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Case Report

Perioperative Management of an 86-year Female with Left Ventricular Aneurysm with Severe Mitral Regurgitation and LV dysfunction with COPD and Acute Renal Dysfunction A Case Report and a Systematic Review

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Abstract

The aged population can frequently suffer from several comorbidity-like cardiovascular lesions (CAD, hypertension, valvular lesions, ventricular dysfunction, AF, CHF, peripheral vascular disease), respiratory diseases (COPD, asthma), obesity, DM, and renal dysfunctions. The presence of this heavy comorbidities burden can increase the risk of perioperative complications like MI, arrhythmias, CHF, low cardiac output syndrome (LCOS), stroke, respiratory failure and AKI, prolonged hospital stays, and even mortality. Therefore, a thorough preoperative assessment of the severity of each associated lesion and its optimization plays a crucial role in the successful perioperative management in elderly patients undergoing non-cardiac surgery. The guidelines for perioperative anaesthetic management for isolated cardiac and the other mentioned lesions are well described in the literature. However, guidelines for perioperative anaesthetic management of an elderly with a heavy comorbidity burden involving multiple high-risk cardiac disabilities, respiratory, and renal lesions have not been well described and so pose a big challenge. A balanced general anaesthesia (GA) technique with ASA standard and advanced monitoring with PAC and TEE should be tailored to the severity of the patient's comorbidity and the surgical procedure, to provide hemodynamic stability with proper guidance of fluid, inodilators or vasopressor therapy. We describe a successful perioperative anaesthetic management of an 86-year-old female with COPD, left ventricular aneurysm, severe mitral regurgitation and severe LV dysfunction with akinetic basal inferior LV wall undergoing posterior decompression with fixation, and laminectomy with discectomy and fusion. The successful surgical outcome necessitates, a multidisciplinary approach involving a cardiologist, cardiac anaesthetist, neurologist, neurosurgeon, radiologist, and intensivist, along with the expert's consensus due to lack of guidelines for these coexisting multiple systemic lesions, and advanced monitoring including PAC and TEE to guide fluid and drug therapy and to assess and optimize the cardiac functions. Even the availability of an expert cardiac anaesthetist with good knowledge of the interpretation of parameters drawn from PAC and TEE and heightened vigilance is crucial for a successful surgical outcome.

Kew Words: old age; Copd; Lv aneurysm; severe Mr; aki; systolic dysfunctions; ga; spine surgery; tee; pac

Introduction

Elderly patients undergoing non-cardiac surgery might have several comorbidities involving cardiovascular conditions like hypertension, coronary artery disease, and heart failure, valvular lesions, as well as respiratory conditions like COPD and asthma with cor pulmonale, diabetes, kidney dysfunctions, and cognitive impairment.[1,2] These associated comorbidities have a significant impact on the cardiac as well

as non-cardiac surgical outcome.[3] Among these cardiovascular diseases particularly increase the risk of perioperative complications like myocardial ischemia (MI), congestive heart failure(CHF) atrial fibrillation (AF) of valvular or nonvalvular origin (CHA2 DS2 Vasc stroke score), cardiogenic shock, and stroke and mortality.[3] Whereas COPD can cause respiratory failure during or after the surgery, and acute kidney injury may be further aggravated with the use of nephrotoxic drugs like aminoglycosides and NSAIDS. Such patients are at increased risk of perioperative complications, prolonged hospital stays, and even mortality. Overall, the perioperative major adverse cardiac events (MACE) have declined recently to 6.9% in elderly of > 85 years but increases in perioperative ischemic stroke warrant further study [4]. These data highlight risks of non-cardiac surgery among the elderly that warrant increased attention to improve perioperative outcomes. Therefore, thorough preoperative assessment and management of these lesions are crucial for optimizing outcomes.

Since there are no high-grade recommendations available on the perioperative anaesthetic management of elderly patients with LV aneurysm and reduced left ventricular ejection fraction (LVEF), with severe ischemic mitral regurgitation (MR), and COPD and acute kidney injury (AKI) undergoing major spine surgical procedure. The anaesthesiologists must extrapolate data from non-surgical studies to manage these patients. Even fluid administration guidelines with respect to the body weight, fasting period, and severity of surgical procedures cannot be followed very strictly as inadequate preload can decrease cardiac output (CO), and over volume can precipitate heart failure and lung congestion. We describe the successful perioperative Anaesthetic management of an 86-year female with a weird combination of multiple comorbidities including left ventricular aneurysm, severe Mitral regurgitation, and LV dysfunction with akinetic basal wall, and COPD and AKI, who has undergone a posterior decompression with fixation, and laminectomy with discectomy and fusion after obtaining an informed consent. As there is a lack of recommendations in the literature on the perioperative management of the elderly with such a heavy comorbidity burden, the clinical presentation and pathophysiology of the individual lesion were considered, and experts' consensus and a multidisciplinary approach played a vital role in the successful surgical outcome. A balanced general anaesthesia (GA) technique with ASA standard and advanced monitoring with pulmonary artery catheter (PAC) and trans-oesophageal echocardiography (TEE) were employed to achieve perioperative hemodynamic stability. [5,6].

Case Presentation

An 86-year-old female, weighing 47 kg diagnosed as Prolapsed Intervertebral Disc (PIVD) for the last 3 months was posted for an urgent posterior decompression with fixation, and laminectomy with discectomy and fusion. The patient's history was suggestive of back pain radiating to both lower limbs and unable to stand and even affecting bowel and bladder control. She was also a known case of hypertension for 2 years but not compliant with any medication. She was a regular smoker for the last 15 years. She was in New York Heart Association (NYHA) Class III cardiovascular disability for the last 3 months, as she had dyspnoea (shortness of breath) and fatigue, palpitations, and angina with less than ordinary physical activity, but did not seek any medical advice. She was also hard of hearing, and MET was <4 suggestive of an increased risk of cardiac complications or arrest during surgery. She was referred to neurosurgery department.

On examination, pulse rate was 84beat/min and blood pressure (BP) was 138/88 mmHg. She was edentulous and airway assessment revealed MPG class 2, with adequate neck movements and mouth opening of more than 3 fingers. The examination of the cardiorespiratory system revealed bilateral diffuse rhonchi without any added sounds, and the apex beat was in the 6th ICS in the midaxillary line, and auscultation of the heart revealed a holosystolic murmur at the apex of the heart suggestive of mitral regurgitation with enlarged left ventricle (LV). Her muscle power in upper limbs and lower limbs were 5/5 and 2/5 respectively.

She was a patient of PIVD of the lumber region with a very heavy comorbidity burden including cardiovascular, respiratory and renal system. So, a thorough preoperative evaluation was required. Laboratory investigations revealed HB of 10.3gm%, total leucocyte count of 10260 cells/cumm,

platelets of 2.43 lacs/cumm, HBA1c- 6.5%, total bilirubin/direct/ indirect were 0.2/0.1/0.1mg/dl respectively and SGOT/PT 20/12U/L respectively, ALP of 85 u/l, serum sodium/ potassium were 136/ 5.1 Meq /L respectively, total proteins were 5.65 gm/L, and serum calcium was 9.4 mg/dl, serum creatinine 1.43 mg/dl and blood urea 76.2 mg/dl and international normalized ratio(INR) was 0.97 and PT was 12.1 seconds. Electrocardiogram (ECG) showed LVH and LV strain (ST/T changes), and left bundle branch block (LBBB) (M pattern QRS) with frequent VPCs. [**Figure-1**] Chest X-ray AP view showed mild cardiomegaly with suspected focal lateral bulge arising from the left heart border suggestive of LV aneurysm. and normal lung fields. [**Figure-2**] she was not subjected to the breath-holding time and pulmonary function test due to a risk of fatal arrhythmias in the presence of severe cardiac disability. Even cardiac cath study and CT angiography to reveal coronary artery lesions could not be performed due to renal dysfunction, bronchospasm and low EF. Transthoracic echocardiography revealed Mild LVH, moderate mitral regurgitation, LV aneurysm and thinning of the posterior, inferior wall and at the basal segment, and akinesia of the basal, inferior and posterior wall, and hypokinesia of the inferior wall and a regional wall motion score index (WMSI) >1.7 suggestive of impending heart failure. In addition, EF was 30% and mitral inflow velocity revealed E<A, suggestive of grade 1 diastolic dysfunction and there was no thrombus in the LV aneurysm.

