Mitral Valve Replacement: A Case Report

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Mitral regurgitation is commonly encountered in anesthesia clinical practice. Knowledge of the pathophysiology and proper anesthetic management is crucial to achieving optimal outcomes. Surgical advancements and early intervention have led to improved outcomes.

An ASA class III, 58-year-old woman with mitral regurgitation secondary to rheumatic fever, presented for repair or replacement of the mitral valve. A graded induction with low-dose narcotic, isoflurane, and phenylephrine was required to maintain acceptable cardiovascular parameters during induction and throughout the case. Additional interventions included adequate preload, normal heart rate, and decreased afterload, to maintain a mean arterial pressure of 65 mm Hg. Ampicillin and gentamicin were administered according to American Heart Association guidelines for prophylactic management against subacute bacterial endocarditis. Milrinone and epinephrine were required for inotropic support until the left ventricle recovered from ischemic time. Milrinone was an ideal inotrope in this case, as its vasodilator properties allowed an increase in forward flow with minimal impact on pulmonary hypertension.

Goals for the anesthetist include preservation of forward flow with minimal regurgitation and decreased pulmonary congestion. Invasive monitoring and transesophageal echocardiography have improved diagnostics and anesthetic management.

Keywords: Mitral valve, mitral regurgitation, mitral valve replacement.

Mitral regurgitation (MR) is the most commonly encountered valvular lesion in anesthesia clinical practice.[1] With MR, the left atrium (LA) acts as a low-pressure vent during left ventricular (LV) ejection; there is no isovolumetric contraction period because blood is immediately ejected retrograde with the onset of ventricular systole.[2] Consequently, total left ventricular stroke volume consists of the forward flow through the aorta as well as the retrograde flow into the LA.

The onset of rapid atrial fibrillation and congestive heart failure in a patient with MR is a marker for worsened outcome and an indication for surgery. In MR, favorable loading conditions (increased preload, normal afterload) augment ejection fraction and cause an overestimation of muscle function; surgery should occur before ejection fraction decreases to 50% to 60%.[3] If possible, repair of the mitral valve should be considered first, but when repair is unlikely, a more definitive approach with valve replacement should be taken.[1,3] Recent studies suggest that the prognosis of patients with a diagnosis of severe MR is poor when they are treated conservatively.[4] Such patients may remain asymptomatic until after irreversible LV dysfunction has already occurred. Furthermore, surgical advances such as chordal preservation and valve repair or replacement have dramatically improved surgical mortality and morbidity. Thus, a consensus is developing that favors early surgery for severe MR, even in asymptomatic patients.[4] With MR, the goal is to reduce or minimize regurgitant flow across the mitral valve.

Case Summary

A 58-year-old woman presented for repair or replacement of the mitral valve secondary to MR. The patient reported a history of MR for several years, which was secondary to rheumatic fever she experienced as a child. Over the past several months, her symptoms of chronic fatigue, dyspnea with exertion, and orthopnea increased. One week before surgery, she was admitted to the hospital with acute shortness of breath, congestive heart failure, and new-onset rapid atrial fibrillation.

Chest xray on admission showed pulmonary congestion/edema with mild cardiomegaly. The electrocardiogram showed atrial fibrillation with rapid ventricular response, a heart rate of 136/min, and occasional premature ventricular contractions. She was treated with diuretics, antiarrhythmics, and vasodilators, and her condition stabilized. Echocardiography revealed moderate to severe MR of 60% with mild aortic regurgitation. Peak systolic pulmonary artery (PA) pressures were 35 to 45 mm Hg. A cardiac catheterization was performed once the patient was stabilized. Cardiac angiography revealed mild coronary artery disease with 10% to 20% blockage of the circumflex artery and an LV ejection fraction of 45% to 50%.

The medical history included systemic hypertension, mild pulmonary hypertension, and gastroesophageal reflux disease. Medications included diltiazem, furosemide, and pantoprazole (Protonix). Surgical history was remarkable for laparoscopic cholecystectomy and 2 cesarean deliveries with no anesthetic complications.

