Heart Sci J 2023; 4(1): 35-39

Contents list available at www.heartscience.ub.ac.id

Heart Science Journal

Journal Homepage : www.heartscience.ub.ac.id

Case Report

Perioperative myocardial infarction after coronary artery bypass grafting: How to Identify?

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ARTICLE INFO

Keywords:
Perioperative Myocardial Infarction;
Coronary artery bypass surgery (CABG).

ABSTRACT

Background: Perioperative myocardial infarction (PMI) associated with the surgical revascularization, Coronary artery bypass surgery (CABG) occurs in about 3–5% of patients. Myocardial necrosis and ischaemia after CABG are caused by direct cardiac trauma from manipulation, reperfusion injury, incomplete revascularization, hypotension, bleeding, ventricular arrhythmia, acute graft closure, inadequate perioperative myocardial protection and others.
Case Illustration: The introduced case report explains the rupture of right ventricle result in periprocedural myocardial infarction following the surgical myocardial revascularization. 62-year-old man has undergone the coronary bypass surgery with arterial graft of left mammary artery (LIMA) to left anterior descending artery (LAD) and savenous graft to left circumflex coronary artery (LCx). Early in the post-surgery period, a perioperative myocardial infarction (PMI) developed, with laboratory correlation of cardio-specific enzymes elevation and ECG changes in terms of ischaemia in the diaphragmatic region. Echocardiography showed akinesia of the apex, apical septal and apical inferior segments accompanied by the decrease in ejection fraction (EF) of the left ventricle.
Conclusion: Early detection of PMI may therefore, prompt institution of therapeutic measures to relieve the ischaemia and decrease the incidence and the size of PMI

1. Introduction

Coronary Artery Bypass Graft (CABG) is a surgery used to manage diffuse coronary artery disease, and the indications for this surgery have recently improved. Due to the existence of an enhance various for avoiding myocardial damage, CABG remains a challenging and high-risk procedure. Perioperative PMI, also known as type-5 MI, is the most prevalent cause of postoperative morbidity and death in CABG.[1,2] It is also the primary cause of prolonged ICU and hospitalization. As there are no diagnostic gold standards and a variety of diagnostic criteria are utilized, the prevalence of PMI ranges from 3% to 30%.[3] There are difficulties in the interpretation and diagnosis of post-operative PMI; as a result, the accurate and sensitive measurement of ischemia, such as a tenfold rise in cardiac troponin in conjunction with coronary angiography, echocardiography, and ECG shifts (e.g., new Q wave or LBBB), have become significantly relevant. ECG shifts can be determined using postoperative troponin concentration.[4]

CABG can lead to myocardial damage in up to a quarter of patients. The patients and the sources of the injury varied considerably. In the postoperative setting, cardiac enzyme tests, echocardiography, and electrocardiography are commonly performed to screen for these acute ischemia episodes changes, as well as to separate them from planned procedure-related occurrences. It's not clear where normal postoperative alterations end and clinically important instances begin. Myocardial ischemia and necrosis can sometimes occur following CABG as a consequence cardiovascular injury from manipulation, reperfusion injury, incomplete revascularization, hypotension, bleeding, ventricular arrhythmias, acute graft closure, inadequate myocardial protection, and cardioplegia.[5,6] The majority of studies have shown a similar mortality risk associated with post-CABG enzyme elevations as seen after PCI. The Guard During Ischemia Against Necrosis (GUARDIAN) study in 2332 CABG patients found that large CK elevations (those above 10 ULN) were associated with the highest mortality risk at 6-month follow-up. Another registry of 3667 CABG patients with 5-year follow-up found a graded reduction in survival associated with post-CABG CK-MB ((CK-MB ULN: 80 percent ; CK-MB 1 to 3 ULN: 78. Every unit increase in CK-MB was linked with an increased risk of death (odds ratio; 95 percent CI, 1.04 [1.009 to 1.062]; P=0.0007)). The condition is outlined in the case study, which provides detail about a patient who had perioperative myocardial infarction during CABG.[7]