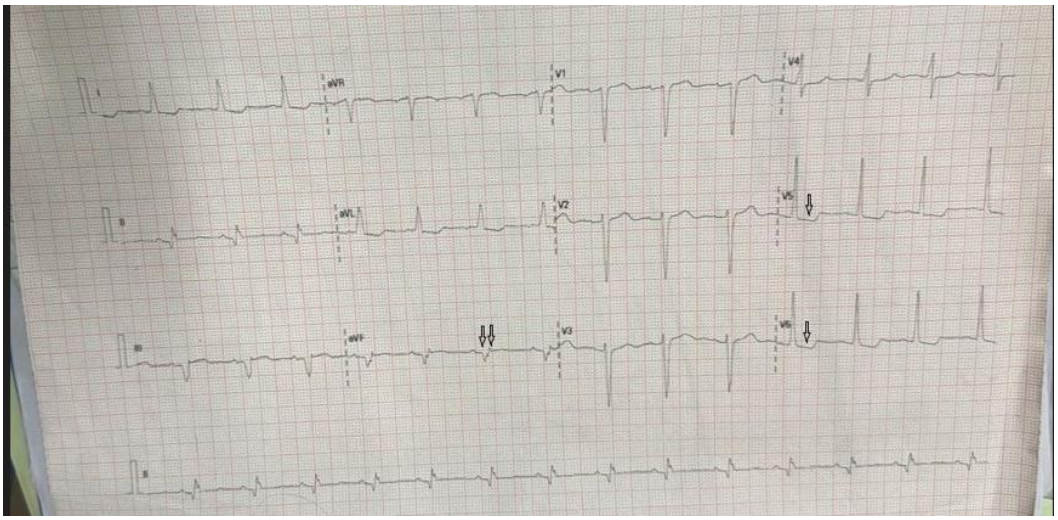
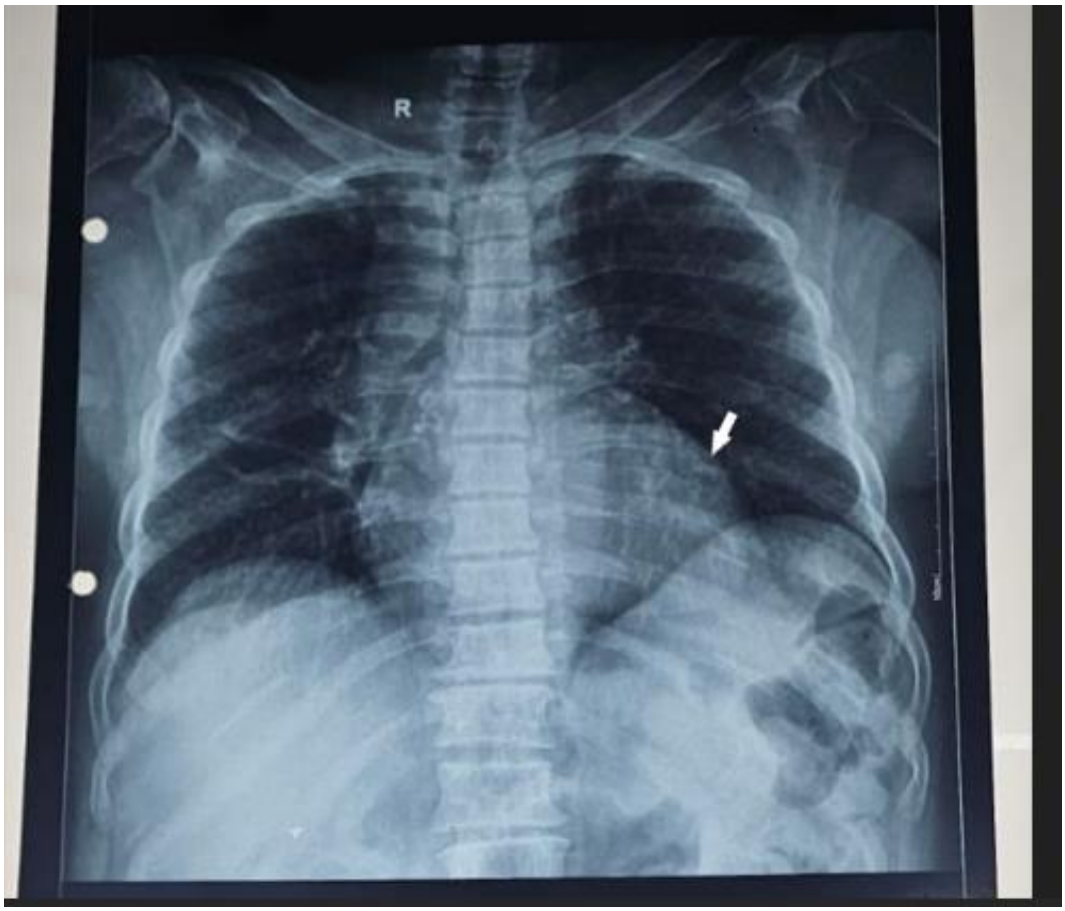


Figure-1: 12 Lead Electrocardiogram (ECG) shows generalised ST, T changes and Left bundle branch block (M pattern of QRS) (2 arrows), The ST/T strain changes) (one Arrow), most likely a combination of LVH and MI.



LVH- left ventricular hypertrophy, LV- left ventricle

Figure-2: Chest X-ray AP view shows mild cardiomegaly with suspected focal lateral bulge arising from the left heart border (arrow) suggestive of LV aneurysm. The carina is on the right side, but lung fields are normal.

Ideally, the patient should be referred for cardiac surgery for the LV aneurysm as posterior or postero-basal aneurysm endoventricular patchplasty using a ventriculotomy or through a transatrial approach with bovine pericardium, along with the mitral valve repair or replacement, and coronary revascularization before non -cardiac surgery, or even a combined cardiac and spine procedure to avoid the cardiac complications or sudden death.[7] However, considering the old age (86 year), heavy comorbidity burden (COPD, AKI) the cardiac surgery was denied by the several tertiary care cardiac centres. MRI dorsolumbar spine revealed spondylitis changes at multiple levels, described as grade-2 anterolisthesis

of L4 over L5 vertebrae with bilateral partial interarticularis defects of L4 vertebrae suggestive of spondylolisthesis. In addition, there was a spinal canal narrowing with ligamentum flavum hypertrophy, multilevel intervertebral disc desiccation and degeneration and compression of the spinal cord at D10 – D11 level with compressive myelopathy at this level. Given the severe progressive neurological dysfunction, it was decided to post her for the emergency posterior decompression with fixation with laminectomy with discectomy and fusion. Detailed counselling was done and informed high-risk consent was obtained from her and relatives in view of the heavy comorbidity burden and old age.

According to some authors, dexmedetomidine and budesonide potentially prevent bronchospasm during procedures and reduce cough, improving patient comfort and tolerability of the surgical procedure and so, on the day of surgery, she received a pretreatment with nebulization with budesonide and dexmedetomidine (0.5mcg/kg) prior to the induction of general anesthesia. [8,9]

In the operation room (OR), standard ASA monitoring was instituted with pulse oximetry, ECG with automated ST segment analysis, non-invasive BP monitor, and ETCO2. Her basal HR was 96bpm with few ventricular premature contractions (VPCs) and arterial oxygen saturation was 93% on room air that improved to 99% on preoxygenation. A 20 G cannula was inserted in the right radial artery for invasive arterial pressure and serial blood gas analysis. Infusions of dobutamine (5mcg/kg/min) and nitroglycerine (NTG) (0.5 mcg/kg/min) were started as cardiac support to maintain cardiac contractility and reduction in systemic vascular resistance(SVR) and pulmonary vascular resistance (PVR) before induction of anaesthesia. GA was induced with etomidate 0.3 mg/kg body weight, midazolam 2mg, fentanyl 2mcg/kg, and vecuronium 0.1 mg/kg body weight was used to facilitate endotracheal intubation with Flexometallic 7.5 mm, ID tube, fixed at 20cm. Lignocaine 60 mg intravenous was given to attenuate laryngoscopy stress response. Magnesium sulphate (2gm) in normal saline was infused over 30 min as a prophylaxis against arrhythmias. 7 FG, pulmonary artery catheter inserted via the right internal jugular vein to assess the PAP, MPAP, CVP, PCWP, CO, CI, PVR, SVR and PVRI and SVRI and left ventricular stroke work index (LVSWI), right ventricular stroke work index (RVSWI), and mixed venous oxygen saturation(SvO2) to guide the fluid and vasoactive agents and to assess the efficacy of the instituted therapy. The perioperative hemodynamics were well maintained as shown in **Table-1.**

PCWP also revealed a large A wave suggestive of mitral regurgitation. A gastric tube was inserted to aspirate the stomach secretions and air and removed, and an adult TEE probe (GE, 6TC-RS, Vivid T9 v205, Norway) was inserted. Anesthesia was maintained with 60% oxygen in the air, sevoflurane 0.75- 1.5 % and vecuronium infusion (2mg/hr) with intermittent fentanyl and midazolam boluses. [9] She was positioned prone for the surgical procedure. External defibrillator pads were applied before turning the patient prone, and all resuscitation and antiarrhythmic drugs were kept ready to tackle an inadvertent arrhythmic event. The TEE was performed in the supine position and continued in the prone position with a little technical difficulty. TEE revealed an EF of 36%, fractional shortening (FS) of 17% after inodilator support, severe MR with central jet, MR flow velocity > 5 m/sec and vena Contracta of 0.8mm(Figure 3a,b) and CO of 2.65 L/M^2, mitral valve area of 4.3cm2 by planimetry, LA -31mm. (Figure 4) In addition LV basal wall aneurysmal, hypokinesia of basal, posterior and inferior LV wall, and no thrombus in LV(Figure 5a,b) and LV type 1, diastolic dysfunction (E<A) were also revealed on mitral inflow velocity interrogation, **(Figure 6**) but the RV functions and IVC size and collapsibility on respiration were normal.

