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Original Article / Özgün Makale

Usefulness of troponin I for prediction of atrial fibrillation after coronary artery bypass grafting

Koroner arter baypas greftleme sonrası atriyal fibrilasyonun öngörülmesinde troponin I'nın kullanılabilirliği

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ABSTRACT

Background: This study aims to evaluate the ability of cardiac troponin I to predict high-risk for AF development after coronary artery bypass grafting.

Methods: Between September 2013 and November 2013, hemodynamically stable and preoperatively in sinus rhythm 74 consecutive patients (65 males, 9 females; mean age 62.1±9.5 years; range, 44 to 75 years) who underwent elective and isolated coronary artery bypass grafting were included in the study. Blood samples were prospectively taken the day before and the next day after surgery to measure cardiac troponin I levels. Atrial fibrillation was documented by electrocardiogram daily and when necessary, until the hospital discharge. The patients were divided into two groups as atrial fibrillation group and sinus rhythm group.

Results: Atrial fibrillation was detected in 15 patients (20%). There was no significant difference between the mean ages of the atrial fibrillation group and sinus rhythm group (p=0.114). Neither cross-clamp time nor cardiopulmonary bypass pump time significantly varied across between two groups (p=0.861, p=0.468, respectively). The incidence of hypertension, hyperlipidemia, and diabetes mellitus were not significantly different between the groups (p=0.225, p=0.385, p=0.318, respectively). There was no significant difference in the length of hospital and intensive care unit stay between two groups (p=0.929, p=0.186, respectively). Preoperative and postoperative cardiac troponin I levels were not associated with postoperative atrial fibrillation development (p=0.763, p=0.336 respectively).

Conclusion: Our study results suggest that cardiac troponin I is not a definite predictor of postoperative atrial fibrillation in patients undergoing coronary artery bypass grafting surgery.

Keywords: Atrial fibrillation; coronary artery bypass grafting; troponin I.

ÖZ

Amaç: Bu çalışmada, kardiyak troponin I'nın koroner arter baypas greftleme sonrası yüksek riskli atriyal fibrilasyon gelişimini öngörebilme özelliği değerlendirildi.

Çalışma planı: Eylül 2013-Kasım 2013 tarihleri arasında, hemodinamik olarak stabil ve ameliyat öncesi sinüs ritminde olan, elektif ve izole koroner arter baypas greftleme yapılan 74 ardışık hasta (65 erkek, 9 kadın; ort. yaş 62.1±9.5 yıl; dağılım 44-75 yıl) çalışmaya alındı. Kardiyak troponin I düzeylerini ölçmek için kan örnekleri, prospektif olarak ameliyattan bir gün önce ve bir sonraki gün alındı. Atriyal fibrilasyon elektrokardiyografi ile taburculuğa kadar her gün ve gerektiğinde kaydedildi. Hastalar, atriyal fibrilasyon grubu ve sinüs ritim grubu olmak üzere iki gruba ayrıldı.

Bulgular: Atriyal fibrilasyon 15 hastada (%20) tespit edildi. Atriyal fibrilasyon grubu ve sinüs ritim grubunun ortalama yaşları arasında anlamlı bir fark yoktu (p=0.114). İki grup arasında kros-klemp süresi ve kardiyopulmoner baypas pompa süresi açısından anlamlı bir fark gözlenmedi (sırasıyla, p=0.861, p=0.468). Hipertansiyon, hiperlipidemi ve diabetes mellitus insidansı açısından gruplar arasında anlamlı bir fark yoktu (sırasıyla p=0.225, p=0.385, p=0.318). Hastanede ve yoğun bakım ünitesinde yatış süresi açısından da iki grup arasında anlamlı bir fark yoktu (sırasıyla, p=0.929, p=0.186). Ameliyat öncesi ve ameliyat sonrası kardiyak troponin I düzeyleri, ameliyat sonrası atriyal fibrilasyon gelişimi ile ilişkili bulunmadı (sırasıyla, p=0.763, p=0.336).

