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Postoperative Delta Troponin I as a Predictor of 30-Day Major Adverse Cardiovascular Events after Coronary Artery Bypass Grafting: A Retrospective Cohort Study from an Indonesian Tertiary Referral Centre

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ABSTRACT

Introduction: Myocardial injury is almost universal after coronary artery bypass grafting (CABG), yet the prognostic significance of the postoperative rise in cardiac troponin I remains uncertain, particularly in Southeast Asian populations. To evaluate the association and prognostic performance of postoperative delta (Δ) troponin I for 30-day major adverse cardiovascular events (MACE) after elective CABG.

Methods: This retrospective cohort study enrolled 60 consecutive patients who underwent elective CABG at Haji Adam Malik General Hospital, Medan, Indonesia, between January 2025 and May 2026. Δ troponin I was calculated as postoperative (≤72 h) minus preoperative (≤24 h) troponin I. MACE comprised malignant arrhythmia, acute heart failure, stroke, type 5 myocardial infarction, and cardiovascular death within 30 days. Analyses included Mann–Whitney tests, multivariable logistic regression, and receiver operating characteristic (ROC) curves.

Results: The mean age was 59.7 ± 7.5 years, and 71.7% were men. MACE occurred in 36 patients (60.0%). Median Δ troponin I was higher in patients with MACE than in those without (7.45 vs. 0.81 ng/mL; p = 0.002). Δ troponin I yielded an area under the curve of 0.742 (95% CI 0.610–0.875); a cut-off of 2.225 ng/mL provided 77.8% sensitivity, 79.2% specificity, and positive and negative predictive values of 84.8% and 70.4%, respectively. Postoperative troponin I independently predicted MACE (odds ratio 1.231 per ng/mL; 95% CI 1.087–1.395; p = 0.001).

Conclusion: Postoperative Δ troponin I showed moderate discrimination for 30-day MACE after elective CABG and may complement early postoperative risk stratification pending external validation in larger prospective cohorts.

Keywords

Cardiac Troponin I, Coronary Artery Bypass Grafting, Major Adverse Cardiovascular Events, Perioperative Myocardial Injury, Prognosis, Risk Stratification

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INTRODUCTION

Coronary artery disease (CAD) remains the leading cause of cardiovascular death and disability-adjusted life years worldwide, and its burden is shifting disproportionately towards low- and middle-income countries [1]. Across the Association of Southeast Asian Nations, the Global Burden of Disease Study 2021 recorded approximately 36.8 million prevalent cases of cardiovascular disease and 1.66 million cardiovascular deaths, with ischemic heart disease as the principal contributor [2]. In Indonesia, the clustering of smoking, hypertension, dyslipidemia, and diabetes sustains a rising incidence of CAD that frequently presents with

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diffuse, multivessel involvement [3]. The 2023 Indonesian Health Survey reported a national CAD prevalence of 0.85% [4]. CABG volumes at regional referral centers in North Sumatra have grown steadily in parallel [5].

Coronary artery bypass grafting (CABG) is the preferred revascularization strategy for many patients with complex multivessel or left main disease, especially in the presence of diabetes or left ventricular dysfunction, because it achieves more complete revascularization and confers durable symptomatic and prognostic benefits [6]. A meta-analysis of randomized trials including more than 7,000 patients with multivessel CAD confirmed lower long-term mortality, myocardial infarction, and repeat revascularization after CABG than after percutaneous coronary intervention [7]. Nevertheless, early complications remain frequent: malignant arrhythmia, acute heart failure, perioperative myocardial infarction, stroke, and cardiovascular death are major determinants of short-term morbidity, resource use, and long-term survival [8]. Cardiac troponins are the biomarkers of choice for detecting myocardial injury. After CABG, however, troponin release is virtually inevitable because of direct surgical trauma, cardioplegic arrest, ischemia–reperfusion injury, and manipulation of the heart. The Fourth Universal Definition of Myocardial Infarction therefore requires a postoperative elevation exceeding 10 times the 99th percentile upper reference limit, together with supportive electrocardiographic, angiographic, or imaging evidence, to diagnose type 5 myocardial infarction [9]. Release patterns reflect a multifactorial process that includes necrosis, apoptosis, increased membrane permeability, and proteolytic degradation [10]. Beyond diagnosis, the magnitude of postoperative release carries prognostic information: in the multinational VISION Cardiac Surgery study, high-sensitivity troponin I on the first postoperative day was independently associated with 30-day mortality at thresholds considerably higher than those recommended for diagnosing perioperative infarction [11]. A recent systematic review confirmed that postoperative troponin I and T are consistently associated with adverse events, but heterogeneity in assays, sampling times, thresholds, and outcome definitions precluded firm recommendations [12].

