Preoperative Troponin Levels and Outcomes of Coronary Surgery Following Myocardial Infarction

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Abstract

Background: This study evaluates the impact of peak preoperative troponin level on outcomes of coronary artery bypass grafting (CABG) for non-ST-elevation myocardial infarction (NSTEMI). Methods: This was a retrospective review of patients undergoing isolated CABG from 2011-2018 with presentation of NSTEMI. Patients were stratified into low- and high-risk groups based on median preoperative peak troponin (1.95ng/dL). Major cardiac and cerebrovascular events (MACCE) and mortality were compared. Multivariable analysis was performed to model risk factors for MACCE and mortality. Results: This study included 1,211 patients, 607 low- ([?]1.95ng/dL) and 604 high-risk (>1.95ng/dL). Patients were well-matched with respect to age and comorbidity. High-risk patients had lower median preoperative ejection fraction (46.5% [IQR 35.0%-55.0%] vs 53.0% [IQR 40.0%-58.0%]) and higher incidence of preoperative intra-aortic balloon pump (15.9% vs 8.73%). Intensive care unit (47 hours [IQR 26-82] vs 43 hours [IQR 25-69]) and hospital lengths of stay (10 days [IQR 8-13] vs 9 days [IQR 8-12]) were longer in the high-risk group (each P<0.05). Postoperative complications and thirty-day, one- and five-year rates of both MACCE and survival were similar between groups. Peak troponin >1.95ng/dL was not associated with increased hazards for MACCE, mortality, or readmission in multivariable modeling. In sub-analyses, neither increasing troponin as a continuous variable nor peak troponin >10.00ng/mL were associated with increased hazards for these outcomes. Conclusions: Higher preoperative troponin levels are associated with longer lengths of stay but not MACCE or mortality following CABG. Dictating timing of CABG for NSTEMI based on peak troponin does not appear to be warranted.

Introduction

Elevation of blood serum troponin I (cTnI) and other cardiac enzymes is a key feature of cardiac ischemia and myocardial infarction[1,2]. In the event of myocardial infarction, detectable troponin elevation is present within a few hours of the event, and peak levels are reached by 24-48 hours[3]. Presence of acute coronary syndrome with troponin elevation has been demonstrated to signify higher risk of mortality than patients without troponin elevation[4,5]. Furthermore, mortality risk increases following coronary revascularization, both via percutaneous coronary intervention or coronary artery bypass grafting (CABG), when preprocedural troponin levels are elevated[6–12]. It is well-documented that pre-revascularization troponin elevation is associated with decreased survival, however, the significance of peak troponin level on outcomes of revascularization has been more controversial[5,6,13]. The purpose of this study was to evaluate the impact of preoperative peak troponin level on short- and long-term outcomes of isolated CABG for non-ST-elevation myocardial infarction (NSTEMI).

Patients and Methods

Study Population

This was a retrospective single institutional analysis that included adults (18 years or older) that underwent isolated, primary CABG operations at a multi-hospital health system between January 2011 and June 2018. Only patients with cardiac presentation of NSTEMI and troponin leak were analyzed. The highest (peak) measured blood serum troponin in the preoperative period was recorded for each patient. This study was approved by the Institutional Review Board at the University of Pittsburgh (MOD18120143-003, approved 3/9/2020). Due to retrospective nature of the study, patient consent was waived.

Outcomes and Sub-analysis

Patients were stratified into low and high-risk cohorts based on the peak preoperative troponin level, with the threshold for stratification centered around the median troponin level for the study cohort. The primary outcome of this study was long-term major adverse cardiac and cerebrovascular events (MACCE), which is a composite of death, myocardial infarction, stroke, or need for repeat revascularization procedures. Other outcomes investigated included short and long-term survival, postoperative complications, and intensive care unit and hospital length of stay. Predictors of MACCE and mortality were also modeled.

