

Journal of Surgery and Medicine

e-ISSN: 2602-2079

Troponin I levels before bypass surgery after acute myocardial infarction; When to operate?

Akut miyokardiyal infarktüs sonrası bypass cerrahisinden önce troponin I seviyeleri: Ne zaman opere etmeli?

Mihriban Yalçın¹, Eda Gödekmerdan Katırcıoğlu¹, Serkan Yazman¹, Kaptanıderya Tayfur¹, Melih Ürkmez¹

¹ Ordu State Hospital, Department of Cardiovascular Surgery, Ordu, Turkey

ORCID ID of the authors

MY: 0000-0003-4767-0880
EGK: 0000-0003-0724-4051
SY: 0000-0002-6035-1123
KT: 0000-0002-4539-1055
MÜ: 0000-0002-5745-5941

Abstract

Aim: Measurement of cardiac troponin I (cTnI) preop coronary artery bypass surgery (CABG) may be a determinant of surgical risk in patients with myocardial infarction (MI). Our aim is to explain the prognostic value of the preoperative serum levels of cardiac troponin I in patients with a recent acute MI under relatively stable clinical conditions.

Methods: This study was a retrospective single-center study in a small state hospital in Turkey. Included were 65 patients that had undergone a first-time isolated CABG between January 2012 and December 2014 due to acute MI. Samples for preoperative cTnI measurements were collected daily prior to the operation. Patients were enrolled in this study if they had nonST or ST elevation MI and had also undergone an early isolated CABG. We evaluated the association between serum levels of cTnI 24 hours before CABG and postoperative in-hospital patient outcomes.

Results: In cases with mortality troponin was high but it was not statistically significant. There was a significant poor positive correlation between admission troponin and intensive care unit (ICU) stay and the duration of discharge. No statically significant correlation was found between troponin levels and other postoperative in-hospital patient outcomes.

Conclusion: Although there was no significant correlation between troponin values and postoperative data troponin was higher than those without mortality. Therefore we suggest that if the vital signs are stable for patients with AMI preparing to undergo CABG, surgery should be delayed until troponin falls to nearly normal values.

Keywords: Acute myocardial infarction, Coronary artery bypass surgery, Troponin I

Öz

Amaç: Kardiyak troponin I'in (cTnI) preop ölçümü miyokard infarktüsü (MI) olan hastalarda cerrahi riskin belirleyicisi olabilir. Amacımız, son zamanlarda akut miyokard infarktüsü geçirmiş olan hastalarda nispeten stabil klinik koşullar altında kardiyak troponin I'in preoperatif serum düzeylerinin prognostik değerini açıklamaktır.

Yöntemler: Bu çalışma küçük bir devlet hastanesinde yapılmış retrospektif bir çalışmadır. Akut MI nedeniyle Ocak 2012 ile Aralık 2014 arasında ilk kez izole KABG geçiren 65 hasta alındı. Ameliyat öncesi cTnI ölçümleri için numuneler operasyondan önce günlük olarak toplandı. Hastalar, ST veya nonST MI geçirmiş ve erken izole KABG geçirmişlerse bu çalışmaya dahil edildi. KABG'den 24 saat önce cTnI serum seviyeleri ile postoperatif hasta sonuçları arasındaki ilişkiyi değerlendirdik.

Bulgular: Mortalite olan olgularda troponin yüksek olmasına rağmen istatistiksel olarak anlamlı değildi. Giriş troponin ve yoğun bakım ünitesinin (YBÜ) kalış süresi ve taburculuk süresi arasında anlamlı derecede zayıf bir korelasyon vardı. Troponin düzeyleri ile diğer postoperatif hastane içi hasta sonuçları arasında istatistiksel olarak anlamlı bir ilişki bulunmadı.

Sonuç: Troponin değerleri ile postoperatif veriler arasında anlamlı bir korelasyon olmamasına rağmen, troponin mortalite olanlarda olmayanlara göre daha yüksekti. Bu nedenle, KABG'ye girmeye hazırlanan AMI hastaları için yaşamsal belirtiler stabil ise, troponinin neredeyse normal değerlere düşmesine kadar ameliyatın ertelenmesi gerektiğini öneriyoruz.