Parameter (units)	Preoperative Vitals	Intraoperative Vitals	Postoperative Vitals
HR, /min	84	96	81
BP(mmHg)	120/58	100/60 - 108/75	110/68
MAP (mmHg)	78.7	85- 65	82 -65
SpO_2 (%)	92% on RA	98	100%
PAP, (mmHg)	—	40/20 (Mean: 27)	—
CVP, mmHg	—	13–14	—
PCWP,(mmHg)	—	21	—
MPAP,(mmHg)	—	18	—
SVR,(dyn·s/cm^5)	—	1841- 1260	—
CO,(L/min)	—	2.65	—
PVR,(dyn·s/cm^5)	—	272 to 181	—
EF	30%	45%	45%
SvO2	-	65%	-

Table 1: Showing hemodynamic data before, during and after surgery

HR-heart rate, BP-blood pressure, MAP- mean arterial pressure, SPO2-arterial oxygen saturation, PAP-pulmonary artery pressure, CVP- central venous pressure, PCWP- Pulmonary Capillary Wedge Pressure, MPAP-mean pulmonary artery pressure, SVR- systemic vascular resistance, CO-cardiac output, PVR- pulmonary vascular resistance, EF- ejection fraction, RA- room air, SvO2- mixed venous oxygen saturation PCWP also revealed a large A wave suggestive of mitral regurgitation. A gastric tube was inserted to aspirate the stomach secretions and air and removed, and an adult TEE probe (GE, 6TC-RS, Vivid T9 v205, Norway) was inserted. Anesthesia was maintained with 60% oxygen in the air, sevoflurane 0.75- 1.5 % and vecuronium infusion (2mg/hr) with intermittent fentanyl and midazolam boluses. [9] She was positioned prone for the surgical procedure. External defibrillator pads were applied before turning the patient prone, and all resuscitation and antiarrhythmic drugs were kept ready to tackle an inadvertent arrhythmic event. The TEE was performed in the supine position and continued in the prone position with a little technical difficulty. TEE revealed an EF of 36%, fractional shortening (FS) of 17% after inodilator support, severe MR with central jet, MR flow velocity > 5 m/sec and vena Contracta of 0.8mm(Figure 3a,b) and CO of 2.65 L/M^2, mitral valve area of 4.3cm2 by planimetry, LA -31mm. (Figure 4) In addition LV basal wall aneurysmal, hypokinesia of basal, posterior and inferior LV wall, and no thrombus in LV(Figure 5a,b) and LV type 1, diastolic dysfunction (E<A) were also revealed on mitral inflow velocity interrogation**, (Figure 6**) but the RV functions and IVC size and collapsibility on respiration were normal.

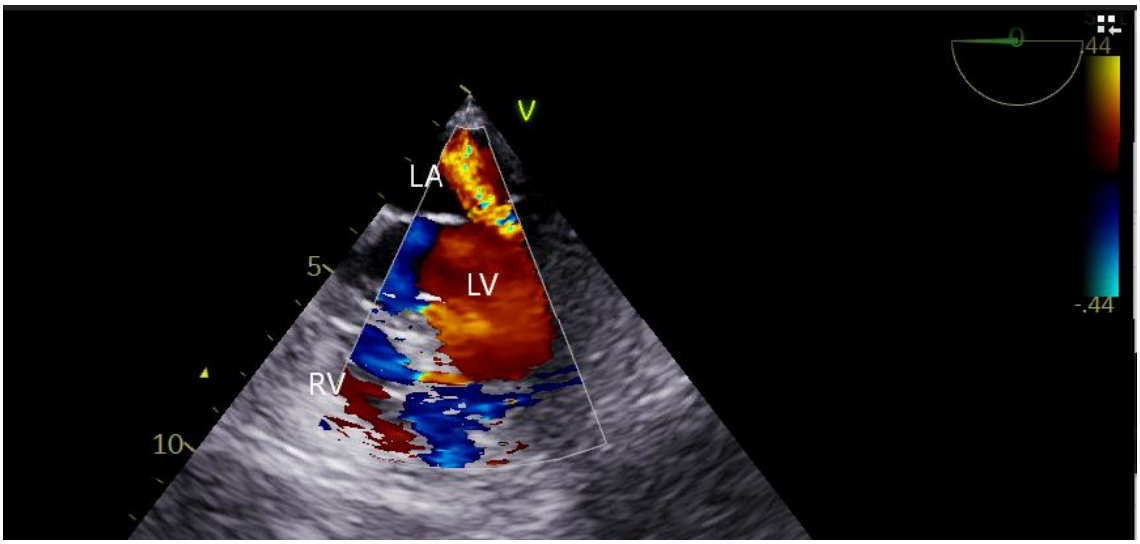


Figure-3a: Colour Doppler of the modified ME 4 C view (0°) of TEE shows central jet of MR reaching to the roof of the LA. The vena contracta of 0.8 cm and LA size of 3.1 cm. As LA is still non-compliant (normal size) the TEE findings are suggestive of acute severe MR. it is to be noted that the tenting height was 1.2 cm, indicating significant leaflet tethering and abnormal leaflet motion during systole as a result of MI. [labelled]

ME4C – mid esophageal 4- chamber view, TEE- trans esophgeal echocardiography, LA- left atrium, MI- myocardial ischemia

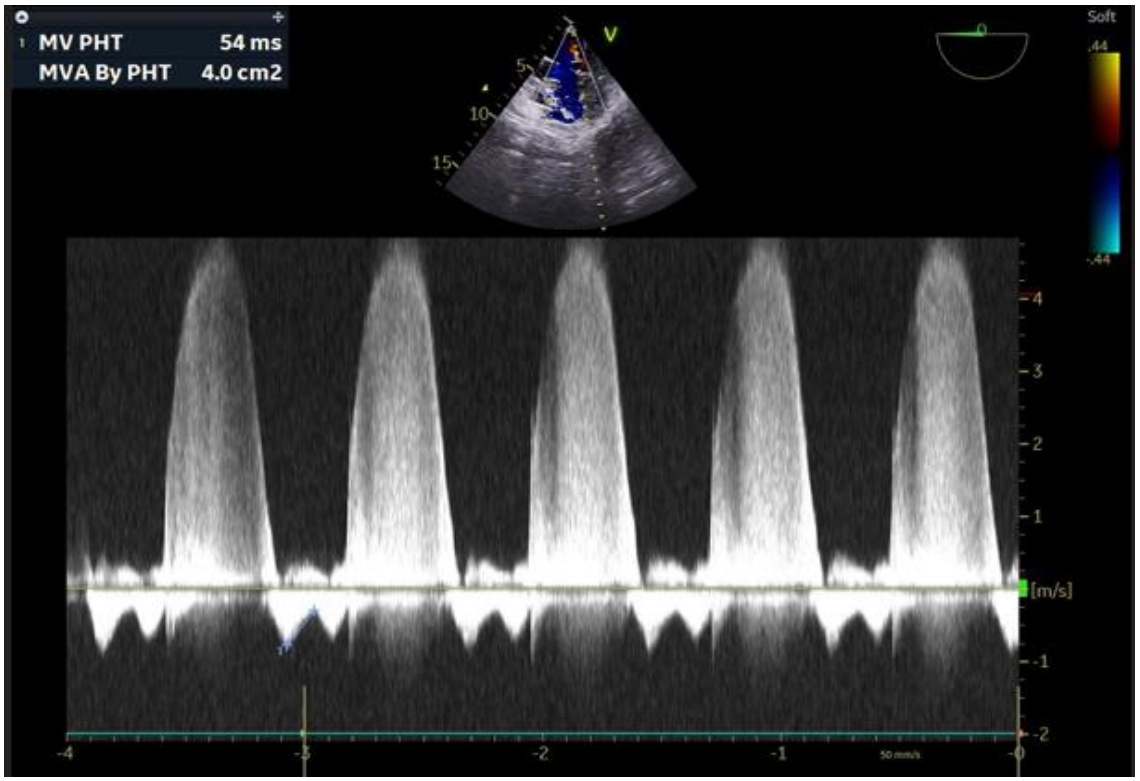


Figure-3b: The ME4C view (0°) 2D, TEE image displays that the CWD of the mitral valve revealed a flow velocity of 5.4 m/sec. and the velocity is thick, well enveloped and triangular suggestive of severe MR.