Preoperative peak troponin levels were also evaluated when modeled as a continuous variable. A secondary sub-analysis was performed with a clinically meaningful troponin threshold of 10.00 ng/mL to define high- and low-risk cohorts as well. In this secondary analysis, long-term MACCE and survival were analyzed and modeled using multivariable analysis.

Statistical Analysis

Continuous data are presented as mean ± standard deviation for Gaussian variables or median [interquartile range (IQR)] for non-gaussian variables and all categorical data as number (percentage). Normality was assessed using the Kolmogorov-Smirnov test. Normally distributed continuous data and categorical data were compared with Pearson's Chi-squared test or Fisher's exact test when 25% of available data points had expected values <5. Non-Gaussian distributions were evaluated using Mann-Whitney U test.

Kaplan Meier analysis was used to compare long-term survival, and cumulative incidence was used to compare incidence of long-term MACCE across cohorts. Multivariable Cox Proportional Hazards modeling was used to identify predictors for 5-year mortality as well as 5-year MACCE. Co-variables were first assessed by univariable model for statistical significance. A threshold of $P<0.2$ was then employed for inclusion into the multivariable model.

Results

Baseline Demographics and Operative Characteristics

A total of 1,211 patients were included in this study, 607 (50.1%) low-risk and 604 (49.9%) high-risk patients. Median age for both cohorts was 66 years, and both cohorts had similar body mass index, as well as distributions of gender and race. Both groups had similar comorbidity burden, but the low-risk group had higher prevalence of cerebrovascular disease (25.04% vs 20.20%, P=0.044). Patients in the high-risk group had higher prevalence of congestive heart failure (25.33% vs 19.77%, P=0.021) and presentation with advanced stage New York Heart Association symptoms (Class III or IV). High-risk patients also had a lower median left ventricular ejection fraction (46.5% [IQR 35.0% to 55.0%] vs 53% [IQR 40.0% to 58.0%], P<0.001) (**Table 1**).

Patients in both cohorts had equal distributions of elective, urgent, and emergent CABG. A higher proportion of patients were bridged to CABG with an intra-aortic balloon pump (15.89% vs 8.73%, P<0.001) in the high-risk group. Society of Thoracic Surgeon predicted risk of mortality was similar between cohorts. There was a higher utilization of cardiopulmonary bypass in the high-risk group (83.11% vs 70.68%, P<0.001), but median perfusion and aortic cross-clamp times were similar (**Table 1**).

Postoperative Complications and Long-Term Survival

Patients in the high-risk cohort had a higher incidence of prolonged mechanical ventilation (12.58% vs 8.24%,

P=0.013), longer median intensive care unit times (47.0 hours [IQR 26.0 to 81.6] vs 43.0 hours [IQR 24.6 to 69.0], P=0.004), and longer hospital length of stay (10 days [IQR 8-13] vs 9 days [IQR 8-12], P=0.013). Rates of operative mortality, reoperation, and need for transfusion were similar between cohorts. Additionally, rates of renal failure, pneumonia, stroke, and sepsis were also similar (**Table 2**).

Thirty-day mortality was similar between high- and low-risk cohorts. Mortality at one (8.94% vs 6.92%, P=0.193) and five years (17.72% vs 14.66%, P=0.149) was also similar. Kaplan Meier survival between cohorts is shown in **Figure 1** .

Cox Proportional Hazards modeling was conducted to identify risk-adjusted predictors for 5-year mortality. In this model, preoperative troponin peak above 1.95 ng/mL was not associated with increased hazards for mortality (HR 1.28, 95% CI 0.94 to 1.72, P=0.113). Similar findings were obtained when preoperative troponin level was modeled as a continuous variable (HR 1.00, 95% CI 0.99 to 1.01, P=0.674) (**Supplemental Table 1).** Risk-adjusted factors associated with increased hazards for mortality included increasing age, history of diabetes mellitus, chronic obstructive pulmonary disease, immunosuppression, cerebrovascular disease, bridge with intravenous inotropes, and increasing preoperative serum creatinine. Factors associated with decreased hazards for mortality included increasing body surface area and increasing preoperative left ventricular ejection fraction (**Table 3**).