Evidence on the incremental value of baseline troponin levels is conflicting. Preoperative high-sensitivity troponin T was associated, in a dose-dependent manner, with perioperative complications and long-term mortality in elective CABG [13]. However, no independent association between preoperative troponin and outcome was observed in patients undergoing CABG after myocardial infarction [14]. In Indonesia, a single-center cohort at the National Cardiovascular Centre Harapan Kita reported that postoperative troponin I ≥5 ng/mL independently predicted 30-day MACE, with an adjusted odds ratio of 6.88 [15]. However, a single absolute postoperative value conflates pre-existing myocardial injury with procedure-related injury. Expressing the postoperative change relative to baseline (Δ troponin I) may better isolate the surgical injury, and early longitudinal changes after off-pump CABG have been linked to adverse events [16]. Standardized perioperative endpoint definitions now facilitate such comparisons across centers [17]. To our knowledge, the prognostic value of Δ troponin I after CABG has not been examined in an Indonesian population, in which assay platforms, sampling protocols, and patient profiles differ from those of Western cohorts. We therefore aimed to evaluate the association between postoperative Δ troponin I and 30-day MACE in patients undergoing elective CABG at a tertiary referral hospital in North Sumatra and to determine its discriminative performance and optimal prognostic threshold.

METHODS

This analytical observational study used a retrospective cohort design and was conducted at the Integrated Cardiac Centre of Haji Adam Malik General Hospital, Medan, a national referral and teaching hospital affiliated with the Faculty of Medicine, Universitas Sumatera Utara. Medical records of patients who underwent CABG between January 2025 and May 2026 were reviewed. The study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement.

Consecutive patients were eligible if they (i) had CAD treated with elective CABG, (ii) had troponin I measured both before and after surgery, and (iii) had complete clinical data to ascertain 30-day MACE. Patients were excluded if they underwent emergency CABG, had previous CABG, had advanced chronic kidney disease (estimated glomerular filtration rate [eGFR] <30 mL/min/1.73 m2), required dialysis, had infection,

sepsis, or another inflammatory condition that could raise troponin independently of myocardial ischemia, or had incomplete records. The sample size was calculated using the formula for comparing two independent means, with a two-sided α of 0.05 and a power of 80%. Assuming a minimum clinically relevant difference in troponin I of 0.94 ng/mL and a pooled standard deviation of 1.20 ng/mL derived from a previous cohort, at least 26 patients per group (52 in total) were required. Sixty patients were enrolled in the study.

Baseline data were extracted using a standardized form and comprised demographic characteristics, cardiovascular risk factors and comorbidities, coronary anatomy, New York Heart Association (NYHA) functional class, left ventricular ejection fraction (LVEF), EuroSCORE II, Society of Thoracic Surgeons (STS) risk score, and SYNTAX score. Operative variables included surgical technique (on-pump or off-pump), cardiopulmonary bypass (CPB) time, aortic cross-clamp (AOX) time, use of an intra-aortic balloon pump, and intensive care unit (ICU) and hospital length of stay. Preoperative laboratory parameters comprised hemoglobin, hematocrit, leukocyte and platelet counts, urea, creatinine, eGFR, liver enzymes, albumin, glycated hemoglobin (HbA1c), and serum electrolytes. Preoperative troponin I was measured within 24 hours before surgery and postoperative troponin I within 72 hours after surgery as part of routine clinical care in the hospital central laboratory [assay name, manufacturer; limit of detection; 99th percentile upper reference limit]. The upper reportable limit of the assay was 15.0 ng/mL. Δ troponin I was defined as postoperative minus preoperative troponin I (ng/mL). The primary outcome was MACE within 30 days after CABG, a composite of malignant arrhythmia, acute heart failure, stroke, type 5 myocardial infarction, and cardiovascular death, defined in line with standardized perioperative endpoints. Malignant arrhythmia was defined as ventricular tachycardia, ventricular fibrillation, or any other arrhythmia requiring resuscitation or immediate intervention. Acute heart failure was defined as new or worsening signs and symptoms requiring intravenous therapy, ventilatory support, or intensive care. Stroke was defined as a focal or global neurological deficit lasting ≥24 hours, confirmed clinically and/or radiologically. Type 5 myocardial infarction was defined according to the Fourth Universal Definition of Myocardial Infarction. Cardiovascular death included death due to myocardial infarction, heart failure, fatal arrhythmia, stroke, or other cardiovascular causes. Outcomes were ascertained from inpatient and outpatient records and, when necessary, confirmed by telephone contact. Patients could experience more than one component; for the composite endpoint, each patient was counted once.