ME4C- mid esophageal 4 chamber view, CWD- continuous wave doppler, MR- mitral regurgitation

Figure 4: Deep TG LAX view of TEE showing the PWD interrogation of the aortic valve and LVOT and tracing the spectral display and measuring the VTI (green thick line) and R-R distance (green dotted lines). The machine calculates the SV and CO based on the entered value of the LVOT diameter and VTI and multiplying VTI and HR (R-R distance) on the machine and so CO and CI are calculated automatically by the TEE computer. In this patient the CO is 2.62 L/min and SV was 31 ml suggestive of severely reduced CO

CO= SV x HR,

SV= π x (LVOT D/2)2 x VTI

OR SV= CSA LVOT x VTI LVOT

TG LAX- trans gastric long axis, TEE -tranesophageal echocardiography, PWD- pulse wave doppler, LVOT- left ventricle out flow tract, VTI- velocity time integral, SV- stroke volume, CO- cardiac output, CI- cardiac index

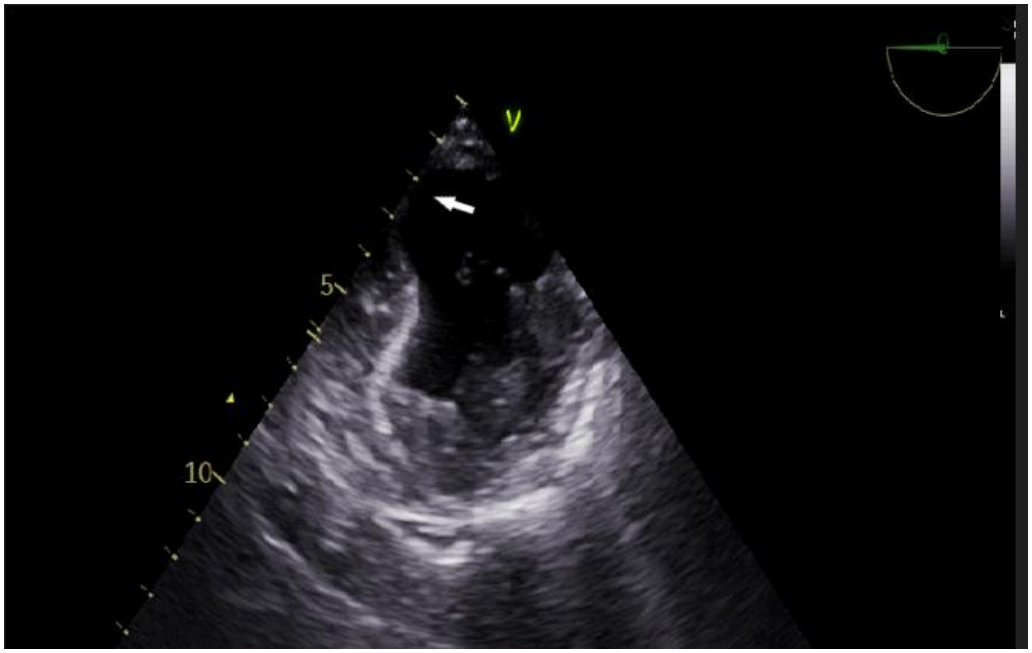


Figure-5a: TG basal LV SAX view, 2D- TEE imaging shows an inferior- posterior wall LV aneurysm (white arrow) with wall thinning and MV in systole.

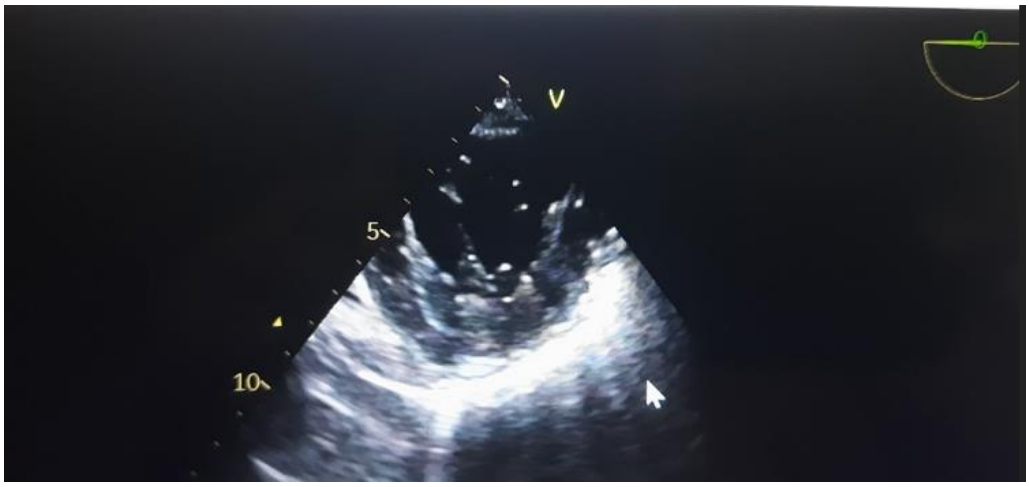


Figure-5b: Video of TG- basal LV SAX view – 2D TEE imaging shows the thin and aneurysmal inferior wall of the LV, most likely due to CAD involving PDA branch of RCA or LCx. In addition, mild to moderate concentric LVH is also confirmed on TG mid papillary view.

TG basal LV SAX- trans gastric basal left ventricle short axis, 2D- 2 -dimensional, CAD- coronary artery disease, PDA- posterior descending artery, RCA- right coronary artery, LCx – left circumflex artery

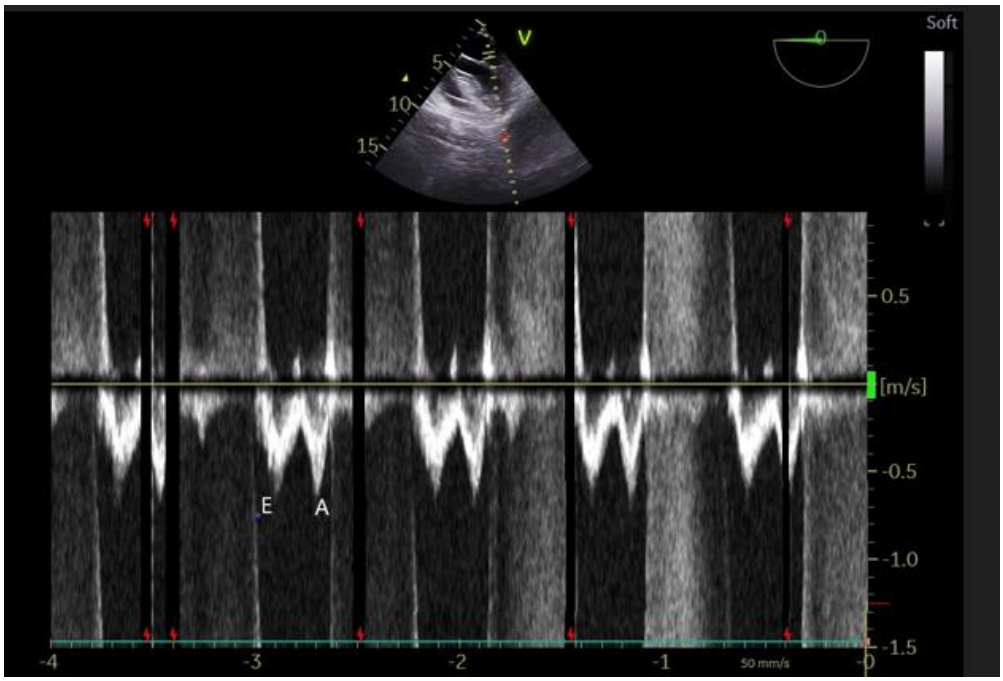


Figure-6: Mitral inflow velocity revealed E wave < A wave suggestive of type 1 diastolic dysfunction. in this patient diastolic dysfunction can occur due to CAD, hypertension, concentric LVH, IMR or LV aneurysm as well.

CAD- coronary artery disease, LVH- left ventricular hypertrophy, IMR- ischemic mitral regurgitation, LV- left ventricle.

