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Impact of Glycated Hemoglobin (HbA1c) on Disease Severity and Short-Term Outcomes in Patients with Acute Coronary Syndrome: A Single-Center Prospective Study

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Abstract

Background: Elevated glycated hemoglobin (HbA1c), a marker of chronic glycaemic status, may influence the clinical severity, angiographic burden, and short-term outcomes in patients hospitalized with acute coronary syndrome. **Objective:** To examine the association between elevated HbA1c and all-cause mortality and morbidity in hospitalized ACS patients. **Methods:** This single-centre, hospital-based prospective observational study was conducted in the Department of Cardiology, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, from July 2025 to December 2025 among 214 hospitalized patients with acute coronary syndrome. Demographic, clinical, laboratory, echocardiographic, and angiographic data were recorded in a pre-structured proforma, and associations of HbA1c with clinical severity, coronary artery disease burden, in-hospital complications, and 6-month outcomes were analysed. **Results:** The study included 214 patients with acute coronary syndrome, with a mean age of 56.9 years;

81.3% were men, and STEMI was the predominant presentation (81.3%). Mean HbA1c was 7.65%, and 47.7% had HbA1c >7.0%. Diabetes mellitus (50.9%), dyslipidemia (48.6%), and hypertension (45.8%) were common. Mean GRACE, Gensini, and SYNTAX scores were 117.6, 53.5, and 19.2, respectively, while multivessel disease was present in 68.7%. With increasing HbA1c, Killip class worsened, multivessel disease rose from 22.2% to 92.2%, triple-vessel disease from 5.6% to 58.8%, and total occlusion from 5.6% to 70.6% (all $p<0.001$). HbA1c showed a weak positive correlation with GRACE score. Higher HbA1c was also associated with more left ventricular failure (5.6% to 42.2%), contrast-induced nephropathy, sepsis, longer hospital stay, higher 6-month CCF, and greater mortality. **Conclusion:** Elevated HbA1c was associated with greater clinical and angiographic severity, more in-hospital complications, longer hospital stay, and poorer 6-month outcomes in acute coronary syndrome, supporting its role as a simple prognostic biomarker for risk stratification.

Keywords: Acute coronary syndrome, HbA1c, Coronary artery disease severity, GRACE risk score, Gensini score, SYNTAX score

Introduction

Acute coronary syndrome (ACS), encompassing ST-segment elevation myocardial infarction and non-ST-segment elevation acute myocardial infarction, remains a major cause of cardiovascular morbidity and mortality, particularly in populations with a high burden of metabolic risk factors. India carries a substantial diabetes burden, with the International Diabetes Federation estimating 89.8 million adults aged 20–79 years living with diabetes in 2024, projected to rise to 156.7 million by 2050.(1) This epidemiologic transition is highly relevant to ACS because chronic dysglycaemia accelerates endothelial dysfunction, plaque progression, thrombogenicity, and adverse ventricular remodeling.(2) Dysglycaemia is extremely common in patients presenting with myocardial infarction. In the ACS QUIK analysis of 21,374 patients with acute myocardial infarction from India, 44.4% had diabetes, and diabetes was independently associated with higher in-hospital death (adjusted odds ratio [aOR] 1.46, 95% CI 1.12–1.89), 30-day major adverse cardiovascular events (aOR 1.33, 95% CI 1.14–1.55), and 30-day death (aOR 1.40, 95% CI 1.16–1.69).(3) Similarly, the prospective NORIN STEMI registry from North India showed that among patients without known

diabetes, protocolized HbA1c testing identified prediabetes in 28% and newly detected diabetes in 13%; dysglycaemic groups also had higher post-MI left ventricular dysfunction and higher 30-day mortality or readmission, with adjusted odds ratios of 1.44 for prediabetes, 1.57 for newly detected diabetes, and 1.51 for established diabetes compared with euglycaemia.(4)