Major Adverse Cardiac and Cerebrovascular Events

One- and five-year rates of MACCE were similar between high- and low-risk groups (**Figure 2**). Multivariable modeling did not find the high-risk cohort to be associated with increased hazards for 5-year MACCE (HR 1.11, 95% CI 0.87 to 1.38, P=0.488). Similarly, when modeled as a continuous variable, increasing preoperative troponin levels did not correlate with rates of MACCE (HR 1.00, 95% CI 1.00 to 1.01, P=0.967) (**Supplemental Table 2**). Factors associated with increased hazards for MACCE included history of diabetes mellitus, cerebrovascular disease, peripheral vascular disease, and increasing serum creatinine. Increasing preoperative left ventricular ejection fraction was associated with decreased hazards for MACCE (**Table 4**).

Post Hoc Sub-analysis

Post hoc sub-analysis was performed by stratifying patients by their preoperative peak troponin level using a clinically meaningful threshold of troponin >10.00 ng/mL to define the high-risk and [?]10.00 ng/mL for the low-risk cohorts. This resulted in 957 (79.0%) low-risk and 254 (21.0%) high-risk patients. In this analysis, there were no differences in intensive care unit or hospital length of stay (**Supplemental Table 3**). Additionally, both 30-day, 90-day, one-, and five-year survival were similar (**Figure 3**). Cox proportional hazards modeling did not find peak troponin above 10.00 ng/mL to be a significant predictor of five-year mortality in univariable (HR 1.11, 95% CI 0.80 to 1.55, P=0.543) or multivariable (HR 0.94, 95% CI 0.66 to 1.33, P=0.713) analysis (**Supplemental Table 4**). Furthermore, peak troponin >10.00 ng/mL was also not found to be a significant predictor of 5-year MACCE in univariable (HR 0.90, 95% CI 0.70 to 1.17, P=0.427) or multivariable (HR 0.80, 95% CI 0.61 to 1.05, P=0.110) models (**Supplemental Table 5**).

Discussion

The principal finding of this study was that a higher level of preoperative peak troponin was associated with increased ICU time and hospital length of stay. However, there was no association with peak troponin level and short- and long-term MACCE or mortality. In sub-analysis, peak troponin >10.00 ng/mL was also not associated with increased hazards for MACCE or mortality.

Previous studies have demonstrated associations with serum troponin elevation with size of myocardial infarction, degree of coronary artery disease, and/or short term morbidity and mortality in patients presenting with acute coronary syndromes[5,8,9]. Furthermore, preoperative troponin elevation has been associated with increased operative morbidity and mortality, as well as increased rates of complications and/or hospital length of stay following CABG[11–15]. In our study, patients with high and low peak troponin levels were fairly well-matched with respect to age and preoperative comorbidity. However, we noticed that the high

serum troponin group had a lower median preoperative left ventricular ejection fraction. Additionally, they required bridging to CABG with intra-aortic balloon pump counterpulsation more frequently, and also had a higher incidence of preoperative congestive heart failure with advanced New York Heart Association class symptoms (III or IV) in comparison to the low troponin group. Based on these findings, it is possible that higher peak troponin may indicate a larger infarction with higher degree of myocardial stunning, which may account for lower preoperative ejection fractions, higher incidence of heart failure, and need for mechanical support. However, it may be possible that patients with history of preexisting heart failure with decreased ejection fraction are more prone to higher serum troponin leakage after NSTEMI, representing a combination of infarction as well as heart failure. In this theory, these patients with worse baseline cardiac function may require intra-aortic balloon pump bridging more frequently after an equivalent cardiac insult as would a patient with normal baseline function and greater cardiopulmonary reserve.