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https://doi.org/10.21776/ub.hsj.2023.004.01.6
Received 9 October 2022; Received in revised form 30 November 2023; Accepted 15 December 2022
Available online 1 January 2023

2. Case Illustration

A 61-year-old male patient diagnosed with coronary artery disease (CAD) scheduled for CABG. The patient had a history of chest pain and dyspnea on exertion (DOE) of New York heart association classification II (NYHA II). He complained about chest pain, stabbing like sensation, < 5 minutes while doing moderate activity and relieved by rest since 2017. He suffered from DOE while doing moderate activities, that relieved by rest since December 2018. He had history of hypertension since 5 years ago. He had history of myocardial infarction at October 2018, coronary angiography revealed three vessel coronary disease with Chronic Total Occlusion (CTO) in Left Main Artery, LAD, and LCx. Due to the nature of coronary artery disease the patient was indicated for surgical revascularization (CABG).

General physical examination revealed a pulse rate of 68/min with a blood pressure of 122/74 mmHg in sitting position. His respiratory rate was 18/min and systemic oxygen saturation was 98% on room air. No murmur was heard on auscultation. The patient's initial ECG showed Sinus rhythm ,normoaxis, with Biphasic T wave at V2-V5. Echocardiography showed preserved left ventricular systolic function (LVEF 52%), with regional wall motion abnormalities at anterior and anteroseptal at mid level, and no haemodynamically significant valvular disease. Before CABG was performed patient was cardiopulmonary compensated, normotensive, with cardiospecific enzymes within the normal range. Preoperative hemoglobin level was 12.6 g/dL.Preoperative Chest X-ray and blood investigations were found to be normal.

On 16-Feb 2021 coronary artery bypass graft surgery was performed (LIMA to LAD, saphenous venous graft to LCX) under cardiopulmonary bypass (CPB). Due to non significant lesion on RCA, grafting of the RCA was not performed. In the operating room (OR) cannulation of the right radial artery, and the right internal jugular vein was accomplished under local anesthesia. Anesthesia was induced with intravenous bolus of midazolam, fentanyl, relaxation was achieved with pancuronium bromide, and anesthesia was maintained with isoflurane and supplemental intravenous pancuronium. After that transducer (6VT) transesophageal probe was inserted orally. A Transthoracic Echocardiogram (TEE) was performed using a Philips Afinityechocardiography system. The RV was normal in structure and function. There were no wall motion abnormalities of the RV free wall and no tricuspid regurgitation, reflecting essentially normal RV function. There was no thrombus or mass in the LV, RV, left atrium, or left atrial appendage.

Surgery proceeded through a median sternotomy incision and included dissection of the left internal mammary artery and endoscopic harvest of the great saphenous vein (SV) from the left leg. Heparin (15,000 IU) was administered to achieve a target activated coagulation time (ACT) of > 400 seconds. Coronary artery grafting was initiated after the ACT had exceeded 400 seconds (first postheparin ACT was 424 seconds). The aorta and right atrium were cannulated and the patient was cooled to 31°C. The initial of antegradecardioplegia wasn't achieved because of aortic regurgitation. Patient got ventricular fibrillation during operation.

After reinsertion of cardioplegia drugs, arrest was achieved with antegradecardioplaegia. The first anastomosis was SVG that grafted to the distal of LCx. There was no complication after anastomosis. The 2nd anastomosis was LIMA to distal LAD, during 2nd anastomosis, it was noted that there was increasing bleeding around the LV apex. Closer examination revealed a 0.2- to 0.5-cm tear on the apical surface of the RV. This tear was a small defect in a low-pressure. Repair was performed using pericardial patch. The 2nd graft was continued after RV repair. Haemodynamic stability was maintained with intravenous norepinephrine infusion (0.01-0.1 μg/kg/min) and intravenous fluid therapy with lactated Ringer's solution. A total of 3 L of crystalloid solution, 500 mL of autologous salvaged blood, and 350 mL of homologous packed red cells were administered in the operating room. The total estimated blood loss was 0.8 L.

