, and impaired autoregulation. Exploratory findings from the DEVOTE programme similarly identified

hyperglycaemia and hypertension as important factors associated with kidney disorders in patients with type 2 diabetes and high cardiovascular risk (21). Reviews have also described the diabetic kidney as particularly vulnerable to acute haemodynamic and metabolic insults superimposed on chronic structural and functional abnormalities (22).

Renal comparisons demonstrated a significant difference in eGFR between diabetic and non-diabetic participants, although the corresponding effect size was small. Rapid deterioration in kidney function has previously been associated with an increased risk of heart failure in patients with type 2 diabetes, emphasizing the close relationship between renal decline and cardiovascular outcomes (23). Renal impairment is also clinically important in non-diabetic populations, where cardiorenal events may independently worsen cardiovascular prognosis (24). The current findings therefore support renal assessment in all post-MI patients with AKI rather than restricting intensified monitoring to those with known diabetes.

The interpretation of eGFR during AKI requires caution because commonly used creatinine-based equations assume relatively stable renal function. During acute and rapidly changing serum creatinine concentrations, calculated eGFR may not accurately represent the instantaneous filtration rate. The equation used to calculate eGFR was also not specified. Consequently, the statistically significant group difference should be interpreted as a comparative renal-function signal rather than definitive evidence that eGFR was diagnostically more reliable than serum creatinine during the acute episode.

Absolute serum creatinine categories differed significantly by diabetes status, with diabetic participants more frequently represented in the higher concentration categories. In contrast, the Mann–Whitney comparison of continuous serum creatinine values did not reach statistical significance. These findings are not necessarily equivalent because categorization and rank-based continuous analysis examine different aspects of the distribution; however, the apparent discrepancy requires verification against the original participant-level data. Category cut-offs, group coding, tied observations, missing values, and the continuous creatinine output should be audited before publication. Importantly, the reported absolute creatinine categories must not be interpreted as KDIGO stages because KDIGO staging is principally based on change from baseline and renal replacement therapy rather than isolated concentration bands.

Initial troponin concentrations differed significantly between the groups, with a small effect size. Diabetes is associated with accelerated atherosclerosis, endothelial dysfunction, chronic inflammation, microvascular disease, and a greater burden of cardiovascular comorbidity, all of which may influence the extent and consequences of myocardial injury (25). However, the direction and clinical magnitude of the troponin difference cannot be fully evaluated without group-specific medians, interquartile ranges, assay type, reference limits, and sampling time. The finding should therefore be reported as a statistically significant difference in troponin distribution rather than proof of independently greater infarct severity among diabetic patients.

Left ventricular ejection fraction did not differ significantly between the groups. This suggests that the measured distribution of left ventricular systolic function was broadly comparable, but it does not establish equivalent overall cardiac dysfunction. LVEF does not capture infarct size, right ventricular function, diastolic dysfunction, cardiogenic shock, valvular abnormalities, or dynamic haemodynamic status. The absence of a statistically significant LVEF difference should therefore not be used to infer that both groups experienced the same degree of cardiovascular compromise.

The study had several strengths. It included a comparatively large and equally sized diabetic and non-diabetic sample recruited from two major tertiary-care hospitals. It evaluated renal, metabolic, and cardiac parameters within the same clinical cohort and used non-parametric tests in response to non-normal continuous data. The revised analysis also demonstrated the magnitude of group differences

through effect-size estimates, showing that statistical significance did not necessarily correspond to a large clinical separation.

Several limitations materially affect interpretation. The cross-sectional analytical framework did not establish temporal or causal relationships and included only patients who had already developed AKI. The method used to determine baseline creatinine was not clearly documented, despite its importance for KDIGO classification. Urine-output criteria were unavailable, which may have led to incomplete staging. Group-specific AKI-stage counts were not supplied, preventing a direct comparison of KDIGO severity between diabetic and non-diabetic patients. Purposive equal-group sampling may have introduced selection bias and prevents estimation of the natural prevalence of diabetes or AKI in the source population.

Diabetes classification relied on documented clinical status, and three non-diabetic participants had HbA1c above 6.5%. The timing of random glucose, troponin, creatinine, and LVEF measurements was not specified. Exposure to contrast media, coronary intervention, CABG timing, cardiogenic shock, medication use, duration of diabetes, hypertension, and other relevant confounders were not consistently captured. The distinction between chronic dialysis and dialysis initiated for Stage 3 AKI was also unclear. Analyses were primarily unadjusted, so the identified variables should be regarded as associated factors rather than independent predictors. No follow-up was available to determine renal recovery, recurrent cardiovascular events, dialysis dependence, or mortality. Prediction models in diabetic AKI populations have shown that sustained metabolic and clinical abnormalities may influence short-term renal recovery, but the present study did not evaluate such longitudinal outcomes (26).

Overall, the findings indicate that diabetic and non-diabetic post-MI patients with AKI may demonstrate distinct metabolic and clinical profiles. Diabetic participants showed different eGFR, troponin, HbA1c, and absolute serum creatinine distributions, while participants classified as non-diabetic demonstrated a high frequency of acute hyperglycaemia. These observations support comprehensive renal and glycaemic assessment irrespective of documented diabetes status. Prospective studies enrolling all patients with myocardial infarction, applying complete KDIGO criteria, confirming diabetes status, recording treatment and haemodynamic exposures, and using adjusted longitudinal analyses are required to determine whether diabetes independently influences AKI development, severity, recovery, and clinical outcomes.

CONCLUSION

Among patients who developed acute kidney injury following myocardial infarction, diabetic and non-diabetic groups demonstrated significant differences in eGFR, initial troponin, random blood glucose, HbA1c category, and absolute serum creatinine distribution, while continuous serum creatinine, urea, blood urea nitrogen, and left ventricular ejection fraction did not differ significantly. Acute hyperglycaemia was frequent among participants classified as non-diabetic, whereas chronic glycaemic elevation was more common among those with documented diabetes. These findings support careful renal, glycaemic, and cardiac assessment in post-MI patients with AKI regardless of known diabetes status, but they do not establish diabetes as an independent cause of greater AKI severity.

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