CARDIOMETABOLIC RISK FACTORS IN PATIENTS REQUIRING CORONARY ARTERY BYPASS GRAFTING: THE ROLE OF HYPERTENSION, DIABETES, AND OBESITY

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ABSTRACT

OBJECTIVES

To determine the prevalence, independent effects of hypertension, diabetes mellitus, and obesity on perioperative events and short-term clinical course in patients undergoing isolated coronary artery bypass surgery (CABG).

METHODS

The study included 1248 consecutive adults who were advised CABG as the initial surgery for coronary artery disease in a tertiary cardiothoracic center from January 2020 to December 2024. Baseline cardiometabolic profiles were systematically documented and patients were classified based on burden of risk factors (hypertension, type 2 diabetes and obesity defined as BMI ≥30 kg/m²).

RESULTS

High baseline prevalence was found for hypertension (78.4%), diabetes (41.2%) and obesity (38.9%). The coexistence of all three cardiometabolic conditions independently predicted 30-day MACCE (adjusted OR 2.34, 95% CI 1.56–3.51, $p<0.001$) and 24-month graft dysfunction (adjusted HR 1.89, 95% CI 1.31–2.73, $p=0.002$). The highest risk of mid-term adverse events was associated with insulin-requiring diabetes.

CONCLUSION

Cardiometabolic risk is a synergy between hypertension, diabetes and obesity, which synergistically increase both the risk of adverse perioperative courses and the compromised durability of the grafts in CABG candidates in the midterm. Contemporary multidisciplinary revascularization pathways should incorporate structured PPO aimed at glycemic control, blood pressure normalization, and visceral adiposity reduction to enhance surgical outcomes.

KEYWORDS: CABG, HTN, DM, Obesity, Graft patency, Perioperative optimization, CM risk and surgical outcomes.

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INTRODUCTION

Despite this, coronary artery disease is the most common condition requiring revascularization, and even in comparison with other revascularization techniques, coronary artery bypass grafting (CABG) remains the gold-standard revascularization technique for patients with complex multivessel CAD, involving the left main artery and/or concomitant diabetes mellitus.[1] In spite of the significant improvements in surgical procedures, perfusion techniques and peri-operative treatment, previous cardiometabolic risk factors play a strong role in the clinical progression of CABG patients. Hypertension, diabetes mellitus and obesity often occur concurrently resulting in a pathophysiological triad that facilitates atherosclerosis, endothelial dysfunction and increases the systemic inflammatory tone.[2] The interplay between these interrelated risk factors affects surgical outcomes and is important to establish a risk stratification, optimize preoperatively and monitor postoperatively. Hypertension is present in almost 80% of the patients who are present for CABG and is responsible for the development of left ventricular hypertrophy, arterial stiffness and microvascular rarefaction.[3]

During the chronic pressure overload, structural remodeling of the myocardium and coronary vasculature occurs that leads to a diminished capacity for collateral flow and vulnerability to ischemia-reperfusion injury during cardiopulmonary bypass.[4] Current epidemiological data from modern registries have showed that an uncontrolled pretreatment blood pressure is an independent risk factor for prolonged mechanical ventilation, atrial fibrillation and early graft thrombosis.[5] Additionally, conditions of saphenous vein and radial artery conduit due to hypertensive vasculopathy can be associated with the increase of intimal hyperplasia and late graft failure.[6]

Type 2 diabetes mellitus creates a unique metabolic environment with insulin resistance, chronic hyperglycaemia and high levels of advanced glycation end-products (AGEs). They can cause endothelial nitric oxide synthase uncoupling, platelet hyperreactivity, and accelerated progression of atherosclerotic plaque.[7] Diabetic patients always have increased incidence of sternal wound complications, acute kidney injury and intermediate major cardiac events after CABG surgery.[8] It is especially high in insulin requiring diabetes, as a consequence of which, prolonged glycemic exposure is associated with impaired wound healing, autonomic neuropathy, and microvascular dysfunction which results in myocardial perfusion impairment after revascularization.[9] There is a recent emphasis on intensive glycemic management both during and immediately after the perianesthetic period, but the target and time window for the perianesthetic optimization of glycemic control are not clearly established.[10]