Anahtar kelimeler: Akut miyokard infarktüsü, Koroner arter bypass cerrahisi, Troponin I

Corresponding author / Sorumlu yazar:
Mihriban Yalçın
Address / Adres: Sahincili Mah. Ordu Devlet Hastanesi, 52200, Ordu, Türkiye
E-mail: mihribandemir33@hotmail.com

Ethics Committee Approval: Ethics committee approval was not received because of retrospective design of the study.
Etik Kurul Onayı: Etik kurul onayı çalışmamızın retrospektif dizaynından dolayı alınmamıştır.

Conflict of Interest: No conflict of interest was declared by the authors.

Çıkar Çatışması: Yazarlar çıkar çatışması bildirmemişlerdir.

Financial Disclosure: The authors declared that this study has received no financial support.

Finansal Destek: Yazarlar bu çalışma için finansal destek almadıklarını beyan etmişlerdir.

Received / Geliş Tarihi: 17.04.2018

Accepted / Kabul Tarihi: 31.05.2018

Published / Yayın Tarihi: 31.05.2018

Copyright © 2018 The Author(s)

Published by JOSAM

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives License 4.0 (CC BY-NC-ND 4.0) where it is permissible to download, share, remix, transform, and buildup the work provided it is properly cited. The work cannot be used commercially without permission from the journal.



Introduction

Cardiac troponins (I and T) are highly sensitive and serologic biomarkers specific to myocardial tissue that are used for the definition of acute myocardial infarction (AMI). They improve the detection criteria for AMI [1]. Myocardial infarction is now defined as a troponin concentration above the 99th percentile of a healthy reference population in the setting of myocardial ischemia [2]. Because troponins are truly cardio-specific, they are the gold standard for biochemical testing for myocardial cell injury. Cardiac troponin I (cTnI, molecular mass 22 500 Da) is a regulatory protein unique to heart muscle. cTnI is the only TnI isotype located in the myocardium and an inhibitor of the troponin-tropomyosin regulatory complex that confers calcium sensitivity to actomyosin [3]. Cardiac troponin T may cross-react with troponin found in other muscles, producing false positive or increased results when there is no myocardial damage [4]. Because cTnI is not found in skeletal muscle, cTnI is highly sensitive and specific for myocardial necrosis [5]. cTnI estimates infarct size after reperfusion. It is a specific marker of cardiac damage during coronary artery bypass surgery [6]. In addition, cTnI has no cross-reactivity with skeletal muscle isoforms and does not increase in healthy people, even under excessive muscular activity or as the result of non-cardiac operations.

After CABG, risk factors for death or cardiac events have been described. To predict postoperative outcomes, several models have developed [7]. Cardiac troponins have been routinely measured as part of preoperative preparation at hospitals for several years.

The primary aim of the present study was to verify the influence of preoperative cTnI values on the incidence of major postoperative complications and mortality of patients who had experienced an AMI within the previous two weeks before undergoing CABG in stable clinical conditions.

Materials and methods

The study population consisted of 71 consecutive patients who had been admitted via the emergency department of Ordu State Hospital with an AMI. From January 2012 to December 2014, the 65 patients, 14 females and 51 males, underwent elective, isolated, first-time CABG operations with cardiopulmonary bypass. The angiogram for patients with AMI revealed more severe coronary artery disease requiring CABG. But these patients did not require an immediate operation. Samples for preoperative cTnI measurements were collected daily prior to the operation. We evaluated the association between serum levels of cTnI at admission and 24 hours before CABG and postoperative in-hospital patient outcomes. Low cardiac output syndrome, IABP necessity and mechanical ventilation lasting longer than 72 hours, acute renal failure, and in-hospital death are considered major postoperative complications

If any of the following preoperative criteria were present, patients were excluded from the study: (1) reoperations, (2) any concomitant heart surgery besides CABG, or (3) any concomitant AMI mechanical complications. Among the 71 patients, 6 excluded because of these criteria. A cTnI value

above 0.3 ng/mL was considered abnormal (Reference range 0.00-0.30 ng/mL). In all patients, standard anesthetic and monitoring techniques were used. Internal thoracic artery and saphenous vein grafts were used as graft conduits. A standard cardiopulmonary bypass was achieved by ascending aortic and two-stage venous cannulation. Myocardial protection was achieved by using antegrade crystalloid cardioplegic arrest and additional topical cooling.

