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Anaesthesia Management of an Elderly Patient with a Rare "Left Ventricular Pseudo-Aneurysm" Posted for Non-Cardiac Surgery -Case Report

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Abstract: Patients with Left ventricular pseudoaneurysm posted for noncardiac surgery is rare. Anaesthesiologists should know the pathophysiology, complications and anaesthesia challenges in these patients to manage the case. Here, we managed an elderly patient with a left ventricular pseudoaneurysm posted for ileostomy closure. The primary goals are to maintain hemodynamic stability, avoid excessive stress on the myocardium, and prevent any further injury to the affected region of the heart.

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I. INTRODUCTION

A left ventricular aneurysm is a localised, abnormal dilation of left ventricular wall, often resulting from myocardial infarction (MI), though it can also be caused by other forms of heart disease, such as chronic hypertension or ischemic cardiomyopathy. This condition can lead to significant hemodynamic alterations, increase the risk of arrhythmias, heart failure, thromboembolism, and sudden cardiac death. Anaesthetic management of LV aneurysm is complex due to the associated cardiovascular instability and the potential for serious perioperative complications. Given the altered myocardial contractility and the presence of abnormal ventricular wall dynamics, careful consideration must be given to intraoperative management, including choice of Anaesthesia, fluid management, and monitoring of cardiac function. The primary goals are to maintain hemodynamic stability, avoid excessive stress on the myocardium, and prevent any further injury to the affected region of the heart.

True LV aneurysm is caused by thinning and weakening of myocardial tissue after infarction, involves all layers of the myocardial wall. Low risk of rupture and mainstay of treatment is medical management.

Left ventricular pseudo-aneurysm is caused by rupture of the myocardial wall with pericardial containment. Only the endocardium is involved. High risk of rupture, which may lead to cardiac tamponade or haemorrhage. Surgical Intervention is usually required due to the high risk of rupture. We presenting a rare case of LV pseudoaneurysm, with no prior history of coronary artery disease, myocardial infarction, or trauma.

II. CASE HISTORY

A 73-year-old elderly man came for Pre Anaesthetic Checkup (PAC) for Ileostomy closure. Pt was an operated case of open appendicectomy with diversion Ileostomy under Spinal Anaesthesia (SA)three months back, and the procedure went uneventful. On Preanaesthetic examination, despite physiological changes in the elderly, He was having tachycardia of 112-124 beats/min. further investigation showed a Thrombosed aneurysm/pseudo-aneurysm of the cardiac apex of the left ventricle on CT thorax. After which 2D ECHO was done, which showed the evidence of Ischemic Heart Disease- apical, septal, anterolateral, hypokinesia, LVEF-50%. Patient had no comorbidity. Patient has been a chronic smoker for the past 30 years with 10-15 cigarettes/day. His preoperative hemoglobin was 11.8mg/dl and other blood investigations were normal. Inside the operation Theatre, monitors including ECG, NIBP, Pulse oximetry, temperature and urine output monitoring done. All emergency cardiac drugs and defibrillator were kept ready. Baseline HR- 86/min, BP-122/84mmHg and oxygen saturation was 98% at RA. Patient was started with Ringer's. Inj. Ondansetron (0.1mg/kg) slow iv was given. Patient planned for Spinal anaesthesia with Epidural Anaesthesia (EA). Spinal Anaesthesia(SA) at the level of L3-4, Inj. Bupivacaine (0.5%) heavy 2.6cc with Inj. Fentanyl 25 mcg was given. In Epidural Anaesthesia, epidural catheter is inserted at the L1-2 space with the loss of resistance technique catheter fixed at 10cm. Adequate level of analgesia and anaesthesia attained till T6. Intraoperatively, mean arterial pressure and heart rate were maintained in the 65-75mmHg and 70-80 beats/min, respectively. After Epidural activation, 2 top-ups of 5 cc of 0.125% bupivacaine were given at an interval of 45 minutes. We encountered a single episode of hypotension (82/44mmHg) and was managed using a bolus of injection phenylephrine 100 mcg. The Ileostomy closure was completed in 90 minutes, during which total blood loss was 100ml, and 500ml of Ringer's+ lactate was infused. The postoperative period was uneventful with heart rate stayed at 80-90/min. The urine output remained adequate throughout the post-operative period. Patient was given 6 hourly epidural top-up with Inj. Bupivacaine (0.125%) with VAS score maintained under 4/10 and monitored in ICU setup. The patient was discharged 8 days post-surgery and a cardiology referral was given.