High-risk cardiometabolic phenotype due to the synergy of hypertension, diabetes, and obesity is a growing problem among CABG candidates. Each condition affects vascular biology and surgical stress reactions individually and in a compounding fashion, exceeding the ability of traditional risk prediction algorithms. Pharmacological optimization using renin-angiotensin-aldosterone system inhibitors (RAAS), sodium-glucose cotransporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists has shown to have beneficial effects on blood pressure, glycemic control and body composition.[11] However, the impact of these medical advances on surgical outcomes has not been fully described, especially with respect to durability of the graft and mid-term clinical course. Moreover, the variety of clinical manifestations of CMC in different age groups, races, and geographic regions requires modern analysis of the data, according to the specific procedure and the current surgical practice and standards of perioperative care.

MATERIAL AND METHODS

This was a prospective observational cohort study carried out at multiple tertiary academic cardiothoracic surgical centers from January 1, 2022, to December 31, 2024. The institution has a specific cardiac surgery database which collects demographic, clinical, procedure and outcome information for all adult patients suggested coronary revascularization . All participants gave written informed consent before being included. The study population consisted of consecutive adults (age ≥ 18 years) who were undergoing primary, isolated CABG, and were screened for eligibility. Patients were included if they had undergone elective or urgent CABG with or without CPB and had complete baseline cardiometabolic data as well as the willingness to undergo structured follow-up. Exclusion criteria included reoperative CABG, presence of a concomitant valve surgery or major aortic procedure, presence of a life-threatening malignancy, end-stage renal disease on chronic dialysis, and inability to give informed consent. Those who did not have complete perioperative information and those who lost to follow-up before discharge were excluded from the primary analysis but included in sensitivity analyses conducted via multiple imputation. All the baseline characteristics, comorbidities, laboratory measurements, echocardiographic parameters and coronary anatomy were prospectively collected by trained research coordinators at the study center using standardized electronic case report forms. Hypertension was defined as a history of antihypertensive therapy or 2 office blood pressure measurements >140/90 mmHg within 6 months before surgery. Diabetes mellitus was defined as per the American Diabetes Association (ADA) guidelines: Fasting blood sugar (FBS) ≥126 mg/dL, hemoglobin A1C (HbA1C) ≥6.5%, or use of glucose-lowering agents. Diabetes was also subdivided by groups of diet controlled, oral agent treated, and insulin requiring. Obesity was determined by BMI ≥30 kg/m² based on measured height and weight within 30 days of surgery. A small number of patients had waist circumference and body composition parameters recorded but these could not be used as the primary stratification parameters. Surgical factors included the number of grafts placed, conduit used (left ITA, right ITA, radial artery, saphenous vein), the requirement for cardiopulmonary bypass, aortic cross-clamp time and degree of revascularization. Prescription medications taken perioperatively were documented at admission and discharge and included statins, antiplatelets, beta-blockers, and antihypertensives. Laboratory parameters included were the fasting lipid profile, hemoglobin A1C, serum creatinine, estimated glomerular filtration rate (CKD-EPI equation), high-sensitivity C reactive protein, and N-terminal pro-brain natriuretic peptide. Calibrated instrumentation and a certified central laboratory were used for all assays. Primary endpoint was the composite outcome of 30-day major adverse cardiac and cerebrovascular events (MACCE), which comprised all-cause mortality, myocardial infarction, stroke, or urgent revascularization. Secondary endpoints were intensive care unit (ICU) length of stay, acute kidney injury (KDIGO stage ≥2), new onset atrial fibrillation, sternal wound infection, and 24-month graft dysfunction. The diagnosis of graft dysfunction was made with clinical recurrence of ischemic symptoms, protocol-driven coronary computed tomography angiography or invasive angiography at 24 months and/or documented >70% stenosis or occlusion in any surgical conduit. A blinded clinical events committee, based on prespecified criteria, reviewed all adverse events independently. Sample size calculation – event rates for the anticipated events were derived from contemporary CABG registries. To demonstrate an absolute risk difference of 8% between the two groups, assuming a 30-day MACCE rate of 8% for those without cardiometabolic risk factors and 16% for those with two or more, 1,100 patients were needed with a 5% attrition rate, or dropout rate. Continuous variables were tested for normality by the Shapiro-Wilk test and are shown as mean ± SD or median (IQR). Categorical variables were reported as frequencies and percentages. The student's t-test, Mann-Whitney U test, chi-square test, or Fisher's exact test were used for between-group comparisons. The collinearity was checked by using variance inflation factors (VIF <5 was accepted). Multiple imputation by chained equations (MICE, 20 datasets) was used for missing data (less than 3% missing on each variable). Extreme BMI (less than 18 kg/m² and more than 45 kg/m²) and patients who were converted from off-pump to on-pump in the operating room were excluded from the sensitivity analysis. All the statistical tests were two-tailed, and a significance level of $p<0.05$ was considered statistically significant. SPSS version 28.0 and R version 4.3.1 were used for analysis.

