Institutional report - Cardiopulmonary bypass

Coronary graft patency after perioperative myocardial infarction: a study with multislice computed tomography

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Abstract

 A total of 55 consecutive patients who experienced perioperative myocardial infarction (MI) after coronary artery bypass grafting were studied using multislice computed tomography (MSCT) angiography to evaluate for graft patency. The MSCT detected acute graft occlusion in 23% grafts. Of the 55 patients, 40% patients had occluded grafts and perioperative MI in the area of the grafted vessels; remaining 60% had patent grafts with infarction in the area of the grafted vessels. Compared with the patients with patent grafts, those with occluded grafts had a higher blood sugar level. In addition, graft occlusion was higher in grafts with severe distal disease. Among the patients with patent grafts, luminal stenosis of the native vessels supplying the infarcted myocardium was higher than that in the native vessels supplying the non-infarcted myocardium. In conclusion, MSCT is feasible for the assessment of graft patency in the setting of perioperative MI. Graft occlusion is detected in less than half of the cases and usually occurs in the grafts with severe distal involvement and the patients with uncontrolled hyperglycemia. In patients with patent grafts, the severity of luminal stenosis of the native grafted vessel is the main predisposing factor for perioperative MI.
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1. Introduction

 Coronary artery bypass grafting (CABG) is the main revascularization modality in the treatment of the patients with multivessel coronary artery disease (CAD). However, CABG may be complicated by the perioperative myocardial infarction (MI). Perioperative MI, as defined by new Q waves on the postoperative electrocardiogram (ECG), occurs in 4–5% of patients, with a range among hospitals of 0–10% [1, 2]. Acute ischemic injury of the myocardium is primarily caused by the limitations of myocardial protection during the procedure.

 MI following CABG was associated with a significant increase in intensive care unit time, hospital length of stay, and overall costs, which contributed to greater hospital and physician service costs [3]. Predictive risk factors and the incidence of graft closure in this setting have not been clearly established. The aim of the present study, therefore, is to determine the incidence and the predictors of graft closure in the setting of perioperative MI following CABG using multislice computed tomography (MSCT).

2. Materials and methods

2.1. Study population

 Between March 2008 and June 2010, 55 consecutive patients who experienced perioperative MI within the first week after CABG were included in this study. The enrolled patients underwent 16-slice computed tomography (CT) angiography to evaluate for graft patency. The study protocol was approved by the institution's Ethics Committee and written informed consent was obtained from all patients. Exclusion criteria were: serum creatinine >1.5 mg/dl (>133 μmol/l), allergy to contrast material, hyperthyroidism, and inability to give informed consent.

2.2. Multidetector computed tomography angiography protocol

 Patients were scanned using a 16-section multidetector CT scanner (SOMATOM Sensation 16, Siemens, Forchheim, Germany). Patients were positioned in the gantry supine position and feet first, with electrocardiographic leads placed on the anterior thorax to enable a retrospectively gated scan. Scan parameters were 140 kV, 0.4-s rotation speed, 400 mA, and 10×0.75 detectors. Pitch, which was dependent on the heart rate, averaged 0.3. The CT system

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automatically recommended a pitch value to optimize the temporal resolution by the number of sectors reconstructed from each scan. Scans were performed in the caudal to cephalic direction, with a scan range from the thoracic inlet through the lung bases. The proximal subclavian arteries were also included. To familiarize the patient with the protocol, the examination, including breath-holding, was practiced in advance. β-Blockers (propranolol or esmolol) were injected intravenously for heart rates exceeding 70 beats/min, unless underlying contraindications, such as asthma were present. A non-ionic, iodinated, low-osmolar contrast medium was injected intravenously in doses ranging from 120 to 150 ml, without direct variation with respect to patient weight. In addition, 20 ml saline flush (saline chaser) was used to optimize the graft visualization.

2.3. Definitions

The perioperative MI is diagnosed based on the diagnostic criteria of creatinine kinase isoenzyme MB (CK-MB or CK) at least five times the upper limits of normal at <24 hours postoperatively and if >24 hours, one of the following: evolutionary ST segment elevation; new Q-waves in two or more contiguous leads or new left bundle branch block; or CK-MB (or CK) at least three times the upper limits of normal [4].

2.4. Data analysis

Variables are expressed as mean±standard deviation (S.D.) for the continuous variables and as absolute or relative frequencies for categorical variables. χ^2-test was used for categorical data and Fisher's exact test for cell count <5. Patient characteristics were compared by means of Student's *t*-test in case of continuous variables. Otherwise, a non-parametric test of Mann–Whitney *U*-test was used. A stepwise logistic regression analysis was used to determine the independent predictive factors for early graft patency. A two-tailed $P<0.05$ was considered statistically significant. The software SPSS version 15.0 (SPSS Inc, Chicago, IL, USA) was used for statistical analysis.