Data were analyzed using Microsoft Excel and IBM SPSS Statistics [version]. Normality was assessed using the Kolmogorov–Smirnov test. Continuous variables are presented as mean ± standard deviation (SD) or median (minimum–maximum), and categorical variables as frequencies and percentages. Between-group comparisons used the independent-samples t test or the Mann–Whitney U test for continuous variables, and the $\chi 2$ test or Fisher's exact test for categorical variables, as appropriate. Multivariable logistic regression with backward stepwise elimination was performed to identify independent troponin-based predictors of MACE; because Δ troponin I is an arithmetic derivative of the preoperative and postoperative values, the two-component measurements were entered into the model rather than Δ troponin I, to avoid perfect collinearity. Results are reported as odds ratios (OR) with 95% confidence intervals (CI). Discriminative performance was assessed using receiver operating characteristic (ROC) curves and the area under the curve (AUC); optimal cut-off values were identified by the maximum Youden index, and sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated. A two-sided p value <0.05 was considered statistically significant. This study received ethical approval from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sumatera Utara, and permission to conduct the research was obtained from Haji Adam Malik General Hospital. All data were obtained from patient medical records and kept confidential using identification codes, without names or other personal identifiers. The data were used solely for research purposes and are presented in aggregate form.

RESULTS

A total of 60 patients fulfilled the eligibility criteria and were included in the analysis. The mean age was 59.71 ± 7.50 years, and 43 patients (71.7%) were men. The most prevalent risk factors were smoking (71.7%),

hypertension (68.3%), and type 2 diabetes mellitus (41.7%). Multivessel disease without left main involvement was present in 39 patients (65.0%), and with left main involvement in 21 (35.0%). Most patients were in NYHA class II (83.3%), and the median preoperative LVEF was 55.5% (23–68%). The median EuroSCORE II and STS scores were 1.96% and 2.10%, respectively, and the mean SYNTAX score was 29.86 ± 6.24, indicating intermediate anatomical complexity. Off-pump CABG was performed in 38 patients (63.3%), and no patient required an intra-aortic balloon pump (Table 1).

Table 1. Baseline clinical and perioperative characteristics according to 30-day MACE

Characteristic	Total (n = 60)	MACE (n = 36)	No MACE (n = 24)	p value
Demographics				
Age, years	59.71 ± 7.50	61.88 ± 7.41	56.45 ± 6.50	0.005[a]
Male sex	43 (71.7)	22 (61.1)	21 (87.5)	0.026[c]
Female sex	17 (28.3)	14 (38.9)	3 (12.5)	
Risk Factors and Comorbidities				
Smoking	43 (71.7)	22 (61.1)	21 (87.5)	0.026[c]
Type 2 diabetes mellitus	25 (41.7)	15 (41.7)	10 (41.7)	1.000[c]
Hypertension	41 (68.3)	26 (72.2)	15 (62.5)	0.174[c]
Preoperative atrial fibrillation	8 (13.3)	5 (13.9)	3 (12.5)	0.877[c]
Peripheral artery disease	6 (10.0)	4 (11.1)	2 (8.3)	0.725[c]
COPD	13 (21.7)	9 (25.0)	4 (16.7)	0.443[c]
Coronary Anatomy				
Multivessel disease with left main	21 (35.0)	12 (33.3)	9 (37.5)	0.740[c]
Multivessel disease without left main	39 (65.0)	24 (66.7)	15 (62.5)	
Functional Status and Risk Scores				
NYHA class II	50 (83.3)	27 (75.0)	23 (95.8)	0.034[c]
NYHA class III	10 (16.7)	9 (25.0)	1 (4.2)	
Preoperative LVEF, %	55.5 (23–68)	54 (23–66)	56 (33–68)	0.740[b]
EuroSCORE II, %	1.96 (0.50–4.60)	1.96 (0.88–4.10)	1.94 (0.50–4.60)	0.020[b]
STS score, %	2.10 (0.70–12.0)	2.75 (0.90–12.0)	1.94 (0.70–6.37)	0.008[b]
SYNTAX score	29.86 ± 6.24	29.91 ± 5.95	29.79 ± 6.79	0.940[a]
Perioperative Characteristics				
On-pump CABG	22 (36.7)	17 (47.2)	5 (20.8)	0.038[c]
Off-pump CABG	38 (63.3)	19 (52.8)	19 (79.2)	
CPB time, min	0 (0–233)	0 (0–233)	0 (0–194)	0.058[b]
Aortic cross-clamp time, min	0 (0–172)	0 (0–172)	0 (0–158)	0.089[b]
ICU length of stay, days	3 (1–6)	3 (1–6)	3 (2–4)	0.705[b]
Hospital length of stay, days	10 (2–34)	10 (2–34)	10 (5–25)	0.739[b]