Sonuç: Çalışma sonuçlarımız, koroner arter baypas cerrahisi yapılan hastalarda ameliyat sonrası atriyal fibrilasyon gelişimi için kardiyak troponin I'nın kesin bir öngördürücü olmadığını göstermektedir.

Anahtar sözcükler: Atriyal fibrilasyon; koroner arter baypas greftleme; troponin I.

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Atrial fibrillation (AF) is still a frequently encountered arrhythmia after coronary artery bypass grafting (CABG).[1] Among patients undergoing CABG, AF incidence has been reported to vary between 10% and 30%.[2,3]

Although AF is a benign and often self-limiting disorder, it has significant adverse effects on the recovery which may lead to hemodynamic instability, renal insufficiency, prolonged requirement for ventilatory support, thromboembolic complications, and stroke.[4,5] As, the length of hospital stay and cost of care may increase in this case,[6] many investigations have addressed to identify patients at a higher risk for AF to improve postoperative recovery and reduce the cost.[4,6]

To date, several predisposing factors have been claimed to be causes of postoperative AF such as age, intraoperative manipulation with the heart, myocardial injury, atrial distension, inflammation, and structural heart diseases.[6,7] Among these factors, we focused on myocardial injury to evaluate the possible association between cardiac troponins and postoperative AF development. Myocardial injury detection by measurement of cardiac troponins has been well-established.[8] Many clinical studies have demonstrated that cardiac troponins are reliable biomarkers for the prediction of short and long-term prognosis in patients underwent cardiac surgery.[8] In the present study, we used cardiac troponin I (cTnI) as cTnI has been shown to be a marker of myocardial ischemia and damage with higher sensitivity and specificity compared to other biochemical markers such as troponin T, creatine kinase MB isoenzyme, and myoglobin.[2,9,10]

In this study, we aimed to evaluate whether cTnI can be used as a predictor biomarker to identify patients at a higher risk for AF development after CABG.

PATIENTS AND METHODS

Between September 2013 and November 2013, 74 patients (65 males, 9 females; mean age 62.1±9.5 years; range, 44 to 75 years) in sinus rhythm (SR) undergoing elective, isolated and first-time CABG surgery under cardiopulmonary bypass (CPB) were prospectively enrolled in this study. Patients with a prior history of AF, renal or liver failure, combined surgical procedures (valve surgery, aortic procedures, and carotid surgery) and urgent CABG surgery were excluded.

After standard median sternotomy, the left internal thoracic artery was harvested and the ascending aorta and right atrial cannulation were performed for all patients. Heparin was administered aiming at an activated clotting time above 480 seconds. Systemic hypothermia (28°-30 °C) was employed and intermittent antegrade cold (4 °C) blood cardioplegia was used in every 20 minutes. Both distal and proximal anastomoses were performed under cross-clamping ascending aorta. When the hemodynamic stability and normothermia were maintained, weaning from CPB was initiated.

All patients were continuously monitored for rhythm during the intensive care unit (ICU) stay. After ICU discharge, the patients were monitored once daily by electrocardiography (ECG) until the hospital discharge. The occurrence of AF was defined as an episode of AF lasting longer than 10 minutes in any of ECG recording within the hospital stay. Then, the patients were divided into two groups including AF group and SR group, according to their postoperative rhythm.

A written informed consent was obtained from each patient. The study protocol was approved by the institutional ethical committee. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Preoperative blood samples were obtained the day before surgery from peripheral veins and postoperative blood samples were obtained 24 hours after ICU admission from the central vein catheter and they were collected in standard tubes with sodium citrate. The cTnI levels were measured by using the Abbott ci4100 equipment (Abbott Diagnostics, Chicago, IL, USA). The diagnostic cut-off value of this assay, indicating significant myocardial injury based on our institution reference ranges, is 0.05 ng/mL.