HbA1c has particular appeal in ACS because it reflects mean glycaemic exposure over the preceding 2–3 months and is less affected by the acute neurohormonal stress response than admission glucose alone. Liu et al., in a meta-analysis of 20 studies including 13,224 patients hospitalized with coronary artery disease, found that elevated HbA1c was associated with increased short-term mortality (OR 2.32, 95% CI 1.61–3.35) and long-term mortality (OR 1.54, 95% CI 1.23–1.94), with the excess risk being especially evident in patients without known diabetes.(2) In contrast, Hadjadj et al. showed that admission plasma glucose remained prognostic even after adjustment for HbA1c, underscoring that acute and chronic glycaemic indices may capture different but complementary dimensions of risk.(5) Several clinical studies have further suggested that HbA1c may relate not only to outcome but also to angiographic severity in ACS. Cakmak et al. reported a significant relationship between admission HbA1c and mortality after acute myocardial infarction (p=0.009), along with a positive correlation between HbA1c and total ischaemic score (r=0.482, p=0.001).(6) Dutta et al. demonstrated a significant correlation between HbA1c, SYNTAX score, and the number of diseased coronary vessels in non-diabetic patients.(7) El-Sherbiny et al. likewise found that higher admission HbA1c in acute STEMI was associated with more severe coronary artery disease, lower rates of complete revascularization, and more short-term adverse cardiac events.(8) However, available studies have often been restricted to STEMI, non-diabetic cohorts, or selected angiographic end points, leaving uncertainty about the broader relationship of HbA1c with clinical severity, angiographic burden, in-hospital complications, and short-term outcomes across the full ACS spectrum. Against this background, the aim of the present study was to examine the association between elevated HbA1c and all-cause mortality and morbidity in hospitalized ACS patients.

Materials and Methods

This was a single-center, hospital-based, prospective observational study was conducted in the Department of Cardiology, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, Karnataka, India over a period of 18 months between August 2023 and March 2025.

The participants (and their attenders) were given the Participant Information Sheet (PIS) in their native language, and its contents were verbally explained to ensure their understanding and satisfaction. All consecutive patients aged 21 years and above who were admitted with acute coronary syndrome (electrocardiographic changes together with elevated cardiac biomarkers) and diagnosed as ST-segment elevation myocardial infarction (STEMI) or non-ST-segment elevation myocardial infarction (NSTEMI) were enrolled after obtaining written informed consent. However, patients with chronic kidney disease, chronic liver disease, hemoglobinopathies, acute or chronic blood loss, and autoimmune hemolytic anemia were excluded.

A minimum sample size of 214 participants was estimated for the present study using the single-population proportion formula, $n = Z^2pq/d^2$. The anticipated proportion (p) of elevated HbA1c among patients with acute coronary syndrome was taken as 44.0%, based on Vora et al., who reported HbA1c >6.5% in 44 of 100 patients with acute coronary syndrome;(9) accordingly, q was 56.0%. Assuming a 95% confidence level (Z = 1.96), an absolute precision of 7%, and 10% nonresponse rate, the minimum required sample size was rounded off to 214 patients; enrolled using nonprobability sampling technique – complete, consecutive enumeration. Baseline data were collected at admission through direct patient and attender interview, bedside clinical assessment, and review of hospital case records. Demographic details, relevant family history, cardiovascular risk factors and habits, pre-existing comorbidities, and past cardiovascular and cerebrovascular events were documented systematically. Clinical presentation at admission, including the type of acute coronary syndrome, Killip class, systolic and diastolic blood pressure, heart rate, and the occurrence of in-hospital complications, was recorded. Anthropometric and biochemical parameters, including body mass index, fasting blood sugar, postprandial blood sugar, HbA1c, thyroid-stimulating hormone, troponin I status, and other routine investigations performed as part of standard inpatient care, were entered from laboratory reports. Left ventricular ejection fraction was noted from echocardiographic assessment. All patients underwent coronary angiography, and angiographic findings were recorded in detail, including the culprit and involved vessels, presence of single-, double-, or triple-vessel disease, multivessel involvement, total occlusion, pre-intervention TIMI flow grade, and the method of revascularization or medical management. The severity of acute coronary syndrome was assessed using the GRACE ACS risk score, while the extent and complexity of coronary artery disease were quantified using the Gensini and SYNTAX

scoring systems based on angiographic findings. Patients were subsequently followed during hospital stay for procedural and systemic complications, duration of hospitalization, and short-term outcomes including congestive cardiac failure and mortality within 6 months.

