Case Report

Anesthetic Management of a Patient with Ischemic Heart Disease Posted for Open Reduction Internal Fixation of the Upper Limb

Nikhila Rajendra, Ravi Madhusudhana

Department of Anaesthesiology, Sri Devaraj Urs Medical College, Kolar, Karnataka, India

Abstract

Patients undergoing noncardiac surgery may develop cardiac complications. Perioperative myocardial infarction (PMI) may be an important predictor of short- and long-term morbidity and mortality associated with noncardiac surgery. The etiology of PMI can be multifactorial; hence, it is indicated that one single intervention will not successfully improve cardiac outcome following noncardiac surgery and multifactorial stepwise approach. Perioperative management of ischemic heart disease (IHD) patients undergoing noncardiac surgery requires careful teamwork and communication between the patient, primary care physician, anesthesiologist, and surgeon.

Key words: Anesthesia, myocardial ischemia, noncardiac surgery, perioperative myocardial infarction

Introduction

Perioperative myocardial infarction (PMI) is the most common cause of morbidity and mortality in patients who have had noncardiac surgery, but diagnosis can be difficult.[1] Management of anesthesia for a cardiac patient who has to undergo noncardiac surgery has always been challenging.[2] More than one half of postoperative deaths are caused by cardiac events, most of which are ischemic in origin.[3] The goal of anesthesia is to keep myocardial oxygen supply greater than the demand to avoid ischemia.[2] Meticulous planning of the technique of anesthesia and pain relief is essential for the prevention of PMI in the perioperative period.

Myocardial ischemia is defined as a dual state composed of inadequate myocardial oxygenation and accumulation of anaerobic metabolites and it occurs when myocardial oxygen demand exceeds the supply. Myocardial infarction (MI) is defined as the death of myocardial myocytes due to prolonged ischemia.[3] The risk of PMI in those with a history of recent MI has declined from 27.3% to 2.1% and in those without a history of recent MI has declined from 2.8% to 0.3%.[4,5]

Access this article online

Quick Response Code:

Website: www.karnatakaanaesthj.org

DOI: 10.4103/2394-6954.163083

Case Report

A 70-year-old male patient following a road traffic accident had sustained a fracture of the right ulna for which he was posted for open reduction and internal fixation. On preanesthetic evaluation, the patient did not have any symptoms suggestive of ischemic heart disease (IHD); electrocardiogram (ECG) showed ischemic changes and echocardiogram (echo) showed IHD with mid and apical segments of anteroseptal and anterior wall that were hypokinetic, mild pulmonary artery hypertension (PAH) (45 mmHg), and severe systolic dysfunction with ejection fraction (EF) of 30–35%. Other routine investigations were normal.

When the patient was taken up for emergency surgery [after 3 h of renal tubular acidosis (RTA)] for open reduction and internal fixation of the open fracture of both bones of the forearm, his heart rate was between 44 bpm and 48 bpm with no angina or dyspnea. Injection glycopyrrolate 0.2 mg intravenous (IV) was given before IV fentanyl for pain relief so that the heart rate would not reduce further. The heart rate did not increase and the patient underwent open reduction internal fixation (ORIF) under brachial plexus block with a nerve stimulator (15 mL 2% plain lidocaine and 15 mL 0.5% bupivacaine plus fentanyl 50 μg; the patient weighed 72 kg); the intraoperative period was uneventful and the surgery lasted for 90 min. Intraoperatively oxygen was given by mask and

Address for correspondence: Dr. Ravi Madhusudhana, Department of Anaesthesiology, Sri Devaraj Urs Medical College, Kolar, Karnataka, India.
E-mail: ravijaggu@hotmail.com

ECG lead 2 and 5 and saturation were monitored noninvasive blood pressure (BP) was monitored and the patient was stable hemodynamically. After the procedure, the patient was shifted to the surgical intensive care unit (ICU) for further monitoring. In the evening (3 h after the block), the patient developed breathlessness for which he was shifted to the main ICU, he had a heart rate of 80 bpm and BP of 150/90 mm of Hg with no signs of congestive cardiac failure (CCF). His ECG showed lateral wall changes and he was diagnosed with acute coronary syndrome (ACS) with lateral wall MI [ST elevation MI (STEMI)] and moderate systolic dysfunction on echo. He was taken up for coronary angiogram, which revealed thrombotic lesions in the left circumflex artery. He was treated on the medical line of management. Troponin levels done on the next day (could not be done immediately) showed positive results [1079 ng/L and creatinine kinase-MB (CKMB 79 units/L)]. He was treated with anticoagulants by the cardiologist (injection heparin), diuretics, and other supportive medication and the patient symptomatically improved and was shifted to the general ward the next day. He was monitored and treated by the cardiologist for 5 days; after he was stable, he was discharged on aspirin, clopidogrel, long-acting nitrates, and angiotensin-converting enzyme (ACE) inhibitors with a plan for further follow-up and intervention by cardiologists at a later date.