Data are presented as n (%), mean ± SD, or median (minimum–maximum). [a]Independent-samples *t* test; [b]Mann–Whitney *U* test; [c]χ^2 or Fisher's exact test. CABG, coronary artery bypass grafting; COPD, chronic obstructive pulmonary disease; CPB, cardiopulmonary bypass; ICU, intensive care unit; LVEF, left ventricular ejection fraction; MACE, major adverse cardiovascular events; NYHA, New York Heart Association; STS, Society of Thoracic Surgeons.

Compared with patients without MACE, those with MACE were older (61.88 ± 7.41 vs. 56.45 ± 6.50 years; p = 0.005), more frequently women (38.9% vs. 12.5%; p = 0.026), more often in NYHA class III (25.0% vs. 4.2%; p = 0.034), had higher EuroSCORE II (p = 0.020) and STS scores (p = 0.008), and more often underwent on-pump surgery (47.2% vs. 20.8%; p = 0.038). Smoking was less frequent among patients with MACE (61.1% vs. 87.5%; p = 0.026). Anatomical pattern, LVEF, SYNTAX score, CPB and AOX times, and length of stay did not differ significantly between the groups. Among the laboratory parameters, patients with MACE had lower hemoglobin levels (12.75 ± 1.54 vs. 13.62 ± 1.69 g/dL; p = 0.044) and eGFR (68.2 ± 20.9 vs. 72.2 ± 22.4 mL/min/1.73 m2; p = 0.030). Hematocrit, leukocyte, and platelet counts; renal and liver function; albumin; HbA1c; and electrolytes did not differ significantly (Table 2).

Table 2. Preoperative laboratory parameters and troponin I concentrations according to 30-day MACE