The timing of CABG following acute coronary syndrome has remained controversial[16–19]. Patients presenting with STEMI or ongoing ischemia in NSTEMI require immediate revascularization. Often times these patients can present with arrhythmias, hemodynamic instability, and end-organ dysfunction. As a result, emergent immediate revascularization in these scenarios is often met with a high likelihood of morbidity and mortality, especially between 6 and 24 hours of symptom onset[16,19]. In the setting of NSTEMI or smaller infarctions with no ongoing active ischemia where cardiac function is preserved or mildly depressed, surgical timing can vary between centers. Some groups advocate for prompt revascularization[20,21], whereas other centers advocate for a delay prior to surgery[22]. This delay can allow for the stable patient to undergo preoperative testing and risk-assessment, and also allow any high-dose antiplatelets and/or anticoagulants the patient may have received at time of initial presentation to be metabolized, which may allow for decreased bleeding complications. Other advantages of delaying surgery in some surgeons' opinion is that tissues and coronary vessels are less edematous and easier to handle, and that there is less myocardial stunning which allows the heart to tolerate the operation better. In our series of NSTEMI patients, we did not observe increasing time from peak troponin level to surgical revascularization to have any significant influence on mortality in multivariable modeling. It is likely that timing of surgery in this patient population can be tailored to the individual circumstances and needs of the patient, and that revascularization does not necessarily need to occur within a specific "surgical window". There is always a theoretical risk that delaying surgery too long can lead to lethal arrhythmias, cardiac arrest, or worsening cardiac function due to expanding infarction.

In our series, we observed a small but significant increase in intensive care unit time (47.0 hours [IQR 26.0 to 81.6] vs 43.0 hours [IQR 24.6 to 69.0], P=0.004) and hospital length of stay in patients with higher peak troponin levels (10 days [IQR 8 to 13] vs 9 days [IQR 8 to 12], P=0.013). However, in our sub-analysis, with patient cohorts stratified by a troponin level of 10.00 ng/mL, we did not observe differences in intensive care unit (47.1 hours [IQR 25.3 to 85.5] vs 45.0 hours [IQR 25.0 to 72.0], P=0.217) or hospital length of stay (10 days [IQR 8 to 13 vs 10.0 hours [IQR 8 to 12], P=0.124) (**Supplemental Table 3**). Though there is a possibility for increased intensive care and/or hospital times with increased peak troponin, the absolute difference is small and the clinical and logistical relevance are likely limited.

Lastly, our findings support the notion that preoperative peak troponin levels have little predictive value on long-term MACCE or mortality following surgical revascularization. These findings are corroborated by those of Beller and colleagues. In their analysis of 1,272 urgent or emergent CABG procedures, they discovered that presence of preoperative troponin elevation to be associated with higher risk of morbidity, 1-year, and long-term mortality, when compared to no troponin elevation. However, when evaluating the actual troponin levels, increasing levels did not have association with postoperative morbidity or survival following CABG[6]. Our study did not observe a significant difference in mortality when peak troponin levels were above the median value across patients ($>$ 1.95 ng/mL). Because this median value seemed to be low from a clinical perspective, we elected to perform a post hoc secondary analysis, and evaluate a clinically meaningful cutoff of 10.00 ng/dL. Despite this, high and low troponin cohorts did not have any significant differences in 5-year MACCE or mortality. Similarly, these findings held true when troponin was modeled as a continuous variable. Furthermore, peak troponin >10.00 ng/ML was not associated with increased hazards for MACCE or mortality in our multivariable models. Collectively, these data suggest that the strategy of

basing timing of CABG in NSTEMI on normalization of troponin or having the troponin levels downtrend to a certain level does not appear to be warranted.

Limitations

The current study has several limitations. This study was retrospective and observational in nature, and as such, may be subject to selection bias. In order to account for such possible confounding and selection bias, we did perform multivariable modeling to adjust for such preoperative risk factors. However, it is possible that there may be some unmeasured confounders that remain despite these measures. Additionally, there is no specific protocol in which serial troponin levels are drawn from patients presenting with acute coronary syndrome. As such, collected troponin measurements may not always represent the true "peak" troponin level, which may influence this study's findings. The timing of CABG in these patients was at the discretion of the individual surgeon and was not standardized across patients.