The patient required ventilatory support in the intensive care unit for 12 days. On admission to the cardiac intensive care unit, the heart rate increased to 115 bpm, blood pressure (BP) varied in a wide range with systolic BP from 82 to 105 mmHg, and serum lactate was 2.8 mmol/L. A quick cardiac ultrasound scan showed a severely reduced LVEF of around 30% and a significant mitral regurgitation, which had been noticed on postoperative echocardiography. The intra-aortic balloon pump (IABP) was planned for inserted, dobutamine was increased from 5 to 10 mcg/kg/min, norepinephrine was added at a dose of 0.025 mcg/kgbw/min. The clinical and hemodynamic picture continued to deteriorate. Of note, significant mid and apical hypokinesia and nearly normal basal contraction were observed. The management plan would be fluid expansion, inotrope uptitration, and cautious vasopressor uptitration, among other adjustments. Since the left ventricular systolic function was poor, and the mitral regurgitation was severe, pulmonary edema might be engendered, which contradicted the above mentioned approach. Lung ultrasound was performed, showing an A-profile ruling out significant pulmonary congestion. Subsequently, 500 ml normal saline was given over 45 minutes, dobutamine was continued, and norepinephrine was increased to 0.1 mcg/kg/min. All these interventions were carried out under close hemodynamic monitoring, echocardiography, and lung ultrasound.

After fluid administered, general physical examination revealed a pulse rate of 108/min, a blood pressure of 92/64 mmHg with dobutamin 8 mcg/kgbw/min and NE 0.1 mcg/kgbw/min. His respiratory rate was 20/min and systemic oxygen saturation was 98% on ventilator PCAC setting. Neither murmur nor rales was heard on auscultation. A subtle ST segment elevations (up to 4 mm) in leads V2-V5 were present on the ECG, one hour after CABG. Echocardiography examination was subsequently performed with the finding of left ventricle EF decreased to 32%, with new akinesia of apex, apical septal and apical inferior segments. The peak values of cardiospecific enzymes were registered on the first postoperative day (cTnT 0.8 7.6 17,4, CK 1134 mg/dl, CK-MB 149 mg/dl). Haemoglobin value on the first postoperative day was 8.6 g/dL. During the bed-side ECG monitoring, sinus rhythm at a frequency of 102BPM was registered, no significant arrhythmias were detected. The follow-up ECG documented the development of Q wave in leads V2-V4, and significantly ST elevation in leads V2-V4. The condition was evaluated as perioperative myocardial infarction (ESC type 5).Considering of this condition, He got loading ASA 160mg and heparin infusion was initiated. He was planned for an emergency surgery ahead of diagnostic modality such as angiography.

On postoperative day 2, he had presented with breath sounds were greatly diminished on left side of the chest. A postero-anterior chest X-Ray was done that showed large left sided pleural effusion. The production of this drain post-operative was diminished (120 cc/24 hours). Postoperative day 3 he developed a Ventilator Associated Pneumonia and failed to weaning from ventilator support. This was treated with intravenous antibiotics. The patency of chest drain was evaluated, Pleural drain was re-inserted. After that, its production was increase (420cc/ 24 hours). Patient developed Post operative Atrial Fibrillation (POAF), with HR 160-170x/minute. The management plan would be fluid expansion, amiodarone 150mg, continued with maintenance dose and lidocaine. This patient required ventilatory support in the intensive care unit for 13 days. On post-extubation he developed delirium, likely due to selective serotonin reuptake inhibitors withdraw

al, which improved after reintroduction of her medications. The patient was discharged on the 16th postoperative day.

