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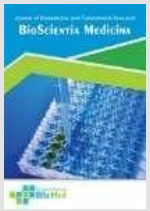

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Anesthetic Management of High-Risk Mitral Valve Replacement in a Young Adult with Posterior Leaflet Prolapse, Biventricular Dysfunction Risk, and Pulmonary Hypertension: A Case Report

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A B S T R A C T

Background: Mitral valve disease with concurrent pulmonary hypertension and biventricular dysfunction represents a complex surgical challenge requiring meticulous perioperative management. This case report presents the anesthetic approach to a 43-year-old male with severe mitral regurgitation secondary to posterior leaflet prolapse, Grade III diastolic dysfunction, and intermediate probability pulmonary hypertension undergoing elective mitral valve replacement. **Case presentation:** The patient presented with 6-month progressive dyspnea, chronic cough, bilateral lower-limb edema, and abdominal distension. Transthoracic echocardiography revealed severe mitral regurgitation with an effective regurgitant orifice area of 2.7 cm^2, bilateral atrial dilation, moderate tricuspid regurgitation, reduced tricuspid regurgitation jet velocity suggesting intermediate pulmonary hypertension probability, and preserved left ventricular ejection fraction of 68% with severely restrictive diastolic filling pattern. The patient underwent uncomplicated elective mitral valve replacement under general anesthesia with cardiopulmonary bypass. Intraoperative management emphasized hemodynamic stability through judicious fluid administration, careful anesthetic agent selection, and appropriate pulmonary vascular protection strategies. Cardiopulmonary bypass time was 125 minutes with an aortic cross-clamp time of 57 minutes. The postoperative course was uneventful with prompt extubation and discharge from intensive care on postoperative day three. **Conclusion:** This case illustrates the importance of comprehensive preoperative optimization, multimodal monitoring, and tailored intraoperative management in patients presenting with the complex intersection of severe organic mitral valve disease, pulmonary hypertension, and advanced diastolic dysfunction. The use of sevoflurane-based anesthesia, preservation of systemic vascular resistance, and lung-protective ventilation strategies contributed to favorable perioperative outcomes. This case highlights unique management considerations that may not be extensively detailed in standard anesthetic textbooks and demonstrates successful outcomes despite significant preoperative cardiac compromise.

1. Introduction

Mitral valve disease remains one of the most frequently encountered valvular pathologies in clinical practice, with significant perioperative implications for the anesthesiologist.[1] The etiology of mitral regurgitation encompasses both structural (organic) valve pathology and functional mechanisms secondary to ventricular remodeling and annular dilation. Primary mitral regurgitation resulting from posterior leaflet prolapse represents a mechanically

defined lesion requiring surgical intervention when hemodynamically significant and associated with progressive cardiac dysfunction.

The pathophysiology of mitral regurgitation fundamentally alters cardiac hemodynamics through the creation of a low-resistance pathway during ventricular systole. This regurgitant flow reduces effective forward cardiac output while increasing preload to the left ventricle.[2] Over prolonged periods, chronic mitral regurgitation results in eccentric hypertrophy of the left ventricle with maintenance of systolic function—a pattern distinct from the concentric hypertrophy observed in pressure-overload states such as aortic stenosis or chronic hypertension. However, the secondary effects of sustained regurgitant flow extend beyond the left ventricle to include atrial enlargement, changes in diastolic filling properties, and eventual transmission of elevated pressures into the pulmonary circulation.

The coexistence of pulmonary hypertension with mitral valve disease creates a particularly complex clinical scenario. Chronic elevation of left atrial pressure from mitral regurgitation results in reactive pulmonary hypertension characterized by pulmonary vascular remodeling and increased pulmonary vascular resistance.[3] This secondary pulmonary hypertension differs mechanistically from idiopathic pulmonary arterial hypertension yet shares similar implications for right ventricular afterload and perioperative risk. The tricuspid regurgitation often accompanying pulmonary hypertension further complicates hemodynamic assessment and surgical planning.

Diastolic dysfunction, particularly when graded as advanced restrictive filling patterns, indicates significant impairment of left ventricular relaxation and compliance. While systolic ejection fraction may remain preserved in mitral regurgitation, the diastolic properties reflect intrinsic myocardial disease and are associated with increased perioperative morbidity and mortality. The combination of severe organic mitral regurgitation with advanced diastolic dysfunction suggests myocardial involvement beyond simple mechanical valve pathology.[4]

The preoperative optimization of patients with severe mitral valve disease requires a comprehensive approach addressing multiple physiologic derangements. Current evidence supports preoperative assessment using transthoracic echocardiography to quantify the degree of regurgitation, assess chamber dimensions, evaluate diastolic function, estimate pulmonary pressures, and determine right ventricular function.[5] Cardiac magnetic resonance imaging may provide additional quantification of ventricular volumes and ejection fraction. Invasive hemodynamic monitoring via right heart catheterization remains reserved for specific clinical scenarios with diagnostic uncertainty or in patients with severe pulmonary hypertension.

The anesthetic management of patients undergoing mitral valve replacement requires consideration of several fundamental principles.[6] First, agents that reduce systemic vascular resistance may paradoxically worsen hemodynamics in mitral regurgitation by increasing the degree of regurgitant flow and reducing effective forward output. Second, excessive preload may increase atrial pressure and pulmonary congestion while minimally improving ventricular contractility in restrictive physiology. Third, adequate anesthesia for patients with pulmonary hypertension must avoid acute increases in pulmonary vascular resistance through careful agent selection and appropriate respiratory management. Fourth, cardiopulmonary bypass itself carries increased risk in patients with pulmonary hypertension through the development of pulmonary vascular reactivity and right ventricular dysfunction.

Sevoflurane possesses several properties that render it advantageous for cardiac anesthesia in complex valvular disease. Unlike desflurane, sevoflurane does not cause dose-dependent increases in sympathetic tone and systemic vascular resistance. Sevoflurane maintains diastolic function to a greater degree than propofol or thiopental, making it a favorable maintenance agent for patients with restrictive physiology. The volatile anesthetic provides

myocardial preconditioning and cardioprotection through activation of adenosine triphosphate-sensitive potassium channels and mitochondrial signaling pathways. Additionally, sevoflurane demonstrates less elevation of pulmonary vascular resistance compared to desflurane when used in hypoxic conditions, rendering it a more appropriate choice in pulmonary hypertension.[7]

The perioperative management of pulmonary hypertension in the operating room requires strategies aimed at reducing pulmonary vascular resistance while avoiding systemic hypotension. Maintenance of adequate oxygenation, normocapnia, and normothermia represents fundamental management. Inhaled pulmonary vasodilators such as inhaled nitric oxide may be required, though availability may be limited in resource-constrained settings.[8] Judicious fluid administration becomes critical to balance the competing demands of preload optimization in restrictive physiology against the risk of pulmonary edema in elevated left atrial pressure states.