Postoperatively, patients were admitted to the intensive care unit (ICU). Postoperative management of patients was standardized. If patients were hemodynamically stable, they were discharged from the ICU to the general cardiothoracic ward, usually on the second postoperative day. Patients were generally discharged from the hospital on postoperative day 7–10. The patients were followed during hospitalization for incidence of death and postoperative complications. The median time from arrival to CABG for AMI patients was approximately 170 hours (about seven days).

Statistical analysis

The data obtained in this study were analyzed using SPSS (Statistical Package for the Social Sciences) program 22.0 for Windows. In the evaluation of the data for descriptive statistical methods, number, percentage, mean, and standard deviation were used. A t-test was used to compare continuous quantitative data between two independent groups. A one-way analysis of variance (ANOVA) test was used to compare continuous quantitative data between more than two independent groups. After the ANOVA test, the Scheffe test was used to determine differences as a complementary post-hoc analysis. A correlation between continuous variables in this study was performed. The findings are evaluated to be in the 95% confidence interval and at the 5% significance level.

Results

Of the 51 (78.5%) male and 14 (21.5%) female patients, 44 (67.7%) were nonST MI, 11 (16.9%) had anterior MI, and 10 (15.4 %) had inferior MI. Preoperative characteristics of the patients are seen on table 1.

CABGX2 was performed for 13 (20.0%) patients; CABGX3 for 30 (46.2%) patients; CABGX4 for 18 (27.7%) patients; and CABGX5 for 4 (6.2%) patients. Mortality was 9.4% (6 patients). Four patients (6.2%) had wound infections 6 (9.4 %) had ARF; 2 (3.1%) had lasting mechanical ventilation; 4 (6.2%), bleeding revision; and 1 (1.6%) LCOS. IABP was used in 8 (12.3%) patients.

Figure 1 shows with Roc analysis to maintain a cut-off value. Shown are 58 positive values, six negative values and one missing value. The area under the ROC curve was not statistically significant ($p > 0.05$). Because the ROC curve was not meaningful, a cut-off value could not be obtained. Figure 2 shows ROC analysis for troponin at admission.

Table 2 shows the mean values of troponin at admission and 24 hours before operation. According to the ANOVA test, the mean of troponin at admission, values were statistically significant ($F=154.906$; $p=0<0.05$) as expected. A complementary post-hoc analysis was conducted to determine the sources of the differences. The first troponin values of patients with anterior MI (17.869 ± 5.747) were higher than of

those with NonST MI (2.493 ± 1.156) and the first troponin values of inferior MI (9.470 ± 2.458) were higher than the first troponin values (2.993 ± 1.156) of NonST MI. After all, the mean of troponin values 24 hours before operation was not statistically significant ($p > 0.05$) compared to the average of the groups.

There was no statistically significant difference between the number of bypasses and troponin at admission according to the ANOVA test. ($F=1.506$; $p=0.222$; >0.05). IABP was used in 8 patients. There was no statistically significant ($p=0.188$ for troponin at admission and $p=0.894$ for troponin 24 hours before) difference for values of troponin in the use of IABP.

Table 3 shows that the result of the t-test to determine whether there was a difference between the mortality and the mean values of troponin variable was not statistically significant ($p > 0.05$). In cases with mortality troponin was high but it was not statistically significant. Table 4 shows, as expected, there was a significant poor positive correlation ($r=0.317$; $p=0.011$; <0.05) between ICU stay and discharge time period. There was a significant poor positive correlation between admission troponin and ICU stay ($r=0.249$; $p=0.045$; <0.05) and the duration of discharge ($r = 0.312$; $p=0.012$; <0.05). The relationship between other variables is not statistically significant ($p > 0.05$).