In addition, the perioperative hemodynamic parameters and cardiac functions and volume status were also assessed using IVC size (1.7 cm) with >50% collapsibility in inspiration, biventricular filling and CO, which remained stable throughout the procedure and the heart rate was maintained between 80-90 beats/min in view of severe MR. TEE also confirmed the improvement in LV contractility and EF following the administration of dobutamine and NTG. Table -1 and (Figure 5b-video 1) The intraoperative ABG analysis revealed a pH- 7.31, PCO2- 51 mmHg, Pao2- 118 mmHg, SaO2 99%, Na+ -135 mmol/L, K+ 4.5meq/l, Ca+ 0.89mmol/L, glucose-118mg/dl, Hct- 46%, HCo3- 21.9mmol/l, BE-8.4 mmol/l and the values were acceptable in view of the COPD. The procedure lasted for 6hrs. Total blood loss was 450 ml, and 1.5 L of normal saline and one unit of fresh packed red blood cell transfusion were given, and total urine output was 1.5 L. At the end of the surgery, she was turned to supine, the neuromuscular block was reversed with standard doses of neostigmine and glycopyrrolate and she also required sugamadex (100 mg) for complete recovery and was smoothly extubated in OR. Inotropes were continued and tapered slowly over 6-8 hours and PAC was also removed in the PICU after 2 hours but the sheath was left in situ. Postoperative multimodal pain management was achieved using tramadol, i.v. paracetamol (1gm), fentanyl (25 mcg) boluses, transdermal buprenorphine patch (5mg). The patient was observed overnight in the postanaesthetic care unit and discharged on the 7th postoperative day with the instructions to attend the OPD for follow-up after one week.

Discussion:

The guidelines on the perioperative anaesthetic management of patients with such a complex clinical scenario with multiple comorbidities are lacking. The clinical spectrum of such a patient is highly complex and variable, so it is not possible to be prescriptive. Often, the anesthetic management is based on clinical experience with the technique and availability of the resources like PAC and TEE and the institutional protocol. Perioperative management is driven by the expert's consensus, involving a multifaceted- patient-centred approach, tailoring the anesthetic plan to individual needs and risk factors and the complexity of the procedure, and potential adverse events, and also balancing the benefits and risks of different techniques to optimize patient outcome.[10,11] The pathophysiology and the clinical presentation of the individual lesion should be considered for planning the anesthesia technique to achieve the hemodynamic goals. Therefore, the expert's consensus plays a vital role in successful perioperative management. Finally, the management involves a multidisciplinary approach comprisingcardiologist, neurosurgeon, radiologist, pulmonologist, cardiac surgeon and cardiac anesthetist, and intensivist for the postoperative hemodynamic management. It is worth describing here the impact of each lesion on the hemodynamics and the patient's outcome including major adverse cardiac events (MACE) and mortality.

Preparation of the OR

In preparation of the OR, several key steps are crucial based on the thorough patient clinical assessment, type of surgical procedure, and preparation of the emergency equipment and drugs. First and foremost is the availability of an expert in dealing with such patients. In addition to standard ASA monitors, arrangements for advance monitoring including intra-arterial blood pressure and CVP and PAC, and TEE or non-invasive CO are essential for the assessment of the cardiac functions, hemodynamics and to identify the complications, and BIS for depth of anaesthesia. [1,2,3] A pacemaker facility to counter complete heart block and defibrillator for SVT, AF or VT or VF should be available. Monitoring with evoke potential SSEM, and MEP for spine surgery is essential as this patient already had decreased power in lower limbs. Essential drugs and equipment to manage the hemodynamics and antiarrhythmics should be available like beta- blockers(metoprolol, atenolol, esmolol), calcium channel blockers (diltiazem, verapamil), ivabradine, amiodarone, lignocaine, magnesium sulphate, calcium gluconate, dexmedetomidine, and inotropes and vasopressors like dobutamine, milrinone, levosimendan, epinephrine, norepinephrine, vasopressin, phenylephrine, or even nitroglycerine or sodium nitroprusside, and labetalol to control the hypertension. Lusitropic agents promote cardiac relaxation and efficiently fill the LV during the diastole including, NTG, dobutamine, milrinone, levosimendan and CCB are essential to attenuate diastolic dysfunctions and PAH and play a vital role in maintenance of cardiovascular stability.[15]

Lastly, there should be a backup plan for mechanical circulatory support like intra-aortic balloon counter pulsations (IABP), left ventricular assist devices (LVADs), right ventricular assist devices (RVADS) and extracorporeal membrane oxygenator (ECMO) to manage the refractory hemodynamic deterioration due to LV systolic and diastolic dysfunctions or even severe MR or LV aneurysm despite the goal-directed medical therapy (GDMT).[16,17,18]

Impact of ischemic Mitral Regurgitation (IMR):

Patients with ischemic mitral regurgitation (IMR) experience a higher risk of perioperative congestive heart failure (CHF), arrythmia, perioperative MI, cardiogenic shock and mortality.[19,20] LV basal, inferior wall aneurysms secondary to MI can result in clinically significant MR. IMR develops due to outward displacement of the papillary muscles causing tethering and systolic tenting preventing the coaptation of the leaflets due to apical shifting of the coaptation point away from the mitral annulus, or due to dilatation of the mitral annulus, or direct distortion of the leaflets, or even reduced contractility of the posterior mitral annulus due to scarring of the basal LV myocardium.[19] The IMR is triggered by advanced age, posterior-inferior MI, spherical LV, triple vessel CAD, large LA, Low EF and remodelling of the LV due to MI. [19,19]The perioperative IMR might get deteriorated due to the fresh MI precipitated by arrhythmias, impaired myocardial oxygen imbalance due to increased oxygen demand with systemic hypertension, wall stress, tachycardia or with sudden increase in contractility, preload and afterload (systemic vascular resistance), or due to decreased myocardial oxygen supply in anaemia (blood loss), systemic hypotension causing reduction in coronary perfusion pressure, coronary stenosis with lack of collaterals.[19] The Severe MR can further compromise LV function, decrease CO result in systemic hypotension impacting the systemic perfusion, increase LA pressure, PAH, RV or biventricular dysfunction, and worsen heart failure or even sudden death.[20]

LV Volume overload due to severe MR or intravascular volume overload can further dilate the mitral annulus thus increasing the effective regurgitation orifice area (EROA), regurgitation fraction (RF) and regurgitation volume (RV) and worsening the IMR.[19] Effective management requires addressing both the LV aneurysm and the MR to optimize patient outcomes. The presented patient also developed severe MR with central Jet as a result of valve tenting and mitral annular dilatation due to an aneurysm, though morphologically leaflets appeared normal. The perioperative hemodynamic management focuses on increasing forward CO and minimising the regurgitant volume (<30 ml) by avoiding bradycardia, maintaining higher HR (80-90bpm), maintaining LV contractility and reduction in SVR to around 900 dynes/sec/cm5 and PVR to 90 dynes/sec/cm5 with administration of milrinone or dobutamine. A further focus is to optimize the LV preloading, as LV overloading may exaggerate MR by increasing the EROA and RV resulting in a further increase in preexisting PAH and RV dysfunctions, and hemodynamic deterioration due to decreased forward CO. Hemodynamic monitoring and management are crucial for the IMR undergoing non-cardiac surgeries. These include CVP, intraarterial BP, PAC, or even TEE for quantification of MR. [6,7] Therefore, the availability of cardiac anaesthetist with good knowledge of TEE must be ensured during the perioperative management of such patients. In the presented patient TEE was used even in the prone position as it is the most vital tool to assess the intraoperative hemodynamic effects of MR, and helped in guiding the management strategies to optimize the preload, cardiac index, SVR, PVR and for the optimum utilization of inodilators, or even vasopressors. However, TEE evaluation in the prone position is a technical challenge and needs extra vigilance and careful probe manipulation. 2024 AHA/ACC /ACCP guidelines advocate repair only for the severe and symptomatic IMR. Surgical interventions for IMR have centred around the coronary revascularization, repair or replacement of the valve for severe MR using criteria as EROA>0.4cm2, RV >60 ml, regurgitation fraction >50%, and VC >8mm[21] The valve interventions should be considered in very symptomatic severe MR before the elective NCS to reduce the perioperative risk.[22] The transcatheter edge-to-edge repair (TEER) can be considered and following mitral TEER, it is reasonable to perform NCS after a successful TEER.[1], The presented elderly patient with heavy comorbid burden has a higher perioperative surgical risk of morbidity and mortality, making it unsuitable for surgical interventions and denied at several tertiary care centers, even TEER could not be performed due to financial constraints and so increasing the additional challenge for perioperative hemodynamic management during spine fixation surgery. On the contrary, asymptomatic patients with good LV function with PA pressures < 50 mmHg, and EF >55% are reasonably suitable for elective non-cardiac surgery.[23] In the IMR it is prudent to understand the aetiology, severity and hemodynamic consequences of MR prior to the NCS, as low EF, AF and PAH may contribute to the increased perioperative risk. In addition, postoperative anticoagulants should be considered in patients with new-onset AF in NCS to prevent increased long-term stroke and mortality. [24,25]