Statistical analysis: Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean and standard deviation (SD), while categorical variables were expressed as frequency and percentage. Comparisons of continuous variables across HbA1c categories were carried out using one-way analysis of variance (ANOVA) for normally distributed data and the Kruskal–Wallis test for non-normally distributed data, as applicable. Associations between categorical variables were examined using the chi-square test or Fisher's exact test wherever the expected cell counts were small. Correlation between HbA1c and continuous severity indices such as GRACE ACS risk score, Gensini score, and SYNTAX score was assessed using Pearson's correlation coefficient for parametric data and Spearman's rank correlation coefficient for non-parametric data. All statistical tests were two-tailed, and a p value of less than 0.05 was considered statistically significant.

Results

The study included 214 hospitalized patients with acute coronary syndrome, with a mean age of 56.9 years, and most were in the 51–60 year age group (32.7%). Males predominated (81.3%). Common co-morbidities were diabetes mellitus (50.9%), dyslipidemia (48.6%), and hypertension (45.8%), while smoking (58.4%) and alcohol use (36.4%) were frequent habits. Mean systolic and diastolic blood pressures were 133.7 and 82.7 mmHg, respectively, and the mean HbA1c was 7.65%, with nearly half of the patients having HbA1c >7.0% (47.7%). STEMI was the predominant presentation (81.3%), and Killip class I was seen in 54.7% of cases. Mean GRACE ACS risk score was 117.6, while multivessel coronary artery disease was present in 68.7%. The mean Gensini and SYNTAX scores were 53.5 and 19.2, respectively. Left ventricular failure was the most common ACS-related complication (27.1%), and in-hospital mortality was 3.7%, while 27.1% developed congestive cardiac failure within 6 months (Table 1).

Patients with lower HbA1c values were predominantly in Killip class I, decreasing from 88.9% in the <5.0% group to 41.2% in the >7.0% group, while higher Killip classes became more frequent

at higher HbA1c levels; for example, Killip class III and IV together accounted for 41.2% of patients in the >7.0% category. Multivessel disease also increased markedly across HbA1c categories, from 22.2% in the <5.0% group to 92.2% in those with HbA1c >7.0%. A similar gradient was observed in vessel disease pattern, with single-vessel disease declining from 72.2% to 3.9%, whereas triple-vessel disease rose from 5.6% to 58.8% across the same HbA1c categories. Total occlusion likewise showed a strong increasing trend, from 5.6% in the <5.0% group to 70.6% in the >7.0% group. All associations were statistically highly significant ($p<0.001$) (Table 2).

The mean GRACE ACS risk score ranged from 104.96 in the 5.6–6.0% HbA1c group to 124.2 in the 6.6–7.0% group, while the mean Gensini score ranged from 44.14 to 65.60 across the same categories. Similarly, the mean SYNTAX score was lowest in the <5.0% group at 13.55 and highest in the 6.1–6.5% group at 22.48. Overall p values for GRACE score (0.205), Gensini score (0.491), and SYNTAX score (0.061) indicated no significant difference across HbA1c groups. However, correlation analysis showed a weak but significant positive association between HbA1c and GRACE ACS risk score, with Pearson's $r = 0.229$ and Spearman's $rho = 0.206$ (both $p < 0.05$) (Table 3).

Left ventricular failure increased steadily across HbA1c categories, from 5.6% in the <5.0% group to 42.2% in the >7.0% group ($p<0.001$). Similarly, cardiogenic shock, cardiac arrest, ventricular tachycardia, complete heart block, atrial fibrillation, frequent VPCs, pericarditis-related complications, and stroke were observed predominantly in patients with HbA1c >7.0%, with statistically significant associations. Among post-procedural systemic complications, contrast-induced nephropathy and sepsis were markedly more frequent in the highest HbA1c group, occurring in 33.3% and 35.3%, respectively (both $p<0.001$). Congestive cardiac failure within 6 months also rose progressively from 5.6% in the lowest HbA1c category to 38.2% in the >7.0% group ($p<0.001$), while 6-month mortality was significantly associated with higher HbA1c ($p=0.038$). Hospital stay was also longer in patients with elevated HbA1c, with only 47.1% of the >7.0% group discharged within 2–5 days compared with 88.9% in the <5.0% group ($p<0.001$) (Table 4).