Cardiopulmonary bypass in patients with significant pulmonary hypertension poses specific risks related to pulmonary vascular reactivity upon initiation and cessation of bypass. The transition from beating heart to total circulatory support may unmask right ventricular dysfunction previously compensated by increased sympathetic tone. Post-bypass separation may be complicated by acute right ventricular failure, necessitating inotropic support and potentially mechanical circulatory support. The application of lung-protective ventilation strategies with moderate positive end-expiratory pressure, low tidal volumes, and permissive hypercapnia remains important in the postoperative period.[9]

The present case presents a relatively uncommon but clinically important scenario of severe organic mitral regurgitation with posterior leaflet prolapse complicated by intermediate probability pulmonary hypertension and restrictive diastolic dysfunction in a young adult. While mitral valve disease and pulmonary hypertension occur commonly, the specific constellation of findings in this case—including the degree of diastolic dysfunction severity, the preservation of systolic ejection fraction, and the particular anesthetic challenges posed by this pathophysiology—represents a valuable addition to the literature. Furthermore, the detailed documentation of intraoperative hemodynamic parameters, fluid management decisions, and postoperative outcomes provides insights into successful management that may not be extensively detailed in standard reference materials.[10] This case report aims to illustrate best practices in anesthetic management of complex mitral valve disease with concurrent pulmonary hypertension and to highlight the pathophysiologic principles that guide perioperative decision-making in this challenging patient population.

2. Case Presentation

Informed consent and ethical considerations

The patient provided informed written consent for both the surgical intervention and for participation in this case report. The consent process involved detailed discussion of the cardiac diagnosis, the rationale for surgical intervention, alternative management options (including medical management with diuretics and cardiac medications), the specific procedure planned (mitral valve replacement with mechanical prosthetic valve), potential complications and their expected frequencies, the postoperative recovery process, and long-term management with warfarin anticoagulation. The patient was given the opportunity to ask questions and time to consider the information before providing consent. Given that this case report describes successful perioperative management of a unique clinical scenario, the publication of this report was deemed to contribute valuable educational information to the medical literature. All personal identifiers have been removed to protect patient privacy, and appropriate approval from institutional review authorities was obtained.

Patient information and demographics

A 43-year-old male patient with no significant past medical history of hypertension, diabetes mellitus, or chronic obstructive pulmonary disease presented with a 6-month progressive clinical course characterized by exertional dyspnea, persistent cough, bilateral lower-limb edema, and progressive abdominal distension. The patient reported recent completion of antituberculous therapy one month prior to presentation with documented negative sputum samples, rendering active tuberculosis unlikely as the primary etiology of the cardiac findings. The patient denied prior cardiac interventions, rheumatic fever, or family history of sudden cardiac death. Social history was notable for the absence of smoking, alcohol use, or intravenous substance abuse. The patient was admitted for a comprehensive cardiac evaluation and surgical intervention, detailed in Table 1.

Table 1. Patient characteristics and demographics.

Parameter	Value
Age	43 years
Gender	Male
Chief complaint	6-month progressive dyspnea, chronic cough
Associated symptoms	Bilateral lower-limb edema, abdominal distension
PMH	Previous anti-TB therapy (1 month, discontinued; sputum negative)
Comorbidities	None (no HTN, DM, COPD)
ASA score	III
NYHA functional class	III
BMI	Not recorded

Clinical findings and diagnostic assessment

Physical examination upon hospital admission revealed hemodynamic parameters consistent with compensated heart failure. Blood pressure measured 92/70 millimeters of mercury with a heart rate of 97 beats per minute. Respiratory rate was 20 breaths per minute with oxygen saturation of 97 percent on ambient air. Cardiovascular examination notably revealed normal heart sounds without auscultated murmurs despite significant mitral regurgitation on subsequent echocardiography, likely reflecting the acute onset and relatively pure regurgitant physiology. Pulmonary examination demonstrated symmetrical breath sounds bilaterally without crackles, indicating the absence of acute pulmonary edema despite elevated left atrial pressure. Peripheral examination confirmed bilateral pitting edema of the lower extremities and abdominal distension consistent with systemic venous congestion. These clinical findings suggested chronic adaptation to the hemodynamic load imposed by significant mitral regurgitation. Chest radiography demonstrated cardiomegaly with significant enlargement of the cardiac silhouette consistent with bilateral atrial and ventricular dilation. A right-sided pleural effusion was noted, likely secondary to chronic elevation of pulmonary venous and right atrial pressure from mitral regurgitation. These findings supported the clinical diagnosis of congestive heart failure and provided objective evidence of chronicity of the underlying valvular disease, detailed in Table 2.

Table 2. Laboratory results.

Test	Result	Normal range	Status
Hemoglobin (Hb)	13.9 g/dL	13.5–17.5 g/dL	Normal
WBC count	9.44 ×10³/μL	4.5–11.0 ×10³/μL	Normal
Platelets	240 ×10³/μL	150–400 ×10³/μL	Normal
Renal function	Normal	Normal	Normal
Liver function	Normal	Normal	Normal
HIV serology	Non-reactive	Non-reactive	Normal
HBV serology	Non-reactive	Non-reactive	Normal
Prothrombin time	Normal	Normal	Normal

Electrocardiography revealed a normal sinus rhythm without evidence of atrial fibrillation, acute ischemic changes, or ventricular hypertrophy patterns. The absence of electrocardiographic evidence of left ventricular hypertrophy despite significant left ventricular enlargement on imaging reflected the nature of eccentric hypertrophy in mitral regurgitation, which does not typically produce increased electrocardiographic voltage.

Transthoracic echocardiography represented the cornerstone of diagnostic assessment and surgical planning. This examination revealed bilateral atrial dilation with marked enlargement of the left atrium secondary to chronic mitral regurgitation. The mitral valve pathology was characterized by posterior leaflet prolapse with severe mitral regurgitation. Quantitative assessment demonstrated an effective regurgitant orifice area of 2.7 square centimeters, indicating the severity of the regurgitant jet. The regurgitant volume was correspondingly elevated. Moderate tricuspid regurgitation was noted in the setting of right atrial dilation, reflecting secondary (functional) tricuspid regurgitation from annular dilation in the context of pulmonary hypertension and elevated right atrial pressure.

The left ventricle demonstrated a preserved ejection fraction of 68 percent, indicating that systolic function remained intact despite years of hemodynamic stress from mitral regurgitation. However, detailed assessment of diastolic function revealed Grade III (restrictive) diastolic dysfunction, characterized by elevated mitral inflow early diastolic velocity, shortened deceleration time, elevated early diastolic mitral annular velocity ratio, and elevated left atrial pressure. This restrictive pattern indicated intrinsic myocardial involvement with impaired relaxation and reduced compliance, distinct from the simple mechanical effects of the regurgitant orifice. The restrictive diastolic physiology has significant implications for perioperative management, particularly regarding fluid administration and ventilatory management, detailed in Table 3.

Table 3. Echocardiographic findings.