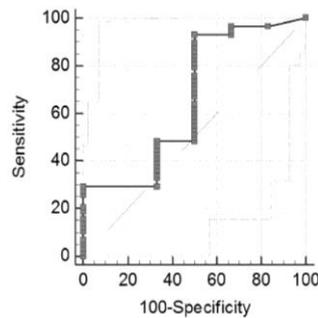
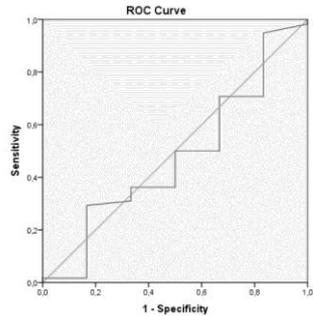


Figure 1: Roc analysis for troponin

Figure 2: Roc analysis for troponin at admission

Table 1: Descriptive parameters

Parameters	Groups	n	%	
Gender	Male	51	78.5	
	Female	14	21.5	
	Total	65	100.0	
Myocardial Infarction	Nonst MI	44	67.7	
	Anterior MI	11	16.9	
	Inferior MI	10	15.4	
	Total	65	100.0	
Cardiovascular risk Factors	DM	40	61.5	
	HT	55	84.6	
	HL	52	80.0	
	Family History	52	80.0	
	Obesity	40	61.5	
Comorbidity	Stroke	5	7.7	
	COPD	22	33.8	
	PAD	10	15.4	
	Total	65	100.0	
Operations	CABGx2	13	20.0	
	CABGx3	30	46.2	
	CABGx4	18	27.7	
	CABGx5	4	6.2	
	Total	65	100.0	
Mortality	No	58	90.6	
	Yes	6	9.4	
	Total	64	100.0	
Complications	None	47	73.4	
	Wound Infection	4	6.2	
	ARI	6	9.4	
	ARDS	2	3.1	
	Revision for bleeding	4	6.2	
	LCOS	1	1.6	
	Total	64	100.0	
	IABP	No used	57	87.7
		Used	8	12.3
		Total	65	100.0

ARDS: acte respiratory distress syndrome, ARI: acute renal insufficiency, CABG:coronary artery bypass graft, COPD: chronic obstructive pulmonary disease, DM: diyabetes mellitus, HL: hyperlipidemia, HT: hypertension, IABP: intra aortic balloon pump, LCOS: low cardiac output syndrome, PAD: peripheral arterial disease

Table 2: The mean value of troponin according to myocardial infarction

Group	n	Mean	Sd	F	p	Diff.
Troponin at admission	NonST MI	44	2.993	1.156		
	Anterior MI	11	17.869	5.747	154.906	0.000
	Inferior MI	10	9.470	2.458		
Troponin 24 hours before operation	NonST MI	44	2.570	10.492		
	Anterior MI	11	4.473	4.198	0.304	0.739
	Inferior MI	10	1.541	2.286		

MI: myocardial infarction, Diff: Difference

Table 3: The mean value of troponin according to the mortality

	Mortality	N	Mean	Sd	t	p
Troponin at admission	yes	6	11.467	10.650		
	no	58	5.671	5.763	-2.146	0.243
Troponin 24 hours before operation	yes	6	3.077	5.870		
	no	58	2.726	9.205	0.091	0.928

Table 4: The relationship between troponin and other parameters

	Age	ICU stay	Discharge from hospital	Troponin at admission	Troponin 24 hours before operation	
Age	r	1.000				
	p	0.000				
ICU stay	r	0.189	1.000			
	p	0.131	0.000			
Discharge from hospital	r	-0.170	0.317*	1.000		
	p	0.180	0.011	0.000		
Troponin at admission	r	-0.016	0.249*	0.312*	1.000	
	p	0.900	0.045	0.012	0.000	
Troponin 24 hours before operation	r	0.066	-0.022	-0.068	0.049	1.000
	p	0.602	0.862	0.594	0.697	0.000

ICU: intensive care unit

Discussion

Nowadays coronary angiography is conducted for an increasing number of patients following a recent AMI. The number of patients referred for CABG after a failed angioplasty or due to left main/multi-vessel disease is increasing. Surgeons therefore now face the difficult decision of the optimal timing of CABG operations for clinically stable patients that have had a recent AMI. Even with improvements in myocardial protection and anesthetic management, however, patients with a recent AMI still have a high mortality rate after CABG operations. A retrospective examination shows that CABG operations should be postponed whenever possible for at least three days after the onset of the AMI [8]. In our study, the median time from arrival to CABG for AMI patients was approximately 170 hours (about seven days). Risk of mortality and postoperative complications may be detectable with the extent of myocardial necrosis, so measuring troponin I before the operation is one noninvasive technique for predicting prognosis after CABG.

