New onset diabetes in patients undergoing CABG: A single center registry analysis

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Abstract

Background: Diabetes mellitus is prevalent in 40–50% of patients undergoing coronary artery bypass grafting (CABG). While impaired glycosylated hemoglobin (HbA1C) is common in this group, the incidence of new-onset diabetes after CABG remains uncertain.

Objectives: To determine the incidence of new-onset diabetes following CABG and its impact on intensive care unit (ICU) and hospital stay in a Pakistani cohort.

Methods: We analyzed retrospective data from hospital registry for consecutive adult patients undergoing CABG at a single tertiary care center from January 2025 to August 2025. Patients with known diabetes were excluded. Descriptive and comparative analyses were performed.

Results: Of 1,559 patients (1,355 men, 204 women), 933 without pre-existing diabetes were included in the analysis. During hospitalization, 57 patients (6.1%) developed persistently elevated glucose levels at discharge, yielding an incidence of 61 ± 5 per 1,000. Compared with those who remained non-diabetic, patients with new-onset diabetes had similar age and body mass index but a higher prevalence of preoperative impaired HbA1C (44.6% vs. 13.7%). They also had longer ICU stay (102.0 ± 75 vs. 80.2 ± 29 hours) and hospital stay (11.7 ± 5.7 vs. 9.6 ± 2.4 days) ($p < 0.001$).

Conclusion: New-onset diabetes is unmasked in a notable proportion of non-diabetic patients following CABG, particularly among those with preoperative impaired HbA1C levels. Its occurrence is associated with prolonged ICU and hospital stay, highlighting the need for closer perioperative glucose surveillance in this high-risk population.

Keywords: CABG surgery, Diabetes, Impaired HbA1C, Intensive care unit

Background

Diabetes mellitus is a major cardiovascular risk factor, and its prevalence among patients undergoing coronary artery bypass grafting (CABG) surgery is estimated at 30–40% [1]. In Pakistan and India, this proportion is even higher, with reports suggesting that nearly 40–50% of CABG patients have diabetes [2]. The prevalence of impaired glucose tolerance (IGT) and impaired glycosylated hemoglobin (HbA1C) is also considerable in South Asian populations, with progression rates to type 2 diabetes mellitus (T2DM) ranging from 13% to 52% and an annual conversion rate of 1–6% [3]. Diabetes is associated with poorer clinical outcomes and prolonged hospitalization following cardiac surgery, compared with non-diabetic patients [4,5]. These individuals experience a higher incidence of wound infections, ischemic cardiovascular events,

neurological complications, renal dysfunction, and increased morbidity and mortality [6]. Moreover, hyperglycemia during the immediate and early postoperative period, even in non-diabetic patients, has been linked with adverse outcomes. However, the risk of new-onset diabetes in non-diabetic patients undergoing CABG remains unclear. To address this gap, we conducted a registry-based study to determine the incidence of new-onset diabetes in a large cohort of CABG patients.

Methods

We prospectively analyzed retrospective registry data of consecutive adult patients who underwent on-pump CABG surgery between January 2025 and August 2025 at a single tertiary care center. Clinical and biochemical parameters were recorded, along with details of perioperative drug therapy. Duration of intensive care unit (ICU) stay (hours) and total hospital stay (days) were documented. Postoperatively, all patients received intravenous insulin and epinephrine infusion for 24–48 hours in the ICU. Patients requiring ongoing therapy were transitioned to a basal-bolus insulin regimen upon transfer to the ward. Those who continued to require insulin at discharge were considered to have developed new-onset diabetes, defined by the need for insulin to maintain fasting plasma glucose <120 mg/dl and 2-hour postprandial glucose <180 mg/dl. Categorical variables were reported as percentages, and continuous variables as mean ± standard deviation (SD). Inter-group comparisons were performed using the t-test for continuous data and chi-square test for categorical data. Logistic regression was applied to identify associations between baseline risk factors, ICU stay, hospital stay, and the development of new-onset diabetes. Results are presented as odds ratios (OR) with 95% confidence intervals (CI).

