Management of an Intraoperative Inferior Myocardial Infarction

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LEARNING OBJECTIVES

- Define myocardial infarction (MI)
- Discuss associated complications and explain the pathophysiology behind a MI
- Discuss the intraoperative management of a perioperative MI

INTRODUCTION

A myocardial infarction (MI) is defined as the rise and/or fall in cardiac biomarkers (typically troponin) in the setting of myocardial ischemia: cardiac symptoms, EKG changes, or imaging findings [4]. MI may be caused by coronary obstruction, decreased oxygen carrying capacity, or decreased coronary perfusion pressure. Typical presentation includes chest pain/tightness localized behind the sternum, sweating, pain that radiates to the arm, neck, abdomen or jaw, usually associated with an increase in physical activity or emotional stress [3].

PREOPERATIVE ASSESSMENT

A 70 y/o, 73 kg female with metastatic breast cancer with malignant stricture of the colon presents for a cystoscopy and an open left colectomy with colostomy reversal and possible distal pancreatectomy. Past medical history was significant for asthma, atrial fibrillation, GERD, HTN, and T2DM. Home medications were as follows: Amlodipine, Furosemide, Losartan, Melatonin, Metoprolol, Xarelto and Zofran. Allergies included morphine, tape, ceftin and ciprofloxacin. Preop lab values and vitals all WNL. The patient had several successful surgeries over the past few decades without complication. However, the patient recently experienced complications shortly after induction for her scheduled left colectomy with colostomy takedown. The patient was induced and intubated without issue. A right arterial line attempt was made and aborted when HR was noted to be in the 140's then up to the 170's, treated with esmolol back down to 80-110's. MAPs were reportedly in the 40's, treated aggressively with pressors. Arterial line and central line were placed. A TEE was performed and concern for a right atrial clot. The procedure was aborted, and the patient was transported to the ICU intubated and sedated.

ANESTHETIC PLAN

The anesthesia plan for this patient was to conduct a general anesthetic with an endotracheal tube. A Glidescope was elected for use due to prior airway history revealing a small mouth opening. A standard induction was planned with the use of Lidocaine, Propofol and Rocuronium. ASA standard monitors and an asleep arterial line were planned for monitoring. An additional IV was planned for after induction.

OPERATIVE COURSE

The patient was induced with 2 mg Versed, 100 mcg Fentanyl, 60 mg Lidocaine, 100 mg Propofol, 70 mg Rocuronium. IV medications were given through a 22G PIV placed pre-operatively. Mask ventilation was successful, and the patient was intubated with a Glidescope and a size 7.0 ETT. After a smooth induction and intubation, TAP blocks were performed on the patient for post-operative analgesia. Simultaneously, a right radial arterial line was placed. Within 1-2 minutes of 0.5 mg IV ceftriaxone administration and initiation of the cystoscopy, the patient's HR rapidly increased to 130's to 180's with MAPs in the 40's. 40 mg of Esmolol, 400 mcg of phenylephrine and 2 units of Vasopressin were administered with minimal response. MAP's trended down to the 20's and peripheral pulses were not palpable. A code blue was called, and chest compressions were started at a rate of 100 RPM. A second 14G PIV was placed, along with a femoral central line. An ABG was obtained, and all values were WNL. Epi and other pressors were given to support blood pressure. The code cart was brought into the room and pacing pads were placed onto the patient. A synchronized cardioversion was performed at 120J and was unsuccessful. 300 mg of Amiodarone was given, and compressions continued. A second attempt to cardiovert at 200J was more successful, patient was now in PEA. A TEE was performed which showed some wall motion abnormalities, which improved with stabilization. The patient received a total of 2.5 L of crystalloid throughout the course of resuscitation. ROSC was obtained after roughly 35 minutes. The decision was made to transport the patient to SICU intubated and sedated, and to have cards evaluate. When compared with a previous 12-lead EKG from a few months prior, an EKG taken that night revealed that the patient had an inferior myocardial infarction.