3. Results

The study population comprised of 55 consecutive patients with perioperative MI who completed the MSCT protocol. There were 40 males and 15 females with a mean age of 60±9 years. The majority of the patients (*n*=47) had three-vessel involvement in preoperative coronary angiography. A total of the 177 grafts, consisting of 111 venous grafts and 66 arterial grafts were evaluated, all of which could be assessed for patency and occlusion using MSCT. The MSCT detected acute graft occlusion in 41 (23%) grafts. Of the 55 patients, 22 (40%) patients had occluded grafts and perioperative MI in the territory of the grafted vessels; the remaining 33 (60%) had patent grafts with infarction in the territory of the grafted vessels. All patients had elevated cardiac biomarker (CK-MB and troponin-I) levels.

3.1. Patency of arterial grafts

Of the 66 arterial grafts, 60 (90%) grafts were anastomosed to the left anterior descending artery (LAD). The arterial grafts to LAD consisted of 52 left internal mammary artery (LIMA) grafts and eight radial artery (RA) graft. The remaining arterial grafts included four right internal mammary arteries (RIMA) to the right coronary artery (RCA) and two RAs to the left circumflex artery (LCX) grafts. Based on the MSCT, seven (11%) of the arterial grafts were classified as occluded and 59 (89%) were patent. Six of the 52 LIMA grafts to LAD and one of the four RIMA grafts to the RCA were found to be occluded.

3.2. Patency of venous grafts

Of the 111 venous grafts, 28 were anastomosed to the LAD (and diagonal branches), 48 to the LCX (and obtuse marginal branches), and 35 to the RCA (and posterior descending artery). Acute graft occlusion was detected in 34 (31%) venous grafts, including 10 grafts (36%) in the LAD territory, 15 grafts (31%) in the LCX territory, and nine grafts (26%) in the RCA territory.

3.3. Electrocardiographic characteristics

Of the 55 patients, evolutionary ST-segment elevation was observed in 17 (31%) patients in precordial leads V1–V6. Eight patients (14.5%) showed evolutionary ST-segment elevation in the inferior limb leads (II, III, aVF). New Q-waves in at least two inferior limb leads were detected in the four (7%) patients. In six (11%) patients, new Q-waves were observed in at least two adjacent precordial leads. No patients showed new left bundle branch block.

3.4. Risk factors of graft closure

The baseline clinical and demographical characteristics were similar between the patients with patent and occluded coronary grafts (Table 1). The only differences observed were related to the baseline fasting blood sugar (FBS) and quality of the target artery. Compared with the patients with patent grafts, those with occluded grafts had higher blood sugar level [135±67 vs. 117±23 mg/dl (7.5±3.7 vs. 6.5±1.3 mmol/l), $P=0.03$]. In addition, graft occlusion was higher in grafts with severe distal disease (100% vs. 48%, $P=0.003$).

3.5. Risk factors of perioperative MI in patients with patent grafts

Among the patients with patent grafts, luminal stenosis of the native vessels supplying the infarcted myocardium was higher than that in the native vessels supplying the non-infarcted myocardium (85±10% vs. 57±9%, $P=0.001$).

4. Discussion

The major findings of the present study are as follows: (1) most of the perioperative MIs occurring after CABG are not caused by the graft occlusion; (2) the severity of the luminal stenosis in the bypassed native vessels is the main factor responsible for the perioperative MI in the patients with patent grafts; (3) the severe distal disease and the hyperglycemia are the major predisposing factors for graft occlusion after CABG.

Table 1. Characteristics of the patients with patent and occluded grafts

Variable	Patent graft ($n=33$)	Occluded graft ($n=22$)	P-value
Age (mean±S.D.)	60±9	60±8.5	0.98
Male gender (%)	79	68	0.38
Smoker (%)	54	55	0.96
Left main artery disease (%)	17	19	0.79
Mean LVEF before surgery (%)	44	46	0.32
Graft no. (mean±S.D.)	3.1±0.7	3.3±0.7	0.36
CPB time (mean±S.D., min)	121±52	120±53	0.76
Fasting blood sugar [mean±S.D., mg/dl (mmol/l)]	117±23 (6.5±1.3)	135±67 (7.5±3.7)	0.03
Severe distal disease in target vessel (%)	48	100	0.003
Aspirin 325 mg immediately after surgery (%)	92	87	0.60
LIMA graft (%)	96	94	0.70

LVEF, left ventricular ejection fraction; CPB, cardiopulmonary bypass; LIMA, left internal mammary artery.

The present study demonstrates that MSCT consistently provides high-quality angiograms of bypass grafts that accurately delineate the presence of graft occlusions or stenoses. Five studies have thus far reported on the accuracy of MSCT to detect graft occlusions/stenoses. The foregoing studies, having evaluated 225 patients via MSCT, compared the results with those of conventional angiography and reported a sensitivity of 85.7–100% and specificity of 94–100% for MSCT [5–10]. A higher sensitivity (100%) and specificity (98–100%) was reported for bypass graft occlusion.

The present study also confirms the previous publications on graft patency rate in perioperative MIs associated with CABG. McKay et al. [8] found a 70% patency rate in 10 patients with perioperative MI using MSCT. In 18 patients with perioperative MI soon after CABG, Brindis et al. [11] reported a 78% graft patency rate. The present study with larger patient population similarly showed that 60% of the patients with perioperative MI had patent coronary grafts in the territory of the infarcted myocardium. Even, late (one year) angiographic study showed graft failure in 62% of patients with perioperative MI [12]. Taken together, these data indicated that and mechanisms other than graft failure accounted for a substantial proportion of perioperative MIs following CABG.