Pre-op TnI levels may be useful to guide ICU management of CABG patients after AMI. Troponin

measurement can help identify a high-risk patients suited for additional therapeutic and mechanical interventions (e.g., intra-aortic balloon pump) prior to CABG surgery. In patients with acute coronary syndromes (ACSs), cardiac troponins were revealed as valuable biomarkers for the prognoses. In addition, for PCI, elevated cardiac troponins are related to an increased risk of mortality [9].

Cardiac troponins were also studied in several non-cardiac [10] and cardiac [11,12] surgeries. Most of these studies showed a relationship between postoperative troponin elevations and short- or long-term outcomes. After noncardiac surgery, preoperative cTnT concentrations were significantly associated with postoperative MI and long-term mortality [13].

Carrier et al. reported that patients undergoing elective CABG with preoperative positive >0.02 ng/L serum levels of cTnI were more likely to experience postoperative complications and postoperative MI [14]. Thielman et al. [15] reported that higher rates of mortality and higher incidence of major adverse cardiac events depend on preoperative cTnI serum elevation. Montgomery et al. [16] reported increased mortality rates after open-heart surgery in infants with higher preoperative cTnI concentrations. An increased risk of short-term mortality and morbidity after major vascular surgery is associated with elevated cTnI levels [17]. Patients with postoperative cTnI levels greater than 1.5 ng/mL have six times the mortality risk of patients with levels below 1.5 ng/mL.

Several studies also have reported that increased troponin release after cardiac surgery is associated with mortality and adverse outcomes [18,19]. This relationship between elevated cTnI and adverse outcomes may be explained by more severe and unstable coronary artery stenosis. In our study, preoperative cTnI values were not significantly associated with a higher incidence of low cardiac output syndrome, intra-aortic balloon pump necessity, mechanical ventilation >72 hours, acute renal failure, or in-hospital mortality. Several studies have reported that CABG performed soon after an AMI carries a higher risk of postoperative mortality and morbidity than an operation performed at a later date [20]. A retrospective multicenter analysis of 44,365 patients who underwent CABG after a transmural or nontransmural AMI revealed that in-hospital mortality decreased in all patients that had increased the waiting time between their AMI and surgery. They reported similar postoperative outcomes in patients with both transmural and non-transmural but when CABG was performed within seven days following an AMI, the mortality rate was higher in patients that had had a transmural AMI [21].

Weiss and colleagues [20] broadcasted the results of 40,159 patients hospitalized for AMI who underwent subsequent CABG. Patients were stratified by the timing of CABG into "early" (<2 days from AMI) and "late" (>3 days from AMI) groups. Mortality rates were higher in the early-group patients therefore indicating that CABG ought to be postponed in nonurgent patients for at least three days after an AMI.

Braxton et al. [22] evaluated the timing of CABG after non-Q wave myocardial infarction (MI) and reported that perioperative MI was greater in the non-Q wave MI group when surgery was performed less than 48 hours from admission. Parikh et al. [23] reported that late CABG patients received more

red blood cell transfusions and had longer hospital stays. We also found a significant poor positive correlation between admission troponin and ICU stay and the duration of discharge. Paparella et al. [24] reported that those patients with cTnI >0.15 ng/ml at the time of surgery had more complications and worse survival rates than patients with cTnI <0.15 ng/ml.

In the ACTION Registry-GWTG, they reported no difference in ischemic outcomes, including death between patients with NonST MI undergoing CABG early (≤ 48 hours) vs late (>48 hours). On the other hand, delaying surgery in patients with NonST MI might increase resource use and increase the risk of recurrent ischemia/MI in those waiting for surgical revascularization [21].