Parameter	Total (n = 60)	MACE (n = 36)	No MACE (n = 24)	p value
Haematology				
Haemoglobin, g/dL	13.10 ± 1.64	12.75 ± 1.54	13.62 ± 1.69	0.044[a]
Haematocrit, %	39.16 ± 4.93	38.21 ± 4.61	40.58 ± 5.14	0.067[a]
Leucocytes, $\times 10^3/\mu L$	8.50 (3.74–24.8)	8.01 (3.74–16.1)	9.06 (4.91–24.8)	0.386[b]
Platelets, $\times 10^3/\mu L$	242 ± 58.4	243 ± 45.8	241 ± 74.5	0.872[a]
Renal and hepatic function				
Urea, mg/dL	31.5 (8.0–82.0)	37.0 (14.4–82.0)	31.0 (8.0–52.0)	0.068[b]
Creatinine, mg/dL	1.09 (0.59–2.75)	1.04 (0.59–2.75)	1.18 (0.67–2.10)	0.291[b]
eGFR, mL/min/1.73 m^2	74.1 ± 21.4	68.2 ± 20.9	72.2 ± 22.4	0.030[a]
AST, U/L	25 (10–86)	23.5 (10–52)	25 (17–86)	0.141[b]
ALT, U/L	22 (0.87–56)	21 (6–56)	23 (0.87–53)	0.212[b]
Albumin, g/dL	4.50 (3.13–5.09)	4.47 (3.30–5.09)	4.54 (3.13–5.07)	0.287[b]
Metabolic and electrolytes				
HbA1c, %	6.0 (4.9–11.5)	5.8 (4.9–11.5)	6.1 (5.3–9.8)	0.427[b]
Sodium, mmol/L	144.5 ± 3.23	144.9 ± 3.05	143.8 ± 3.44	0.195[a]
Potassium, mmol/L	4.50 ± 0.56	4.41 ± 0.50	4.61 ± 0.64	0.179[a]
Calcium, mg/dL	9.26 (7.86–10.2)	9.26 (7.98–10.2)	9.30 (7.86–10.2)	0.640[b]
Magnesium, mg/dL	2.04 (1.48–3.42)	2.04 (1.48–3.42)	2.03 (1.58–2.76)	0.502[b]
Troponin I				
Preoperative troponin I, ng/mL	0.01 (0.01–0.17)	0.01 (0.01–0.17)	0.01 (0.01–0.03)	0.034[b]
Postoperative troponin I, ng/mL	4.14 (0.01–15.0)	7.49 (0.01–15.0)	0.82 (0.01–15.0)	0.001[b]
Δ troponin I, ng/mL	4.13 (0.00–14.99)	7.45 (0.00–14.99)	0.81 (0.00–14.99)	0.002[b]

Data are presented as mean ± SD or median (minimum–maximum). [a]Independent-samples *t-test*; [b]Mann–Whitney *U* test. Δ troponin I = postoperative minus preoperative troponin I. ALT, alanine aminotransferase; AST, aspartate aminotransferase; eGFR, estimated glomerular filtration rate; HbA1c,

During the 30-day follow-up, MACE occurred in 36 patients (60.0%). Malignant arrhythmia was the most frequent component (22 patients, 36.7%), followed by type 5 MI (16, 26.7%), acute heart failure (14, 23.3%), cardiovascular death (10, 16.7%), and stroke (5, 8.3%). Fifteen patients (25.0%) experienced a single MACE component, whereas 21 (35.0%) experienced more than one (Table 3).

Table 3. Distribution of 30-day major adverse cardiovascular events after CABG (n = 60)

Outcome	n (%)
No MACE	24 (40.0)
Any MACE	36 (60.0)
Individual components[a]	
Malignant arrhythmia	22 (36.7)
Type 5 myocardial infarction	16 (26.7)
Acute heart failure	14 (23.3)
Cardiovascular death	10 (16.7)
Stroke	5 (8.3)
Number of MACE components	
Single component	15 (25.0)
More than one component	21 (35.0)

[a]Patients could experience more than one component; therefore, the sum of components exceeded the number of patients with MACE (n = 36). MACE, major adverse cardiovascular events.

The median preoperative troponin I level was 0.01 (0.01–0.17) ng/mL, the median postoperative troponin I level was 4.14 (0.01–15.0) ng/mL, and the median Δ troponin I level was 4.13 (0.00–14.99) ng/mL. Troponin concentrations did not differ significantly between on-pump and off-pump procedures (all $p > 0.05$). Patients who developed MACE had significantly higher postoperative troponin I (7.49 vs. 0.82 ng/mL; $p = 0.001$) and Δ troponin I (7.45 vs. 0.81 ng/mL; $p = 0.002$) than those who did not (Table 2). When individual MACE components were examined, postoperative and Δ troponin I levels were significantly higher in patients who died and in those with type 5 MI, whereas no significant differences were observed for malignant arrhythmia, acute heart failure, or stroke (Table 4).

Table 4. Troponin I concentrations according to individual 30-day MACE components

