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Anesthetic management of intraoperative myocardial infarction during emergency spine surgery: A case report

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Abstract
Aim: To understand the anesthesia management in a case of an adverse cardiac outcome intra-operatively
Background: Peri-operative Myocardial Infarction (PMI) is associated with a very high rate of morbidity and mortality, especially in patients posted for high-risk surgery with unknown cardiac status. We report a case of acute myocardial infarction in a patient without cardiac risk factors during decompression and instrumentation of spine.
Case Description: A 49-year-old, 58 kg and 178 cm man, with a raised blood pressure of 160/100 mm Hg, with no previous history of hypertension or cardiovascular diseases was posted for Emergency Decompression and Instrumentation surgery. Intra-operatively, patient developed sudden hypotension and ST segment elevation which was corrected with intravenous fluids, packed red cell, DAPT and anticoagulants. Patient improved clinically within 2 weeks and was discharged without complications.
Conclusion: Peri-operative Myocardial Infarction (PMI) requires close monitoring, prompt detection, correction of hemodynamics and team effort.
Clinical Significance: To outweigh the benefits of conducting a high-risk surgery in a patient with uncontrolled hypertension, to initiate timely symptomatic therapy, administering drugs that will maintain the coronary blood flow and giving definitive measures for the same.

Keywords: Peri-operative myocardial infarction, intra-operative MI, STEMI, general anaesthesia, cardiac risk factors

Introduction
Peri-operative Myocardial Infarction is an emergency and anesthetic challenge which often occurs in patients with poor preoperative cardiac conditions undergoing high risk surgery [1]. This is a case report on successful management of a 49-year-old man, with undiagnosed hypertension and no cardiovascular diseases, posted for emergency decompression and instrumentation spine surgery, who developed acute myocardial infarction intra-operatively.

Case description
A 49-year-old male patient, previously diagnosed with D6-D9 myelopathy secondary to Koch's had developed gait instability and urinary urge incontinence since 1 day. He was posted for Emergency Decompression and Instrumentation surgery. Patient was on Category I anti-tubercular treatment for 6 months, chronic alcoholic and cigarette smoker for more than 10 years. Patient's height was ~ 178 cm, weight ~ 58 kg, Pulse: 78/min and Blood pressure: 160/100 mm Hg. Airway and Systemic examination of the Respiratory, Cardiovascular and Gastrointestinal and Central Nervous system was normal. Spine examination showed no obvious external deformity, no visible swelling, sinus or fistula and lowr limb power 4/%
Complete blood count, Serum Electrolytes, Coagulation profile, Renal and Liver function tests, Random blood sugar, Chest X-ray and Pre-operative ECG (Figure 1) was normal. Pre-operative 2D-Echo showed a Left ventricular ejection fraction of 60% with Left ventricular hypertrophy.
Previous Plain MRI of Dorsal spine showed destruction of D8 and D9 vertebral body and obliteration of posterior CSF space causing compression over thecal sac suggestive of infective etiology like Koch's.

Considering the urgency of the surgery in view of bladder involvement and myelopathic malacia of the cords, patient was counselled regarding the surgical and anesthetic complications in view of hypertension, increased risk of bleeding requiring blood transfusion, post-operative ICU monitoring, and cardiac risk factors. Patient was given fitness under ASA IIE. Inside the operation theatre, two large bore intravenous cannulas were secured. Multipara monitors like Temperature, ECG with ST segment analysis, Pulse oximetry, Non-Invasive blood pressure cuff and EtCO2 were attached. Baseline readings showed Spo2: 100% on room air, Pulse: 82/minute, BP: 160/100 mm Hg and Mean arterial pressure: 126 mm Hg. Antibiotic was given pre-operatively. Patient was pre-oxygented with 100% oxygen, pre-medicated with Injection Glycopyrrolate 0.2 mg i/v, Midazolam 1 mg i/v, Fentanyl 100 mcg i/v and induced with Injection Propofol 150 mg i/v and Vecuronium 6 mg i/v. intubate with 8 mm endotracheal tube after 3 minutes, followed by insertion of a 16G Nasogastric tube and throat pack. Patient was given prone position, dependent parts adequately padded, maintained on Oxygen, Air, Sevoflurane 1-2%, Vecuronium and an infusion of Injection Dexmedetomidine was started with a loading dose given over 10 minutes followed by 0.5 ug /kg/hr maintenance. Patients vital parameters were stable.And before incision Inj.Fentanyl60 ug and Inj.Paracetamol 1 gm iv given.