Preoperative cardiac troponin levels are more accurate than days for the evaluation of surgery timing. In our study, we performed CABG when troponin levels decreased if a patient's vital signs were stable. This generally took one week. In cases with mortality troponin was not statistically significant, although it was high.

As we described above, if clinical conditions or coronary anatomy of a patient with elevated cTnI values require CABG operation, then surgeons ought to be aware that these patients may be under a higher risk of perioperative myocardial damage and, a higher risk of postoperative adverse events

In contrast to the literature, in our study we could not find any statistically significant relationship between troponin and mortality and postoperative complications. First of all, it can be because of the small numbers of our patient group. And in our clinic, we wait until the troponin decreases if the patient situation is stable.

Therefore, in this study there were no really high values of troponin. There was no statistically significant difference between the number of bypasses and troponin at admission. We only found that there is a significant poor positive correlation between admission troponin and ICU stay and the duration of discharge.

The main limitations of our study were its retrospective design and small numbers of patient group. Therefore, the generalizability of our findings may not extend to all of the clinical centers performing CABG surgery. For clinical decision-making, relative cut-off level and the usefulness of troponin need to be established by large-scale prospective studies.

Finally, patients in our clinic were followed up until troponin levels decreased as low as possible and then operated. There was no correlation between mortality and postoperative complications with troponins. Although there was no significant correlation, troponin elevation was present in those with mortality. Therefore, we believe that these results are due to the fact that normalization of cTnI values before CABG operations seems the way for best postoperative results.

Acknowledgement

We acknowledge the medical writing assistance provided by American manuscript editors (www.americanmanuscripteditors.com) for the final draft of the manuscript.