Structure/Function	Finding	Severity/Status
Left atrium	Dilated	Dilated
Right atrium	Dilated	Dilated
Mitral valve	Severe regurgitation, posterior leaflet prolapse	Severe
EROA (Mitral)	2.7 cm²	Severe
Tricuspid valve	Moderate regurgitation	Moderate
LVEF	68%	Preserved
Diastolic function	Grade III dysfunction	Abnormal
TR velocity	2.94 m/s	Moderate
Est. PA gradient	34 mmHg	Intermediate PH
RV contractility	Preserved	Preserved
PA systolic pressure (est.)	~44 mmHg	Intermediate PH

Pulmonary hypertension assessment based on tricuspid regurgitation jet velocity of 2.94 meters per second yielded a systolic right ventricular pressure to right atrial pressure gradient of 34 millimeters of mercury. This value falls within the intermediate probability range for pulmonary hypertension, indicating elevated pulmonary pressures secondary to chronic elevation of left atrial pressure from mitral regurgitation rather than primary pulmonary arterial hypertension. The right ventricle maintained adequate contractile function despite the increased afterload, as evidenced by preserved systolic excursion and tissue Doppler imaging. The tricuspid annular plane systolic excursion was 18 millimeters, indicating preserved right ventricular function.

Anesthetic management and monitoring

Preoperative optimization was conducted over a period of 48 hours prior to surgical intervention. The patient received continuation of beta-blocker therapy and judicious diuretic administration to maintain clinical euvolemia while avoiding excessive preload reduction that could compromise cardiac output in the setting of mitral regurgitation. Antithrombotic prophylaxis was initiated, and baseline coagulation parameters were confirmed. The patient was counseled regarding the surgical procedure, anesthetic plan, and potential complications, including the need for postoperative mechanical ventilation and intensive care unit monitoring.

Intraoperative monitoring was comprehensive and included electrocardiography with a five-lead configuration and computerized ST-segment analysis, pulse oximetry, capnography, and core temperature monitoring via esophageal probe. Invasive hemodynamic monitoring was established including a right radial arterial catheter (20-gauge) for continuous arterial pressure monitoring and blood sampling, a left subclavian central venous catheter (18-gauge) for fluid and medication administration, and a Swan-Ganz pulmonary artery catheter (7.5-French, inserted via right internal jugular vein) for measurement of central venous pressure, pulmonary artery pressure, pulmonary capillary wedge pressure, and mixed venous oxygen saturation. An additional 18-gauge peripheral intravenous catheter was placed for rapid fluid infusion if required. This multimodal monitoring strategy provided a comprehensive hemodynamic assessment necessary for safe management in the context of complex valvular disease and pulmonary hypertension.

Transesophageal echocardiography was performed following anesthetic induction and served multiple functions: baseline assessment of valve anatomy and function, confirmation of the posterior leaflet prolapse anatomy, evaluation of the degree of mitral regurgitation before and after cardiopulmonary bypass, assessment of right ventricular function throughout the procedure, and detection of any residual regurgitation or complications related to the prosthetic valve placement. The transesophageal echocardiographic findings confirmed the transthoracic echocardiographic findings of severe mitral regurgitation with posterior leaflet pathology and demonstrated good left ventricular contractile function. The pulmonary artery pressure estimated by transesophageal echocardiography using tricuspid regurgitation jet velocity remained consistent with intermediate pulmonary hypertension probability, detailed in Table 4.

The anesthetic induction strategy was chosen specifically for this high-risk patient with depressed hemodynamics and pulmonary hypertension. Premedication was deliberately avoided to prevent further reduction in systemic vascular resistance and to maintain spontaneous breathing during the period before intubation. Fentanyl 200 micrograms was selected as the primary induction agent due to its cardiovascular stability, analgesic properties, and favorable hemodynamic profile in patients with depressed baseline blood pressure and elevated pulmonary vascular resistance. Midazolam 5 milligrams was administered for amnestic and anxiolytic effects without inducing significant additional vasodilation.

Table 4. Anesthetic management and monitoring.

Component	Specifications
Premedication	None (avoided to maintain hemodynamic stability)
Induction agent	Fentanyl 200 μg + Midazolam 5 mg
Neuromuscular blocker	Rocuronium 30 mg
Endotracheal tube	8.0 mm internal diameter
Maintenance agent	Sevoflurane in 100% oxygen
Maintenance MAC	0.8-1.0 MAC (titrated to response)
Ventilation mode	Volume-controlled, 6-8 mL/kg ideal body weight
PEEP target	5 cm H_2O

Rocuronium 30 milligrams provided neuromuscular blockade with minimal histamine release and without significant cardiovascular effects. Endotracheal intubation was accomplished with an 8.0-millimeter internal diameter tube, and immediately following intubation, the head of the operating table was elevated to 15 degrees to optimize pulmonary blood flow and reduce airway edema given the elevated pulmonary venous pressures.

Sevoflurane was selected as the volatile anesthetic maintenance agent based on several specific advantages in this clinical context. First, sevoflurane demonstrates less sympathomimetic activity compared to desflurane, making it preferable in patients with baseline systemic hypotension where preservation of systemic vascular resistance is desirable in mitral regurgitation. Second, sevoflurane causes less elevation in pulmonary vascular resistance in hypoxic conditions compared to other volatile anesthetics, rendering it superior in patients with pulmonary hypertension. Third, sevoflurane maintains diastolic function more effectively than alternative agents, an important consideration in the patient with Grade III restrictive diastolic dysfunction. Fourth, sevoflurane provides myocardial preconditioning through activation of adenosine triphosphate-sensitive potassium channels, offering neuroprotection during periods of myocardial ischemia during aortic cross-clamping. The sevoflurane concentration was titrated to maintain a minimum alveolar concentration equivalent of 0.8 to 1.0 based on movement and hemodynamic response, with reductions during periods of unstable hemodynamics and increases during periods of sympathetic response.

Mechanical ventilation was managed with a volume-controlled mode using a tidal volume of 6 to 8 milliliters per kilogram of ideal body weight to implement lung-protective ventilation strategies and minimize ventilator-induced lung injury in the context of elevated pulmonary venous pressures. The respiratory rate was set at 12 breaths per minute to maintain normocapnia (target end-tidal carbon dioxide of 35 to 40 millimeters of mercury). Positive end-expiratory pressure was set at 5 centimeters of water, a level chosen to provide mild alveolar recruitment and improve oxygenation while avoiding excessive intrathoracic pressure that could impair venous return and worsen pulmonary hypertension. Higher levels of positive end-expiratory pressure were avoided due to the potential to increase pulmonary vascular resistance through direct compression of alveolar vessels and indirectly through increases in intrathoracic pressure, reducing venous return and increasing right atrial pressure, thereby increasing the driving pressure for pulmonary vascular resistance.

Intraoperative course and cardiopulmonary bypass management

The intraoperative course was characterized by hemodynamic stability with appropriate response to anesthetic and surgical stimuli. Pre-incision baseline pulmonary artery pressure measured approximately 19 millimeters of mercury (systolic), central venous pressure measured 14 centimeters of water, and systemic arterial pressure measured 108/63

millimeters of mercury with a calculated mean arterial pressure of 73 millimeters of mercury. Heart rate ranged from 97 to 124 beats per minute, and oxygen saturation remained between 98 and 99 percent throughout. These hemodynamic parameters reflected the appropriate balance achieved through anesthetic technique, adequate preload, and judicious use of vasoconstrictive agents.