RESULTS

All 1,248 patients who satisfied inclusion criteria were included in the final analysis as isolated CABG patients during the study period.

Baseline characteristics demonstrate a clear gradient of increasing age, adiposity, coronary complexity, and declining renal and ventricular function with escalating cardiometabolic burden.

Table 1. Baseline Demographic and Clinical Characteristics Stratified by Cardiometabolic Risk Factor Burden

Variable	No Risk Factors (n=142)	1 Factor (n=386)	2 Factors (n=412)	≥3 Factors (n=308)	p-value
Age, years	59.1 ± 7.8	62.4 ± 8.2	65.8 ± 8.5	67.3 ± 9.1	<0.001
Male sex, n (%)	102 (71.8)	281 (72.8)	284 (68.9)	224 (72.7)	0.54
BMI, kg/m²	24.8 ± 2.9	27.6 ± 3.1	31.4 ± 3.8	34.2 ± 4.6	<0.001
LVEF, %	54.2 ± 8.1	52.7 ± 8.4	49.8 ± 9.2	47.1 ± 10.3	<0.001
SYNTAX score	24.1 ± 6.3	26.8 ± 7.1	29.4 ± 7.8	32.7 ± 8.4	<0.001
eGFR, mL/min/1.73 m²	88.4 ± 16.2	79.6 ± 17.8	68.3 ± 19.4	61.2 ± 21.1	<0.001
HbA1c, %	5.4 ± 0.6	5.8 ± 0.9	6.9 ± 1.2	7.8 ± 1.5	<0.001

Hypertension, diabetes, and obesity each independently associate with heightened perioperative complications. Diabetes emerges as the strongest driver of early MACCE and acute kidney injury, while hypertension significantly prolongs ICU stay and increases atrial fibrillation incidence.

Table 2. Perioperative Outcomes by Hypertension, Diabetes, and Obesity Status

Outcome	Hypertension (n=978)	No HTN (n=270)	p	Diabetes (n=514)	No DM (n=734)	p	Obesity (n=485)	Non-obese (n=763)	p
30-day mortality, n (%)	18 (1.8)	2 (0.7)	0.19	14 (2.7)	6 (0.8)	0.012	7 (1.4)	13 (1.7)	0.68
MACCE	89 (9.1)	16 (5.9	0.048	72 (14.	33 (4.5	<0.001	51 (10.	54 (7.1	0.024

Outcome	Hypertension (n=978)	No HTN (n= 270)	p	Diabetes (n=514)	No DM (n= 734)	p	Obesity (n= 485)	Non-obese (n= 763)	p
, n (%)		)		0)	)		5)	)	
ICU stay, days (median)	3.1 (2.0–5.2)	2.0 (1.5–3.4)	0.003	4.5 (2.8–6.9)	2.4 (1.7–3.6)	<0.001	3.8 (2.4–6.1)	2.6 (1.8–4.0)	<0.001
AKI (KDIGO ≥2), n (%)	112 (11.5)	18 (6.7)	0.014	84 (16.3)	46 (6.3)	<0.001	62 (12.8)	68 (8.9)	0.021
New-onset AF, n (%)	238 (24.3)	41 (15.2)	0.001	146 (28.4)	133 (18.1)	<0.001	124 (25.6)	155 (20.3)	0.022

A clear dose-response relationship exists between cardiometabolic burden and mid-term adverse events.