Results of the preoperative examination the morning

of surgery showed a well-developed, well-nourished female who appeared her stated age and was in no acute distress. She had no known allergies. Her height was 167 cm, and weight was 80 kg. The vital signs were as follows: blood pressure, 122/63 mm Hg; oral temperature, 36.7°C; heart rate, 67/min and irregular; and respiratory rate, 16/min. Bilateral breath sounds were clear to auscultation, with diminished sounds bilaterally in the bases. Oxygen saturation on 2 L/min nasal cannula was 98%. Respirations were regular and unlabored. A grade III systolic murmur was heard at the fifth intercostal space midclavicular line, with radiation to the left chest border. Her airway was classified as a Mallampati class II, with all teeth intact and an adequate oral opening. There was full range of motion of the cervical spine. Heparin was infused at 600 U/h via a 20-gauge intravenous (IV) catheter in her right arm. Preoperative laboratory values were as follows: prothrombin time, 15.2 seconds; international normalized ratio, 1.3; and partial thromboplastin time, 35 seconds. All other values were within normal limits. The patient was classified ASA class III. She had had nothing by mouth since the previous night (11 PM).

Consent was obtained for mitral valve repair or replacement, invasive monitoring including arterial and PA catheters, transesophageal echocardiogram (TEE), blood products, and general anesthesia. Midazolam (Versed), 1 mg, was administered, the heparin infusion was discontinued, and the patient was transported to the operating room. The patient was assisted onto the operative table and noninvasive monitors and oxygen were applied. Within 1 hour of incision, 2 g of ampicillin and 80 mg of gentamicin were administered IV. An additional 1 mg of midazolam and 100 µg of fentanyl were administered IV. Placement of invasive lines proceeded, which included an 8.5 Fr introducer into the right internal jugular vein and PA catheter, which was secured at approximately 50 cm. A left radial arterial line was also placed and secured. Baseline cardiac measurements were as follows: heart rate, 86/min in atrial fibrillation; blood pressure, 118/58 mm Hg; PA pressures, 38/22 mm Hg; and central venous pressure, 14 mm Hg. Cardiac output measured at 3.5 mL/min, with the cardiac index at 2.7 L/min per square meter.

After preoxygenation with 100% fraction of inspired oxygen, anesthesia was induced with 500 µg of fentanyl, 3 mg of midazolam, and 12 mg of etomidate to maintain hemodynamic stability. Also, 10 mg of pancuronium was administered for muscle relaxation, for the added benefit of increased heart rate, counteracting the opioid-related bradycardia.

The trachea was intubated with a 7.5-mm endotracheal tube, and placement was confirmed with end-tidal carbon dioxide measurement and auscultation of bilateral breath sounds. Isoflurane was initiated at 0.5%, and 240 µg of phenylephrine was administered in divided doses to maintain mean arterial pressure greater than 65 mm Hg. A TEE probe was placed, and a +2 to +3 MR with mild aortic insufficiency was noted.

After a test dose of 10,000 kallikrein-inhibiting units (KIU) of aprotinin was administered without negative effects, a loading dose of 2 million KIU was infused over 20 minutes. A continuous infusion of aprotinin was then initiated at 500,000 KIU/h and continued throughout the surgery. An additional 250 µg of fentanyl was administered, and the surgeon proceeded with the chest incision. A heparin bolus of 24,000 U was administered before cardiopulmonary bypass, and the initial activated clotting time (ACT) was maintained at more than 700 seconds. Cardiopulmonary bypass was instituted after retrograde priming, and the perfusionist maintained blood pressure with intermittent boluses of phenylephrine. Cardioplegia was induced, and cardiac arrest was achieved.

After the native valve was deemed irreparable, the surgeon proceeded with replacement of the mitral valve with a porcine tissue valve. During cardiopulmonary bypass, an additional 15,000 U of heparin was administered to maintain the ACT longer than 700 seconds. Midazolam, 5 mg, was administered for an increasing bispectral index (BIS) value. Pancuronium was given to maintain a train-of-four stimulation at 0 to 1 of 4 twitches. Also, 2 U of packed red blood cells were transfused for diminished hemoglobin. Urine output was adequate during bypass.