Conclusions

In our analysis of 1,211 adult patients undergoing isolated CABG following NSTEMI, we found higher level of preoperative "peak" troponin (>1.95 ng/mL) to be associated with increased ICU time and hospital length of stay. However, there was no association with peak troponin level and short- and long-term MACCE or mortality. In sub-analysis, peak troponin >10.00 ng/mL was not associated with increased hazards for MACCE or mortality. These data argue against the utility of using preoperative troponin levels as a guide for timing of CABG in NSTEMI.

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Disclosures

Ibrahim Sultan, MD receives institutional research support from Atricure and Medtronic and serves as a consultant for Medtronic Vascular. Arman Kilic, MD is on the medical advisory board for Medtronic, Inc. These affiliations do not create direct conflicts with the content of this manuscript.

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Table 1. Preoperative demographics and operative characteristics

	Low-Risk ([?]1.95 ng/mL)	**High-Risk (>1.95 ng/mL)**	**P-Value**
	N = 607	**N = 604**	
Age, years	66.00 (59.00-74.00)	66.00 (59.00-74.00)	0.588
Body mass index, kg/m^2	29.51 (25.81-33.59)	29.40 (25.79-33.21)	0.683
Body surface area, m^2	2.04 ± 0.26	2.05 ± 0.28	0.693
Female	185 (30.48%)	158 (26.16%)	0.095
Race			
White	559 (92.09%)	550 (91.06%)	0.258
Black	32 (5.27%)	28 (4.64%)	
Other	16 (2.64%)	26 (4.30%)	
Diabetes mellitus	287 (47.28%)	303 (50.17%)	0.315
Dialysis dependency	15 (2.47%)	22 (3.64%)	0.236
Chronic obstructive pulmonary disease	131 (21.58%)	125 (20.70%)	0.706
Hypertension	529 (87.15%)	516 (85.43%)	0.384
Immunosuppression	35 (5.77%)	25 (4.14%)	0.192
Family history of coronary artery disease	151 (24.88%)	161 (26.66%)	0.479
Cerebrovascular disease	152 (25.04%)	122 (20.20%)	0.044
Peripheral vascular disease	114 (18.78%)	107 (17.72%)	0.631
Myocardial infarction timing			
Not specified	8 (1.32%)	9 (1.49%)	0.673
Less than 6 hours	6 (0.99%)	5 (0.83%)	
6 to 25 hours	38 (6.26%)	31 (5.13%)	
24 hours to 7 days	542 (89.29%)	539 (89.24%)	
Greater than 7 days	13 (2.14%)	20 (3.31%)	
Previous myocardial infarction	607 (100.0%)	604 (100.0%)	0.631
Congestive heart failure	120 (19.77%)	153 (25.33%)	0.021
NYHA class symptoms			
I	491 (80.89%)	457 (75.66%)	0.043
II	18 (2.97%)	15 (2.48%)	
II	50 (8.24%)	56 (9.27%)	
IV	48 (7.91%)	76 (12.58%)	
Prior cardiac arrhythmia	101 (16.64%)	102 (16.89%)	0.908
Number of diseased coronary vessels			
1	16 (2.64%)	12 (1.99%)	0.317
2	129 (21.25%)	111 (18.38%)	
3	462 (76.11%)	481 (79.64%)	
Antiplatelet within 5 days of surgery	573 (94.40%)	579 (95.86%)	0.237
Anticoagulation within 5 days of surgery	493 (81.22%)	536 (88.74%)	<0.001
Intravenous inotropes	13 (2.14%)	22 (3.64%)	0.119
Cardiopulmonary bypass utilization	429 (70.68%)	502 (83.11%)	0.000
Perfusion time, min	98.00 (79.00-117.0)	99.00 (79.00-121.0)	0.440
Cross-clamp time, min	68.00 (53.00-87.00)	68.50 (53.00-88.00)	0.627
Preoperative intra-aortic balloon pump	53 (8.73%)	96 (15.89%)	0.000
Bilateral IMA harvest	90 (14.83%)	67 (11.09%)	0.053
Serum creatinine, mg/dL	0.94 (0.80- 1.13)	1.00 (0.80- 1.20)	0.132
Total albumin, g/dL	3.40 (3.20- 3.80)	3.40 (3.10- 3.70)	<0.001
Total bilirubin, mg/dL	0.60 (0.40- 0.70)	0.60 (0.40- 0.80)	0.042