The present study also demonstrated that the severity of the coronary obstruction in the grafted native vessel was the main determinant of perioperative MI in the patients with patent grafts. Similar results have been reported by Brindis et al. and Yau et al. [11, 12]. These findings suggest that acute ischemic injury of the myocardium is primarily caused by inadequate myocardial protection during the procedure. Because coronary blood flow is absent during aortic cross-clamping for surgery utilizing CABG, protection against the perioperative MI depends directly on the ability to reduce myocardial oxygen requirements to a negligible level.

Although the graft occlusion is responsible for the perioperative MI in a small group of the patients, this complication is more common in the grafts with severe distal disease and the patents with uncontrolled hyperglycemia. The association of hyperglycemia with early graft occlusion may be related to a hypercoagulable state observed in poorly controlled hyperglycemia. Boden et al. [13] demonstrated that acute normalization of hyperglycemia with insulin resulted in significant reduction of the tissue factor procoagulant activity. Oysel et al. [14] provided important evidences that support the hypothesis that coagulation influences perioperative ischemia. In latter study, the patients with perioperative MI had a lower serum levels of protein C (101.2±26% vs. 124.7±31.4%, $P<0.05$) and tissue plasminogen activator (322±580 vs. 2307±2830 IU/ml, $P<0.01$). In our previous study, the patients with patent coronary grafts had lower serum FBS level (6.6±1.6 vs. 7.8±3.6 mmol/l, $P=0.02$) and longer partial thromboplastin time (PTT) (34±11 vs. 30±2 s, $P=0.04$) [15]. These findings again highlighted the importance of technical factors, good myocardial protection, and good metabolic preparation in reducing the incidence of perioperative MI occurring early after CABG.

5. Conclusions

MSCT is feasible for the assessment of graft patency in the setting of perioperative MI associated with CABG. The causes of perioperative MI in CABG patients are almost certainly multifactorial, although not well understood. Graft occlusion is detected in less than half of the cases and usually occurs in the grafts with severe distal involvement and the patients with uncontrolled hyperglycemia. In patients with patent grafts, the severity of the luminal stenosis of the native grafted vessel is the main predisposing factor.

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eComment: Perioperative myocardial infarction following coronary artery bypass grafting

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This well-designed study [1] concerning perioperative myocardial infarction (MI) and graft patency, offers us the opportunity to add some data regarding this challenging complication. Coronary artery bypass grafting (CABG) is of considerable benefit for those in need of revascularization; however, it may be associated with significant perioperative and postoperative myocardial damage and necrosis, which may occur in varying degrees. Multiple mechanisms have been proposed to explain myocardial injury after CABG. Intraoperative injury may result from cardiac manipulation, inadequate myocardial protection, and intraoperative defibrillation, while postoperative myocardial injury may be associated with acute loss of bypass grafts [2]. The incidence of perioperative MI varies considerably, from 3% to 30%, because of different diagnostic criteria and variable patient populations [3]. However, troponin values more than five times the 99th percentile of the normal reference range during the first 72 hours following CABG, when associated with the appearance of new pathological Q-waves or new left bundle-branch block (LBBB), or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium, should be considered as diagnostic of a CABG related MI [4].

The PREVENT IV study identified such intraoperative risk factors for perioperative MI as prolonged cardiopulmonary bypass or aortic cross-clamp times, perioperative myocardial ischemia, and inadequate revascularization. Moreover, patients with perioperative MI also had longer surgery durations, although it was unclear whether this longer duration was a cause of or caused by perioperative myocardial ischemia. Other well-established risk factors for perioperative MI included age, left main coronary artery disease and three-vessel disease, impaired left ventricular function, unstable angina, recent MI, and emergent operations [3]. According to the PREVENT IV study, both 30-day and two-year clinical outcomes were worse in patients suffering perioperative MI, as they had longer postsurgery ventilation times and intensive care unit and hospital stays. Although rates of angiographic vein graft failure were higher in patients with a perioperative MI, one-third of patients with perioperative MI had patent vein grafts at one-year, which suggests that a substantial portion of perioperative MI was not caused by early vein graft failure and that global or regional myocardial ischemia, possibly related to CABG or worse coronary anatomy, may have an important role [3].

Finally, reperfusion injury occurring with restoration of blood flow to ischemic tissue is associated with myocardial cell death and apoptosis, and microvascular injury. Three to 20% of patients experience MI associated with reperfusion injury after CABG [5]. Adenosine, being a powerful inducer of ischemic preconditioning, has been shown to improve postischemic ventricular function, reduce neutrophil accumulation/activation and reduce further myocardial necrosis. Mangano et al. [5], reported that treatment with acadesine, an adenosine regulating agent, significantly reduced mortality by 4.3-fold, with the principal benefit occurring over the first 30 days after MI. Acadesine is the first therapy proven to reduce the severity of acute postreperfusion MI, substantially reducing the risk of dying over the two years after infarction.

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