With rewarming of the patient after mitral valve replacement, a 4-mg loading dose of milrinone was administered, and an epinephrine infusion was initiated at 0.05 µg/kg per minute. After administration of 500 mg of calcium and 2 g of magnesium, the cardiopulmonary bypass was discontinued at 2 hours and 35 minutes. Total aortic cross-clamp time was 1 hour and 25 minutes. An additional 1 g of ampicillin was also infused. After bypass, the cardiac output was 4.6 mL/min and cardiac index was 3.5 L/min per square meter. The TEE showed marked improvement in MR, with trace aortic regurgitation remaining. Protamine, 250 mg, was administered, and 1 U of packed red blood cells and 4 units of fresh frozen plasma were transfused to obtain hemostasis. The patient's chest was closed, while cardiac output remained adequate at 3.8 mL/min and cardiac index was 2.9 L/min per square meter.

The patient was transferred to the intensive care unit intubated, on full monitors with epinephrine infusing. She was extubated 6 hours postoperatively, and slowly weaned off the epinephrine infusion while adequate cardiac output was maintained.

Discussion

The mitral valve is composed of 4 components: the annulus, 2 leaflets, chordae tendinae, and papillary muscles.[3] Alteration in the proper functioning of the mitral valve leads to MR. Primary (structural) MR results

from an abnormality of any one of the mitral valve components.[3] This can occur as a result of myxomatous degeneration with mitral valve prolapse and/or chordal rupture, rheumatoid heart disease, fenfluramine intake, or endocarditis.[5] Secondary (functional) MR is the consequence of abnormalities of the LV, which modify the position of the subvalvular apparatus and determine incomplete leaflet closure.[6] These abnormalities are primarily the result of myocardial muscle failure caused by ischemia, infarction, or dilated cardiomyopathy.[1,5]

The spectrum of MR ranges from acute forms, in which rapid deterioration of myocardial function can occur, to chronic forms, which have indolent presentations.[7] Acute MR leads to a sudden increase in left atrial volume. As a result of the volume overload, left atrial, and eventually pulmonary pressures, are increased. Because LV ischemia is a common cause, acute MR often presents as biventricular failure[7] and is poorly tolerated. Studies have shown that mortality rates are higher in patients with ischemic MR and are correlated with the severity of regurgitation.[6]

Chronic MR is a progressive disorder causing a steady increase in regurgitant volume of approximately 7 to 8 mL per beats per year.[1] Severity can be classified into the following 3 stages: stage 1, mild MR (<30% of total LV stroke volume); stage 2, moderate MR (30% to 60% of LV stroke volume); and stage 3, severe MR (>60% of LV stroke volume).[7]

Mild MR may elicit no symptoms despite the eccentric hypertrophy that occurs.[7] Dilatation allows the LV to pump increased volume to compensate for that which is regurgitated, while preserving a relatively normal LV end diastolic pressure. Forward cardiac output is preserved by the overall increase in LV stroke volume. Resultant LA enlargement, from increasing regurgitant volumes, eventually leads to atrial fibrillation. The patient becomes symptomatic as the MR progresses to moderate severity. The LV dilatation and hypertrophy continue until forward stroke volume is compromised and symptoms of heart failure begin to appear. Once the regurgitant fraction reaches 60%, congestive heart failure occurs, as was evident in this case. An ejection fraction of 50% or less indicates substantial ventricular dysfunction. When the regurgitation becomes severe, terminal failure occurs. Continued LV dysfunction leads to increased pulmonary pressures and eventually right ventricular failure. Irreversible LV dysfunction eventually occurs and is not correctable despite surgical intervention on the mitral valve.