Findings

Baseline characteristics of the study population (n = 1,559; men 1,355 [86.9%], women 204 [13.1%]) are presented in Table 1. The mean age was 60.6 ± 8.7 years, and mean body mass index (BMI) was 25.4 ± 4.1 kg/m². A total of 626 patients (40.1%) had known diabetes and were excluded, leaving 933 patients (59.9%) without pre-existing diabetes for analysis. (Table-1)

Among these, 57 patients (6.1%) developed persistently elevated glucose levels at the time of hospital discharge (mean 7.0 ± 4.0 days post-surgery), corresponding to an incidence rate of 61 per 1,000 (95% CI: 50–85). Compared with those who remained non-diabetic, patients with new-onset diabetes had higher BMI (25.9 ± 4.5 vs. 24.9 ± 4.0 kg/m²) and lower left ventricular ejection fraction (46.9 ± 9.2% vs. 50.0 ± 9.3%), though these differences did not reach statistical significance. No group differences were observed in the prevalence of smoking, hypertension, or preoperative pharmacological therapy.

Preoperative fasting plasma glucose was significantly higher among patients who developed new-onset diabetes (154.9 ± 67 mg/dl vs. 115.3 ± 27 mg/dl; $p < 0.001$). Furthermore, 44.6% of these patients had a preoperative glucose ≥140 mg/dl, compared with only 13.7% of those who remained non-diabetic ($p < 0.001$). Intraoperative and immediate postoperative management, including the use of adrenergic vasopressors and anti-arrhythmic agents, did not differ between groups.

Patients who developed post-CABG diabetes had significantly longer ICU stay (102.0 ± 75.2 vs. 80.2 ± 28.5 hours) and total hospital stay (11.7 ± 5.7 vs. 9.6 ± 2.4 days; $p < 0.001$) Table-2.

Logistic regression analysis identified preoperative fasting plasma glucose ≥140 mg/dl as the only independent predictor of new-onset diabetes, with an unadjusted odds ratio (OR) of 3.20 (95% CI: 1.93–5.32) and an adjusted OR of 5.40 (95% CI: 3.04–9.63; $p < 0.001$) after controlling for age, sex, BMI, and baseline medication use.

Table 1.

Comparison of anthropometric, biochemical, pharmacological and outcome variables in patients without and with post-CABG diabetes.

Variables	**No diabetes (n = 877)**	**Post-CABG diabetes (n = 57)**	***p* value**
Age (years)	60.3 ± 9.1	61.0 ± 8.0	0.585
Body mass index (kg/m^2)	24.9 ± 4.0	25.9 ± 4.5	0.070
Current smoker	195 (22.2)	09 (16.1)	0.462
Hypertension	434 (49.4)	37 (64.9)	0.015
Preoperative pharmacological treatments:			
Anti-platelets	163 (18.5)	14 (24.5)	0.234
Renin-angiotensin system blockers	134 (15.3)	15 (26.3)	0.013
Beta blockers	194 (22.1)	16 (28.1)	0.268
Diuretics	36 (4.1)	03 (5.2)	0.648
Calcium channel blockers	22 (2.5)	02 (3.5)	0.607
Statins	161 (18.3)	13 (22.8)	0.366
Plasma glucose (random, mean, mg/dl)	115.3 ± 27.2	154.9 + 67.0	<0.001
Preoperative random blood glucose ≥140 mg/dl	120 (13.7)	25 (44.6)	<0.001
Left ventricular ejection fraction (mean%)	50.0 ± 9.3	46.9 ± 9.2	0.015
Triple vessel disease	643 (73.3)	44 (77.1)	<0.001
Intra and postoperative treatments:			
Adrenaline, noradrenaline and other adrenergic drugs	877 (100.0)	54 (100.0)	1.00
Immediate post-operative hyperglycemia (≥200 mg/dl)	455 (51.8)	37 (64.9)	0.009

Table-2: Invasive ventilation and duration of hospital stay of both groups

Variables	No diabetes (n = 877)	Post-CABG diabetes (n = 57)	*p* value
Duration of invasive ventilation (hours)	16.0 ± 7.6	19.9 ± 14.9	0.001
Intensive care unit stay (hours)	80.2 ± 28.5	102.0 ± 75.2	<0.001
Overall hospital stay (days)	9.6 ± 2.4	11.7 ± 5.7	<0.001

Numbers ± are 1 SD; Numbers in parentheses are percent.