Surgery was uneventful up to one hour when Dexmedetomidine infusion was tapered and stopped due to low blood pressure in view of ongoing blood loss. After ~ 30 minutes, nearing the end of the surgery, BP was 80/50 mm Hg, pulse 66/minute and ST segment showed elevation in Lead II and blood loss was ~ 2000 ml. It was treated with100% oxygen, 200 ml bolus of Ringer's Lactate fluid and Injection Ephedrine graded doses (12 mg) and one Packed red blood cell transfusion till completion of surgery. Patient was immediately turned supine, and a post-operative 12-lead ECG (Figure 02) established the diagnosis of Anterior Wall ST segment elevation Myocardial Infarction (AWMI). As per cardiology advice, a loading dose of DAPT was administered through the nasogastric tube. Blood samples were sent for ABG, CBC, Cardiac biomarkers, and serum electrolytes. Patient was extubated and complained of mild chest pain post-operatively. Injection Fentanyl 60 mcg i/v and Oxygen supplementation @ 4L/min was administered in propped position. Post-operatively, patient was monitored in the Intensive care unit in view of hemodynamic instability and intraoperative MI and cardiac monitoring. DAPT was continued, post-operative 2D Echo suggestive of LVEF: 35% with Anteroseptal distal interventricular septum hypokinesia, elevated Troponin I > 0.2, normal CK-MB and LDH, ABG showing mild metabolic acidosis (pH: 7.32, pCO2: 30.8 mm Hg, pO2: 97.3 mm Hg, HCO3-: 15.8 mmol/L, BE: 5 mmol/L) and a normal CBC, electrolyte and sugar profile. Coronary Angiography was performed 48 hours after the surgery. It showed a 100% blockage in the Left Anterior Descending (LAD) Artery and thus Percutaneous Transluminal Coronary Angioplasty (PTCA) to LAD was performed. Patient was monitored for a week in the CCU and discharged without any further complications.

Discussion

The incidence of Peri-operative Myocardial Infarction (PMI) is rare and varies depending on patient's co-morbidity and type of surgery. It is reported to be 0.6% of all non-cardiac surgeries and 11% or more in high risk groups. Most perioperative myocardial ischemic events occur within the first 72 hours after surgery, up to 20% occur intraoperatively [3]. The incidence of perioperative myocardial infarction (PMI) is between 0.3% and 36% depending on the population, study design, and definition used. [4] The mortality for PMI and for myocardial injury (troponin elevation alone) was 22% at 1 year in a recent study.

Table 1: Possible approach to management of STEMI based on bleeding risk as a function of the timing of the STEMI after surgery

Bleeding risk	Delay after surgery		
	0 to 24 hours	**24 to 48 hours**	**48 to 72 hours**
High	Bespoke protocol 1	Bespoke protocol 2	NICE Guideline
Intermediate	Bespoke protocol 2	NICE Guideline	NICE Guideline
Low	NICE Guideline	NICE Guideline	NICE Guideline
Bespoke protocol 1			
No immediate aspirin and clopidogrel			
Short-acting antiplatelet agent (tirofiban, eptifibatide, cangrelor)			
Consider balloon angioplasty followed by deferred conventional PCI at the appropriate time if risk of bleeding is very high			
Follow-on aspirin and ticagrelor once definitive stent/procedure undertaken.			
Bespoke protocol 2			
No immediate aspirin and clopidogrel.			
Immediate PCI.			
Short-acting antiplatelet agent (tirofiban, eptifibatide, cangrelor).			
Follow-on aspirin and ticagrelor.			
For both bespoke protocols, periprocedural heparinisation as appropriate			

In STEMI, the cause is acute thrombotic occlusion of an epicardial coronary artery, where early recanalisation of the occluded artery can salvage the critically ischaemic myocardium, improving left ventricular function and survival [5].

The exact pathophysiology of PMI is not clearly understood. The postmortem studies showed that it is either due to mismatch of oxygen supply and demand or plaque rupture [18]. STEMI is usually caused by platelet aggregation, vasoconstriction, and thrombus formation at the site of an atheromatous plaque in a coronary artery [9].

The main risk factors for PMI are poor cardiac status, diabetes mellitus, chronic renal insufficiencies, cerebrovascular disease, hypertension, smoking, dyslipidemia, anemia, major surgeries, significant intraoperative blood loss and blood transfusion, peri-operative hypotension [6], recent PCI and stent implantation, Improper perioperative analgesia [7]. Our patient being a chronic smoker, hypertensive, and complicated further due to intra-operative blood loss causing significant hypotension intra-operatively during a major spine surgery. This initiated the cascade of major adverse cardiac events and precipitation of myocardial ischemia.