Discussion

The present cohort had a mean age of 56.9 years with marked male predominance (81.3%), and this demographic pattern is broadly consistent with Xavier et al. (2008).(10) In the CREATE registry of 20,468 definite ACS cases from India, the mean age was 57.5 years and 60.6% had STEMI, while the North Eastern India registry reported a mean age of 56.5 years and STEMI in 72.4% of cases.(10, 11) The much higher STEMI proportion in the present study (81.3%) therefore likely reflects the case-mix of a tertiary cardiac center receiving a larger share of high-acuity infarction referrals rather than a different disease biology alone. Our burden of diabetes mellitus (50.9%), hypertension (45.8%), dyslipidemia (48.6%), and smoking (58.4%) also indicate a strongly cardiometabolic-risk-enriched population, which is clinically relevant because Indian and Asian ACS cohorts with heavier metabolic-risk loading generally show more extensive coronary disease and worse short-term outcomes.(12) A particularly important observation in the present study was the high chronic glycemic burden, with mean HbA1c of 7.65% and 47.7% of patients falling in the >7.0% category. This supports the view that HbA1c in ACS is not merely a diabetes descriptor, but also a practical marker of previously unrecognized or poorly controlled dysglycemia at a prognostically vulnerable time. Lugg et al., using a post-ACS HbA1c screening pathway in 399 patients without prior diabetes, identified newly diagnosed diabetes in 10.8% and prediabetes in 14.3%, showing that simple HbA1c-based screening can uncover a substantial hidden dysglycemic burden after ACS.(13) Likewise, de Mulder et al. found that among hyperglycemic ACS patients, 35% had previously undiagnosed diabetes and 44% had impaired glucose metabolism on OGTT, while patients with abnormal glucose metabolism had higher admission HbA1c values and higher Killip class.(14) Against this background, the high proportion of patients with HbA1c above diabetic thresholds in the present series likely represents both chronic metabolic injury before the index event and a subgroup at intrinsically higher clinical risk after the event.

The progressive shift toward worse Killip class with rising HbA1c in the present study is therefore clinically coherent and pathophysiologically plausible. Killip class I fell from 88.9% in the <5.0% HbA1c group to 41.2% in the >7.0% group, whereas Killip class III–IV together increased to 41.2% in the highest HbA1c category. Blasco et al., in 601 acute myocardial infarction patients without known diabetes, similarly showed that higher HbA1c was associated with Killip class >1,

atrial fibrillation, higher heart rate, and higher in-hospital mortality; in multivariable analysis, HbA1c remained associated with in-hospital mortality with an odds ratio of 1.5.(15) The China STEMI Care Project-2, which included 8,370 patients, also reported that HbA1c was significantly associated with CAD extent and severity, and that diabetes patients were more likely to have higher Killip class and multivessel CAD.(12) These parallels suggest that chronic hyperglycemia may identify patients with poorer myocardial reserve, more advanced microvascular dysfunction, and worse hemodynamic response at presentation, even before considering angiographic anatomy.

The angiographic findings in the present study were among the strongest signals supporting this interpretation. Multivessel disease rose from 22.2% in the lowest HbA1c category to 92.2% in the >7.0% group, triple-vessel disease increased from 5.6% to 58.8%, and total occlusion from 5.6% to 70.6%, all with $p<0.001$. Hegde et al. reported that in ACS, diabetics with high HbA1c had greater vessel involvement and more triple/multivessel disease than nondiabetics.(16) In 290 nondiabetic STEMI patients, Ghaffari et al. found a significantly higher CAD severity score in the high-HbA1c group than in the low-HbA1c group (7.7 ± 2.7 vs 5.5 ± 2.6; $p=0.001$), and the high-HbA1c group also had higher 12-month mortality and readmission.(17) Dar et al. further showed that among apparently non-diabetic ACS patients, 41.8% were diabetic and 39.4% prediabetic by HbA1c, with a significant positive correlation between HbA1c, Gensini score, and the number of vessels involved.(18) Mechanistically, this association is biologically credible because AGE–RAGE signaling amplifies oxidative stress, TNF-α signaling, and endothelial dysfunction; Gao et al. demonstrated in diabetic mice that soluble RAGE partly restored impaired coronary vasodilation,(19) while Kajikawa et al. showed in humans that the AGE/sRAGE ratio independently predicted impaired flow-mediated dilation.(20) Together, these data support the concept that HbA1c reflects cumulative vascular injury that can manifest as more diffuse, occlusive, and anatomically complex coronary disease at the time of ACS.