Impact of LV systolic and diastolic dysfunctions:

The LV systolic and diastolic dysfunctions triggering is multifactorial including CAD, systemic hypertension, valvular heart lesions, malignant arrhythmias, and myocardial disease i.e. hypertrophic or dilated cardiomyopathy and myocardial inflammation and infection(endocarditis) [26,27] Systolic dysfunction is always a risk factor for the development of perioperative MACE including cardiogenic shock, LCOS, MI, arrhythmias and sudden death.[28,29] The risk factors for perioperative morbidity and mortality are recent MI, CHF, peripheral vascular disease, angina pectoris, diabetes mellitus, hypertension, hypercholesterolemia, dysrhythmias, obesity, smoking, and renal dysfunctions.[30,31]

Treating the underlying cause is of extreme importance as some etiological conditions for systolic dysfunctions may be reversible like CAD, cardiomyopathies induced by alcohol, tachycardia, and control of blood pressure can also help prevent further deterioration.[32,33,34] In patients with the acute coronary syndrome (ACS) for elective NCS, ideally, coronary revascularization is recommended and NCS should be deferred to prevent the perioperative MACE, also the chronic CAD with left main coronary >50% lesion and even hemodynamically stable, it's appropriate to consider revascularization and deferral the elective NCS to avoid the MACE. However, in non-left main CAD for elective NCS, revascularization is not recommended to reduce the MACE. [34,35]

Usually, patients with severely reduced EF or diastolic dysfunctions are put on the combination of angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) with a beta blocker (carvedilol, metoprolol). If symptoms don't improve with a maximal dose of ACE inhibitor or ARBs, an angiotensin receptor-neprilysin inhibitor may be substituted. Other medications like hydralazine, nitrates, and mineralocorticoid receptor antagonists such as spironolactone, ivabradine, and digoxin in refractory patients also be required.[36] Recently, sodium-glucose Co transporter 2 inhibitors(SGLT2) inhibitors have become the cornerstone of heart failure management. They reduce the risk of cardiac vascular death and hospitalization for heart failure. Even other adverse outcomes with or without type-2 diabetes or even in patients with chronic renal diseases are prevented. The most likely mechanism is improved cardiac energy metabolism, decreased inflammation, better renal functions and positive erythropoiesis. In addition, these block the reabsorption of sodium in the renal proximal tubules resulting in natriuresis and osmotic diuresis due to glucosuria with blood pressure lowering effect. Sota-gliflozin (200-400 mg/day) with a combined SGLT 1&2 inhibition; 2 action leads to excretion of glucose in urine and 1 delays the intestinal glucose absorption. However, these should be discontinued for 3-4 days before the surgery to avoid metabolic acidosis and euglycemic ketoacidosis, prolonged hospital stay and death.[37,38] All cardiac medications should be continued on the day of the surgery except ACE inhibitors or ARBs to potentially reduce the risk of intraoperative hypotension which might drive postoperative complications such as acute kidney injury, myocardial injury, or stroke.[38,] Regional anaesthesia technique is often a viable option with reduction of afterload and stress response of the surgery and so preferred in severe LV dysfunction. The key considerations in low EF or systolic dysfunctions are to maintain cardiac contractility with an infusion of milrinone or dobutamine or /and levosimendan, and optimization of the preload(CVP 0f 7-8 mmHg and PCWP of 10-12), and sinus rhythm at HR of 60-70 bpm for adequate LV filling and afterload (SVR) should be reduced to minimize the LV workload and avoid conditions that could exacerbate ischemia like tachycardia, hypertension as well as hypotension, anaemia, surgical stress and AKI and COPD.[40,41]

In addition, maintain the adequate myocardial oxygen supply, with the lowest feasible myocardial oxygen demand to avoid the MI and further deterioration of the LV contractility and stiffness. Therefore, it is imperative to maintain adequate analgesia, depth of anaesthesia and volume status, and additional use of beta blockers and vasopressors.[32] If general anaesthesia is necessary then a balanced anaesthesia technique comprising etomidate (0.2-0.3 mg/kg) or ketamine (2mg/kg), midazolam (0.05 mg/kg), fentanyl (2 mcg/kg) or remifentanil (2-5mcg/kg), and sevoflurane (1-1.5 MAC) and a neutral muscle relaxant like vecuronium (0.1mg/kg) or atracurium (0.5 mg/kg or cisatracurium (0.2 mg/kg) should be preferred. In addition, the use of isoflurane and sevoflurane also has myocardial protection effect via the pharmacological cardiac preconditioning and thus reduces myocardial stunning, attenuates inflammatory responses, and improves post-operative cardiac function. [12,3,4,5] Some patients with overloaded ventricles may require diuretic therapy. The intraoperative monitoring is determined by the severity of LV dysfunction and the surgical procedure. The standard ASA monitoring (ECG, SPO2, BP, ETCO2, temperature) as per case is sufficient or may require extensive monitoring including PAC with CVP, invasive arterial BP, BIS, NIRS and even TEE. The presented patient was also suffering from severe LV systolic and grade 1 diastolic dysfunction and managed with the mentioned anaesthesia technique and monitoring. Echocardiography, as per AHA/ACC guidelines is the single most useful diagnostic modality in the evaluation of a patient with systolic/ diastolic dysfunctions. However, TEE has been increasingly used as a perioperative monitoring tool in patients with high-risk factors or undergoing high-risk noncardiac procedures.[45] TEE provides real-time detailed imaging of the heart, and allows for continuous monitoring of cardiac output, filling pressures, and ventricular function, which are vital for guiding fluid management and drug therapy during surgery in this subset of patients.[46,47]

S. No	Parameter	Normal	Mild	Moderate	Severe	Notes
1.	EF	55–70%	41–49%	30–40%	<30%	
2.	FAC	>35%	25–35%	20–25%	<20%	
3.	FS	25%	16–25%	15-19%	<15%	
4.	SV	70-100 ml				
5.	MAPSE	12-15 mm		10–14 mm	<8 mm with LVEF <50%	Specificity= 82% Sensitivity =95%
6.	GLS	>18%			<16%	
7.	dp/dt [mmHg/s]	>1200	800-1200	<800	<500	

Table 2: Echocardiography Parameters Used for Assessment of LV Systolic Dysfunction [47,48,49]

*Formulas/Notes:

*EF (Ejection Fraction) = (LVEDV - LVESV) / LVEDV × 100

*FAC (Fractional Area Change) = (EDA - ESA) / EDA × 100

*FS (Fractional Shortening) = (LVEDD - LVESD) / LVEDD × 100

*MAPSE = Mitral Annular Plane Systolic Excursion

*GLS = Global Longitudinal Strain via speckle tracking

Systolic index of contractility= dp/dt (P2 − P1 × 1000/Δt (time for velocity to rise to 3 m/s from 1 m/s) by MR flow velocity

SV (Stroke Volume) = CSA × VTI (velocity time integral [VTI]) & (LVEF) represents SV as a percentage of end-diastolic volume.

LV diastolic dysfunctions or heart failure with preserved EF(HFpEF) is called as diastolic heart failure with reduced LV relaxation or increased stiffness in the diastole that limits the proper filling of LV. Diastolic dysfunctions can be adequately assessed by TEE using the following parameters; mitral inflow velocity, mitral annular tissue doppler interrogation(TDI) for e' and a' prime, LA volume, TR velocity, pulmonary venous flow velocity.[48] The criteria to determine the severity of diastolic dysfunctions have been described in Table-3.[49,50,51] However, it's not possible to provide the details of these diastolic dysfunction parameters in this current review. It has been reported that regional anesthesia or neuraxial block reduces the 0-30 days mortality in patients undergoing surgery with an intermediate or higher cardiac risk. [6] The important hemodynamic goals are to maintain MAP > 65 mmHg, low HR [60-70], and maintain SVR and contractility, sinus rhythm and normovolemia, and avoid tachycardia and myocardial ischemia. Treatment with beta-blockers or non-dihydropyridine calcium-channel blockers has been proposed to prevent tachycardia and improve LV diastolic filling. Patients with severe diastolic dysfunction may require more aggressive management, including inotropic support, vasopressors, or mechanical ventilation and TEE can be useful in early diagnosis and management of diastolic dysfunctions.[51] The lusitropic agents like calcium channel blockers, ACE inhibitors, inhalational agents or inodilators (milrinone, dobutamine, levosimendan) and NTG can be utilised to ameliorate the perioperative diastolic dysfunction. [53,54]

Echocardiography Parameter	Description
Average E/E'	> 14
Septal E' / Lateral E'	< 7 cm/s or < 10 cm/s
Left Atrium Volume Index (LA Volume Index)	> 34 ml/m²
A-ar Duration (A)	> 30ms
L-wave Velocity &TR velocity	> 20 cm/s & > 2.8 m/s
Pulmonary Vein S/D Ratio (PV-S/D)	< 1
Mitral E-deceleration Time	< 150ms

Grade of Diastolic Dysfunction	Description
Grade I (Impaired Relaxation)	E/A < 1, normal Left Atrium Pressure
Grade II (Pseudo normal)	E/A 0.8-1.5, appears normal reverses with Valsalva
Grade III (Reversible restrictive)	E/E' > 2.0, E-deceleration < 150ms, E/A < 1 with Valsalva
Grade IV (Irreversible restrictive)	E/A > 2.0 even with Valsalva, high filling pressure

E = Mitral Inflow Velocity; A = Mitral Inflow Velocity (A-wave);

S/D = Pulmonary Vein Flow Velocity systolic and diastolic; PV = Pulmonary Veins Flow Velocity, ar – atrial reversal wave, E,- e prime wave on mitral annular TDI

Table 3: Echocardiography Parameters used to Diagnose LV Diastolic Dysfunction and Grade of Diastolic Dysfunction, 50% of the above are suggestive of Diastolic Dysfunction. [50,51,52,53,54].