	Low-Risk ([?]1.95 ng/mL)	**High-Risk (>1.95 ng/mL)**	**P-Value**
Left ventricular EF, %	53.00 (40.00-58.00)	46.50 (35.00-55.00)	<0.001
Operative status			
Elective	1 (0.16%)	5 (0.83%)	0.245
Urgent	572 (94.23%)	568 (94.04%)	
Emergent	34 (5.60%)	31 (5.13%)	
STS predicted risk of mortality, %	1.52 (0.77- 3.43)	1.75 (0.83- 3.88)	0.129
Time from peak troponin to surgery, days	2.48 (1.28- 4.00)	2.56 (1.09- 4.03)	0.668

EF, ejection fraction

IMA, internal mammary artery

NYHA, New York Heart Association

STS, Society of Thoracic Surgeons

Table 2. Postoperative Complications and Outcomes

	Low-Risk ([?]1.95 ng/mL)	**High-Risk (>1.95 ng/mL)**	**P-Value**
	N = 607	**N= 604**	
Operative mortality	14 (2.31%)	22 (3.64%)	0.171
Reoperation	30 (4.94%)	41 (6.79%)	0.172
Blood product transfusion	202 (33.28%)	191 (31.62%)	0.538
Prolonged mechanical ventilation	50 (8.24%)	76 (12.58%)	0.013
Pneumonia	20 (3.29%)	18 (2.98%)	0.753
Renal failure	15 (2.47%)	25 (4.14%)	0.104
Stroke			
Undetermined	6 (0.99%)	6 (0.99%)	0.683
Hemorrhagic	0 (0.00%)	1 (0.17%)	
Ischemic	5 (0.82%)	3 (0.50%)	
Transient ischemic attack	1 (0.16%)	2 (0.33%)	0.560
Sepsis	3 (0.49%)	6 (0.99%)	0.312
Intensive care unit time, hours	43.00 (24.58-69.00)	47.00 (26.00-81.63)	0.004
Hospital length of stay, days	9.00 (8.00-12.00)	10.00 (8.00-13.00)	0.013

Table 3. Multivariable Cox Proportional Hazards model for 5-year mortality

	Hazard Ratio	**95% Hazard Ratio Confidence Interval**	**95% Hazard Ratio Confidence Interval**	**P-Value**
Peak troponin >1.95 ng/mL	1.28	0.94	1.72	0.113
Diabetes mellitus	1.50	1.10	2.04	0.011
Dialysis dependency	2.36	0.99	5.64	0.053
Chronic obstructive pulmonary disease	1.40	1.01	1.94	0.044

	Hazard Ratio	**95% Hazard Ratio Confidence Interval**	**95% Hazard Ratio Confidence Interval**	**P-Value**
Hypertension	0.72	0.45	1.14	0.164
Immunosuppression	2.35	1.45	3.79	0.001
Family history of CAD	0.76	0.52	1.11	0.153
Cerebrovascular disease	1.68	1.22	2.32	0.002
Peripheral vascular disease	1.36	0.97	1.91	0.072
Intravenous inotropes	2.34	1.23	4.43	0.009
Age, increasing, years	1.04	1.02	1.05	<.001
Body surface area, increasing, per m^2	0.52	0.28	0.94	0.030
Serum creatinine, increasing, per mg/dL	1.17	1.02	1.33	0.025
Left ventricular EF, increasing, per 10%	0.76	0.67	0.85	<.001
Total bilirubin, increasing, per mg/dL	0.60	0.39	0.94	0.025
Cardiopulmonary bypass usage	0.76	0.54	1.06	0.103
Preoperative troponin peak timing, increasing, per day	1.04	0.99	1.10	0.157