3. Discusion

Perioperative MIs occur in more than 6% of the over 200,000 patients who undergo CABG surgery annually.[8] Despite this relatively high frequency, their clinical significance is not clear. Studies are approximately evenly divided into those showing an impact on morbidity and mortality and those showing no impact. There are many possible reasons for these disparate results, including the use of suboptimal diagnostic criteria for perioperative MI in some studies and inadequate numbers of patients in others.[9] However, the major methodological problem with most of the studies is the statistical analyses used. Most of the studies have simply used survival analyses to determine the clinical significance of perioperative MI. This approach does not control for the differences in baseline (preoperative) patient characteristics that may affect the survival rate in those who do and those who do not develop a perioperative MI.[10]

CABG-related MI is characterized randomly as an increase in cTn levels over the 99th percentile URL in patients with normal baseline cTn values. In populations with a high pre-procedure cTn who have steady (20% fluctuation) or decreasing cTn levels, the post-procedure cTn must rise by more than 20%. The absolute post-procedural value, however, must be greater than ten times the 99th percentile URL. Furthermore, one of the following components is required: a, Angiographic recorded new graft occlusion or new native coronary artery occlusion; imaging evidence of new loss of viable myocardium or new regional wall motion abnormalities in a pattern associated with an ischemic aetiology.[11]

In this case, the diagnosis of MI type-5 was fulfilled criteria with development new pathological q wave at anterior segment, increase of cTn> 10 times the 99th percentile URL, and new wall motion abnormality according to TTE and TEE. The diagnosis of MI, remains a clinical challenge since patients are unable to express classic clinical symptoms. Early diagnosis and re-intervention have to be made rapidly to ventricular reserve function and improve the patient's outcome after CABG surgery. Routine ECG combined with cardiac biomarker is very useful in diagnosing MI post CABG. The universal definition of MI recommended cardiac troponins as MI marker in the early postoperative period after CABG surgery and was superior in sensitivity and specificity compared to creatinine kinase myocardial band (CKMB).[4] Surgical trauma and the usage of a cardiopulmonary bypass (CPB) machine could cause ECG changes and elevated biochemical markers, which make those non-invasive markers have less diagnostic value to diagnose MI in post CABG patients. Transthoracic and transesophageal echocardiography were also not stated as the recommended tool due to its lack of detailed information about coronary arteries.[6]

Patients who develop perioperative MI have been reported to have more extensive coronary artery disease worse angina, more extensive surgical procedures, longer bypass times or ischemic (cross-clamp) times, fewer coronary artery collaterals, greater incidence of previous MI, and worse left ventricular function as evidenced by cardiomegaly on the chest radiogram.[12] Our patient with perioperative MI also differed from those without in two important preoperative characteristics: They were older and longer bypass or ischemic time due to RV rupture during surgery.

There have been few research on identifying and treating patients with early MI after CABG surgery. Despite MI has been the most frequent cause of early graft failure after CABG, percutaneous coronary angiography (PTCA) proved helpful in determining graft patency. Although percutaneous coronary intervention (PCI) is recommended in cases of anastomosis with stable hemodynamics, redo CABG is more beneficial in saving infarcted myocardium in patients with unstable hemodynamics. Redo surgery is also more effective in discovering additional issues linked with the anastomotic location.[13]

Emily et al, 2021 found that study of 114,871 patients performed isolated CABG surgery, 57% underwent TEE, Its study confirmed that TEE was associated with lower 30-day mortality (3.7% vs 4.9%, $p<0.0001$) and lower incidence of stroke (4.5% vs 5.6%, $p<0.0001$).(14) When performing revascularization surgery, the use of intraoperative TEE must be investigated in order to determine the preliminary diagnosis, effectively detect undiagnosed disease, manage both anesthetic and insertion of cardioplegic correctly, and assess surgical outcomes. In addition, it is appropriate to consider using TEE to evaluate RWMA, ventricular function hemodynamic status and valvular function in patients having CABG surgery.[15]

