

Perioperative management of a patient with left ventricular dysfunction and anomalous coronary arteries

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Abstract

Anesthetic management of patients with coronary artery disease undergoing noncardiac surgery is quite challenging. Such patients are at increased risk of perioperative cardiac complications and death. We report an illustrative case of a 62-year-old male with ischemic heart disease and anomalous coronary arteries for obstructed paraumbilical hernia repair.

Key words: Anomalous coronary arteries, coronary artery disease, obstructed paraumbilical hernia repair, perioperative cardiac morbidity

Introduction

Patients with coronary artery disease (CAD) or multiple risk factors for CAD are at a higher risk of perioperative myocardial infarction and mortality.^[1]

An association between anomalous origin of coronary arteries and fatal cardiac events has been reported.^[2] The proposed mechanism is stress induced mechanical compression of anomalous coronary arteries between great vessels.^[2]

We report a case of an elderly man with double vessel disease, poor left ventricular (LV) function and concomitant anomalous origin of left anterior descending (LAD) artery from the right coronary artery (RCA) for repair of irreducible, obstructed paraumbilical hernia.

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Case Report

A 62-year-old, 180 cm, 90 kg male with a history of unstable angina, old inferior wall myocardial infarction and irreducible, obstructed paraumbilical hernia presented for resection of gangrenous loop of small bowel with anatomical repair of hernia. He also had long-standing diabetes, hypertension and was on multiple medications such as regular insulin, aspirin 75 mg, clopidogrel 75 mg, metoprolol 25 mg, atorvastatin 20 mg daily and nitrocontin 2.6 mg twice daily.

On examination, he was obese with poor effort tolerance. There was no pallor or edema feet and no signs of heart failure. His resting heart rate (HR) was 70/min and blood pressure measured 130/80 mmHg.

Most of his preoperative blood investigations including coagulation profile were within normal limits except random blood sugar which was 164 mg/dl. Electrocardiography (ECG) showed Q waves in leads II, III and aVF and ST segment depression in leads V₁-V₆. The preoperative chest X-ray was normal. Two-dimensional echo done on admission revealed akinetic inferior and mid-posterior wall; hypokinetic basal, posterior wall, mid and distal interventricular septum; mildly dilated LV and left ventricular ejection fraction (LVEF) <25%.

Two months ago, a detailed cardiac work up of the patient had been done wherein a coronary angiogram revealed anomalous origin of the LAD from RCA and absent left main coronary artery (Figure 1). Thallium scan also showed



Figure 1: Coronary angiography: Total occlusion of right coronary artery (RCA) and left circumflex artery. Left anterior descending artery originating from RCA

LVEF of 25% at rest tomography angiography showed a common ostium for RCA and LAD (Figure 2).

Chronic total occlusion of RCA (after giving off LAD) as well as a short, narrowed out left circumflex (LCx) artery resulted in complete loss of viable myocardium in their corresponding territories. Thus, the myocardium had its blood supply from LAD and partly from LCx artery.

The patient was on medical management as coronary revascularization could not be considered owing to the semi-emergent nature of surgery and that most of the myocardium was already compromised. The patient's relatives were initially reluctant to give their consent for the procedure, thereby causing delay in surgical intervention. Until then, the patient was managed conservatively on continuous Ryle's tube aspiration, intravenous fluids and antibiotics.

All the prescribed medications were continued preoperatively except aspirin and clopidogrel, which were stopped at admission. Low molecular weight heparin (enoxaparin 60 mg subcutaneously twice daily) was started. It was discontinued 24 hours prior to surgery. The patient was hemodynamically stable and co-operative. Therefore, sequential epidural combined with local infiltration of the surgical site with 0.25% bupivacaine was selected as the anesthetic technique. Central venous pressure (CVP), arterial blood pressure (ABP), blood gas analysis and continuous cardiac output (CO) monitoring (Edwards Life sciences FloTrac monitor) were used along with routine monitors. The baseline ABP, HR, CVP, CO were 122/72 mmHg, 90/min, 7 mmHg and 7 L/min respectively. An epidural catheter was inserted in T₁₀-T₁₁ interspace with the patient in sitting position. This provided sufficient analgesia and motor blockade for surgery. The surgical site was also infiltrated with 10 ml of 2% lignocaine. The patient was on spontaneous ventilation

