

## ANAESTHETIC MANAGEMENT OF STRANGULATED HERNIA WITH TRIPLE VESSEL CORONARY ARTERY DISEASE FOR LAPAROTOMY AND PROCEED

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### Abstract

The presentation of acute abdomen in the elderly with comorbidities is not uncommon. Sepsis and toxemia influence haemodynamics and ventricular function. Low ejection fraction with severe global hypokinesia in the elderly presenting for anaesthesia would be very challenging to the anaesthesiologist because of high risk of peri-operative complications. However, 63 yrs old patient with old, coronary artery disease with poor ejection fraction tolerates general anaesthesia conducted appropriately and evolved into a successful outcome. This case is presented to project that a patient with age, sepsis, toxemia, dehydration, poor ventricular function, and drug-induced coagulation derangement would recover appropriately by dealing actively with the aetiology of the acute condition.

## INTRODUCTION

The presentation of acute abdomen in the elderly with comorbidities is not uncommon. Sepsis and toxemia influence haemodynamics and ventricular function. Low ejection fraction with severe global hypokinesia in the elderly presenting for anaesthesia would be very challenging to the anaesthesiologist because of high risk of peri-operative complications.<sup>[1]</sup> We report the case management of an elderly patient with background coronary artery disease- triple vessel disease who had a strangulated hernia for emergency laparotomy under general anaesthesia.

## CASE REPORT

A 63years old female presented at the emergency department with a history of pain around the umbilical region for the past 4 days. She also suffered from vomiting, constipation, and decreased urine output. She had a history of triple vessel disease and has been on anti-coagulant therapy for the past 5 years.

On examination, she was in painful distress, pallor, and dehydrated with sunken eyes. She had an enlarged heart and the first, second and fourth heart sounds were heard. Her haemoglobin concentration was 9 g/dl with a deranged coagulation profile. Chest X-ray revealed cardiomegaly, unfolding of the aortic knuckle with calcific rim and homogenous

opacities with air bronchogram noted in both lower lung zones. ECG revealed left bundle branch block with intraventricular conduction block, ST depression, and Tall T waves in limb leads. Echocardiography shows severe global hypokinesia with a dilated left ventricle and a 30% ejection fraction.

The patient gave a history of stoppage of anti-coagulant therapy for the past three days. She was scheduled for emergency laparotomy because of gangrenous bowel. Pre anaesthetic evaluation was done to plan the management of cardiac illness during the peri-operative period.<sup>[3]</sup> Airway examination showed Mallampati grading 2 and ASA 4E (American society of anaesthesiologist) assessment. After explaining the anaesthetic procedure to the patient and relatives, high-risk informed consent was obtained. Blood and blood products (fresh frozen plasma) were made ready. ECG, non-invasive blood pressure, pulse rate, and oxygen saturation were monitored at the surgery. Intravenous access was obtained in both arms using an 18 G cannula. Preoperative vital signs were: pulse- 68-74 / min, BP of 100/60 mmHg, sPo<sub>2</sub> around 97% in room air. Pre-loading has done with 300 ml crystalloids. Preoxygenation was done with 100% oxygen; induction of anaesthesia was done with fentanyl 2mcg/kg, etomidate 0.2mg/kg and muscle relaxation were achieved by atracurium 0.5mg/kg; stress response was attenuated with 45mg Xylocard. Using a 7.0mm endotracheal tube, intubation was facilitated with a C-Mac video

laryngoscope and controlled ventilation. Anaesthesia was maintained with oxygen/ air (50% each).

Laparotomy proceeded and the gangrenous bowel underwent resection anastomosis. Meanwhile, 100% oxygen was given during anastomosis. Throughout the procedure, the total fluid transfused over 4 hrs was 1100ml (500 ml crystalloid, 200 ml of FFP, one packed cell), and blood loss was around 250 ml; high coloured urine output of 200ml was recorded. Analgesia was supplemented with aliquots of fentanyl (50mcg) throughout the procedure. Stable haemodynamics were maintained throughout the procedure. Smooth extubation was done after adequate reversal and thorough suctioning. Postoperatively, the patient was monitored for mean arterial pressure, CVP, urine output, and analgesia with fentanyl infusion.<sup>[5]</sup> The patient was started on graded oral feeds after the 8th postoperative day and discharged on the 10th day.

## DISCUSSION

A cardiac patient undergoing emergency non-cardiac surgery always presents great challenges to anesthesiologists. There must be careful preoperative evaluation, optimization of fluid and good collaboration with surgeons to define the surgical procedure.

DCM (Dilated Cardiomyopathy) is characterised by left ventricular or biventricular dilatation and impaired ventricular contractility. Anaesthetic management of these patients is quite challenging because of the associated global ventricular dysfunction and predisposition to malignant arrhythmias perioperatively, both of which are aggravated by the myocardial depressant effect of the anaesthetic drugs and surgical stress.<sup>[2]</sup>

The major concerns in the anaesthetic management of this patient are to improve cardiac output & prevent cardiac decompensation. A fine balance between pre-load and after load is mandatory to obtain better outcome and avoid perioperative morbidity.<sup>[1]</sup> Soon after induction, these patients have hypotension which we avoided to an extent by using etomidate as an induction agent (transient hypertension produced due to activation of alpha-2 adrenergic receptors).

The anaesthetics, which were supposed to decrease cardiac output by their cardio depressant action, helped improve the cardiac output by reducing Systemic vascular resistance and suppressing baroreceptor reflex-induced tachycardia. We anaesthesiologists must take care to evaluate intra-operative haemodynamics, risks of bleeding, and new cardiac signs suggesting further deterioration of the pre-existing cardiac disease. We infer that multiple comorbidities and cardiac decompensation are not limitations to take up a patient who deteriorated due to a treatable cause such as sepsis.

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