Pulmonary hypertension often is associated with both acute and chronic MR. In some studies, it was found that even in the presence of preserved LV systolic function, chronic MR is associated with pulmonary hypertension (usually mild) in as high as 76% of cases.[8] In the presence of MR, right ventricular performance deteriorates with an increase in PA pressures; deterioration of right ventricular ejection fraction was proposed as a useful predictor of progressive deterioration in cardiac function.[8]

Volume status of the LV plays a role in the amount of regurgitant flow. Some studies indicate the annular size with MR is dynamic and can be greatly altered with LV loading conditions.[1] In the absence of calcification, the size of the mitral valve regurgitant orifice parallels ventricular size. Consequently, the degree of MR parallels the changes in preload and afterload.[1]

It can be extremely challenging to maintain cardiovascular stability with optimal hemodynamic parameters and adequate systemic perfusion pressure during the anesthetic management of patients with MR. A thorough understanding of the factors affecting myocardial function and the patient's disease is essential. The preoperative evaluation will assist the anesthesia team to distinguish between acute and chronic MR as well as compensated vs decompensated status, and will reveal the patient's underlying (baseline) status. The physical examination findings seek to elicit signs of cardiac decompensation such as an S3 gallop, rales, jugular venous distention, or pulsatile liver. Routes for vascular access should be assessed, both venous and arterial, and a careful evaluation of the airway should be completed.[2]

An extensive preoperative evaluation of our patient revealed decompensating chronic MR resulting from rheumatic fever contracted as a child. She presented with classic signs and symptoms of atrial fibrillation, pulmonary congestion, cardiomegaly, fatigue, and acute shortness of breath.

Currently, quantification of MR usually is performed with Doppler echocardiography. By providing clear, noninvasive images of the structure and function of the mitral valve, 2-dimensional echocardiography is invaluable in the evaluation of MR.[8] The uses of Doppler echocardiography to establish the severity of MR include: (1) visual examination of the jet: assumes an increase in jet size and reflects an increase in severity of MR; (2) jet mapping: jet size is measured and expressed in relation to the left atrial size; (3) proximal isovelocity surface area (pica) method: scientific approach where flow equals velocity multiplied by the area; (4) vena contracta method: evaluates jet size as it leaves the regurgitant orifice area; and (5) quantitative echocardiographic Doppler cardiography: total flow is calculated as that which crosses the mitral valve in diastole.[1,3,6] This last method is the most accurate but time-consuming method for evaluation of MR.

Because of the close proximity of the ultrasound probe to the mitral valve, higher resolution, and multiplane capabilities, TEE is particularly well suited to identify the underlying mechanism of MR.[2] Findings from one study concluded that systematic TEE examination of the mitral valve improves identification of mitral segments, enables precise localization of pathologies, and may improve the diagnosis of the mechanism of MR.[9] This is important in

determining an approach to mitral valve repair or replacement and its feasibility.

In this case, TEE accurately identified +2 to +3 MR with mild aortic insufficiency and later confirmed marked improvement after the mitral valve was replaced.

Intraoperative monitoring of patients with hemodynamically significant MR also includes an arterial line, central venous pressure, and PA catheter as well as standard monitors. Consequently, all of these monitors were used in this case. Pulmonary artery catheters are extremely helpful in guiding fluid management. Particular attention is paid to the presence of a ventricular wave (v wave) when the PA catheter is wedged, reflecting regurgitant flow into the LA and pulmonary veins.[8] The size of the regurgitant wave or "giant v wave" depends on the compliance of the LA, the compliance of the pulmonary vasculature, the amount of pulmonary venous return, and the regurgitant volume.[7] Aortic balloon counterpulsation frequently is employed in hypotensive patients with MR because it increases forward output and mean arterial pressure, while the regurgitant volume and LV filling pressure are decreased.[10]

Pharmacologically, the therapeutic goal in acute MR is to stabilize the patient by diminishing the degree of MR, increasing forward cardiac output, and reducing pulmonary congestion.[10] Factors that determine regurgitant flow in MR are the systolic pressure gradient between LV and LA, the size of the mitral orifice, and the duration of ventricular systole; hence, the classic approach of "faster, fuller, and vasodilated" when describing management.[8]