Statistical analysis

Statistical analysis was performed using the SPSS for Windows version 10.1 software (SPSS Inc., Chicago, IL, USA). The Kolmogorov-Smirnov test was used to analyze normally distributed continuous variables. Categorical variables are presented in counts (percentages), while continuous variables are presented in mean ± standard deviation (SD) or median (25^{th}-75^{th} percentiles). The continuous variables were compared using a t test for unpaired samples or the Mann-Whitney U test. The categorical data were tested with chi-square test or Fisher's exact test. Logistic regression analyses were performed to analyze the association between the AF development and various variables and cTnI levels. The receiver operating characteristic curves (ROC) were used for the assessment of the cut-off cTnI value of AF occurrence in the postoperative period. A *p* value of $p<0.05$ was considered statistically significant.

RESULTS

Baseline characteristics of the patients are shown in Table 1. Atrial fibrillation was detected in a total of 15 patients (20%), of those 13 were males (86.6%). The mean age of the patients who developed postoperative AF was 62.1±9.5. There were no statistically significant differences in the mean age (p=0.114) and the incidence of hypertension (p=0.225), hyperlipidemia (p=0.385), and diabetes mellitus between the AF and SR groups (p=0.318). Neither cross-clamp time nor CPB pump time significantly varied across between the two groups (p=0.861, p=0.468, respectively). No statistically significant relationship was found between the postoperative AF and left atrial anteroposterior diameter above 40 mm (p=0.446) and right coronary artery (RCA) revascularization (p=0.999). The length of ICU and hospital stays was not significantly different between the groups (p=0.186, p=0.929 respectively). Preoperative and postoperative cTnI levels were not associated with postoperative AF development (p=0.763) (Table 2).

The ROC analysis showed no cut-off point for cTnI levels measured before and after surgery. In addition, based on the logistic regression analysis, none of the variables were found to be predictors of postoperative AF development (Table 3).

DISCUSSION

In this study, we hypothesized that perioperative myocardial injury, which can be detected by cTnI, plays an important role in the development of postoperative AF. However, we did not observe an association between the cTnI levels and postoperative AF development.

Despite the improvements in surgical techniques, anesthesia, and myocardial protection, the incidence of AF remains a frequent complication after CABG.[4] Consistent with previous data, we observed an incidence of 20% of AF events after CABG. Several studies[11] found that the incidence of AF was higher in patients who underwent CABG, when combined valve procedures were performed; however, such patients were excluded. In addition, several studies have shown that AF occurrence peaks between the second and fourth days after cardiac surgery.[12,13]

The pathogenesis of postoperative AF is multi-factorial and includes preoperative, perioperative, and postoperative factors such as gender, age, hypertension, DM, hyperlipidemia, chronic renal failure, ejection fraction, number of grafts performed, left atrial enlargement, acute atrial dilatation, atrial injury caused by cannulation, RCA stenosis, CPB run length, cross-clamping time, electrolyte imbalance, and pericarditis.[2,5,14-17] However, in this study, we found no association between these variables and postoperative AF development. Among these factors, age was documented to be the most reliable predictor for postoperative AF.[18] Gökşin et al.[13] reported that age was an independent risk factor for postoperative AF, whereas Polat et al.,[19] similar to our results, found no correlations between gender

Table 1. Baseline characteristics of the patients

Clinical or demographic variable	%	Mean±SD	Median	IQR
Age (year)		59.2±8.1		
Gender				
Male	88			
Hypertension	53			
Hypercholesterolaemia	12			
Diabetes mellitus	38			
Ejection fraction (%)		53.5±8.6		
Left atrial anterior-posterior diameter (>40 mm)	17.6			
Totally bypassed coronary artery count			3	2-3
Right coronary artery revascularization	62.2			
Preoperative troponin level			0.04	0-1.2
Postoperative troponin level			3.33	2.06-7.29
Cardiopulmonary bypass pump time (min)			95	77-123
Cross-clamp time (min)			60	45-84
Intensive care unit stay (day)			1	1-1
Total hospital stay (day)			7	7-9

SD: Standard deviation; IQR: Interquartile range.