Outcome / troponin I (ng/mL)	Event	No event	p value
Malignant arrhythmia (event n = 22; no event n = 38)			
Preoperative	0.01 (0.01–0.10)	0.01 (0.01–0.17)	0.146
Postoperative	6.82 (0.01–15.0)	1.40 (0.01–15.0)	0.155
Δ troponin I	6.80 (0.01–14.99)	1.39 (0.00–14.99)	0.193
Acute heart failure (event n = 14; no event n = 46)			
Preoperative	0.01 (0.01–0.17)	0.01 (0.01–0.03)	0.957
Postoperative	8.21 (0.09–15.0)	2.23 (0.01–15.0)	0.181
Δ troponin I	8.20 (0.08–14.99)	2.22 (0.00–14.99)	0.188
Stroke (event n = 5; no event n = 55)			
Preoperative	0.01 (0.01–0.01)	0.01 (0.01–0.17)	0.307
Postoperative	15.0 (0.17–15.0)	2.36 (0.01–15.0)	0.130
Δ troponin I	14.97 (0.13–14.99)	2.35 (0.00–14.99)	0.145
Cardiovascular death (event n = 10; no event n = 50)			
Preoperative	0.01 (0.01–0.10)	0.01 (0.01–0.17)	0.199
Postoperative	11.1 (6.7–15.0)	1.70 (0.01–15.0)	0.010*
Δ troponin I	11.0 (2.35–14.99)	1.69 (0.00–14.99)	0.013*
Type 5 myocardial infarction (event n = 16; no event n = 44)			
Preoperative	0.01 (0.01–0.12)	0.01 (0.01–0.17)	0.041*
Postoperative	15.0 (0.90–15.0)	1.10 (0.01–15.0)	<0.001*
Δ troponin I	14.9 (0.89–14.99)	1.09 (0.00–14.99)	<0.001*

Data are presented as median (minimum–maximum); Mann–Whitney *U* test. *p < 0.05. Δ troponin I = postoperative troponin I − preoperative troponin I.

In backward stepwise logistic regression, including preoperative and postoperative troponin I levels, preoperative troponin I was eliminated (p = 0.354). Postoperative troponin I levels remained independently associated with 30-day MACE (β = 0.208; OR 1.231; 95% CI 1.087–1.395; p = 0.001), indicating a 23.1% increase in the odds of MACE for each 1 ng/mL increase in postoperative troponin I. The final model was logit(MACE) = −0.645 + 0.208 × postoperative troponin I (ng/mL). ROC analysis (Table 5)

Table 5. Prognostic performance of troponin I for 30-day MACE after CABG

Troponin I	Cut-off (ng/mL)	AUC (95% CI)	p value	Sens (%)	Spec (%)	PPV (%)	NPV (%)
Preoperative	0.015	0.605 (0.463–0.747)	0.170	25.0	95.8	90.0	46.0
Postoperative	2.235	0.749 (0.619–0.879)	0.001*	77.8	79.2	84.8	70.4
Δ troponin I	2.225	0.742 (0.610–0.875)	0.002*	77.8	79.2	84.8	70.4

Optimal cut-off values were determined using the maximum Youden index. *p < 0.05. AUC, area under the receiver operating characteristic curve; CI, confidence interval; NPV, negative predictive value; PPV, positive predictive value; Sens, sensitivity; Spec, specificity.

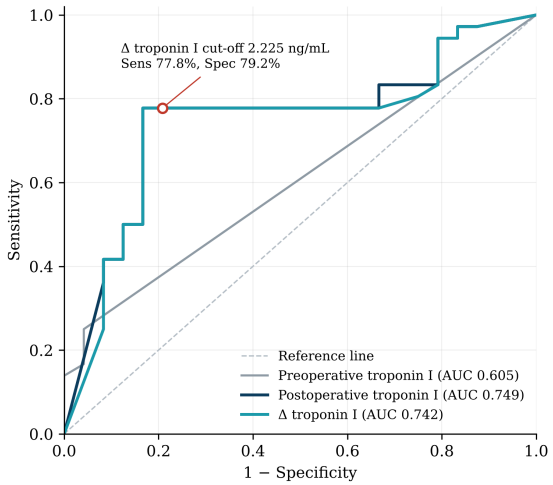


Figure 1. Receiver operating characteristic curves of preoperative troponin I, postoperative troponin I, and Δ troponin I for predicting 30-day MACE after CABG. The circle marks the optimal Δ troponin I cut-off (2.225 ng/mL). The dashed diagonal line indicates no discrimination.