CAD, coronary artery disease

EF, ejection fraction

Table 4. Multivariable Cox Proportional Hazards model for five-year MACCE

	Hazard Ratio	**95% Hazard Ratio Confidence Interval**	**95% Hazard Ratio Confidence Interval**	**P-Value**
Peak troponin >1.95 ng/mL	1.11	0.87	1.38	0.488
Female	1.12	0.89	1.40	0.328
Diabetes mellitus	1.29	1.04	1.60	0.019
Dialysis dependency	1.43	0.71	2.87	0.317

	Hazard Ratio	**95% Hazard Ratio Confidence Interval**	**95% Hazard Ratio Confidence Interval**	**P-Value**
Chronic obstructive pulmonary disease	1.09	0.85	1.40	0.498
Cerebrovascular disease	1.45	1.15	1.83	0.002
Peripheral vascular disease	1.42	1.11	1.83	0.006
NYHA class symptoms				
I	Reference	Reference	Reference	Reference
II	1.27	0.74	2.21	0.388
III	1.37	0.99	1.90	0.060
IV	1.17	0.84	1.64	0.350
Cardiac arrythmia	1.15	0.89	1.49	0.298
Intravenous inotropes	2.22	1.38	3.57	0.001
Cardiopulmonary bypass usage	0.84	0.66	1.08	0.172
Bilateral IMA harvest	0.81	0.57	1.14	0.223
Serum creatinine, increasing, per mg/dL	1.16	1.05	1.29	0.006
Total albumin, increasing, per g/dL	0.88	0.70	1.11	0.281
Preoperative troponin peak timing, increasing, per day	1.03	0.99	1.07	0.108
Left ventricular EF, increasing, per 10%	0.91	0.83	0.99	0.030