Table 2. Demographic and clinical characteristics of the groups

Variable	AF group				SR group				
	%	Mean±SD	Median	IQR	%	Mean±SD	Median	IQR	*p*
Age (year)		62.1±9.5				58.4±7.6			0.114
Gender									
Male	86.6				88.1				0.876
Hypertension	66.6				49.1				0.295
Hypercholesterolemia	6.6				15.2				0.385
Diabetes mellitus	26.6				40.6				0.318
Ejection fraction (%)		51.7±9.6				54.0±8.3			0.352
Left atrial anterior-posterior diameter (>40 mm)	26.7				15.3				0.446
Totally bypassed coronary artery count			3	2-3			3	2-3	0.344
Right coronary artery revascularization	60.0				62.7				0.999
Preoperative troponin level			0.04	0-1.41			0.03	0-1.06	0.763
Postoperative troponin level			2.53	1.76-3.86			3.46	2.07-8.38	0.336
Cardiopulmonary bypass pump time			81	76-123			95	77-124	0.468
Cross-clamp time			57	45-85			61	45-84	0.861
Intensive care unit stay			1	1-1			1	1-1	0.186
Total hospital stay			7	7-9			7	7-9	0.929

AF: Atrial fibrillation; SR: Sinus rhythm; SD: Standard deviation; IQR: Interquartile range.

and age and the development of postoperative AF. Also, there are other studies showing no relationship between postoperative AF in patients undergoing CABG and gender, DM, hypertension, and hypercholesterolemia.[17,18] Furthermore, Leal et al.[14] found no association between postoperative AF and age, gender, ejection fraction, left atrial dimension, the number of grafts with the exception of CPB run length and cross-clamping time. Similarly, Koletsis et al.[1] and Sezai Hata et al.[2] did not found any relation between postoperative AF and CPB run length and cross-clamping time in patients undergoing CABG. In addition, there are several studies showing no significant association between the occurrence of postoperative AF and the number of bypassed vessels.[15,19]

Table 3. Logistic regression analysis for the prediction of atrial fibrillation occurrence

Variable	Odds ratio	95% CI	*p*
Age (year)	1.101	0.998-1.226	0.079
Gender			
Male	0.572	0.051-6.391	0.650
Hypertension	2.648	0.495-14.176	0.255
Hypercholesterolemia	0.2	0.015-2.611	0.220
Diabetes mellitus	0.411	0.087-1.952	0.264
Ejection fraction (%)	0.937	0.857-1.024	0.152
Left atrial anterior-posterior diameter (>40 mm)	1.805	0.447-7.286	0.407
Totally bypassed coronary artery count	0.484	0.133-1.768	0.272
Right coronary artery revascularization	0.965	0.295-3.153	0.953
Cardiopulmonary bypass pump time	0.968	0.909-1.032	0.320
Cross-clamp time	1.047	0.971-1.13	0.233
Preoperative troponin level	1.019	0.9-1.155	0.764
Postoperative troponin level	0.793	0.597-1.052	0.108

CI: Confidence interval.

Although, the effect of left atrial enlargement on the pathogenesis of AF is well-known, its association is under debate in patients undergoing CABG without a valvular disease. In our study, we assessed only left atrial anteroposterior diameter exceeding 40 mm due to the limitations of our database. However, Hernández-Romero et al.[20] calculated left atrial volume according to the ellipsoid model and indexed this calculation to body surface area and showed an association between the left atrial volume and AF after cardiac surgery. The authors included not only patients undergoing CABG, but also those with valvular heart diseases. Knayzer et al.[21] also analyzed isolated CABG patients and found that postoperative AF was significantly associated with the left atrial diameter above 40 mm. On the other hand, consistent with several studies, our study showed no correlation between postoperative AF and the left atrial diameter.[1,2,19]

Furthermore, the association between the atrial injury by cannulation with postoperative AF has been evaluated by some authors.[16] However, in our study population, none of the patients had such a type of injury. In addition, diseased atrioventricular and sinoatrial arteries were proposed as a cause of AF after CABG.[1,16] With the fact that these two branches are most commonly originating from the RCA, Koletsis et al.[1] reported that the patients with proximal stenosis of RCA showed an increased incidence of AF. In our study, we evaluated RCA revascularization whether it was associated with postoperative AF; however, we did not find an association. Similarly, Polat et al.[19] investigated the effect of RCA revascularization on the occurrence of postoperative AF; however, they found no association, either.

