



INTRA OPERATIVE MANGEMENT OF ACUTE MYOCARDIAL ISCHAEMIA

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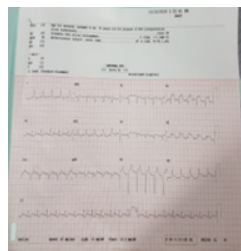
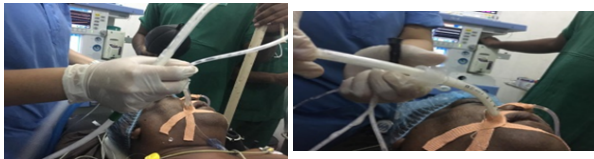
KEYWORDS :

INTRODUCTION

Intra operative myocardial ischemia is rare but is associated with very high mortality and morbidity. It is an emergency anesthetic crisis which poses a unique management challenge for the anesthetist. The case presented here illustrates the interaction of multiple perioperative factors that might be responsible for the ischemia in this patient.

CASE SUMMARY

63 year old male ,diagnosed to have cholelithiasis and Hydatid cyst of liver. Patient is known case of type 2 DM on oral hypoglycemic agents under control and Hypertension past 8 years on regular treatment with T.Amlodipine. Airway assessed mallampatti grade III ,adequate mouth opening, normal neck movement. Pre op ECG –Normal sinus rhythm. ECHO – EF- 65%, no regional wall motion abnormalities. Patient was taken up for Open cholecystectomy and Hydatid cyst excision under ASA-II.



repeat ECG showed anterior wall MI. TROP-I positive and TROP-T positive, with the advice of cardiologist started on Inj Low molecular weight Heparin. Next day morning repeat ECG was normal ,ABG was normal, patient is weaned and extubated . Patient is on follow up with cardiologist for further treatment.

DISCUSSION

The term acute coronary syndrome (ACS) refers to any group of clinical symptoms compatible with acute myocardial ischemia and includes unstable angina, non-ST-segment elevation myocardial infarction and ST-segment elevation myocardial infarction (STEMI). ACS is more frequent in the elderly than in the general population and is associated with very high morbidity and mortality. The two most significant risk factors for developing coronary artery disease are increasing age and male gender. Other risk factors include cigarette smoking, hypertension, obesity and sedentary lifestyle, high cholesterol, diabetes mellitus, family history of premature coronary artery disease. Diagnosis of ACS in an unconscious patient under general anesthesia is quiet challenging.

CONCLUSION

Here, the anesthetist has to recall for clinical history, relevant risk factors, physical examination, and intraoperative findings during monitoring [including electrocardiography (ECG) and hemodynamics]. Transesophageal echocardiography (TOE) is very sensitive for detecting acute segmental wall motion abnormalities and can be detected before ECG changes occur.

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ANAESTHESIA TECHNIQUE:

Under aseptic precautions patient in sitting position epidural catheter was secured and tip at T6 level. Patient in supine position induced with Inj propofol ,after ensuring adequate mask ventilation inj Atracurium was given and Intubated with 8.0 size I.D Endotracheal tube ,bilateral air entry checked and tube fixed at 21cm. Depth of anaesthesia was maintained with oxygen (50%)+nitrous oxide(50%)+ Isoflurane (0.5-1 MAC)+ Atracurium in titrated doses. Intra operative analgesia was maintained by epidural with 0.25% bupivacaine. Intra operative vitals were monitored through the procedure. It was 3 hours 30 minutes from the starting of procedure, transient T wave inversions were noted in the ECG, Patient started to desaturate and T wave inversions became prominent on ECG. Pink Frothy secretions were noted to come from endotracheal tube. Thorough suctioning was done and Inj. Furesemide 20mg IV was given initially followed by 20mg IV and Inj Morphine 5mg IV was given. Saturation started to improve and was maintaining at 99%. ABG was done and PH- 7.08 PCO₂ -58 PO₂ -74, Lactate- 2.3 ,HCO₃- 17.2. Inj Sodabarbonate correction done. Procedure duration was 4hours. Patient was shifted to ICU for further management. In ICU ,patient connected to the ventilator and on (S)CMV mode Vt- 480ml, PEEP- 6cmH₂O, FiO₂- 60%. Inj Frusemide 5ml/hr infusion started. Vitals: HR-90/min, Bp-140/90, Spo₂- 99% , CVS-S1,S2+ , RS- B/l basal crepts+ ,urine output was 75ml/hr. Bed side chest x-ray- showed Acute pulmonary edema and