Showed that preoperative troponin I levels had poor and non-significant discrimination (AUC 0.605; 95% CI 0.463–0.747; p = 0.170). In contrast, postoperative troponin I (AUC 0.749; 95% CI 0.619–0.879; p = 0.001) and Δ troponin I (AUC 0.742; 95% CI 0.610–0.875; p = 0.002) demonstrated moderate, statistically significant discrimination. The optimal Δ troponin I cut-off of 2.225 ng/mL yielded a sensitivity of 77.8%, specificity of 79.2%, PPV of 84.8%, and NPV of 70.4%; nearly identical performance was obtained for postoperative troponin I at a cut-off of 2.235 ng/mL. (Figure 1)

DISCUSSION

In this retrospective cohort of patients undergoing elective CABG at an Indonesian tertiary referral center, three principal findings emerged. First, postoperative Δ troponin I was substantially higher in patients who experienced 30-day MACE and showed moderate discrimination (AUC 0.742), with a threshold of approximately 2.2 ng/mL providing balanced sensitivity and specificity. Second, postoperative troponin I was independently associated with MACE, whereas preoperative troponin I added no independent prognostic information. Third, among individual endpoints, troponin elevation was most strongly related to cardiovascular death and type 5 MI, rather than to arrhythmia, heart failure, or cerebrovascular events.

Our findings are consistent with those of previous clinical studies. Li et al. reported that early postoperative high-sensitivity troponin I was independently associated with perioperative MACE after CABG and proposed an updated prognostic cutoff for contemporary assays [18]. Abdelrehim et al. found that postoperative troponin I predicted mortality and MACE after CABG with an AUC of 0.777, comparable to the 0.749 observed here, and that it improved the discrimination of perioperative risk models [19]. In a secondary analysis of the VISION Cardiac Surgery study, high-sensitivity troponin I after both on-pump and off-pump CABG was independently associated with 30-day mortality after adjustment for EuroSCORE II, with a graded relationship between postoperative troponin and early death [20]. In the Indonesian cohort of Nauli et al., a troponin I threshold of 5 ng/mL at 8 hours yielded a sensitivity of 70.6% and a specificity of 68.2% for 30-day MACE [15]. Our optimal threshold was lower (2.225 ng/mL) with somewhat better accuracy. The near-identical performance of Δ troponin I and absolute postoperative troponin I is mechanistically informative. In our elective population, preoperative concentrations were close to the detection limit (median 0.01 ng/mL), so Δ troponin I was effectively equivalent to the postoperative value. In this setting, both measures quantify myocardial injury attributable to the operation itself. Troponin release after CABG is multifactorial: beyond irreversible necrosis due to inadequate myocardial protection, graft failure, or incomplete revascularization, troponin may be released through reversible membrane injury, apoptosis, and proteolytic degradation of the contractile apparatus [21]. Larger postoperative releases nonetheless indicate a greater volume of injured myocardium and a greater likelihood of graft-related or ischemic complications, which plausibly explains the link with mortality. Friedman et al. showed that postoperative high-sensitivity troponin I was markedly higher in patients with type 5 myocardial infarction, but that the conventional 10 × upper reference limit threshold has high sensitivity and low specificity for perioperative infarction [22]. supporting the concept that prognostically relevant thresholds are procedure-specific.

Differences in thresholds across studies likely reflect variations in assay platforms, sampling time, and outcome definitions. High-sensitivity assays permit earlier and more precise quantification of injury [23]. However, their interpretation must remain context-dependent, integrating clinical, electrocardiographic, and imaging findings [24]. The systematic review by Thio et al. reported c-statistics of 0.60–0.81 for postoperative troponin I across studies, while noting moderate-to-high risk of bias and heterogeneous endpoints [12]. Our AUC of 0.74–0.75 falls within this range and indicates acceptable, but not excellent, discrimination. At the 2.225 ng/mL cut-off, the high PPV (84.8%) suggests that values above the threshold should prompt intensified surveillance, whereas the modest NPV (70.4%) indicates that values below it cannot reliably exclude subsequent events. The lack of independent prognostic value of preoperative troponin I contrasts with reports in which baseline elevation predicted adverse outcomes [25,26]. Our cohort consisted predominantly of stable patients undergoing elective surgery with near-undetectable baseline troponin, which limited variability and statistical power; in this context, baseline troponin primarily confirms the absence of ongoing acute injury.

This interpretation is consistent with Hess et al., who found no independent association between preoperative troponin and outcomes after CABG [14].