Many studies have shown that the patients with postoperative AF need an additional treatment and longer hospital stay.[15,20] However, we did not find an increased length of ICU and hospital stay in the AF group. This may be explained by the fact that is all patients in our study recovered early and without any AF complication.

Moreover, several structural myocardial proteins such as CK-MB, troponin T (TnT), high-sensitive troponin T (hsTnT), troponin I, and also metabolites such as cardiac lactate, N-terminal fragment of the brain natriuretic peptide (NT-pro-BNP), and C-reactive protein (CRP) have been used to show the relationship between them and postoperative AF occurrence.

A significant number of reports have shown the relation of cTnT and cTnI with short and long-term outcome following cardiac surgery.[22-24] Nesher et al.,[9] studied 1915 patients undergoing various types of cardiac operations and observed varying peaks of troponin T and its association with death, myocardial infarction, and low cardiac output syndrome. They found a cut-off value of 0.8 μg/L. Similarly, van Geene et al.[25] studied the cTnI after cardiac surgery and found a cut-off value of 4.25 μg/L to predict the hospital mortality.

Cardiac troponin values drawn at 18 to 24 hours after surgery, compared to the earlier time points, were found to have a stronger association with postoperative adverse outcomes;[26] therefore, we collected blood samples 24 hours after surgery.

Although we found no association between cTnI and postoperative AF and did not detect a cut-off value of cTnI which was able to distinguish the patients at high-risk for AF occurrence, Leal et al.[14] included 95 patients who exclusively underwent CABG showed that postoperative cTnI levels were associated with the AF occurrence after CABG. The authors also found a cut-off value of 0.901 ng/mL of cTnI after surgery, which was able to identify high-risk patients for postoperative AF. In addition, Hernández-Romero et al.[20] included 100 patients who underwent either CABG or valvular surgery and showed that preoperative hsTnT levels were independently predictive of patients developing AF, while postoperative hsTnT levels were not associated. In another study including 215 patients undergoing CABG, Gasparovic et al.[15] reported that preoperative and postoperative TnT values were significantly higher in patients who subsequently developed AF; however, they in the multivariate analysis, cTnT were not found to be statistically significant predictors of AF. Similarly, Koolen et al.[17] evaluated 3,148 patients undergoing elective CABG and reported that preoperative and postoperative cTnT was associated with postoperative AF. However, in the logistic regression analysis, cTnT was not found to be independently associated with postoperative AF without a cut-off value. In contrast to these studies, Knayzer et al.[21] included 156 patients and Sezai Hata et al.[2] included 234 patients of isolated CABG and found no association between cTnI levels and postoperative AF.

Furthermore, some authors evaluated the association of cardiac troponin with AF in another aspect, based on the fact that high level of cardiac troponins may be seen in patients with AF without coronary artery disease (CAD).[20] Bandorski et al.[27] evaluated diagnostic and clinical usefulness of cTnI for the diagnosis of CAD in patients with AF; however, they reported that AF patients with and without CAD showed similar cTnI

levels at admission. In addition, Hijazi et al.[28] and Parwani et al.[29] showed that cTnI was a biomarker of poor prognosis in AF patients.

On the other hand, there are several limitations to this study. First, our study sample was small. Second, we were unable to retrieve any information on any episode of AF occurred after discharge which would probably affect our results. Third, we were unable to assess the potential confounding variables including medications, fluid balance which may affect atrial dilatation, electrolyte imbalance, the amount of postsurgical chest tube drainage, postoperative heart valve disease, pericarditis, and postoperative renal failure.

In conclusion, these results are not able to confirm the usefulness of cardiac troponin I in the prediction of atrial fibrillation occurrence after coronary artery bypass grafting. Therefore, further large-scale and well-designed studies addressing to biomarkers to identify high-risk patients for the development of postoperative atrial fibrillation are required.

Declaration of conflicting interests

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