Case Report

Management of Intraoperative Myocardial Ischemia

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Introduction

Perioperative hypotension is a common problem, however its incidence is largely unknown. There are evidences suggesting that incidence of myocardial adverse events in the postoperative period may be linked to the prolonged episodes of perioperative hypotension. There are few comprehensive resources available in the literature regarding diagnosis and management of these common clinical outcomes, especially in non-cardiac surgery[1]. Perioperative myocardial ischemia is associated with significant morbidity and mortality, 50% of postoperative deaths are due to cardiac complications, ischemia being the major problem amongst them[1]. It is an emergency which poses a unique challenge to the anaesthetist in its management. We report a case of intraoperative myocardial ischemia in an elderly patient with multiple comorbidities who underwent orthopaedic hip surgery. Intraoperative myocardial ischemia was diagnosed on time and managed successfully.

Case Report

A 70 year old male, a known case of diabetes mellitus type 2 on treatment with insulin, systemic hypertension on Metoprolol 12.5 mg OD, Telmisartan 40 mg OD and Furosemide 40 mg BD and coronary artery disease, who underwent coronary artery bypass graft 4 years back, with a pathological intertrochanteric fracture of right femur, was admitted in the intensive care unit with urosepsis and acute on chronic kidney disease. He was managed with IV Meropenam, Dopamine and Noradrenaline infusions, and hemodialysis. The inotropes were tapered and stopped 2 days before surgery. He was posted for right proximal femur nailing after optimisation of his general condition.

Post dialysis lab investigations revealed hemoglobin - 10g%, TLC - 10,500 cells/mm^3, platelet count - 2,15,000 cells/mm^3, serum creatinine - 2 mg/dl, sodium - 134 meq/dl, potassium - 3.5 meq/dl, INR - 1.01. ECG, X-ray chest and 2D Echo were normal. He was advised nil per oral 8 hours prior to surgery. On the morning of surgery, insulin and Telmisartan were avoided, Metoprolol and Furosemide were continued. Patient's heart rate (HR) was 92 bpm, blood pressure (BP) was 130/ 90 mmHg & SpO_2 100% in room air. The anaesthetic plan was general anaesthesia with controlled ventilation and lumbar epidural for analgesia. Under all aseptic precautions, left radial artery and right internal jugular vein were cannulated for invasive blood pressure (IBP) and central venous pressure (CVP) monitoring respectively. Prior to induction, epidural catheter was placed at L2-L3 intervertebral space under aseptic precautions. Intravenous Midazolam 1 mg, Fentanyl 200 mcg were administered while preoxygenating the patient for 3 minutes. He was induced with IV Thiopentone sodium 150 mg, Vecuronium 6 mg, intubated with 8.5 mm ID cuffed endotracheal tube and mechanically ventilated in volume control mode. Anaesthesia was maintained with O_2:N_2O - 1:1, Isoflurane 0.8%, Vecuronium 1 mg intermittent boluses. IV fluids were administered according to CVP (target of 8-10 cms H_2O). HR, IBP and ECG were normal. After 1 hour of surgery, there was sudden blood loss of 1100 ml which presented as tachycardia and hypotension. CVP became zero cms H_2O, HR 148 bpm, IBP 60/30 mmHg and ECG showed ST depression with multiple VPC's. ABG revealed pH-7.21, pCO_2-36.5mmHg, pO_2-60 mmHg, HCO_3-12.2. He was managed with IV fluids- 2000 ml Ringer Lactate and 500 ml Hydroxyethylstarch. Inotropic support (Dopamine @ 10 mcg/kg/min, Noradrenaline @ 0.05 mcg/kg/min) was initiated as the hypotension was refractory to IV fluids alone. Two units of packed red blood cells were transfused. Ventilatory support was continued with 100% O_2. Point of care 2D Echo showed regional wall motion abnormality with global hypokinesia. After 30 minutes, HR settled to 90 bpm and BP improved to 100/60 mmHg. Surgery was deferred and wound closed with sutures. Postoperatively, ABG showed pH-7.321, pCO_2-34.5 mmHg, pO_2-108 mmHg, HCO_3-16.4 and negative cardiac enzymes. Ventilatory support was continued in ICU and gradually inotropes were tapered off. Patient was weaned off the ventilator and extubated on the same day. The fracture was managed conservatively and patient was discharged on the 10th postoperative day.

Discussion

Sudden profound hypotension following acute massive blood loss can result in adverse myocardial events, especially in elderly patients with multiple comorbidities. Prompt recognition and early intervention can prevent fatal outcomes.

Acute coronary syndrome (ACS) term denotes a group of clinical symptoms with acute myocardial ischemia; it also includes ST segment elevation (STEMI), non ST segment elevation (NSTEMI) myocardial infarction and unstable angina. ACS is associated with high morbidity and mortality[2].

The two primary risk factors for ACS are increasing age and male gender. Others include smoking, hypertension, diabetes mellitus, sedentary lifestyle, obesity and a family history of CAD. Perioperative myocardial infarction is also observed in patients with chronic kidney disease and peripheral vascular disease[3,4].

ACS may be precipitated by deleterious effects during anaesthesia and surgery such as hypotension, hypertension, hypoxia, hypothermia, pain, anemia caused by blood loss, electrolyte disturbances causing arrhythmias and acute stress response[4].

In an unconscious patient under general anesthesia, diagnosis of ACS can be challenging. So, a detailed clinical history including relevant risk factors, physical examination, and vigilant intraoperative hemodynamic monitoring are quintessential. ECG monitoring combining the three leads II,V4,V5 increases the sensitivity to detect ischemia to 98%[5,6]. For detecting acute segmental wall motion abnormalities, transesophageal echocardiography (TEE) has more sensitivity and detects even before the ECG changes are noted[7].

Continuous monitoring of heart rate, blood pressure and ECG trends are mandatory for early detection of intraoperative complications,. If myocardial ischemia is suspected, the primary goals of management should include adequate oxygenation, maintaining hemodynamic stability and use of heparin or aspirin and intra aortic balloon pump, whenever needed. The effective treatment depends on early intervention with best cardiac care[8,9].

When myocardial ischemia is because of hypovolemia, hypotension should be primarily managed with IV fluids in the form of crystalloids or colloids and blood products. Inotropic support is required when there is no response to fluid administration[10]. In this case, myocardial ischemic changes were because of hypovolemic shock. Hence by correcting hypotension secondary to acute blood loss, the myocardial ischemic changes were reverted.

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