Discussion

IHD is the leading cause of morbidity and mortality in the world and also of perioperative complications in cardiac patients. Patients with risk of IHD require identification of risk factors by thorough preoperative evaluation and optimization, medical therapy, monitoring, and appropriate anesthetic technique and drugs.[6] The exact nature of perioperative myocardial injury remains elusive. Perioperative cardiac outcome might be improved by preventive measures, if the causes of perioperative myocardial ischemic events could be identified.[7] The etiology of PMI remains poorly understood; long-duration subendocardial myocardial ischemia or acute coronary occlusion as a result of plaque disruption or thrombosis can be the primary mechanism of PMI in the individual patient.[8]

Perioperative cardiac morbidity may be influenced by recent MI, Congestive cardiac failure, peripheral vascular disease, angina pectoris, diabetes mellitus, hypertension, hypercholesterolemia, dysrhythmias, age, renal dysfunction, obesity, lifestyle, and smoking.[6] Myocardial ischemia can be diagnosed by hemodynamic (pulmonary artery capillary wedge or left atrial pressure wave), electrocardiography, functional (echo), metabolic (coronary lactate production), biochemical (release of CKMB isoenzyme and/or troponin), or regional perfusion (scintigram) parameters. The techniques may have considerable limitations regarding sensitivity and specificity.[7] PMI, as defined by a rise in serum troponin, in patients undergoing noncardiac surgery is not uncommon and occurs with increasing frequency as the number of risk factors increases; short- and long-term survival get decreased in patients who have sustained MI.[9]

The goals for anesthesia for a patient with IHD are to maintain stable hemodynamics, optimize myocardial oxygen supply and reduce oxygen demand, monitor for ischemia, treat ischemia or infarction if it develops, and maintain normothermia. Selection of drugs should be with the objective of minimizing demand, and optimum supply of oxygen and few cardiac drugs should be readily available to maintain hemodynamics to prevent and treat ischemia.[6]

The advantage of regional anesthesia over general anesthesia (GA) should be an asset in cardiac patients if the surgery can be performed under regional block. The patient can be premedicated with anxiolytics. Disadvantages of regional anesthesia include hypotension from uncontrolled sympathetic blockade. Care should be taken while giving local anesthetics because larger doses can cause myocardial toxicity and myocardial depression. Tachycardia is the single most common abnormality that is often associated with ischemia, which by causing both an increase in demand and a reduction in supply can jeopardize the myocardium and bring about ischemic changes in susceptible patients.[10] Perioperative tachycardia can be due to a light plane of anesthesia, endotracheal intubation and extubation, hypovolemia, fever, anemia, congestive heart failure (CHF), and postoperative pain. Similarly, hypertension can cause increased demand and hypotension can lead to decreased supply in the perioperative period.

Pain management may be a crucial aspect of perioperative care because the majority of cardiac events in noncardiac surgical patients occur postoperatively; the postoperative period may be the time during which ablation of stress, adverse hemodynamics, and hypercoagulable responses are most critical. Cardiologists usually do treatment of acute pre- or post-operative MI but in the intraoperative setting, the anesthesiologist plays a major role. If diagnosis of acute MI is made, monitoring of the patient is important; pulse oximetry, automated noninvasive BP, and in selected cases a radial arterial line can be inserted to have a continuous monitoring. Also, 100% oxygen should be administered and volatile agent discontinued. As soon as PMI is diagnosed, aspirin 325 mg is administered orally (through Ryles tube if unable to take orally) and is continued thereafter. Tachycardia is treated with IV β blockers like esmolol while nitroglycerine is the drug of choice in the presence of normal to modestly elevated systemic BP.

Postoperative myocardial ischemia can be associated with postoperative anemia, postoperative hypothermia and pain, and increase in myocardial oxygen consumption in the presence of a decrease in delivery.[7] Increased attention should be focused on the postoperative period to reduce perioperative cardiac morbidity.[11]

In our patient, there was no history suggestive of IHD and he was diagnosed with the help of ECG and echo; pain relief and oxygen supplementation were done. The patient

developed MI in the postoperative period, the reason for which could have been pain though he did not complain or the age factor with the traumatic experience of RTA could have precipitated MI.

PMI is a significant issue not only in patients undergoing high risk surgery but also in those with minor surgical interventions and it also can be silent; its ECG changes are frequently transient and minor troponin elevations predict early and late morbidity and mortality.[12] Careful monitoring for ischemia in the perioperative period, a low threshold for treating and preventing tachycardia while avoiding hypotension, and decreased cardiac output or cardiac decompensation help prevent PMI.[13]

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How to cite this article: Rajendra N, Madhusudhana R. Anesthetic management of a patient with ischemic heart disease posted for open reduction internal fixation of the upper limb. Karnataka Anaesth J 2015;1:69-71.

Source of Support: Nil. **Conflict of Interest:** None declared.