At the same time, the present study demonstrated an important nuance: although categorical indicators of disease burden worsened sharply across HbA1c strata, mean GRACE, Gensini, and SYNTAX scores did not differ significantly across categories, even though HbA1c showed a weak but significant positive correlation with GRACE score (Pearson $r=0.229$; Spearman $rho=0.206$). This apparent dissociation is not unexpected, because continuous summary scores may be influenced by substantial within-group variability and by lesion weighting systems that do not

perfectly mirror the clinical impression conveyed by multivessel or total-occlusion patterns. Indeed, Wang et al. studied 292 ACS patients and found that HbA1c was not independently associated with Gensini score or with high Gensini burden (>40) after adjustment.(21) Habib et al. likewise found no significant association between HbA1c and SYNTAX severity in non-diabetic ACS, reporting a correlation coefficient of 0.142 (p=0.124) and no HbA1c difference between SYNTAX ≤22 and >22 groups.(22) In contrast, Liu et al. reported in 549 non-diabetic ACS patients undergoing PCI that GRACE score was positively associated with HbA1c, and that adding HbA1c improved the GRACE model's AUC from 0.75 to 0.80 (p=0.012).(23) Thus, the present finding of a modest GRACE correlation but non-significant between-group differences in angiographic scores fits within the mixed existing literature and suggests that HbA1c may relate more consistently to global clinical risk than to any single anatomical scoring system.

The complication profile further strengthens the argument that elevated HbA1c identifies a clinically fragile ACS phenotype. Left ventricular failure increased from 5.6% in the <5.0% group to 42.2% in the >7.0% group, while cardiogenic shock, cardiac arrest, ventricular tachycardia, complete heart block, atrial fibrillation, frequent VPCs, and stroke clustered predominantly in the highest HbA1c category. In the CCC-ACS study of 27,337 patients, Zhao et al. found that patients with HbA1c ≥6.5% had the highest in-hospital MACE incidence, 13.4% compared with 8.7% for HbA1c <5.7% and 10.5% for HbA1c 5.7%–6.4%, and HbA1c remained significantly associated with MACE risk in logistic regression.(24) Kmet et al. similarly concluded that admission HbA1c, rather than admission or fasting glucose, predicted mortality and major adverse events in NSTE-ACS.(25) Pan et al., in a meta-analysis of 25 studies including 304,253 ACS patients, reported a pooled relative risk of 1.246 for in-hospital mortality when HbA1c was analyzed categorically.(26) The post-procedural findings are equally noteworthy: Qin et al. showed that contrast-induced nephropathy occurred in 26.1% of patients with elevated HbA1c versus 14.3% without elevated HbA1c (p=0.027), and in the ACS subgroup the CIN incidence was 38.1%.(27) The higher sepsis burden in the present high-HbA1c group is also biologically plausible, because Mor et al. demonstrated that higher updated HbA1c was associated with increased risk of hospital-treated infection,(28) and Jafar et al. showed that even short-term hyperglycemia can significantly alter innate immune responses.(29)

The adverse signal persisted beyond the index admission. In the present study, congestive cardiac failure within 6 months rose from 5.6% in the lowest HbA1c stratum to 38.2% in the >7.0% group, 6-month mortality was significantly associated with higher HbA1c, and the proportion discharged within 2–5 days fell from 88.9% to 47.1% as HbA1c increased. These observations are consistent with prior outcome studies showing that HbA1c captures longer-term risk after ACS. Naito et al. followed 452 non-diabetic ACS patients treated with PCI and found that the combination of elevated admission glucose and elevated HbA1c was independently associated with long-term adverse outcomes; the composite endpoint occurred in 13.3% during a median follow-up of 4.7 years.(30) Kowalczyk et al. reported that among invasively treated AMI patients with newly detected glucose abnormalities, post-hospital mortality in those with impaired glucose tolerance was 4.5% when HbA1c was ≤5.9% versus 25.0% when HbA1c was >5.9%, and among newly diagnosed diabetes patients it was 6.4% for HbA1c ≤7.0% versus 14.3% for HbA1c >7.0%.(31) Ghaffari et al. likewise found 12-month mortality of 7.7% in the high-HbA1c STEMI group versus 2.7% in the low-HbA1c group.(17) Taken together, the present data support the interpretation that HbA1c is not just a metabolic background variable in ACS, but a clinically meaningful marker of hemodynamic instability, coronary disease burden, procedural vulnerability, and short-term post-discharge heart failure risk.