Vasodilating drugs such as nitroprusside, hydrazaline, and nitroglycerine are useful and may accomplish all 3 goals. Thus, vasodilator therapy with a variety of agents improves forward output and decreases symptoms in patients with severe MR. Alternatively, vasopressor drugs such as phenylephrine and norepinephrine may be necessary in the presence of papillary muscle dysfunction, ruptured chordae, or dilated cardiomyopathy when modest to severe depression of LV function accompanies severe MR.[7,8] Inotropic agents, such as dobutamine and milrinone, that increase contractility have a tendency to provide increased forward flow and actually decrease regurgitation due to constriction of the mitral annulus.[7] In this case, milrinone was used for its inotropic effect and vasodilating properties to facilitate forward flow and prevent pulmonary congestion.

To summarize, the LV preload should be kept normal, and the best level of preload augmentation must be based on the patient's clinical response to a fluid load. The heart rate should be kept in the normal to elevated range, because bradycardia is harmful, leading to an increase in LV volume, a reduction in forward cardiac output, and an increase in regurgitant fraction. Contractility should be normal, afterload should be decreased, and α-adrenergic drugs should be used with extreme caution or avoided.[2,7,8]

The task confronting the anesthetist is to render the patient undergoing MR surgery analgesic, amnesic, and unconscious, while suppressing the endocrine and autonomic responses to intraoperative stress. Premedication will assist in providing a calm patient; however, anxiolytics must be used judiciously so as not to oversedate, causing hypercapnia and an increase in pulmonary vascular resistance. The patient in this case was cautiously premedicated with 2 mg of midazolam and 100 µg of fentanyl before the placement of invasive lines. Supplemental oxygen was provided, and vital signs were monitored closely to assess for an increase in MR. Risk factors for the development of infective endocarditis include the presence of MR, a systolic murmur, and valvular redundancy,[8] and these warrant prophylactic antibiotics according to the American Heart Association guidelines.[5] According to those guidelines, 2 g of ampicillin and 80 mg of gentamicin were administered over 1 hour as prophylactic antibiotics in this case. Administration of 1 g of ampicillin was repeated after the cessation of cardiopulmonary bypass, according to surgeon preference.

For induction of anesthesia, etomidate causes minimal cardiac depression, hemodynamic change, or alteration in sympathetic tone and may be the best choice in the face of severe cardiac dysfunction.[8] Opioids lack negative inotropic effects and have found widespread use as the primary agents in cardiac surgery because they provide adequate analgesia without substantial myocardial depression[2,8] and can also be used for induction of anesthesia. Muscle relaxants are a part of the anesthetic plan for cardiac surgery because paralysis facilitates endotracheal intubation, prevents movement and shivering, and attenuates skeletal muscle contraction during defibrillation. Anesthesia in our patient was induced with 500 µg of fentanyl, 3 mg of midazolam, and 12 mg of etomidate to maintain hemodynamic stability. Pancuronium, 10 mg, was given for muscle relaxation for the added benefit of increased heart rate, counteracting the opioid-related bradycardia.

For the patient with hemodynamically significant MR, higher levels of volatile agents are not likely to be well tolerated due to myocardial depression. Although smaller concentrations (approximately 0.5 minimum alveolar concentration) of isoflurane, desflurane, or sevoflurane may decrease the regurgitant fraction because of their vasodilatory properties (and minimal cardiac depression), the anesthetist cannot rely on this effect.[8]

In one study[11] fentanyl was compared with isoflurane anesthesia in patients with pulmonary hypertension undergoing mitral valve surgery. Two randomized groups received anesthesia, either induced with thiopental and maintained with isoflurane or induced with a fentanyl bolus and maintained with a fentanyl infusion. The isoflurane technique demonstrated a significantly higher hemodynamic failure rate.[11] In particular, a decrease in systemic arterial pressure was seen. Neither anesthetic

technique was associated with acute improvement of right-sided heart performance or pulmonary hypertension. Although no significant benefits of the vasodilatative properties of isoflurane were observed, the disadvantages of the predominately inhaled technique are not insurmountable. This randomized study suggests that isoflurane-based anesthesia resulted in significantly ($P < .05$) shorter time to awakening and appears to be a suitable choice for patients undergoing mitral valve surgery. Its limitations are defined only by dose-dependent cardiovascular depression.[11]