References

- Alpert JS, Thygesen K, Antman E, Bassand JP. Myocardial infarction redefined -a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. *J Am Coll Cardiol.* 2000;36:959-69.
- The Joint European Society of Cardiology/American College of Cardiology Committee. Myocardial infarction redefined—a consensus document of the Joint European Society of Cardiology/ American College of Cardiology Committee for the redefinition of myocardial infarction. *Eur Heart J* 2000;21:1502–13.
- Adam JE, Bodor GS, Davila-Roman VG, Delmez JA, Apple FS, Ladenson JH, et al. Cardiac troponin I: a marker with big specificity for cardiac injury. *Circulation.* 1993;88:101-6.
- Nesher N, Alghamdi AA, Singh SK, Sever JY, Christakis GT, Goldman BS, et al. Troponin after cardiac surgery: a predictor or a phenomenon? *Ann Thorac Surg.* 2008;85:1348–54.
- Wu AH, Apple FS, Gibler WB, Jesse RL, Warshaw MM, Valdes R Jr. National Academy of Clinical Biochemistry Standards of Laboratory Practice: recommendations for the use of cardiac markers in coronary artery diseases. *Clin Chem.* 1999;45:1104 –21.
- Caputo M, Dihmis W, Birdi I, Reeves B, Suleiman MS, Angelini GD, et al. Cardiac Troponin T and Troponin I release during coronary artery surgery using cold crystalloid and cold blood cardio-plegia. *Eur J Cardio-thorac Surg.* 1997;12:254-60.
- Tu JV, Jaglal SB, Naylor CD. Multicenter validation of a risk index for mortality, intensive care unit stay, and overall hospital length of stay after cardiac surgery. *Circulation.* 1995;91:677-84.
- Paparella D, Scrascia G, Paramythiotis A, Guida P, Magari V, Malvindi PG, et al. Preoperative Cardiac Troponin I to Assess Midterm Risks of Coronary Bypass Grafting Operations in Patients With Recent Myocardial Infarction. *Ann Thorac Surg.* 2010;89:696–703.
- Giannitsis E, Muller-Bardorff M, Lehrke S, Wiegand U, Tölg R, Weidtmann B, et al. Admission troponin T level predicts clinical outcomes, TIMI flow, and myocardial tissue perfusion after primary percutaneous intervention for acute ST-segment elevation myocardial infarction. *Circulation.* 2001; 104:630–35.
- Landesberg G, Shatz V, Akopnik I, Wolf YG, Mayer M, Berlatzky Y, et al. Association of cardiac troponin, CK-MB and postoperative myocardial ischemia with long-term survival after major vascular surgery. *J Am Coll Cardiol.* 2003;42:1547–54.
- Lehrke S, Steen H, Sievers HH, Peters H, Opitz A, Müller-Bardorff M, et al. Cardiac troponin T for prediction of short- and long-term morbidity and mortality after elective openheart surgery. *Clin Chem.* 2004;50:1560–7.
- Thielmann M, Massoudy P, Marggraf G, Knipp S, Schermund A, Piotrowski J, et al. Role of troponin I, myoglobin, and creatine kinase for the detection of early graft failure following coronary artery bypass grafting. *Eur J Cardio-Thorac Surg.* 2004;26:102–9.
- Nagele P, Brown F, Gage BF, Gibson DW, Miller JP, Jaffe AS, et al. High-sensitivity cardiac troponin T in prediction and diagnosis of myocardial infarction and long-term mortality after noncardiac surgery. *Am Heart J.* 2013;166(2):325-32.
- Carrier M, Pelletier LC, Martineau R, Pellerin M, Solymoss BC. In elective coronary artery bypass grafting, preoperative troponin T level predicts the risk of myocardial infarction. *J Thorac Cardiovasc Surg.* 1998;115:1328–34.
- Thielmann M, Massoudy P, Neuhauser M, Knipp S, Kamler M, Piotrowski J, et al. Prognostic Value of Preoperative Cardiac Troponin I in Patients With Non-ST-Segment Elevation Acute Coronary Syndromes Undergoing Coronary Artery Bypass Surgery. *Chest.* 2005; 128:3526-36.
- Montgomery VL, Sullivan JE, Buchino JJ. Prognostic value of pre-and postoperative cardiac troponin I measurement in children having cardiac surgery. *Pediatr Dev Pathol.* 2000;3:53– 60.
- Kim LJ, Martinez EA, Faraday N, Dorman T, Fleisher LA, Perler BA, et al. Cardiac Troponin I Predicts Short-Term Mortality in Vascular Surgery Patients. *Circulation.* 2002; 106(18):2366-71.
- Onorati F, De Feo M, Mastroberto P, Cristodoro L, Pezzo F, Renzulli A, et al. Determinants and prognosis of myocardial damage after coronary artery bypass grafting. *Ann Thorac Surg.* 2005;79:837–45.
- Adabag AS, Rector T, Mithani S, Harmala J, Ward HB, Kelly RF, et al. Prognostic significance of elevated cardiac troponin I after heart surgery. *Ann Thorac Surg.* 2007;83:1744–50.
- Weiss ES, Chang DD, Joyce DL, Nwakanma LU, Yuh DD. Optimal timing of coronary artery bypass after acute myocardial infarction: a review of California discharge data. *J Thorac Cardiovasc Surg.* 2008;135:503–11.
- Lee DC, Oz MC, Weinberg AD, Lin SX, Ting W. Optimal timing of revascularization: transmural versus nontransmural acute myocardial infarction. *Ann Thorac Surg.* 2001;71:198–204.
- Braxton JH, Hammond GL, Letsou GV, Braxton JH, Hammond GL, Letsou GV, et al. Optimal timing of coronary artery bypass graft surgery after acute myocardial infarction. *Circulation.* 1995;92:66–8.
- Parikh SV, de Lemos JA, Jessen ME, Brilakis ES, Ohman EM, Chen AY, et al. Timing of in-hospital coronary artery bypass graft surgery for non-ST segment elevation myocardial infarction patients: Results From the National Cardiovascular Data Registry ACTION Registry–GWTG (Acute Coronary Treatment and Intervention Outcomes Network Registry–Get With The Guidelines). *JACC Cardiovasc Interv.* 2010;3:419–27.
- Paparella D, Scrascia G, Paramythiotis A, Guida P, Magari V, Malvindi PG, et al. Preoperative cardiac troponin I to assess midterm risks of coronary bypass grafting operations in patients with recent myocardial infarction. *Ann Thorac Surg.* 2010;89:696–702.