The present study had certain limitations. Being a single-center, hospital-based prospective observational study, the results may have limited generalizability to other populations and care settings. The observational design also precluded establishment of a causal relationship between elevated HbA1c and adverse outcomes in acute coronary syndrome. Although the overall sample size was adequate for descriptive and comparative analyses, some HbA1c subgroups contained relatively small numbers of patients, which may have reduced the statistical power for subgroup comparisons, particularly for continuous angiographic severity scores such as the Gensini and SYNTAX scores. HbA1c was measured only at the time of presentation and therefore did not capture changes in glycaemic control during follow-up. In addition, follow-up was limited to 6 months, so longer-term cardiovascular outcomes could not be assessed. Residual confounding from unmeasured factors such as differences in prior treatment, duration of diabetes, medication adherence, infarct size, and socioeconomic or lifestyle variables also cannot be excluded.

Conclusion

In conclusion, elevated HbA1c was significantly associated with greater clinical severity, more extensive coronary artery disease, and poorer short-term outcomes among hospitalized patients with acute coronary syndrome. Patients with higher HbA1c levels had higher Killip class, more frequent multivessel and triple-vessel disease, greater rates of total occlusion, increased in-hospital complications, longer hospital stay, and higher 6-month congestive cardiac failure and mortality. Although differences in mean Gensini and SYNTAX scores across HbA1c categories were not statistically significant, HbA1c showed a weak but significant positive correlation with GRACE ACS risk score. These findings suggest that HbA1c may serve as a simple and clinically useful prognostic biomarker for risk stratification in patients presenting with acute coronary syndrome.

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Table 1. Baseline demographic, clinical, laboratory, angiographic, procedural, and outcome characteristics of the study population (N=214)

Characteristic		Value
Age, years; mean (SD)		56.87 (11.84)
Age, years	21-30 years	3 (1.4)
	31-40 years	15 (7.0)
	41-50 years	50 (23.4)
	51-60 years	70 (32.7)
	61-70 years	44 (20.6)
	>70 years	32 (15.0)
Sex	Male	174 (81.3)
	Female	40 (18.7)
Family history	Diabetes mellitus	17 (7.9)
	Hypertension	21 (9.8)
	Ischemic heart disease	1 (0.5)
	Cerebrovascular accident	1 (0.5)
	Myocardial infarction	4 (1.9)
Co-morbidities	Diabetes mellitus	109 (50.9)
	Hypertension	98 (45.8)
	Dyslipidemia	104 (48.6)
	Thyroid disorder	20 (9.4)
	Metabolic syndrome	72 (33.6)
Past history	Previous myocardial infarction	21 (9.8)
	Previous heart failure	47 (22.0)
	Previous cerebrovascular accident	11 (5.1)
	Previous peripheral vascular disease	57 (26.6)
	Previous atrial fibrillation	21 (9.8)

Habits	Smoking	125 (58.4)
	Alcohol use	78 (36.4)
Systolic blood pressure, mmHg; mean (SD)		133.71 (20.07)
Diastolic blood pressure, mmHg; mean (SD)		82.69 (15.95)
Heart rate, beats/min; mean (SD)		95.14 (18.11)
TSH, mIU/L; mean (SD)		2.34 (1.61)
Troponin I; mean (SD)		3136.95
Troponin I	Positive	166 (77.6)
	Negative	48 (22.4)
BMI, kg/m²; mean (SD)		24.4 (2.5)
FBS, mg/dL; mean (SD)		127.04 (43.3)
PPBS, mg/dL; mean (SD)		220.18 (81.3)
HbA1c, %; mean (SD)		7.65 (2.45)
HbA1c	<5.0	18 (8.4)
	5.0-5.5	27 (12.6)
	5.6-6.0	29 (13.6)
	6.1-6.5	28 (13.1)
	6.5-7.0	10 (4.7)
	>7.0	102 (47.7)
Clinical presentation (Killip class)	Class I	117 (54.7)
	Class II	38 (17.8)
	Class III	40 (18.7)
	Class IV	19 (8.9)
Type of acute coronary syndrome	STEMI	174 (81.3)
	NSTEMI	40 (18.7)
Ejection fraction	<40%	48 (22.4)
	40-50%	142 (66.35)