Some of the causes of new RWMAs include ventricular loading, electrolyte abnormalities, blood viscosity, air embolism, degree of inotropic support, hypothermia, and bundle branch conduction abnormalities. Perioperatively, it is difficult to distinguish between insufficient revascularization, continuing ischemia, and shocked myocardium. Improve coronary perfusion pressure, normalize electrolytes and arterial blood gas, and check graft patency in new-onset RWMA. In severe cases, CABG may need to be redone. On-pump treatments are associated with lower mortality and morbidity, according to Savage et al. Intraoperative TEE should be utilized frequently in cardiac surgeries. Based on a second research, intraoperative TEE in CABG can improve volume replacement treatment by 47%. Intraoperative TEE also aided in the administration of inotropes, vasodilators, and volume replacement.[16]

After surgical coronary revascularization, patients who exhibited poorer regional wall motion had a twofold higher risk of mortality or MI, as well as the need for additional revascularization, during the first two years after the procedure was performed. While there is a dynamic nature to wall motion immediately following CPB, individuals who have worsening in wall motion as seen by echocardiography must be regarded to be at greater risk for having later catastrophic cardiac events.[17]

Studies have shown that left ventricular diastolic failure is an immediate, more sensitive marker of myocardial ischemia, and persists longer than systolic dysfunction. According to McKenney et al., disrupted intracellular calcium homeostasis or both can cause diastolic dysfunction following CPB. 2D echocardiography helps us determine the specific coronary artery involved before surgery. According to Smith et al., RWMAs develop sooner and are a more sensitive indication of myocardial ischemia than aberrant ECG alterations. In our situation, TEE RWMA preceded ECG alterations. However, artifacts and inter-observer differences may affect the accuracy of RWMA analysis by echocardiography.[18] RWMA does not distinguish stunned or hibernating myocardial from acute ischemia. Advanced echocardiographic methods, such as tissue Doppler, strain, and strain rate, aid in the identification of myocardial ischemia. TDI or speckle-tracking echocardiography can detect myocardial strain (STE). STE is a non-Doppler, generally image method for evaluating myocardial deformation and LV systolic and diastolic motions.[19]

It also gives a specific and repeatable assessment of LV contractility. Ischemic myocardium has a higher strain value than normal myocardium due to longitudinal shortening.

STE also diminishes subjective and pacing variations. For us, strain analysis indicated the exact site of cardiac involvement. Perioperative strain analysis is thus critical in evaluating regional wall mobility.[15]

Right ventricular rupture was also linked to myocardial infarction, cardiac catheterization, CPR, and surgery. One incidence of RV rupture was documented during surgical dissection to find the LAD. Barnard and coworkers observed RV rupture 12 hours after CABG due to myocardial fatty infiltration. To ensure sufficient heart exposure and immobilization during off-pump cardiac surgery, many tools are used.[20] Under mechanical stress, cardiac tissue may rupture for many reasons. RV rupture has been linked to MI, infection, and steroid usage. This patient had none of these variables. Apical epicardium traction was likely caused by excessive epicardial fatty tissue, which in turn caused over-traction on the apical epicardium. The thin-walled RV may have been more vulnerable to epicardial traction than the LV.[21]

During follow up , this patient got pneumonia at day care of three. Patients undergoing CABG are highly susceptible to pneumonia that 8.9-fold increased risk of mortality and 4.2-fold increased risk of length of stay. Common risk factors among cardiac surgery patients, such as COPD, heart failure, and advanced age, result in a higher risk profile for pneumonia. Moreover, cardiopulmonary bypass, with its effect on systemic inflammatory mediators and its potential for lung injury, may further contribute to the risk of developing pneumonia. Significant fluid shifts in the perioperative setting often leading to pulmonary edema, combined with the frequent need for transfusion of blood products may affect pneumonia risk. Any prolonged use of mechanical ventilation places patients at increased risk for pneumonia.This patient has several risk factor for developing post-operative pneumonia, such as advanced age, blood transfusion, prolong mechanical ventilation and prolong of CPB duration due to mechanical complication during procedure.[22,23]

