Case Report

A rare presentation of perioperative myocardial infarction as a consequence of stent thrombosis in non-cardiac surgery

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Received: 19 May 2021
Accepted: 15 June 2021

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ABSTRACT

Perioperative myocardial infarction (PMI) is a major cause of long term morbidity and mortality associated with non-cardiac surgery. In patients with recent coronary artery stent placement undergoing non-cardiac surgery perioperative management is always challenging. We reported an interesting case of a 67 year old man posted for head neck surgery with recent history of angioplasty with drug eluting stent developing perioperative MI and the challenges faced by an anaesthesiologist.

Keywords: Perioperative myocardial infarction, Drug eluting stent, Plain old balloon angioplasty, Dual antiplatelets therapy

INTRODUCTION

As per the definition of the WHO, in order to detect PMI minimum two of the three criteria must be satisfied. They are classical ischaemic chest pain, raised serum concentration of creatine kinase-MB isoenzyme and classic electrocardiographic findings, along with development of pathological Q-waves.1 Patients with coronary artery diseases having non-cardiac surgery are at an increased risk for perioperative complications like as myocardial ischaemia, MI, cardiac failure, arrhythmias, cardiac arrest and increased morbidity and mortality. The incidence of these complications is greater in patients with history of recent MI or unstable angina requiring emergency or semi emergency surgery. We reported a case of intraoperative MI and stent thrombosis and its management.

CASE REPORT

A 64 year old male with history of hypertension and diabetes since 6 years presented with a lesion in right lower gingivobuccal mucosa abutting the mandible since 2 months was posted for wide local excision with marginal mandibulectomy, neck dissection and reconstruction using primary closure.

Patient had a history of ischemic heart disease 1 month prior to surgery underwent coronary angiography, diagnosed as triple vessel disease with 80% stenosis in left anterior descending artery (LAD) and 90% stenosis in left circumflex artery/obtuse marginal artery (LCX/OM3). Angioplasty was done and 2 drug eluting stents were put in LAD and OM3, dual antiplatelet therapy of tablet ecospirin 75 mg and tablet ticagrelol 90 mg was initiated. General anaesthesia and high risk of anaesthesia consent was taken and patient and relatives were explained about the risk of perioperative cardiac events.

On preoperative evaluation, his heart rate was 73 beats per minute, blood pressure was 123/60 mmhg, room air saturation was 99 percentage, weight was 68.71 kg, height was 170 cm, BMI was 23.80 kg/m². Airway
examination his mouth opening was two fingerbreadth and Mallampati classification grade 4. All the necessary blood investigations were within normal limits. A 12 lead electrocardiogram showed sinus rhythm, 2d echocardiography showed ejection fraction (EF) of 50% with basal inferior and inferolateral wall hypokinesia. Dual antiplatelet was stopped 3 days prior to surgery. Cardiologist had given clearance for surgery under perioperative low molecular weight heparin cover with moderate cardiac risk.

On the day of surgery, 20 g IV cannula was secured and routine induction of general anaesthesia with nasal intubation of north pole tube number 7 was done. After 2 hours into surgery during primary resection, elevated ST segment seen in chest lead V and increased ST depression in lead II III avf was seen, patient started to have fall in BP, injection phenylephrine 50 mcg bolus repeated and noradrenal in 1 amp in 50 ml NS started at 0.1 ug/kg/min, nitroglycerin (NTG) 25 mg per 50 ml NS started at 0.2 ug/kg/min, informed to operating surgeon and right radial arterial line was taken. Cardiologist was informed, a 12 lead electrocardiogram was done suggestive of anterolateral infarct and 2d echo was done in OT by cardiologist and new onset of regional wall motion abnormality was reported with fall in EF to 35%, intra-aortic balloon pump and femoral venous line were secured (Figure 1). Relatives were informed of the same, decision was taken to abandon the flap and primary closure was started. After 15 minutes, ECG showed multiple ventricular premature complexes, injection 2% preservative-free lignocaine 100 mg was given and infusion started at the rate of 1 mg/kg/hr. Patient went into ventricular tachycardia, injection amiodarone 150 mg bolus was given and infusion of NTG was stopped. After 15 minutes, pulseless electrical activity seen, cardiopulmonary resuscitation started. Rhythm changed to ventricular tachycardia 1 DC shock of 200 J given. Rhythm went back to pulseless electrical activity, 5 cycles of CPR given as per ACLS protocol, inotropes titrated as per vitals, injection amiodarone and injection adrenaline infusion started. Sinus rhythm achieved after 10 minutes and patient shifted to angiography suite for further management. Angiography revealed thrombotic occlusion of proximal LAD stent, thrombus suction was done. Plain old balloon angioplasty to LAD stent was done (Figure 2). Adequate flow was established in LAD with good myocardial blush patient shifted to ICU with inotropic supports. Patient extubated on postop day 2 and was stable on minimal inotropic supports. Postop day 4 patient hemodynamically stable shifted to ward.