Impact of concentric LVH:

Severe LVH is always a risk for alterations in diastolic compliance, deterioration of the intrinsic contractile performance of the myocardium, potential myocardial ischemia due to decreased coronary flow reserve, and sub-endocardial ischemia due to reduced blood flow, and potential imbalances in the myocardial oxygen supply and demand relationship.[19] Maintaining adequate preload, and afterload, sinus rhythm, avoiding tachycardia and sympathetic stimulus, and minimizing factors that increase myocardial contractility are crucial for a successful anesthetic outcome. Avoidance of fluid overload and maintaining normovolemia, and appropriate blood pressure control (MAP > 65 mmHg) are essential in LVH with diastolic dysfunction. The hemodynamic goals are like the diastolic dysfunction. [51,55]

Impact of the LV Aneurysm

LV aneurysm can occur due to MI, infective, traumatic, idiopathic or even postpartum. Other causes may predispose to LV aneurysm formation like systemic hypertension, Chagas disease, sarcoidosis and use of steroids, and NSAIDs. [57]

It presents in 30% to 35% of acute transmural MI. The two major risk factors for developing LV aneurysm include total occlusion of the left anterior descending artery (LAD) and failure to achieve revascularization of the infarct site artery. [58,56] Therefore, this patient most likely developed the LV aneurysm due to non-revascularization of the coronary artery. LV aneurysms with potential complications like angina, heart failure, thromboembolism, and ventricular arrhythmias require specific therapies, including surgical reconstruction, anticoagulation, ablation of arrhythmias, implantable devices, and pharmacological therapy for afterload reduction with ACEI and ARBs.[57] In patients with ventricular aneurysms complicated by ventricular tachycardia, implanted cardioverter-defibrillators may reduce the risk of sudden death. Electrophysiological mapping is critical to determining whether to pursue surgical or percutaneous ablation. [58]

However, surgical intervention in the presented patient for aneurysm and severe MR was denied due to old age and heavy comorbid burden, and despite the severity of the cardiac disability even pharmacological therapy was not used by her to optimize the cardiac functions, and therefore it was a unique challenge for the perioperative care providers.

Patients with LV aneurysms secondary to CAD (85-90%) face increased risks during noncardiac surgery, due to the potential for complications like heart failure, hypotension, arrhythmias, thromboembolism, LCOS, ventricular rupture and cardiac tamponade.[61] Specifically, the risk of perioperative mortality and morbidity is higher in patients similar to those

described by us. The concomitant conditions like LV aneurysm, severe MR, LV dysfunctions, and COPD, PAH, AKI, and fluid overloading, surgical or anaesthetic stresses can further increase the perioperative risk of heart failure and arrythmia. Hence, perioperative optimization of cardiac contractility, blood pressure, HR and fluid balance is prime important. Therefore, ECG along with TEE and PAC are the most vital tools for intraoperative monitoring for early detection of cardiac failure, arrhythmias, LCOS, and even MI by detection of newly developed regional wall motion abnormalities (RWMA) and exaggerated diastolic dysfunction and hypokinesia, the early signs of MI. in addition, RWMA scoring systems is validated as a predictor for heart failure and mortality.[59]

The scoring is calculated as follows; normal motion (score 1), Hypokinesia (score 2), Akinesia (score 3), and Dyskinesia (Score 4). The Wall Motion Score Index (WMSI) is calculated for the 17 segments of the left ventricle. The score is calculated by cumulating the number of points (a score for each segment) and dividing this by 17. When all segments move normally, the ratio is 1 (17 divided by 17). In adults, a WMSI >1.7 is indicative of heart failure. [61,60] According to this WMSI score this patient was in heart failure. [63,61] Therefore, availability of the expertise and vigilant perioperative management is crucial to mitigate these risks and ensure the best possible outcome.

Impact of ventricular premature contractions (VPCs):

During surgery, PVCs can also lead to life threatening complications like hemodynamic instability, LV dysfunction, dilated cardiomyopathy, and sustained frequent and multifocal VPCs can result in VT/ VF and sudden death, in patients with underlying structural heart disease like CAD or cardiomyopathy, or valvular heart diseases and so, require urgent clinical attention.[62]

In Patients with frequent VPCs, continuous cardiac monitoring is crucial during surgery to detect and address any changes in heart rhythm or function. In elderly patients with severe cardiac dysfunction like the presented patient the factors like insertion of CVP or PAC, and Hypovolemia, hypoxemia, electrolyte disorders- hypokalaemia, hypomagnesemia, hypercalcemia, and metabolic disorders (acidosis), hypothermia or hyperthermia, tension pneumothorax, tamponade, toxins, drugs, thromboembolism (pulmonary or cardiac) are the major contributing factors to precipitate intraoperative life-threatening arrythmia. This patient also had multiple VPCs and VT during PAC insertion even at a PAC length of 35cm, however, the hemodynamics were stable and sinus rhythm was restored spontaneously on withdrawal of the catheter. [63] During surgery, management of VPCs should focus on addressing underlying causes, suppressing the arrhythmia, and ensuring hemodynamic stability with the use of beta-blockers, CCB (diltiazem, verapamil) and amiodarone. The VPCs rapidly deteriorating into more dangerous arrhythmias might require cardioversion. [64,65]

Impact of Acute Kidney Injury (AKI):

Renal dysfunction in the setting of heart failure is termed as a cardio-renal syndrome (CRS). AKI is significantly more common in patients with LV or biventricular dysfunctions because of low CO leading to decreased renal blood flow and so GFR or even increasing pressures in the renal veins causing congestion. [56,60] The incidence rate of AKI in acute HF and chronic HF is around 36% (95% CI: 31–40%) and 30% (95% CI: 24–35%), respectively.[67]

AKI following surgery is defined by an increase of ≥ 0.3 mg/dl in serum creatinine within 48 h of ICU admission, an increase of ≥ 50% within 7 days of ICU admission, or requiring acute dialysis, in keeping with the kidney disease improving global outcomes guidelines KDIGO.[68,69] AKI during non-cardiac surgery can be triggered by Preexisting kidney disease, old age, diabetes, heart failure, hypertension, hypotension, hypovolemia, blood transfusion, long duration of surgery, particularly emergency procedures and those involving the abdomen or chest, and sepsis, vasoplegic syndrome, inflammation, increased intraabdominal pressure, anaemia, and nephrotoxic agents like NSAIDS, ACEi and ARBs can also contribute to perioperative AKI.[70] In addition, obesity, liver and respiratory diseases can increase the risk of AKI. AKI following major non-cardiac surgery has consistently been associated with substantial long-term morbidity and mortality. [71,72]

Presently, common practice is to discontinue the use of ACEI and ARBs before surgery to avoid perioperative hypotension and so reduce the risk of perioperative AKI. The focus should be on ensuring euvolaemia prior to the surgery. The guidelines suggesting CO monitoring to optimize cardiac stroke volume and/or increase global oxygen delivery, and avoiding hypotension, is known as goal-directed therapy. A Cochrane review and meta-analysis have suggested that goal-directed therapy can reduce the risk of perioperative AKI.[73] The goal-directed therapy was also adopted during the management of this patient including TEE and PAC, invasive arterial blood pressure monitoring for continuous assessment and optimization of biventricular functions, and MAP> 65mmHg, and urine output along with the serial arterial blood gas analysis.[74,75] Caution is advised when using starch-based colloids and 0.9% saline compared to the balanced crystalloids.[78] One RCT has reported no significant difference in a composite outcome of death or postoperative complications within 14 days of major abdominal surgery between patients who received low-molecular-weight hydroxyethyl starch compared with those who received 0.9% saline.[76,77] The consensus is to use of goal-directed haemodynamic therapy in high-risk patients to optimize volume status, blood pressure and CO to reduce the risk of PO-AKI.[78] finally, the authors believe that an integrated approach for such patients with heavy comorbidities for perioperative-AKI management requires proper evaluations of AKI risk, diagnosis, progress and outcomes throughout the perioperative period, which can be achieved by intensive monitoring, maintaining CO and tissue perfusion, and avoiding LCOS, MI, CHF and malignant arrythmia and respiratory failure.