III. DISCUSSION

Transmural MI is the most common cause of LV pseudoaneurysm and can result from cardiac surgery, trauma, infection including tuberculosis or pericarditis and post-MI status[1]. It can also be due to the mitral valve replacement, history of unsuccessful ventriculotomy, inflammation and tumour invasion. The most likely Location of LV true aneurysm- anterior/apical, and most likely Location of Pseudoaneurysm- posterior/inferior. Remarkably, in our patient the location of LV pseudoaneurysm was in the apex, which makes it unique. Higher risk for the development of LV pseudoaneurysm is seen in females, those with older age and hypertension. Interestingly, 12% of the times, LV pseudoaneurysm can be completely asymptomatic on presentation. LV Aneurysm rupture occurs in 4% of patients after acute MI and are seen in 23% of patients at autopsy following MI-related death [2]. The LV True aneurysm has a well-delineated, thin, scarred or fibrotic wall because it arises from a weakened ventricular wall due to ischemic and it enlarges over time. LV Pseudoaneurysm results from rupture of ventricular free wall. The wall of pseudoaneurysm composed of organised hematoma and pericardium and lack elements from the original myocardial wall. Unlike True aneurysm, a pseudoaneurysm contains no endocardium or myocardium and hence, greater tendency to rupture leading to pericardial tamponade and sudden death. It is difficult to diagnose LV Pseudoaneurysm because of the absence of precisely defining symptoms. Transthoracic Echocardiography (TTE), cardiac CT, cardiac MRI and Ventricular Angiography can be used for diagnostic imaging. The most reliable method for diagnosing LV pseudoaneurysm is angiography[3]. Left ventriculogram used to be the gold standard, and now newer modalities are preferred. Transthoracic and transesophageal echocardiograms have a sensitivity of 26% and 75%, respectively. Distinguishing a Pseudoaneurysm from a true aneurysm is important because treatment strategies for the two differ vastly. Urgent surgical resection is needed in the true aneurysm, whereas true aneurysms can be managed

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with medical therapy alone. 30-45% risk of rupture can be there with unmanaged pseudo-aneurysm, with a mortality of almost 50% reported when managed with medical therapy alone[4]. Thromboembolic events are commonly seen with LV pseudoaneurysm, where they arise from the lumen[5]. Both hypertension and hypotension are deleterious to the patient. Hypotension causes an increased risk of pump failure, and hypertension causes LV cavity distension, thereby increasing the risk of rupture [6]. Perioperative anaesthetic management of LV pseudoaneurysm should be focused on preventing rupture of the aneurysm and on avoiding the risk of vital organ ischemia, which is frequently related to complicated MI events. A detailed pre-operative checkup with relation to diagnosis between true and Pseudoaneurysm is crucial. LV pseudoaneurysm is rare and without any history of myocardial infarction or other afore-mentioned risk factors. These patients are prone to rapid hemodynamic changes in the perioperative period, and thus, hemodynamic monitoring should be accurate as these patients have a narrow safety range for hemodynamic parameters[7].

Post operative management plays an important role in the outcome of left ventricular Pseudoaneurysm.

IV. CONCLUSION

Left ventricular pseudoaneurysm possesses significant perioperative mortality. The primary goals are to maintain hemodynamic stability, avoid excessive stress on the myocardium, and prevent any further injury to the affected region of the heart and thereby preventing rupture of the left ventricle.

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