**DISCUSSION**

PMI is challenging as in most of the cases patients are under general anaesthesia and chest pain if present it is usually concealed because of residual analgesic action of anaesthesia drugs. Usually ECG changes are momentary, cardiac enzymes have limited sensitivity and specificity because of coincidental skeletal muscle injury. The major factors which can lead to PMI are either acute coronary syndrome due to plaque rupture or persistent myocardial oxygen supply demand disparity in presence of existing coronary artery disease. The primary treatment in non ST segment elevation myocardial infarction (NSTEMI) is medical management but in case of myocardial infarction with ST segment elevation (STEMI) treatment is revascularization with either fibrinolysis or percutaneous coronary intervention (PCI). The treatment modalities are restricted in PMI due to threat of surgical side bleeding in the immediate postoperative period.

As per 2014 ACC/AHA guidelines it is proposed that patients who needed to undergo PCI and posted for elective non-cardiac surgery in next 12 months, plain old balloon angioplasty (POBA) or use of bare metal stent (BMS) accompanied by 4 to 6 weeks of dual antiplatelet therapy (DAPT) is rational approach. In case of emergency surgical procedures in patients with drug eluting stent (DES) requiring cessation of DAPT, it is recommended to continue aspirin if possible and resume...
P2Y12 inhibitor promptly in postoperative period as early as possible.\textsuperscript{5,6} A study also revealed that occurrence of serious adverse cardiac events was markedly higher (10-15\%) in the course of first 30 days post the stent placement.\textsuperscript{7} The latest perioperative guidelines proposed to continue DAPT in patients undergoing emergency noncardiac surgery during the course of initial 4 to 6 weeks following BMS or DES placement unless the risk of bleeding exceeds the benefit of stent thrombosis prevention.\textsuperscript{5} In our patient a diagnosed case of carcinoma oral cavity, underwent emergency angioplasty for triple vessel disease on coronary angiography (CAG) for acute coronary syndrome with placement of two drug eluting stent (DES) one month prior, now posted for definitive surgery. Hence preoperative cardiologist reference was done and DAPT was stopped (ticagrelol 5 days and aspirin 3 days) before surgery, with low molecular weight heparin used as bridging therapy before surgery.

The diagnosis of MI under general anaesthesia is very challenging as patient is not able to communicate chest pain and changes in vitals may not be easily differentiated from those due to surgical factors but ischemia can be recognized from ECG changes, which we noticed in our case before cardiac arrest, due to vigilant monitoring from the beginning of case.\textsuperscript{8} We picked up ECG changes immediately and cardiologist reference done inside the OT and after discussing with surgical team plan changed to primary closure instead of reconstruction with free flap. In addition, ECG leads that reflect changes may also differ, depending upon location of ischemia. As usually we monitor lead II and V during general anaesthesia diagnosis of MI is challenging.\textsuperscript{10} Hence quick diagnosis and management is critical. If required prompt interruption of surgery, quick cardiac work up and PCI are key to patient’s survival.

After acute MI post ROSC we shifted our patient to cathlab for early PCI suspecting acute MI most likely due to stent thrombosis with timely recognition and prompt treatment along with swift transfer to angiography suite we achieved reperfusion through PCI within 30 minutes after ROSC. Post PCI rest of the postoperative course was uneventful and patient recovered completely without any further complications.

**CONCLUSION**

Diagnosis of acute MI in patients under general anaesthesia is very challenging, being vigilant from the beginning of surgery in patients with associated risk factors allows early diagnosis by careful monitoring of ECG changes along with vital signs and quick decision making with prompt treatment will definitely improve prognosis.

_Funding: No funding sources_  
_Conflict of interest: None declared_  
_Ethical approval: Not required_  

**REFERENCES**


International Journal of Research in Medical Sciences | July 2021 | Vol 9 | Issue 7 | Page 2127