EF, ejection fraction

IMA, internal mammary artery

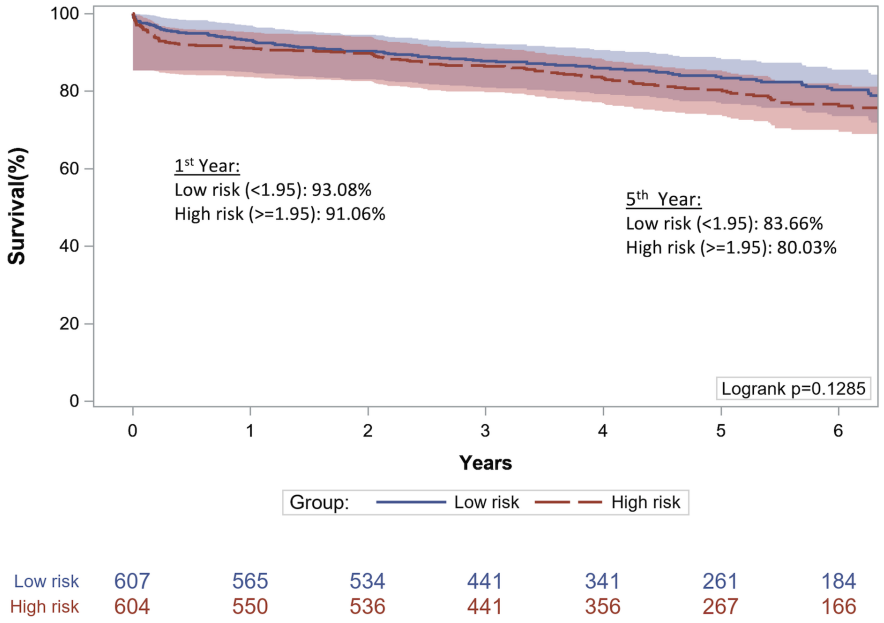
NYHA, New York Heart Association

Figure Legends

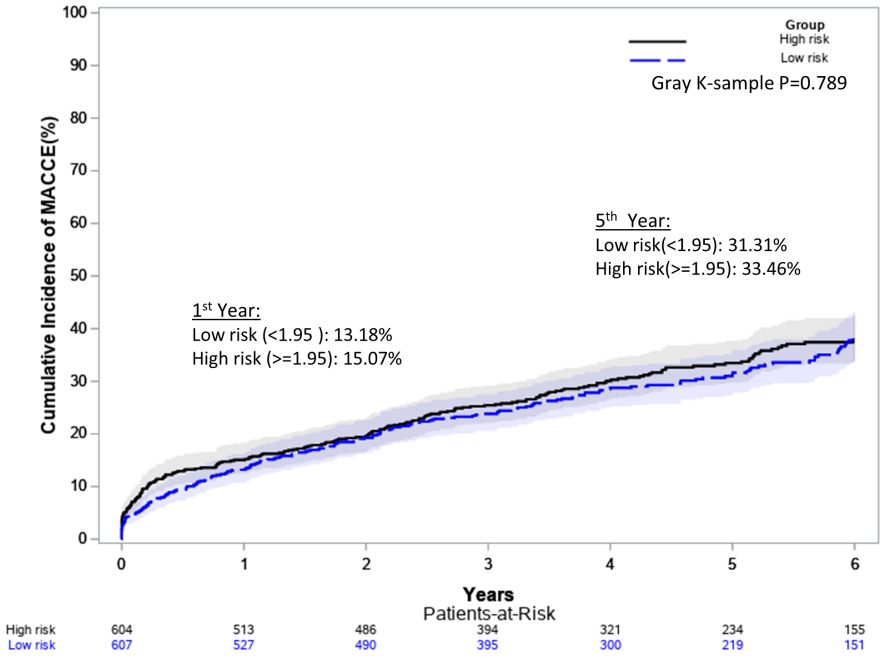
Figure 1. Long-term comparison of overall survival between cohorts according to peak preoperative troponin level

Figure 2. Long-term comparison of MACCE between cohorts according to peak preoperative troponin level

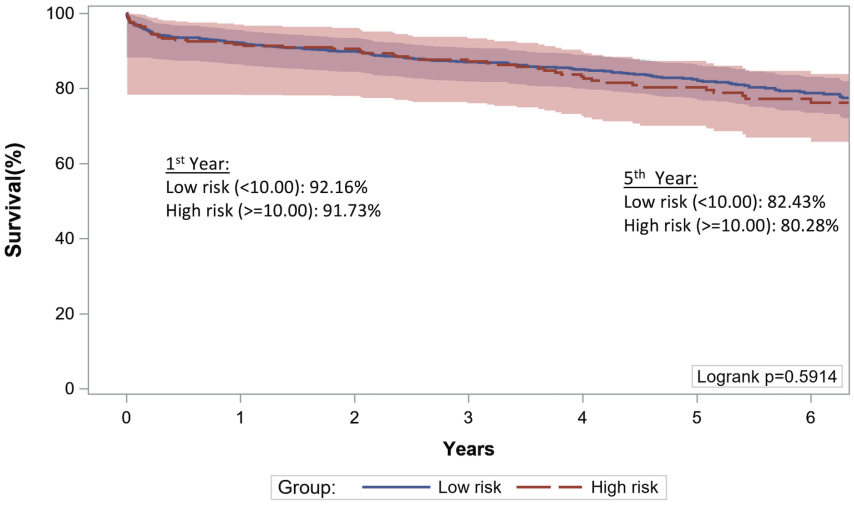
Figure 3. Sub-analysis. Long-term comparison of overall survival between patients with preoperative peak troponin [?] 10.00 ng/mL and >10.00 ng/mL



Low risk	607	565	534	441	341	261	184
High risk	604	550	536	441	356	267	166



High risk	604	513	486	394	321	234	155
Low risk	607	527	490	395	300	219	151



Low risk	957	882	842	692	541	414	274
High risk	254	233	228	190	156	114	76