Cardiopulmonary bypass was initiated following achievement of anticoagulation with heparin 300 units per kilogram, achieving an activated clotting time greater than 400 seconds. The bypass perfusate consisted of a standardized crystalloid composition with targeted flow rates of 2.4 liters per minute per square meter of body surface area at moderate hypothermia of 32 degrees Celsius. The transition to total cardiopulmonary bypass was gradual, with careful observation of right ventricular function via transesophageal echocardiography to detect any acute decompensation. Upon initiation of bypass, pulmonary artery pressure initially decreased to 12 millimeters of mercury as pulmonary vascular resistance decreased with the normalization of left atrial pressure from the removal of the regurgitant flow. This hemodynamic change reflected the fundamental pathophysiology of mitral regurgitation—the pulmonary hypertension was reactive and dependent on the presence of elevated left atrial pressure rather than primary pulmonary vascular disease.

The aorta was crossclamped, and the left atrium was opened, revealing the posterior mitral leaflet prolapse prolapsing into the left atrium during diastole and contributing to the severe regurgitation during systole. The mitral valve was carefully excised, preserving the underlying chordal structures when possible to maintain a relatively normal left ventricular geometry. A mechanical bileaflet prosthetic valve (25-millimeter diameter) was selected based on the patient's age and need for long-term anticoagulation therapy, and was seated in the mitral annulus using interrupted braided polyester sutures. The aorta was then partially uncrossed to allow blood to perfuse the coronary circulation while the left atrium was closed with running monofilament suture, detailed in Table 5.

Table 5. Intraoperative course and cardiopulmonary bypass management.

Time point	MAP (mmHg)	PAP (mmHg)	CVP (cm H_2O)
Pre-incision	73	19	14
Upon CPB initiation	68	12	8
During aortic cross-clamp	71	11	7
Beginning of rewarm	75	13	10
End of rewarming	76	16	12
Weaning from CPB	79	18	13
Post-CPB (chest closure)	82	17	11

Separation from cardiopulmonary bypass was achieved without difficulty. The heart was defibrillated into normal sinus rhythm without the requirement for inotropic support. Transesophageal echocardiographic assessment of the prosthetic mitral valve showed excellent function with normal leaflet motion, appropriate coaptation, and trace physiologic mitral regurgitation (which is normal for mechanical prosthetic valves due to the design of the valve mechanism). The left ventricular function remained robust with an ejection fraction of 68 percent. The tricuspid regurgitation was significantly reduced following the relief of the mitral regurgitation and restoration of normal left atrial pressure, indicating that the tricuspid regurgitation was indeed secondary and would be expected to improve with successful mitral valve replacement. The pulmonary artery pressure rose to 18 millimeters of mercury as cardiopulmonary bypass was terminated and the patient transitioned back to native cardiac output, a

modest increase from the nadir values on bypass but substantially lower than the preoperative baseline of 19 millimeters of mercury, reflecting the reduction in left atrial pressure transmission to the pulmonary circulation.

Total cardiopulmonary bypass time was 125 minutes, with an aortic cross-clamp time of 57 minutes and total myocardial ischemic time of 63 minutes, including the period of aortic cross-clamp and subsequent warm-up period. These times are well within acceptable limits for mitral valve replacement and reflect the technical skill of the surgical team and adequate myocardial protection provided by cold crystalloid cardioplegia. The moderate hypothermia maintained during cardiopulmonary bypass (32 degrees Celsius) provided additional myocardial and systemic neuroprotection.

Fluid management and hemostasis

Fluid management in this patient with restrictive diastolic dysfunction and elevated pulmonary venous pressure required careful balance to maintain adequate intravascular volume for systemic perfusion while avoiding excessive preload that could precipitate pulmonary edema. The principle guiding fluid administration was recognition that in restrictive physiology, the left ventricular pressure-volume curve is steep; small increases in volume produce large increases in pressure without substantial increases in stroke volume, detailed in Table 6.

Table 6. Fluid management and hemostasis.

Fluid type	Volume (mL)	Indication/Timing
Crystalloid (Ringer's Lactate)	~500	Preinduction and bypass prime
Packed red cells	399	Intraoperative blood loss replacement
Fresh frozen plasma	414	Coagulation factor replacement
Platelets (Units)	150 mL	Platelet support post-transfusion
Estimated blood loss	~1000	Total intraoperative loss

The fluid administration strategy proceeded in phases. During the preoperative period (before induction), approximately 500 milliliters of Ringer's lactate was infused to optimize preload while avoiding excessive volume expansion given the patient's baseline elevated pulmonary venous pressure. During induction and before bypass, additional fluid was minimized to preserve the anesthetic effect and maintain the modest degree of relative hypovolemia that optimizes left ventricular performance in mitral regurgitation by reducing the degree of regurgitant flow (afterload reduction in reverse). Once cardiopulmonary bypass was initiated, fluid administration was governed by the bypass pump perfusion parameters and central venous pressure monitoring, with target central venous pressure maintained between 8 and 12 centimeters of water. During cardiopulmonary bypass itself, the standard crystalloid prime of approximately 1500 milliliters provided the volume necessary to avoid excessive hemodilution while maintaining adequate circulating volume. The bypass circuit incorporated a venous reservoir with appropriate volume management.

Intraoperative blood loss totaled approximately 1000 milliliters. Packed red cells (399 milliliters) were transfused to maintain hemoglobin above 7 grams per deciliter and to support oxygen-carrying capacity. Fresh frozen plasma (414 milliliters) was administered based on coagulation monitoring and to provide procoagulant factors following the large-volume blood loss and cardiopulmonary bypass. Platelet transfusion (150 milliliters) was administered when platelet count fell below 50,000 per microliter, as routinely recommended following cardiopulmonary bypass. Cell salvage techniques were employed throughout the procedure to minimize exposure to allogeneic blood products. Heparin anticoagulation was reversed with protamine sulfate (calculated dose of 1 milligram protamine per 100 units of administered heparin) at the conclusion of cardiopulmonary bypass.

Postoperative course and outcomes

At the conclusion of surgery, the patient was transported to the intensive care unit in stable condition with mechanical ventilatory support. Immediate postoperative hemodynamic parameters reflected a good surgical outcome: systolic arterial pressure 111 millimeters of mercury, diastolic pressure 78 millimeters of mercury, heart rate 78 beats per minute, respiratory rate 19 breaths per minute, and oxygen saturation 98 percent on fractional inspired oxygen of 0.4. The patient was deeply sedated with propofol and remifentanil infusions, maintained on mechanical ventilation, and monitored continuously with an arterial line, central venous line, and pulmonary artery catheter. Postoperative laboratory parameters showed hemoglobin of 9.2 grams per deciliter (reflecting hemodilution from transfusion), white blood cell count of 11.8 thousand per microliter (expected postoperative elevation), platelet count of 156 thousand per microliter, and prothrombin time of 16 seconds (normal for the immediate postoperative period in a patient on warfarin prophylaxis), detailed in Table 7.