Impact of Chronic Obstructive Pulmonary Disease (COPD):

Worldwide the most common cause of COPD is cigarette smoking. Smoking cessation has been shown to reduce the deterioration of lung function, and maximum benefit is obtained if smoking is stopped at least 8 weeks before surgery. The mainstay of treatment of COPD is bronchodilation both for maintenance and for exacerbations. Both β-agonists and anticholinergics (ipratropium bromide and tiotropium bromide) are used, and long-term inhaled steroids are usually only indicated in patients with severe COPD and repeated exacerbations. Unfortunately, this patient was not on any bronchodilator therapy and continued with smoking until one day before surgery, and even unsuitable to perform breath holding time or PFT due to severe cardiac disability, so, potential for increased risk of bronchospasm during laryngoscopy and intubation or during emergence from the anaesthesia. COPD patients tolerate a significant degree of chronic hypoxia and often chronic hypercapnia. These patients are commonly very oxygen-sensitive and can rapidly develop type II respiratory failure if given too much supplemental oxygen

During administering the GA, the use of lung-protective ventilation strategies is crucial to prevent complications like hyperinflation and pneumothorax. It includes lower tidal volumes(6-8 ml/kg), I:E ratio (typically to 1:3–1:5) for maximum exhalation, appropriate Positive End-Expiratory Pressure (PEEP), and limiting inspiratory pressures.[71,78] These patients are always at risk of haemodynamic compromise on induction of anaesthesia and initiation of IPPV and therefore, Placement of an arterial catheter should be considered for monitoring of beat-to-beat BP and serial ABG. The existence of cor pulmonale may require aggressive diuresis to reduce peripheral oedema, but peripheral oedema may be present due to heart failure from Severe MR, systolic/ diastolic dysfunction and LV aneurysm and should be managed accordingly. Inhaled therapy provides the mainstay of day-to-day treatment and the

nebulization with budecort (0.5 mg) and dexmedetomidine(0.5mcg/kg) should be performed before administering the GA as a prophylaxis against the bronchospasm. [80,81]

Before extubation, bronchodilator treatment may be helpful. Extubation of the high-risk patient directly to non-invasive ventilation may reduce the work of breathing and air trapping and has been shown to reduce the need for reintubation in the postoperative period after major non-cardiac surgery. [84] one should be aware of the fact that reversal of neuromuscular block with neostigmine in patients with sensitive airways can precipitate the bronchospasm, even reports are available on bronchospasm that developed immediately after sugammadex administration. [82]

Anaesthetic considerations for spine surgery:

Despite the various anaesthesia techniques available, spine surgery is a challenge in view of the prone position, significant blood loss, long duration of procedure, old age, associated comorbidity and complex postoperative pain management.

These patients are potentially difficult airway candidates, particularly for cervical spine procedures. This patient with a severe cardio-respiratory disability was also at risk of Physiologically Difficult Airway which places her at higher risk of cardiovascular collapse and arrest during or immediately after airway approach and conversion to positive pressure ventilation despite the first attempt success. [8,9] These patients might already be in decreased motor or sensory functions as the presented patient also had reduced motor power (2/5) in the lower limbs. During Spine surgery, excessive blood loss is expected as most of the patients are on anticoagulants. So, measures should be taken to minimize the blood loss by surgical positioning, reduce intra-abdominal pressure, electrocautery, topical hemostatic agents(bone wax, collagen foam), attaining a controlled hypotension (MAP 70-80 mmHg), use of antifibrinolytics like tranexamic acid (20mg/kg boluses), Epsilon amino caproic acid (100mg/kg), and infusion of fresh frozen plasma, platelets, cryoprecipitates, fibrinogen, desmopressin(0.3 µg/kg) intravenously, or even recombinant activated factor VII (rFVIIa) 35-90mcg/kg every 2-6 hrs in the first 48 hrs, until bleeding is controlled in refractory cases.[83,84]

Even, avoidance of hypertension is of prime importance in this patient to avoid LV rupture and increase myocardial oxygen demand and even LV failure and MI. There are issues due to prone position like eye injury, nerve injuries of brachial plexus, and ulnar nerve. Pressure on the abdomen should be avoided to prevent respiratory problems, increase in venous pressure including epidural veins that may be the culprit for excessive bleeding or even decreased CO due to IVC compression. [10] Use of CVP/PAC line provides an additional route of intravenous infusions and to remove the air from the RA or RV, if a significant air embolism occurs during spine surgery. Anaesthesia technique specifically employed for the multiple cardiac lesions is also appropriate for spine surgery. However, somatosensory evoked potentials (SSEP) and motor evoked potentials (MEP) should be assessed as spinal cord monitoring, and spinal cord is at risk if the amplitude of SEEP is decreased to <50% of the baseline. [85,86] The total i.v. anaesthesia with propofol is the anesthic technique of choice when the assessment of SEEP and MEP is necessary. However, the primary aim is to maintain the hemodynamic and cardiac functions and to avoid respiratory complications and exaggeration of the preexisting AKI, so a balanced GA technique should be preferred. The PAC and TEE are now more widely used to achieve intraoperative hemodynamic stability. The utility of TEE in detail has been discussed under previous headings, even though the use of PAC has been on the decline over the past decades in cardiac and non-cardiac surgery and medical ICUs. It's worth discussing here the impact of PAC on the management of such patients; The decision to use PAC should be made on a case-by-case basis. In non-cardiac surgery PAC is a valuable tool in high-risk cardiac patients for monitoring and perioperative hemodynamic management by guiding the fluid and drug therapy following appropriate interpretation of the data obtained from it and, even tissue oxygen delivery (DO2) and maximum oxygen consumption (VO2) and mixed venous oxygen saturation can be assessed.[90,91,92] However, it requires expertise to insert the PAC and interpret the data derived from it, as inexperienced anaesthetist can do more harm than benefit to this subset of patients with severe cardiac dysfunction.[95,93] However, Based on SVR, CI, and PADP and PCWP, it will allow the distinction between the low cardiac output and high filling pressure (cardiogenic shock), low cardiac pressure low filling pressure high SVR (hypovolemic shock) or supranormal CO, very low SVR and requiring one or two vasopressors to maintain a MAP >65 mmHg.[86] So, it guides the administration of inotrope (Dobutamine, milrinone, epinephrine, levosimendan) or vasopressors (phenylephrine, norepinephrine, vasopressin) or fluid therapy. However, practical trends of these data have more value than the single value. Patients with pre-existing severe cardiac dysfunctions, PAH, cardiogenic shock, LCOS, and liver and renal failure may benefit from PAC monitoring and its utility to assess the effectiveness of the instituted therapy e.g. inotropes, vasopressors, fluid or even IABP. Likely, the PAC was also utilized for the monitoring and hemodynamic management of the presented patient.

A Multimodal Approach should be adopted for postoperative pain control to minimize opioid use and reduce side effects; including local infiltration of bupivacaine/ ropivacaine at the surgical site, a transcutaneous patch of fentanyl or buprenorphine, systemic analgesics and anti-inflammatory agents like tramadol, PCM, COX-2 specific inhibitors, diclofenac acid, opioids'(fentanyl, morphine, nalorphine) should be used judiciously to avoid the respiratory depression and dexmedetomidine can be used to facilitate the smooth recovery and prophylaxis against nausea and vomiting, as well as nebulization (0.5mcg/kg) to prevent bronchospasm during intubation and should also be included in the protocol for the management of such patients. Even drugs like gabapentin or pregabalin and antidepressants have been used to manage neuropathic pain or enhance the effects of other analgesics. [94,95] extra care should be taken as the use of NSAIDs can exacerbate existing kidney or liver problems.

Conclusion

The guidelines for the perioperative management of elderly with severe cardiac disability including LV aneurysm, IMR, systolic dysfunctions and PAH, along with AKI and COPD are lacking. However, such a patient can be safely managed with multidisciplinary approach, being hypervigilant, using balanced anaesthesia technique along with vasoactive agents to optimize cardiac functions. The presence of the expertise has a crucial role in optimizing the hemodynamic and to perform and interpret the parameters obtained from the advanced monitoring like PAC, TEE, and even of critical arrhythmia on ECG monitoring. The perioperative focus is to avoid complications like physiological difficult airway, deterioration of AKI, MR, PAH, CHF, malignant arrythmias, LCOS, LV rupture, VF and sudden death.

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