The prebypass period may be associated with hypertension and tachycardia due to intense surgical stimulation during sternotomy and will often require deepening the anesthesia. However, care must be taken to avoid hypotension during less stressful periods immediately before cardiopulmonary bypass. Aggressive treatment of hypotension with phenylephrine can lead to an increase in the mitral regurgitant fraction by dramatically increasing systemic vascular resistance. Small amounts of phenylephrine were used in this case to maintain a mean arterial pressure of 65 mm Hg without increasing regurgitant fraction on the TEE. After heparin administration, the cannulas are inserted and adequate levels of anticoagulation are checked to ensure that the patient is ready for the institution of cardiopulmonary bypass.[2] In this case, the ACT was safely maintained at a level greater than 700 seconds before cannulation and during bypass.

When the surgical repair is nearly complete, gradual rewarming of the patient begins and patient awareness becomes a possibility. The potentiation of anesthetic effects due to hypothermia dissipates and additional anesthetics may be necessary to prevent recall. An additional 5 mg of midazolam was given to minimize the potential for awareness during rewarming. A primary concern after mitral valve surgery is maintaining adequate LV function. Therefore, in the postbypass period, LV performance frequently must be augmented using intra-aortic balloon counterpulsation or inotropic support until the LV can adjust to the new hemodynamic state.[7] In this case, a 4-mg milrinone loading dose and epinephrine at 0.05 µg/kg per minute were required for inotropic support. The epinephrine was successfully tapered off in the intensive care unit within several hours postoperatively. All of these factors necessitate careful anesthetic management and planning by the anesthetist.

REFERENCES

1. Johnston W. Anesthesia for patients with valvular heart disease. *ASA Refresher Courses Anesthesiol.* 2004;32:105-119.
2. Skubas N, Lichtman AD, Sharma A, Thomas SJ. Anesthesia for Cardiac Surgery. In: Barash PG, Cullen, BF, Stoelting RK. *Clinical Anesthesia.* Philadelphia, PA: Lippincott Williams & Wilkins; 2006:905-908.
3. Carabello BA. Progress in mitral and aortic regurgitation. *Curr Probl Cardiol.* 2003;28:553.
4. Grayburn PA. Vasodilator therapy for chronic aortic and mitral regurgitation. *Am J Med Sci.* 2000;320(3):202-208.
5. Otto CM. Timing of surgery in mitral regurgitation. *Heart.* 2003; 89(1):100-105.
6. Iung B. Management of ischaemic mitral regurgitation. *Heart.* 2003; 89(4):459-464.
7. Moore RA, Martin DE. Anesthetic Management for Treatment of Valvular Heart Disease. In: Hensley F, Martin D, Gravlee G. *A Practical Approach to Cardiac Anesthesia.* Philadelphia, PA: Lippincott Williams & Wilkins; 2003:318-322.
8. Hanson EW, Neerhut RK, Lynch C III. Mitral valve prolapse. *Anesthesiology.* 1996;85:178-195. Cited by Kleinman B. Should antibiotics be prophylactically administered to a patient with an asymptomatic systolic murmur? *Anesth Analg:* 1996;89(5):1221-1222.
9. Lambert AS, Miller JP, Merrick SH, et al. Improved evaluation of the location and mechanism of mitral valve regurgitation with a systematic transesophageal echocardiography examination. *Anesth Analg.* 1999;88:1205-1212.
10. Ghobashy AM, Barash PG. Valvular Heart Disease. In: Troianos CA. *Anesthesia for the Cardiac Patient.* St Louis, MO: Mosby; 2002:259-261.
11. Howie MB, Black HA, Romanelli VA, Zvara DA, Myerowitz PD, McSweeney TD. A comparison of isoflurane versus fentanyl as primary anesthetics for mitral valve surgery. *Anesth Analg.* 1996;83(5): 941-948.

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