Discussion

Our study demonstrates that although CABG restores coronary perfusion, it can unmask a new cardiovascular risk factor—diabetes mellitus—in a small but clinically important subset of patients. The observed incidence of new-onset diabetes (61 per 1,000) is noteworthy. Several mechanisms may explain this phenomenon. Norepinephrine and epinephrine, used routinely during CABG, inhibit insulin secretion and stimulate glucagon release [7]. Stress hyperglycemia is influenced by pre-existing glucose tolerance, severity of illness, and counter-regulatory hormone activity [8,9]. Clinical situations such as myocardial infarction, infection, and trauma—all associated with catecholamine excess—share similar metabolic alterations, including increased insulin resistance, reduced insulin secretion, and higher glucagon concentrations. Insulin resistance is known to worsen during cardiopulmonary bypass [10]; however, data remain limited for normothermic surgeries such as in our cohort.

In our study, patients who developed new-onset diabetes had longer ICU and hospital stays. This may either reflect the consequence of hyperglycemia itself or indicate more severe underlying illness necessitating prolonged critical care. Stress-induced insulin resistance likely results from catecholamine and cortisol secretion, systemic inflammatory response, and effects of systemic heparinization [11]. All surgeries in our cohort were performed under moderate hypothermia, various previous studies have also reported higher glycemic disturbances during hypothermic CABG, suggesting a multifactorial etiology [11].

Potential contributors to new-onset diabetes in our cohort include pre-existing dysglycemia, inotropic support, surgical stress, and heparinization. Longer ventilatory support and extended ICU stay—conditions often requiring more inotrope use—should alert clinicians to the risk of emergent diabetes. Importantly, nearly 45% of our patients with new-onset diabetes had preoperative glucose values ≥140 mg/dl, emphasizing the role of impaired glucose tolerance as a key predisposing factor. While the incidence rate of 6.1% in our study is comparable to the annual conversion rate of IGT to diabetes (5–6%), the much shorter timeframe suggests that the stress of CABG may accelerate this progression [3]. Long-term follow-up, which was not available in this study, is necessary to confirm whether diabetes persists or regresses. Prior studies, such as those among South African Indians, have shown conversion rates of >50% within four years, while other

cohorts report that <50% of IGT cases progress within a decade, with many reverting to normal glucose tolerance.

Our findings highlight important clinical implications. Failure to detect prediabetes may result in uncontrolled hyperglycemia, underestimation of risk, and missed opportunities for early intervention. While strong evidence supports the association between hyperglycemia and adverse outcomes in the first 48–72 hours post-CABG, data beyond the first week remain scarce. To our knowledge, this is the first study to provide evidence of new-onset diabetes emerging during hospitalization following CABG [13]. We therefore recommend systematic screening of all patients with preoperative IGT and postoperative hyperglycemia for new-onset diabetes, and provision of appropriate glycemic management before discharge. This includes diabetes education, self-monitoring of blood glucose, insulin titration, and hypoglycemia management—similar to established diabetic patients. Preoperative HbA1c testing in non-diabetic patients, particularly those at high risk, may help identify subclinical dysglycemia and guide perioperative glycemic strategies.

Our study has several strengths: a large cohort, uniform surgical technique (moderate hypothermia on-pump CABG), and standardized perioperative management, which minimized confounding. However, limitations include its single-center design and short in-hospital follow-up.

In conclusion, patients undergoing CABG appear to be at increased risk of developing new-onset diabetes, particularly those with preoperative dysglycemia. Routine HbA1c measurement and structured postoperative glycemic management should be incorporated into the preoperative assessment and discharge planning for this population.

Conflict of interest

None

References

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