Table 7. Postoperative course and outcomes.

Parameter	Postop day 0	Postop day 1
SBP/DBP (mmHg)	111/78	128/82
Heart rate (bpm)	78	82
Hemoglobin (g/dL)	9.2	8.9
WBC (x10³/µL)	11.8	13.2
Platelet (x10³/µL)	156	178
Chest tube output (mL)	425	215
Ventilation status	Intubated, sedated	Extubated POD#1

Mechanical ventilation was continued overnight with lung-protective ventilation parameters maintained (tidal volume 6 milliliters per kilogram ideal body weight, respiratory rate 12 breaths per minute, positive end-expiratory pressure 5 centimeters of water, fraction of inspired oxygen 0.4 to maintain oxygen saturation between 95 and 98 percent). Sedation was gradually reduced on postoperative day one, and the patient demonstrated excellent spontaneous respiratory effort and adequate oxygenation. A spontaneous breathing trial was conducted on postoperative day one, during which the patient successfully tolerated 2 hours of pressure support ventilation with minimal support. Arterial blood gas analysis showed partial pressure of oxygen of 92 millimeters of mercury on fraction of inspired oxygen of 0.4, partial pressure of carbon dioxide of 38 millimeters of mercury, and pH of 7.41, indicating good ventilation and oxygenation. The patient was extubated on postoperative day one without complications and continued on supplemental oxygen via nasal cannula at 2 liters per minute.

Cardiac assessment in the immediate postoperative period was favorable. Transthoracic echocardiography performed on postoperative day one demonstrated excellent function of the prosthetic mitral valve with normal leaflet motion, appropriate coaptation, and only trace physiologic mitral regurgitation. Left ventricular ejection fraction remained preserved at 67 percent. Right ventricular function was normal with tricuspid annular plane systolic excursion of 20 millimeters and reduced tricuspid regurgitation (only mild) despite the intermediate probability pulmonary hypertension, confirming that the pulmonary hypertension had been secondary to the mitral valve disease rather than primary. Estimated right ventricular systolic pressure based on tricuspid regurgitation jet velocity decreased to 21 millimeters of mercury (down from the preoperative 34 millimeters of mercury), representing a substantial reduction in pulmonary artery pressure following correction of the mitral disease. The left

atrium measured 46 millimeters in the long axis (down from 52 millimeters preoperatively), reflecting initial remodeling from the elimination of the regurgitant load.

The postoperative course was uncomplicated. The patient maintained stable hemodynamics, did not require inotropic support, and had appropriate diuresis with negative fluid balance achieved on loop diuretics. Chest tube output was 425 milliliters on postoperative day zero and declined to 215 milliliters on postoperative day one, indicating cessation of significant bleeding. The patient was transferred from the intensive care unit to the cardiac step-down unit on postoperative day two with stable vital signs, no arrhythmias, and good functional status. Physical therapy was initiated, and the patient ambulated with assistance on postoperative day two. Oral intake was restarted without difficulty. Warfarin anticoagulation was initiated, given the mechanical prosthetic mitral valve, with a target international normalized ratio of 2.0 to 3.0. The patient was discharged from the hospital on postoperative day six with appropriate medications including beta-blockers, diuretics, warfarin, and aspirin. No perioperative complications occurred, including no myocardial infarction, no stroke, no renal failure, no infection, and no arrhythmias requiring intervention.

Patient perspective

The patient understood that he had a serious heart condition requiring surgery and that the operation carried risks including the need for intensive care monitoring and mechanical ventilation in the immediate postoperative period. He was counseled that the cardiac symptoms he had experienced for six months—progressive difficulty with exertion, shortness of breath, swelling in the legs, and abdominal distension—were caused by his heart not pumping efficiently due to the damaged mitral valve. He learned that the valve would be replaced with a mechanical prosthetic valve, which would require him to take a blood thinner (warfarin) for the rest of his life. He was informed that the surgery carried risks of bleeding, infection, heart attack, stroke, and death, though such complications were expected to be uncommon with appropriate surgical technique and perioperative care. Following surgery, the patient was pleased with the rapid improvement in his cardiac symptoms. His dyspnea resolved within days, his lower-extremity edema resolved completely within two weeks, and his exercise tolerance improved substantially. At follow-up visit one month after surgery, the patient reported feeling better than he had in years and expressed gratitude for the successful surgical intervention. He understood the importance of adherence to warfarin anticoagulation and regular follow-up appointments with his cardiologist.

3. Discussion

This case presentation illustrates a complex clinical scenario of severe organic mitral regurgitation with posterior leaflet prolapse, complicated by reactive pulmonary hypertension and advanced diastolic dysfunction in a previously healthy middle-aged male. The successful perioperative management of this patient required a comprehensive understanding of the pathophysiology underlying mitral valve disease, the hemodynamic consequences of regurgitation, the mechanisms of reactive pulmonary hypertension, and the principles guiding anesthetic decision-making in complex valvular disease.[11]

Primary mitral regurgitation resulting from organic valve pathology represents a fundamental derangement in the normal closure mechanisms of the mitral valve. In the case presented, posterior leaflet prolapse—the pathologic basis for the regurgitation—results from loss of coaptation between the anterior and posterior leaflets during ventricular systole. This may result from degenerative changes in the valve structure (myxomatous degeneration), rupture of papillary muscles, elongation of chordae tendineae, or rarely endocarditis with valve destruction. Regardless of the underlying etiology, the hemodynamic consequence is identical: a portion of the left ventricular stroke volume regurgitates back into the

left atrium during systole rather than being ejected into the systemic circulation.

The fundamental consequence of mitral regurgitation is the creation of volume overload of the left ventricle. During diastole, the left ventricle receives blood from the pulmonary circulation via the mitral orifice.[12] During systole, the regurgitant fraction returns to the left atrium while the remainder is ejected into the aorta. This volume overload mandates increased chamber size to accommodate the greater end-diastolic volume. Chronic mitral regurgitation results in eccentric hypertrophy of the left ventricle—a pattern of enlargement in which wall thickness remains relatively normal or decreases while chamber dimensions increase substantially. This contrasts with the concentric hypertrophy seen in pressure-overload states, where wall thickness increases while chamber dimensions remain relatively normal. The eccentric hypertrophy preserves normal systolic wall stress (according to the Laplace relationship: wall stress is proportional to pressure times radius divided by wall thickness; when both radius and thickness increase proportionally, wall stress remains normal). This is why left ventricular ejection fraction is typically preserved in primary mitral regurgitation despite the severe hemodynamic load.