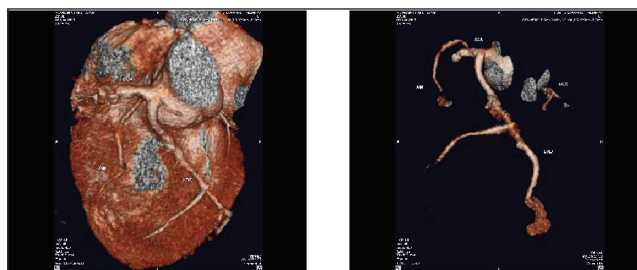


Figure 2: Computed tomography coronary angiography: Common ostium for right coronary artery (RCA) and left anterior descending artery; absent left main artery. Left circumflex artery short in length and significantly narrow in caliber; completely blocked RCA in proximal segment due to dense calcification

with titrated mild sedation with fentanyl and midazolam. Total fluid administered was 1.5 L (1 L of normal saline; 500 ml of colloid). It was guided by CVP, stroke volume (SV), stroke volume variation (SVV) and CO on the Edwards Sciences FloTrac Vigileo monitor. The patient remained hemodynamically stable until about 50 min of the start of surgery when ABP was 86/54 (65mmHg) and CO was 3 L/min probably due to last epidural bolus of bupivacaine. This was not accompanied by any ECG changes. CVP remained normal. Blood loss was minimal. Inotropic support (nor-adrenaline 0.07 µg/kg/min) was then started. Thereafter, ABP stabilized and serial ABC's done during surgery were normal. The surgical procedure included resection of gangrenous segment of bowel, anastomosis and anatomical repair of hernia, which lasted approximately 2 h.

Postoperatively, 12 lead ECG was done, which showed no fresh changes. Trop-T done after 12 h of surgery was also negative. Intensive monitoring (ABP, CVP, ECG, and CO) was continued for 48 h postoperatively. The inotropic support was tapered and discontinued after 36 h of surgery to maintain a mean ABP of 75-80 mm Hg. Analgesia was maintained with epidural infusion of 0.125% bupivacaine with 100 mcg fentanyl in 50 ml of normal saline titrated to effect and hemodynamics, intravenous analgesics (diclofenac 75 mg 8 hourly and tramadol whenever required). All preoperative medications including enoxaparin (60 mg sc twice daily for 2 days) and Aspirin were restarted on the 1st postoperative day (POD) and review done by a cardiologist and an endocrinologist. Epidural catheter was removed on the 2nd POD after which clopidogrel was resumed. The course of stay in the surgical intensive care unit (48 h) and later in the ward was uneventful. The patient was discharged on the 5th POD.

Discussion

Coronary artery anomaly refers to a wide range of congenital abnormalities involving origin, course and structure of

epicardial coronary arteries. It is an incidental finding during 1.3% of conventional cardiac catheterizations.^[3]

In our patient, LAD arose from RCA along with double vessel disease (complete block in RCA and thinned out LCx) and poor LV function.

Patients with CAD undergoing intermediate to high-risk noncardiac surgery are at increased risk of perioperative cardiac complications and death. Most of them presenting for emergency noncardiac surgery cannot undergo prophylactic cardiac catheterization or coronary revascularization. Therefore, perioperative intensification of medical treatment should be more widely considered.^[4]

Care of these patients focuses on a thorough preoperative evaluation; stratifying risks; optimizing medical treatment; intensive monitoring and choosing the appropriate anesthetic technique and drugs in the perioperative period. As per the revised American College of Cardiology/American Heart Association Guidelines^[5] for cardiac patients undergoing noncardiac surgery, our patient was a high cardiac risk scheduled for a high-risk surgery.

The anesthetic goal in these patients should be to minimize hemodynamic instability and interference with myocardial oxygen supply. Hypotension and volume overload may precipitate myocardial ischemia owing to poor cardiac reserve.

A meta-analysis by Beattie *et al.*^[6] reports a reduction in PMI with thoracic epidural analgesia (TEA), which attenuates neuro-humoral stress response through adequate pain management. Tuman *et al.*^[7] showed decreased hypercoagulability with epidural anesthesia that may reduce the incidence of coronary artery occlusion and deep vein thrombosis. Kocket *et al.*^[8] provided insight into mechanisms by which epidural anesthesia may be a therapeutic measure in preventing and treating myocardial ischemia.

Prevention, early detection and treatment of myocardial ischemia are crucial for a favorable cardiovascular outcome. The simplest and most cost effective method of detecting myocardial ischemia is ECG.^[9] Computerized analysis has become standard in modern monitors, which have an average sensitivity and specificity of 74% and 73% respectively.^[10]

Stroke volume variation is an indicator of relative preload responsiveness.^[11] Perioperative fluid management in our patient was guided by CVP, SV, SVV, CO. High variations of SVV and CO were not observed as our patient was on

spontaneous ventilation with mild sedation of fentanyl and midazolam.

The case report highlights perioperative anesthetic management of patients with CAD undergoing noncardiac surgery. The approach to preventing perioperative cardiac events entails a careful risk assessment and modification of patient and procedure related risk factors as far as possible. The most important determinants of cardiovascular stability are an anesthetic technique that obtunds the pathophysiological responses induced by surgery, combined with excellent control of pain, fluid balance and oxygenation under close monitoring.

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