After cardiac surgery, age > 60 is a risk factor for postoperative pneumonia. In 2017, a retrospective research in Japan revealed that 9.8% of 123 senior patients (>60 years old) who underwent heart surgery had post operative pneumonia. According to earlier research, blood transfusion was the second independent risk factor for pneumonia after heart surgery. Transfusions can temporarily weaken the immune system, increasing infection risk. It has been linked to severe sternal wound infections.[23] Allogeneic blood transfusion's immunomodulatory impact is still unknown. Allogenic white blood cells are thought to be involved in the immunological suppression and clonal deletion induced by foreign antigens infusion. Another worry is macrophage dysfunction. After transfusion, macrophages lose chemotactic migratory capacity and generate more prostaglandin E2, reducing antigen-presenting cell activity and interleukin generation.[22]

There is a link among transfused red blood cell storage time and postoperative pneumonia, which occurs less frequently in patients who get fresh red blood cells. Immunosuppressive chemicals from white blood cell granules leak into red blood cell components, causing transfusion-induced immunomodulation. Furthermore, reduced levels of 2,3-diphosphoglycerate and decreased deformability of stored red blood cells may impede oxygen transport to tissues.[23]

As a result of systemic inflammation and ischemia reperfusion damage, CPB reduces pulmonary compliance and increases the risk of atelectasis. Longer CPB was found to be an independent risk factor for postoperative pneumonia in our study, as predicted. The risk of postoperative pneumonia rose 2.98-fold when intraoperative red blood cell transfusion was combined with a CPB duration >60 minutes, according to Allou and colleagues. On average, individuals who received CPB for more than 100 minutes had a 1.71-fold higher risk of pneumonia. Improved clinical results have been linked to the use of minimally invasive CPB.[24]

Previous research suggests that extended weaning may alter the natural respiratory barrier, raising the risk of infection and death. As a result, individuals who have had heart surgery are more likely to suffer Ventilator-associated pneumonia (VAP), as our study found. A favourable prognosis is typically due to early antibiotic therapy of VAP. [25] Previous research has shown that early antibiotic treatment can minimize the need for mechanical breathing and reduce death in individuals with VAP. To prevent VAP in patients who have undergone heart surgery, we propose third- or fourth-generation cephalosporins as the recommended antimicrobial regimen (eg, cefoperazone, ceftazidime, or cefepime). Other pathogen-fighting antibiotics are available. Despite high expense, increased toxicity, and probable drug resistance, combination treatment was formerly thought to improve microbiological and clinical results.[23]

4. Conslusions

This case emphasizes the need for constant and vigilant monitoring of hemodynamics, blood loss, and the surgical field for possible complications during CABG such as RV rupture that can cause PMI. Early detection of PMI may therefore, prompt institution of therapeutic measures to relieve the ischaemia and decrease the incidence and the size of PMI.

5. Declarations

5.1. Ethics Approval and Consent to participate
This study was approved by local Institutional Review Board, and all participants have provided written informed consent prior to involvement in the study.

5.2. Consent for publication
Not applicable.

5.3. Availability of data and materials
Data used in our study were presented in the main text.

5.4. Competing interests
Not applicable.

5.5. Funding source
Not applicable.

5.6. Authors contributions
Idea/concept: DIS, SA. Design: DIS, SA. Control/supervision: SA, AR. Literature search: DIS, SA. Data extraction: DIS, SA. Statistical analysis: KAN SW. Results interpretation: SA, AR. Critical review/discussion: SA, AR. Writing the article: DIS, SA. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

6.7. Acknowledgements
We thank to Brawijaya Cardiovascular Research Center.

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