The diastolic dysfunction observed in this patient represents a separate and distinct pathophysiologic process from the mechanical effects of mitral regurgitation. While all patients with mitral regurgitation have elevation of left atrial pressure and thus develop diastolic dysfunction secondary to impaired left ventricular relaxation and increased stiffness, the Grade III restrictive filling pattern observed in this case indicates advanced intrinsic myocardial dysfunction. The restrictive pattern is characterized by severely elevated mitral inflow velocities, shortened deceleration time, and elevated filling pressures. This pattern indicates that diastolic dysfunction has progressed beyond the simple pressure-related dysfunction to include intrinsic changes in myocardial relaxation and compliance. The presence of advanced diastolic dysfunction in this patient with preserved systolic ejection fraction suggests myocardial involvement beyond the mechanical aspects of valvular disease, potentially indicating early myocardial disease or advanced remodeling.[13]

The pulmonary hypertension in this patient represents a secondary (reactive) form resulting from chronic elevation of left atrial pressure transmitted backward through the pulmonary veins into the pulmonary arterial system. This is distinct from primary pulmonary arterial hypertension, which results from intrinsic pulmonary vascular disease. In secondary pulmonary hypertension from mitral valve disease, the initial mechanism is passive elevation of pulmonary artery pressure due to passive transmission of elevated left atrial pressure.[14] However, with chronic elevation of pulmonary venous pressure, the pulmonary vasculature undergoes remodeling including intimal proliferation, medial hypertrophy, and adventitial fibrosis. Additionally, endothelial dysfunction develops with decreased nitric oxide production and increased endothelin-1 production, rendering the pulmonary vasculature reactive and prone to further pressure elevation. The tricuspid regurgitation observed in this patient resulted from functional (secondary) mechanisms—specifically, right atrial dilation from elevated right atrial pressure and pulmonary hypertension, leading to annular dilation and failure of tricuspid leaflet coaptation. Importantly, functional tricuspid regurgitation from pulmonary hypertension is expected to improve substantially following correction of the mitral lesion and reduction of left atrial pressure, which is precisely what was observed in this case.

The specific tricuspid regurgitation jet velocity of 2.94 meters per second corresponds to a pressure gradient of 34 millimeters of mercury (calculated using the simplified Bernoulli equation: gradient equals 4 times velocity squared). This gradient represents the right ventricular to right atrial pressure difference. With a central venous pressure of 14 centimeters of

water (approximately 10 millimeters of mercury), the calculated right ventricular systolic pressure is 44 millimeters of mercury. Using the American College of Cardiology/American Heart Association criteria, this falls in the intermediate probability range for pulmonary hypertension (pressure between 36 and 50 millimeters of mercury). The intermediate probability classification indicates that right heart catheterization would be indicated if clinical uncertainty existed regarding the need for surgical intervention. However, in this case, the indication for surgery was based on the severe organic mitral regurgitation with hemodynamic consequences rather than the pulmonary hypertension, and the expectation was that successful mitral valve replacement would result in a substantial reduction in pulmonary pressures.

The fundamental principle guiding anesthetic management of mitral regurgitation is the understanding that systemic vascular resistance directly affects the degree of regurgitation.[15] During ventricular systole, regurgitation occurs through the incompetent valve orifice into the low-pressure left atrium. The degree of regurgitation is determined by the pressure gradient between the left ventricle and left atrium and by the area of the regurgitant orifice. By reducing systemic vascular resistance (which is equivalent to reducing left ventricular afterload), the left ventricle ejects more blood into the aorta (which has lower resistance than the regurgitant pathway) and less blood regurgitates backward. Conversely, increasing systemic vascular resistance worsens regurgitation by making the regurgitant pathway relatively more attractive hemodynamically. This principle explains why volatile anesthetics that reduce systemic vascular resistance (such as sevoflurane) are preferable to those that maintain or increase it (such as desflurane), and why vasodilating agents may paradoxically improve hemodynamics despite reducing mean arterial pressure.

The concept of afterload mismatch is central to understanding anesthetic management in mitral regurgitation. In normal cardiac physiology, afterload refers to the resistance against which the left ventricle must contract.[16] In mitral regurgitation, two afterloads exist: the systemic vascular resistance (aortic afterload) and the atrial afterload (the resistance of the regurgitant pathway). The effective afterload in mitral regurgitation should be considered as the relative attractiveness of these two pathways. When systemic vascular resistance is elevated, the regurgitant pathway becomes relatively more attractive, increasing the fraction of stroke volume that regurgitates. When systemic vascular resistance is reduced, the aortic pathway becomes relatively more attractive, increasing effective forward output despite the lower afterload. This explains why a modest reduction in mean arterial pressure that accompanies vasodilation often results in improved cardiac output in mitral regurgitation. In this patient with borderline systemic hypotension at baseline (92/70 millimeters of mercury), aggressive vasodilation was avoided. Instead, the anesthetic approach emphasized preservation of systemic vascular resistance while avoiding excessive sympathetic stimulation. Sevoflurane was chosen over desflurane because it causes less sympathomimetic activity and less elevation of systemic vascular resistance.

Preload management in mitral regurgitation and restrictive diastolic dysfunction requires careful consideration. In normal cardiac physiology, increasing preload increases stroke volume along the steep portion of the Frank-Starling curve. However, in restrictive diastolic dysfunction, the left ventricular pressure-volume curve is steep throughout its physiologic range. Small increases in left ventricular volume produce large increases in left ventricular pressure without substantial increases in stroke volume. Additionally, because of the regurgitant pathway, increasing preload simply increases the volume available to regurgitate. The optimal hemodynamic state in mitral regurgitation is actually modest hypovolemia—sufficient to maintain systemic perfusion but not so excessive as to increase the regurgitant fraction. This principle explains why aggressive fluid preloading before surgery is detrimental in mitral regurgitation. In this case,

limited fluid administration was used preoperatively, with a target central venous pressure of 12 to 14 centimeters of water. This modest preload was sufficient to maintain systemic perfusion while avoiding pulmonary edema.[17]

The choice of induction agents was critical for this patient with baseline hypotension and pulmonary hypertension. Propofol, while widely used, causes a substantial reduction in systemic vascular resistance through mechanisms involving direct myocardial depression and vasodilation. In a patient with a baseline blood pressure of 92/70 millimeters of mercury, propofol-induced vasodilation could result in profound hypotension requiring aggressive vasopressor support. Etomidate, while maintaining systemic vascular resistance better than propofol, causes tachycardia and concerns regarding adrenal suppression despite single-dose use. Thiopental causes myocardial depression and a reduction in systemic vascular resistance similar to propofol. Ketamine maintains airway reflexes and causes sympathomimetic stimulation, but carries the risk of increasing pulmonary vascular resistance through sympathetic activation. Consequently, fentanyl—a mu-opioid agonist with minimal effects on systemic vascular resistance—was selected as the primary induction agent.[18] The addition of midazolam provided amnestic effects while maintaining relatively stable hemodynamics. This combination allowed smooth induction without profound hemodynamic perturbation.