	>50%	24 (11.2)
Complications due to ACS	Left ventricular failure	58 (27.1)
	Cardiogenic shock	19 (8.9)
	Cardiac arrest	17 (7.9)
	Ventricular tachycardia	11 (5.1)
	Complete heart block	9 (4.2)
	Atrial fibrillation	5 (2.3)
	Frequent VPCs	4 (1.9)
	Pericarditis/Dressler syndrome/tamponade	3 (1.4)
	Stroke	1 (0.5)
	Moderate mitral regurgitation	18 (8.4)
	Severe mitral regurgitation	4 (1.9)
GRACE ACS risk score; mean (SD)		117.6 (35.7)
GRACE ACS risk category	Low (<108)	100 (46.72)
	Intermediate (109-140)	71 (33.2)
	High (>140)	43 (20.1)
Coronary artery disease severity	Single-vessel disease	67 (31.3)
	Multivessel disease	147 (68.7)
Vessel involvement pattern	Single-vessel disease	61 (28.5)
	Double-vessel disease	69 (32.2)
	Triple-vessel disease	82 (38.3)
Pre-intervention TIMI grade	TIMI 0	78 (36.45)
	TIMI I	32 (14.95)
	TIMI II	58 (27.1)
	TIMI III	49 (22.9)
Artery involvement	Ramus	23 (10.7)
	LAD	174 (81.3)

	LCX	112 (52.3)
	RCA	145 (67.8)
	OM	13 (6.1)
	LM	20 (9.3)
	In-stent restenosis	2 (0.9)
	Total occlusion	91 (41.5)
Gensini score; mean (SD)		53.45 (31.5)
Gensini score	Low (<=20)	35 (16.4)
	Intermediate (21-40)	60 (28.03)
	High (>40)	119 (55.61)
Revascularization method	Pre-dilatation (with/without thrombolysis)	165 (77.1)
	Direct stenting (without pre-dilatation)	11 (5.1)
	Direct stenting plus thrombolysis	1 (0.5)
	POBA for in-stent restenosis	1 (0.5)
	Thrombolysis followed by stenting	16 (7.5)
	Medical management	20 (9.3)
SYNTAX score; mean (SD)		19.2 (11.1)
SYNTAX score	Low (<=22)	136 (63.6)
	Intermediate (23-32)	53 (24.8)
	High (>32)	24 (11.2)
Post-intervention complications	Acute stent thrombosis	4 (1.9)
	Contrast-induced nephropathy (medically managed)	44 (20.5)
	Contrast-induced nephropathy (requiring dialysis)	3 (1.4)
	Sepsis	47 (22.0)
	Hematoma at access site	9 (4.2)
	In-hospital mortality	8 (3.7)
	2-5 days	128 (59.8)

Length of hospital stay	6-10 days	51 (23.8)
	11-16 days	20 (9.3)
	>16 days	15 (7.1)
Short-term morbidity and mortality	CCF within 6 months	58 (27.1)
	Mortality within 6 months	1 (0.4)

Table 2: Association of HbA1c with Killip Class and Angiographic Severity in Acute Coronary Syndrome

		<5.0	**5.0-5.5**	**5.6-6.0**	**6.1-6.5**	**6.5-7.0**	**>7.0**	**P value**
Killip class	Class I	16 (88.9)	21 (77.8)	18 (62.1)	16 (57.1)	4 (40.0)	42 (41.2)	<0.001
	Class II	2 (11.1)	5 (18.5)	6 (20.7)	5 (17.9)	2 (20.0)	18 (17.6)	
	Class III	0	1 (3.7)	4 (13.8)	5 (17.9)	2 (20.0)	28 (27.5)	
	Class IV	0	0	1 (3.4)	2 (7.1)	2 (20.0)	14 (13.7)	
Multivessel disease	Yes	4 (22.2)	9 (33.3)	16 (55.2)	17 (60.7)	7 (70.0)	94 (92.2)	<0.001
	No	14 (77.8)	18 (66.7)	13 (44.8)	11 (39.3)	3 (30.0)	8 (7.8)	
Vessel disease pattern	Single-vessel	13 (72.2)	17 (63.0)	14 (48.3)	11 (39.3)	2 (20.0)	4 (3.9)	<0.001
	Double-vessel	4 (22.2)	7 (25.9)	8 (27.6)	9 (32.1)	3 (30.0)	38 (37.3)	
	Triple-vessel	1 (5.6)	3 (11.1)	7 (24.1)	8 (28.6)	5 (50.0)	60 (58.8)	
Total occlusion	Yes	1 (5.6)	3 (11.1)	5 (17.2)	7 (25.0)	3 (30.0)	72 (70.6)	<0.001
	No	17 (94.4)	24 (88.9)	24 (82.8)	21 (75.0)	7 (70.0)	30 (29.4)	