Major determinants of myocardial oxygen consumption (MVO2) include heart rate, myocardial contractility and wall stress. An increase in heart rate leads to less time spent

in diastole and therefore less time to perfuse myocardium, particularly the subendocardium. Muscle shortening, activation, and basal metabolic requirements also contribute to MVO2 to a lesser degree [10].

Myocardial oxygen delivery is determined by arterial oxygen content (CaO2) and coronary blood flow. CaO2 is determined by the amount of oxygen bound to hemoglobin and, to a much lesser extent, the amount dissolved in blood [11]. Coronary blood flow is determined by coronary perfusion pressure, the time available for perfusion, and the patency of coronary arteries [12]. Coronary perfusion pressure can decrease due to a decrease in systemic pressure or increase in left ventricular end diastolic pressure. Other determinants of coronary blood flow include extravascular compression by the myocardium, myocardial metabolism, and effects of neurohumoral control [10]. These physiological principles form the basis for the management of acute myocardial ischemia.

The perioperative period induces a wide range of physiological changes in the sympathetic tone, cardiovascular system performance, coagulation, and inflammatory milieu. These changes include unpredictable alterations in the atherosclerotic plaque morphology, function, and the progression. Simultaneous perioperative alterations in homeostasis may trigger myocardial oxygen supply and demand imbalance or better designated as an "Ischemic imbalance". Absence of a timely resolution begets PMI, irrespective of the etiology [13].

According to World health organization (WHO) at least two of the following criteria are required to diagnose PMI: (i) typical chest pain (ii) rise and fall of cardiac biomarkers (iii) typical ECG changes and (iv) echocardiographic wall movement abnormalities [14]. The type of surgery is of vital importance in evaluating cardiac risk and abnormal preoperative ECG (atrial fibrillations, right or left bundle branch block, LVH, pacemaker rhythm, or ventricular ectopic) also increases the risk for the cardiac complications [15]. The cardiac troponins (troponin T and I) are rapidly released into the circulation after myocyte injury with absolute myocardial tissue specificity and a high sensitivity. Intra-operative MI under general anaesthesia often silent, as patients are unable to report chest pain, with transient ischemic changes in the ECG, making its diagnosis difficult. Our patient was diagnosed as having intraoperative MI based on ST elevations of > 1 mm in Lead II of ECG, with significant hypotension. The POISE-2 trial demonstrated that hypotension, a potentially modifiable exposure, was significantly associated with a higher composite of MI and death intraoperatively, postoperatively on the day of surgery, and during the initial 4 postoperative days. It was also an independent predictor of MI [16]. Hence, resuscitative measures target a SPB > 90 mm Hg.

Fluid resuscitation and blood transfusion in view of ongoing blood loss helped stabilise this patient's vital signs.

Targeted management strategies were planned once the diagnosis of Intraoperative MI was confirmed by 12-lead ECG and troponin values. Oxygenation, stabilising hemodynamics with the help of fluid resuscitation and blood transfusion, Cardiac consultation to initiate Dual Anti-Platelet Therapy, pain control relief with intravenous Fentanyl and early invasive procedure was successfully accomplished [13].

Preventive measures for PMI include careful monitoring for ischemia, preventing tachycardia, hypotension, hypoxia, hypercarbia, hypothermia, alleviating pain, anxiety and initiating anti-coagulation treatment [17].

Although Intravenous Nitroglycerin decreases preload, afterload and ventricular wall tension, it was not used in our patient due to intra-operative and post-operative hypotension, with the patient requiring Noradrenaline infusion in the ICU. Although not an absolute indication, Central Venous Catheter and Arterial line placement for guiding the fluid therapy and precise management of blood pressure could have been considered. Furthermore, our patient had stable heart rate throughout the duration of the surgery, and hence there was no role of beta-blockers.

The best reperfusion strategy for perioperative STEMI is depends upon residual bleeding risk of the noncardiac surgery (Table 1 BJA).

As a result of close monitoring of hemodynamics, early and prompt management when PMI was suspected, assessing the benefit versus risk of administering anti-platelet therapy to the patient, in addition to angiographic examination post-operatively, and efficient and co-ordinated team work amongst the Anesthesiologists, Surgeons and Cardiologists led to an overall good prognosis without further complications adding to the successful management in this case of intra-operative myocardial infarction.

Conflict of Interest
Not available

Financial Support
Not available

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