The selection of sevoflurane as the volatile anesthetic maintenance agent was based on multiple pharmacologic and physiologic considerations. Sevoflurane causes dose-dependent reduction in systemic vascular resistance through mechanisms involving peripheral vasodilation and reduced sympathetic outflow. However, compared to desflurane, sevoflurane causes less sympathomimetic activation and less elevation of systemic vascular resistance. Sevoflurane maintains diastolic function better than propofol, thiopental, or etomidate, an important consideration in a patient with Grade III restrictive diastolic dysfunction. Sevoflurane provides myocardial preconditioning through activation of mitochondrial and nuclear signaling pathways, offering potential cardioprotection during the period of aortic cross-clamping. Sevoflurane is rapidly eliminated through the lungs without hepatic metabolism, allowing rapid emergence and quick assessment of neurologic status postoperatively. In hypoxic conditions, sevoflurane causes less elevation of pulmonary vascular resistance compared to desflurane, making it the preferred volatile agent in pulmonary hypertension. Desflurane, in contrast, causes sympathomimetic stimulation with increased heart rate and systemic vascular resistance, potentially worsening pulmonary vascular resistance in hypoxic conditions. The minimum alveolar concentration of sevoflurane at sea level and 37 degrees Celsius is approximately 2 percent; in this case, sevoflurane concentration was titrated to 0.8 to 1.0 minimum alveolar concentration equivalent (approximately 1.6 to 2.0 percent) based on movement and hemodynamic response.[19]

The management of patients with pulmonary hypertension undergoing cardiac surgery presents unique challenges related to the increased pulmonary vascular resistance and the risk of acute right ventricular dysfunction. The fundamental management strategies include maintenance of adequate oxygenation, normocapnia or mild hypocapnia, normothermia, and avoidance of hypoxia, hypercarbia, and acidosis—all of which increase pulmonary vascular resistance. In the operating room, this is accomplished through appropriate volatile anesthetic agent selection (sevoflurane preferred over desflurane), avoidance of nitrous oxide (which increases pulmonary vascular resistance), judicious use of positive end-expiratory pressure, and rapid correction of any hypoxemia.[20] The positive end-expiratory pressure applied in this case (5 centimeters of water) was modest, specifically to avoid excessive intrathoracic pressure, which could compress pulmonary vessels and increase pulmonary vascular resistance. Higher levels of positive end-expiratory

pressure, while improving oxygenation in some conditions, can worsen pulmonary hemodynamics by increasing intrathoracic pressure, compressing pulmonary vessels, and reducing venous return, thereby increasing right atrial pressure and the driving pressure for pulmonary vascular resistance.

The relationship between positive end-expiratory pressure and pulmonary vascular resistance follows a U-shaped curve. Very low positive end-expiratory pressure results in alveolar collapse, hypoxemia, and increased pulmonary vascular resistance through hypoxic pulmonary vasoconstriction. Moderate positive end-expiratory pressure (5 to 8 centimeters of water) recruits alveoli and improves oxygenation while minimally affecting pulmonary vascular resistance. High positive end-expiratory pressure (greater than 10 centimeters of water) compresses alveolar capillaries, increases pulmonary vascular resistance, and reduces venous return. Additionally, excessive intrathoracic pressure from high positive end-expiratory pressure increases right atrial pressure, which directly increases the driving pressure for pulmonary vascular resistance (the gradient between pulmonary artery pressure and left atrial pressure minus the driving pressure, which is pulmonary artery pressure minus left atrial pressure, but right atrial pressure affects this through effects on venous return and subsequent effects on left atrial pressure). The choice of positive end-expiratory pressure of 5 centimeters of water in this patient represented a balance between the need for alveolar recruitment (given the elevated pulmonary venous pressure and risk of pulmonary edema) and the need to avoid excessive intrathoracic pressure that could worsen pulmonary vascular resistance.[21]

Inhaled pulmonary vasodilators such as inhaled nitric oxide and inhaled epoprostenol can reduce pulmonary vascular resistance and improve right ventricular function in patients with pulmonary hypertension. However, these agents are expensive, require specialized equipment, and may not be available in all settings. In this case, inhaled pulmonary vasodilators were not required because the patient maintained reasonable hemodynamics without them, and the moderate reduction in pulmonary artery pressure upon initiation of cardiopulmonary bypass indicated that the pulmonary hypertension was reactive rather than fixed. Inhaled nitric oxide has been shown in multiple studies to reduce pulmonary vascular resistance by 10 to 25 percent in patients with pulmonary hypertension during cardiac surgery. However, the decision to use such agents should be based on clinical hemodynamic evidence of right ventricular dysfunction or inability to maintain adequate systemic perfusion, rather than on the theoretical basis of elevated pulmonary artery pressure alone.

The transition to and from cardiopulmonary bypass represents a critical period in patients with pulmonary hypertension. Upon initiation of bypass, there is typically an initial reduction in pulmonary artery pressure as the mechanical load on the right ventricle is reduced and as left atrial pressure decreases (in the case of mitral valve surgery). However, the cessation of cardiopulmonary bypass can be associated with an acute elevation in pulmonary vascular resistance related to rewarming, separation from mechanical circulatory support, and restoration of mechanical work to the right ventricle. In this case, the pulmonary artery pressure was 19 millimeters of mercury preoperatively, decreased to 12 millimeters of mercury upon initiation of bypass, reached a nadir of 11 millimeters of mercury during aortic cross-clamping, and returned to 18 millimeters of mercury upon weaning from bypass. This pattern is entirely consistent with reactive pulmonary hypertension secondary to mitral regurgitation.[22] The reduction in pulmonary artery pressure postoperatively to 17 millimeters of mercury at the conclusion of surgery reflected the beneficial effect of eliminating the mitral regurgitation and reducing left atrial pressure.

The constellation of findings in this case—severe mitral regurgitation with posterior leaflet prolapse, intermediate probability pulmonary hypertension, and Grade III restrictive diastolic dysfunction—represents a relatively uncommon clinical scenario. While mitral

regurgitation is common, the specific pattern of advanced diastolic dysfunction with preserved systolic ejection fraction is less frequently reported in the literature. Diaz-Rodriguez and colleagues reviewed anesthetic management of patients with pulmonary hypertension undergoing cardiac surgery and identified several key principles that were applied in the current case. Their study emphasized the importance of volatile anesthetic agent selection, maintenance of systemic vascular resistance, and avoidance of perioperative hypoxemia and hypercarbia. The current case aligns with their recommendations and demonstrates successful application of these principles in a patient with intermediate probability pulmonary hypertension and severe valvular disease.

Efremov and colleagues conducted a detailed review of intraoperative hemodynamic management in patients with pulmonary hypertension undergoing valve surgery. Their findings supported the use of sevoflurane over desflurane, maintenance of modest preload (central venous pressure of 8 to 12 centimeters of water), and careful fluid management to avoid both hypovolemia and excessive preload. The current case demonstrated the successful application of these principles with preservation of hemodynamic stability throughout the operative period without the requirement for inotropic support. The systemic vascular resistance was maintained through careful anesthetic agent selection and judicious fluid administration, while right ventricular function remained adequate throughout, avoiding the acute right ventricular dysfunction that can complicate pulmonary hypertension cases.[23]

The topic of diastolic dysfunction in the perioperative setting has received increasing attention in recent literature. McDonagh and colleagues emphasized that Grade III restrictive diastolic filling patterns are associated with substantially increased perioperative morbidity and mortality compared to normal diastolic function or milder degrees of diastolic dysfunction. The present case demonstrated excellent perioperative outcomes despite advanced diastolic dysfunction, likely because the surgical correction of the mitral regurgitation addressed the underlying mechanism driving the pulmonary hypertension and secondary tricuspid regurgitation. The patient's postoperative echocardiography demonstrated immediate improvement in tricuspid regurgitation severity and reduction in estimated pulmonary artery pressure, confirming the functional nature of these findings and their relationship to the mitral pathology.