The 30-day MACE rate in our cohort (60.0%) is high and warrants careful interpretation. Our composite deliberately captured clinically relevant early events, including type 5 myocardial infarction and arrhythmias requiring intervention, which are frequent after cardiac surgery and inflate event rates relative to studies restricted to death, myocardial infarction, and stroke. The absence of a significant relationship between troponin and malignant arrhythmia, acute heart failure, or stroke is biologically plausible. Postoperative arrhythmias are driven largely by systemic inflammation, autonomic imbalance, electrolyte shifts, and atrial remodelling [27,28]. Postoperative stroke is predominantly embolic and related to aortic manipulation, atrial fibrillation, carotid disease, and prior cerebrovascular disease [29]. In addition, the small number of strokes (n = 5) limited the statistical power. Conversely, the strong association with cardiovascular death agrees with large cohorts in which perioperative myocardial injury, renal dysfunction, and reduced ventricular function are dominant determinants of mortality after isolated CABG [30]. Several clinical characteristics were also associated with MACE. Older age is a well-established determinant of adverse outcome owing to age-related vascular stiffening, endothelial dysfunction, and reduced physiological reserve [31]. The higher event rate among women is consistent with the loss of oestrogen-mediated vascular protection after menopause and with the older age and greater comorbidity of women at the time of surgery [32]. The apparently lower MACE rate among smokers most likely reflects confounding by sex, as most smokers were men, rather than any protective effect, given the well-documented endothelial toxicity of tobacco smoke [33]. EuroSCORE II and STS scores were higher in patients with MACE, in keeping with validation studies from Malaysia and Indonesia [34,35]. Lower preoperative hemoglobin and reduced renal function, both recognized predictors of adverse outcome after CABG [36,37]. were also more common in the MACE group. Patients undergoing on-pump surgery experienced more events, although troponin release did not differ by technique; the excess risk may therefore be mediated by factors other than myocardial injury alone, such as the systemic inflammatory response to cardiopulmonary bypass and longer extracorporeal exposure [38]. Preserved LVEF in most of our patients may explain why ventricular function did not discriminate outcomes, in contrast to cohorts with a higher proportion of reduced ejection fraction [39].

This study has several strengths, including consecutive enrolment, a clinically comprehensive composite endpoint, and, to our knowledge, the first evaluation of Δ troponin I after CABG in an Indonesian population. Several limitations must be acknowledged. First, the single-centre retrospective design and modest sample size limit generalizability and precision, as reflected in the wide confidence intervals. Second, with 36 events, the multivariable model could not be adjusted for established clinical predictors such as age, sex, EuroSCORE II, and surgical technique without risking overfitting. Third, troponin sampling was not standardized (any value within 72 hours), so peak concentrations may have been missed, and the assay's upper reportable limit (15.0 ng/mL) produced a ceiling effect that compressed the upper range of values. Fourth, heterogeneity in MACE definitions remains a recognized barrier to comparison across studies, and our inclusion of type 5 MI, which is itself partly defined by troponin, introduces incorporation bias and may inflate the apparent prognostic performance for the composite outcome [40]. Finally, the optimal cut-off values were derived and evaluated in the same dataset and are therefore optimistic; they require external validation before clinical adoption. From a clinical perspective, Δ troponin I is inexpensive, routinely available, and can be obtained within the first postoperative days. Our findings support its use as a pragmatic adjunct to identify patients who may benefit from closer monitoring, earlier echocardiography, and, where appropriate, assessment of graft patency, rather than as a standalone rule-out tool. Prospective multicenter studies with standardized sampling, high-sensitivity assays, and harmonized outcome definitions are required to validate these thresholds and establish their incremental value over established clinical predictors.

CONCLUSION

Postoperative Δ troponin I was significantly associated with 30-day MACE after elective CABG and showed moderate discriminative ability, with a threshold of approximately 2.2 ng/mL, identifying patients at increased

risk. Postoperative troponin I levels were independently associated with MACE and were markedly higher in patients who died, whereas preoperative troponin I levels provided no independent prognostic information. Δ troponin I may serve as a simple and inexpensive adjunct to clinical risk scores for early postoperative risk stratification, pending external validation in larger prospective cohorts.

DECLARATIONS
None

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The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS
C.I.P. Conceptualization, methodology, data curation, investigation, formal analysis, visualization, and writing – original draft. A.H.R. conceptualization, methodology, supervision, validation, and writing – review and editing. A.S. methodology, supervision, validation, and writing – review and editing. All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

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