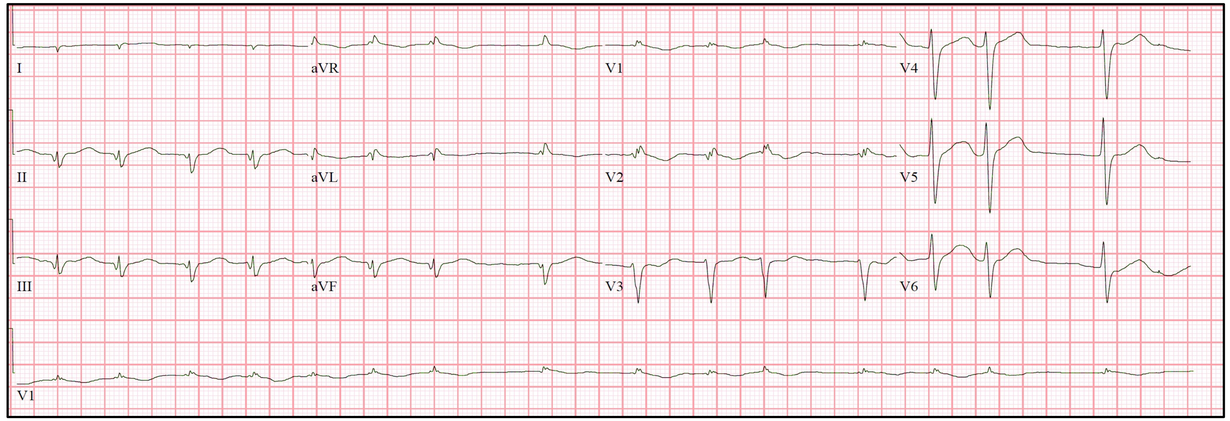

Figure 1. 12-Lead EKG taken 10 hours postoperatively demonstrating atrial fibrillation, right bundle branch block, and an inferior myocardial infarction.

DISCUSSION

The two prominent mechanisms that can lead to a perioperative MI include: Type I MI-acute coronary syndrome-typically a plaque rupture leading to partial occlusion or Type II MI- imbalance between myocardial oxygen supply and demand in the presence of coronary artery disease. Defining a PMI in an anesthetized patient is difficult because certain symptoms are masked, and EKG changes can be subtle and/or transient [1]. Both Type I and Type II MI can be precipitated by external stressors such as those occurring perioperatively. Infarcts can also be classified as anterior, inferior and lateral. In this case, an inferior infarct was identified by ST-elevation in the inferior leads (II, III, aVF). In most patients, the inferior myocardium is supplied by the RCA [6]. In addition to EKG changes, the patient had elevated troponin levels that were trended post-operatively. Both abnormalities resulted in the diagnosis of an acute inferior myocardial infarction.

Perioperative management of a MI includes aggressively treating all causes of tachycardia, hypertension, hypotension, anemia and pain. Treatment of tachycardia associated with HOTN can be challenging and requires understanding of the patient's physiology and comorbidities. It is recommended that vasopressors and beta blockers are used to maintain blood pressure and slow heart rate, while managing the patient's volume status, postoperative pain and respiratory function. Tight perioperative hemodynamic monitoring including echocardiography, arterial line, central venous or possible pulmonary arterial pressure. Emergency cardiology consult for possible thrombolysis or stenting is recommended [2].

CONCLUSIONS

In summary, perioperative myocardial infarctions are a rare, serious complication with a high morbidity and mortality rate. The perioperative period induces unpredictable and significant hemodynamic changes which may result in adverse cardiovascular events. There are prophylactic measures the anesthesia care team can provide such as pharmacologic therapy, pre-operative optimization and careful intraoperative monitoring.

In retrospect, alterations to our initial care plan that could have improved outcomes include awake arterial line placement, large bore IV placement preoperatively, using etomidate for induction, beta blocker therapy prior to surgical stimulation and placing pacing pads on the patient prior to induction.

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