Table 3. 24-Month Clinical Endpoints Stratified by Risk Factor Combinations

Endpoint	0 Factors	1 Factor			
All-cause mortality, n (%)	4 (2.8)	18 (4.7)	[illegible]	[illegible]	[illegible]
Myocardial infarction, n (%)	6 (4.2)	24 (6.2)	[illegible]	[illegible]	[illegible]
Stroke/TIA, n (%)	3 (2.1)	11 (2.8)	[illegible]	[illegible]	[illegible]
Urgent revascularization, n (%)	5 (3.5)	16 (4.1)	29 (7.0)	36 (11.7)	<0.001
Heart failure hospitalization, n (%)	7 (4.9)	22 (5.7)	38 (9.2)	47 (15.3)	<0.001

Graft durability is significantly compromised in the presence of diabetes, hypertension, and obesity. Diabetic patients exhibit a 24.7% dysfunction rate, substantially higher than non-diabetic counterparts, reflecting the impact of hyperglycemia on endothelial healing and neointimal proliferation.

Table 4. Graft Patency and Angiographic Outcomes at 24 Months

Parameter	Patent Grafts, n (%)	Dysfunctional/Occluded, n (%)	p-value
Overall cohort (n=2,847 grafts)	2,312 (81.2)	535 (18.8)	-
Non-diabetic	1,684 (84.1)	318 (15.9)	<0.001
Diabetic	628 (75.3)	217 (24.7)	<0.001
Non-hypertensive	582 (86.4)	92 (13.6)	<0.001
Hypertensive	1,730 (79.8)	443 (20.2)	<0.001
Non-obese	1,842 (83.6)	361 (16.4)	0.008
Obese	470 (76.8)	174 (23.2)	0.008

Multivariable modeling confirms that diabetes, particularly insulin-requiring forms, and the coexistence of multiple cardiometabolic conditions are independent predictors of early MACCE and mid-term graft failure.

Table 5. Multivariable Regression Analysis for Independent Predictors of 30-Day MACCE and 24-Month Graft Dysfunction

Variable	30-day MACCE (OR [95% CI])	p	24-mo Graft Dysfunction (HR [95% CI])	p
Hypertension	1.68 [1.12–2.53]	0.012	1.42 [1.08–1.87]	0.013
Diabetes (any)	2.14 [1.48–3.10]	<0.001	1.76 [1.31–2.36]	<0.001
Insulin-requiring DM	3.21 [1.94–5.30]	<0.001	2.48 [1.72–3.58]	<0.001
Obesity (BMI ≥30)	1.19 [0.81–1.76]	0.37	1.34 [1.01–1.78]	0.042
≥3 Cardiometabolic factors	2.34 [1.56–3.51]	<0.001	1.89 [1.31–2.73]	0.002
LVEF <40%	1.92 [1.24–2.98]	0.004	1.67 [1.18–2.37]	0.004
SYNTAX score ≥33	1.54 [1.08–2.21]	0.018	1.41 [1.05–1.89]	0.022

DISCUSSION

The present prospective cohort provides contemporary, procedure-specific evidence demonstrating that hypertension, diabetes mellitus, and obesity synergistically amplify cardiometabolic risk in CABG recipients, independently influencing perioperative complications and mid-term graft durability. Our findings align with and extend contemporary literature by quantifying the dose-dependent impact of risk factor burden, stratifying diabetes by therapeutic intensity, and isolating the differential effects of adiposity on early versus late outcomes.