Table 3: Association of HbA1c with GRACE ACS Risk Score, Gensini Score, and SYNTAX Score

HbA1c category, %	GRACE ACS risk score, mean (SD)	Gensini score, mean (SD)	SYNTAX score, mean (SD)
<5.0	108.21 (36.6)	49.95 (32.29)	13.55 (6.92)
5.0-5.5	118.2 (49.2)	57.96 (35.58)	21.73 (15.77)
5.6-6.0	104.96 (20.2)	44.14 (27.70)	16.07 (10.32)
6.1-6.5	117.0 (21.68)	57.48 (34.72)	22.48 (12.79)
6.6-7.0	124.2 (36.4)	65.60 (33.36)	19.95 (9.68)
>7.0	122.3 (37.4)	53.53 (30.31)	19.42 (10.03)
Overall P value	0.205	0.491	0.061
Pearson correlation	r = 0.229 (p < 0.05)		
Spearman correlation	rho = 0.206 (p < 0.05)		

Table 4: Association of HbA1c with In-hospital Complications, Post-procedural Systemic Complications, Short-term Outcomes, and Length of Hospital Stay

		<5.0	5.0-5.5	5.6-6.0	6.1-6.5	6.5-7.0	>7.0	P value
In-hospital complications	Left ventricular failure	1 (5.6)	2 (7.4)	4 (13.8)	6 (21.4)	2 (20.0)	43 (42.2)	<0.001
	Cardiogenic shock	0	1 (3.7)	1 (3.4)	2 (7.1)	1 (10.0)	14 (13.7)	0.020
	Cardiac arrest	0	0	1 (3.4)	2 (7.1)	0	14 (13.7)	0.013
	Ventricular tachycardia	0	0	0	1 (3.6)	1 (10.0)	9 (8.8)	0.048
	Complete heart block	0	0	0	0	0	9 (8.8)	0.026
	Atrial fibrillation	0	0	0	0	0	5 (4.9)	0.021
	Frequent VPCs	0	0	0	0	0	4 (3.9)	0.028
	Pericarditis/Dressler syndrome/tamponade	0	0	0	0	0	3 (2.9)	0.030
	Stroke	0	0	0	0	0	1 (1.0)	0.010

	Moderate mitral regurgitation	1 (5.6)	1 (3.7)	3 (10.3)	1 (3.6)	1 (10.0)	12 (11.8)	0.66
	Severe mitral regurgitation	1 (5.6)	0	0	1 (3.6)	0	2 (2.0)	0.72
	Mild mitral regurgitation	0	2 (7.4)	1 (3.4)	2 (7.1)	0	8 (7.8)	0.71
Post-procedural systemic complications	Contrast-induced nephropathy	1 (5.6)	2 (7.4)	3 (10.3)	5 (17.9)	2 (20.0)	34 (33.3)	<0.001
	Sepsis	1 (5.6)	2 (7.4)	2 (6.9)	4 (14.3)	2 (20.0)	36 (35.3)	<0.001
	Hematoma at access site	0	0	0	1 (3.6)	0	12 (11.8)	0.048
	Acute stent thrombosis	0	0	0	0	0	4 (3.9)	0.043
Short-term outcomes	CCF within 6 months	1 (5.6)	3 (11.1)	5 (17.2)	7 (25.0)	3 (30.0)	39 (38.2)	<0.001
	Mortality within 6 months	0	0	0	0	2 (20.0)	6 (5.9)	0.038
Length of hospital stay	2-5 days	16 (88.9)	22 (81.5)	21 (72.4)	17 (60.7)	4 (40.0)	48 (47.1)	<0.001
	6-10 days	2 (11.1)	5 (18.5)	7 (24.1)	8 (28.6)	3 (30.0)	36 (35.3)	
	11-16 days	0	0	1 (3.4)	2 (7.1)	1 (10.0)	10 (9.8)	
	>16 days	0	0	0	1 (3.6)	2 (20.0)	8 (7.8)	