The decision to proceed with mitral valve replacement (rather than repair) in this case reflected the anatomy of posterior leaflet prolapse, which is typically amenable to repair in many cases. However, the specific extent of the prolapse and involvement of the leaflet structure rendered the valve unsuitable for attempted repair. The choice of mechanical (rather than bioprosthetic) valve was appropriate for a 43-year-old patient with life expectancy measured in decades; mechanical valves provide superior long-term durability but require anticoagulation. The prosthetic valve function in this case was excellent with only trace physiologic regurgitation, which is normal for mechanical prosthetic valves.

The postoperative course in this case was notably uncomplicated, with prompt extubation on postoperative day one, no arrhythmias requiring intervention, and no perioperative cardiac, renal, neurologic, or infectious complications. This favorable outcome contrasts with the expected higher morbidity associated with advanced diastolic dysfunction in other disease states.[24] The explanation likely lies in the fact that the patient's diastolic dysfunction was entirely secondary to the chronic mitral regurgitation and the associated elevated left atrial pressure. The surgical correction of the mitral regurgitation eliminated the primary driver of the diastolic dysfunction, allowing rapid hemodynamic improvement and functional recovery. In contrast, patients with diastolic dysfunction secondary to hypertensive heart disease, amyloidosis, or other intrinsic myocardial processes would not be expected to have such rapid improvement following valvular surgery, detailed in Table 8.

Table 8. Literature comparison – this case vs. published case reports.

Parameter	Reckard et al. 2008	Xu et al. 2025	Lowson et al. 2002	Present case
Age & gender	58F	Elderly M	63F	43M
Primary diagnosis	Severe MR (rheumatic)	Severe MR + TR + AF + mod PH	Severe MR + mod-severe PH	Severe MR + CHF + TR + intermediate PH
Procedure	MVR	MVR + TVR	MVR + PH management	MVR
PH severity	Mild-moderate	Moderate-severe	Moderate-severe	Intermediate
Airway difficulty	Not mentioned	Difficult airway reported	Not mentioned	Routine intubation (8.0 ETT)
Inotropic support	Required (milrinone, epinephrine)	Required post-CPB	Required post-CPB	NONE required post-CPB
Inhaled vasodilators	Inhaled nitric oxide	Not mentioned	Inhaled prostacyclin (rescue)	Not required
CPB time	Not specified	Not specified	~180 min (prolonged)	125 min
Outcome	Successful; difficult post-op course	Successful; moderate support needed	Successful with rescue therapy	Excellent; minimal support

This case report provides several important learning points relevant to anesthetic management of complex mitral valve disease. The pathophysiology of mitral regurgitation differs fundamentally from pressure-overload lesions, necessitating distinct management strategies focused on optimization of the balance between systemic vascular resistance and regurgitant fraction. Agents and interventions that reduce systemic vascular resistance (such as volatile anesthetics or vasodilators) may paradoxically improve effective forward cardiac output in mitral regurgitation despite reducing mean arterial pressure. Advanced diastolic dysfunction, particularly the restrictive filling pattern observed in this case, reflects intrinsic myocardial involvement beyond the mechanical effects of valve pathology. Such dysfunction carries increased perioperative risk but may rapidly improve following surgical correction of the underlying valvular lesion that was driving the diastolic impairment. Reactive pulmonary hypertension secondary to chronic mitral regurgitation differs mechanistically from primary pulmonary arterial hypertension and demonstrates substantial hemodynamic improvement following correction of the mitral lesion. The reduction in pulmonary artery pressure observed in this patient upon initiation of cardiopulmonary bypass provided intraoperative confirmation that the pulmonary hypertension was indeed secondary and reactive. Sevoflurane offers specific advantages over desflurane in patients with pulmonary hypertension and mitral regurgitation through its favorable effects on systemic vascular resistance, preservation of diastolic function, and reduced effects on pulmonary vascular resistance in hypoxic conditions. Judicious fluid management with modest preload (rather than aggressive fluid loading) optimizes hemodynamics in mitral regurgitation by reducing the regurgitant fraction while maintaining adequate systemic perfusion. The Frank-Starling relationship is unfavorable in restrictive diastolic dysfunction, such that increases in preload produce excessive increases in pressure without proportional increases in stroke volume. Moderate positive end-expiratory pressure (5 centimeters of water) provides a balance between alveolar recruitment (important in elevated pulmonary venous pressure) and avoidance of excessive intrathoracic pressure (which increases pulmonary vascular resistance). The selection of positive end-expiratory pressure should be individualized based on the patient's specific pathophysiology. The successful perioperative management of complex mitral valve

disease with concurrent pulmonary hypertension requires a comprehensive understanding of underlying pathophysiology, multimodal monitoring including transesophageal echocardiography and right heart catheterization, tailored anesthetic agent selection, and careful fluid management based on hemodynamic principles rather than empiric protocols.[25]

4. Conclusion

This case report presents the successful anesthetic management of a complex patient with severe mitral regurgitation secondary to posterior leaflet prolapse, intermediate probability pulmonary hypertension, and Grade III restrictive diastolic dysfunction undergoing elective mitral valve replacement. The perioperative management emphasized several fundamental principles: preservation of systemic vascular resistance through careful anesthetic agent selection with sevoflurane, judicious fluid administration with modest preload targets, avoidance of excessive positive end-expiratory pressure to minimize pulmonary vascular resistance, and comprehensive hemodynamic monitoring with both invasive and transesophageal echocardiographic assessment. The patient demonstrated excellent perioperative hemodynamic stability without requirement for inotropic support, successful separation from cardiopulmonary bypass, and uncomplicated postoperative course with prompt extubation on postoperative day one. The reduction in pulmonary artery pressure from preoperative value of 19 millimeters of mercury to postoperative value of 17 millimeters of mercury confirmed that the pulmonary hypertension was secondary and reactive, as expected in mitral valve disease. The resolution of tricuspid regurgitation severity in the postoperative period further supported the functional nature of the right ventricular changes. This case illustrates that despite significant preoperative cardiac compromise and advanced diastolic dysfunction, comprehensive preoperative optimization and tailored perioperative management can achieve excellent outcomes in patients undergoing mitral valve surgery. The principles outlined in this case—particularly the understanding of the pathophysiologic basis for decision-making rather than adherence to rigid protocols—may be applicable to the management of other complex valvular disease patients with pulmonary hypertension or advanced diastolic dysfunction. Future research should focus on the long-term outcomes in patients with mitral valve disease and advanced diastolic dysfunction, particularly regarding the degree of reverse remodeling achievable following surgical correction and the impact on symptom resolution and functional capacity.

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