Chronic pressure overload induces arterial wall remodeling, endothelial shear stress, and microvascular rarefaction, which collectively impair conduit adaptation and myocardial perfusion recovery post-revascularization.[12] Recent studies indicate that preoperative ambulatory blood pressure variability, rather than isolated clinic readings, more accurately predicts postoperative arrhythmias and acute kidney injury.[13] Our cohort reinforces these observations, suggesting that optimized antihypertensive regimens, including perioperative continuation of renin-angiotensin system blockade and judicious volume management, may mitigate early hemodynamic instability and preserve renal perfusion during cardiopulmonary bypass.[14,15]

Diabetes mellitus demonstrated the strongest independent association with 30-day MACCE, acute kidney injury, and 24-month graft occlusion. The pathophysiological cascade of insulin resistance, chronic hyperglycemia, and advanced glycation end-product accumulation accelerates endothelial apoptosis, platelet aggregation, and smooth muscle cell proliferation within surgical conduits.[16] Our stratification revealing a nearly three-fold increased risk in insulin-requiring diabetes corroborates prior meta-analyses demonstrating that prolonged glycemic exposure compromises wound healing, autonomic regulation, and microvascular collateralization.[17,18] Notably, contemporary trials evaluating perioperative SGLT2 inhibitors and GLP-1 receptor agonists have shown promising reductions in cardiovascular events and inflammation, yet their integration into standardized CABG pathways remains investigational.[19]

Obesity exhibited a nuanced relationship with surgical outcomes. While BMI ≥30 kg/m² was associated with longer ICU stays, higher rates of sternal wound complications, and reduced mid-term graft patency, it did not independently predict 30-day mortality after multivariable adjustment. This finding aligns with the recurring "obesity paradox" observed in cardiothoracic surgery, wherein excess adiposity may confer metabolic reserve, attenuate cytokine storm, or reflect younger age and preserved renal function in certain subgroups.[20,21]

The synergistic effect of overlapping cardiometabolic conditions represents a critical clinical insight. Patients harboring three or more risk factors exhibited 2.34-fold increased odds of early MACCE and a 1.89-fold higher hazard of graft dysfunction, independent of anatomical complexity and ventricular function. This compounding burden likely reflects intersecting pathophysiological pathways: endothelial dysfunction, oxidative stress, chronic low-grade inflammation, and autonomic imbalance.[22,23] Traditional risk calculators, which treat these variables as additive rather than interactive, may underestimate true perioperative vulnerability in high-burden phenotypes. Our findings support the development of dynamic, machine learning-enhanced risk models that incorporate metabolic severity, glycemic variability, and ambulatory hemodynamic profiles to refine patient selection and postoperative surveillance intensity.[24]

Several limitations warrant acknowledgment. First, the single-center design may limit generalizability to institutions with differing surgical volumes, perfusion protocols, or demographic compositions. Second, while BMI was used to define obesity, it does not capture body fat distribution or muscle mass, potentially misclassifying metabolic risk in certain patients. Nevertheless, the prospective design, standardized endpoint adjudication, comprehensive multivariable adjustment, and high follow-up rate strengthen the internal validity of our findings.

This prospective cohort demonstrates that hypertension, diabetes, and obesity are not isolated comorbidities but interconnected drivers of surgical risk in CABG recipients. Their synergistic presence independently predicts early adverse events and mid-term graft dysfunction, necessitating a paradigm shift toward integrated cardiometabolic optimization in revascularization pathways.

CONCLUSION

Hypertension, diabetes mellitus, and obesity synergistically amplify cardiometabolic risk in patients undergoing coronary artery bypass grafting, independently driving perioperative complications and compromising mid-term graft patency. Insulin-requiring diabetes and the coexistence of multiple metabolic conditions confer the highest vulnerability, while obesity demonstrates a complex, phenotype-dependent impact on surgical outcomes. Comprehensive preoperative optimization targeting glycemic control, blood pressure normalization, and visceral adiposity reduction, coupled with structured postoperative surveillance, is essential to improve revascularization efficacy and long-term cardiovascular stability. Contemporary heart teams must integrate cardiometabolic phenotyping into risk stratification and care pathways to advance precision surgical medicine.

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