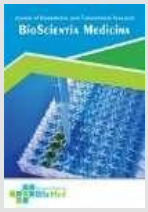

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Anesthetic Management of a Young Adult with Severe Rheumatic Mitral and Aortic Stenosis Undergoing Double Valve Replacement: A Case Report

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A B S T R A C T

Background. Rheumatic heart disease remains a leading cause of valvular pathology in young adults of low- and middle-income countries. The coexistence of severe mitral stenosis (MS) and severe aortic stenosis (AS) confronts the anesthesiologist with directly opposed hemodynamic imperatives and a markedly narrowed margin of safety, particularly during separation from cardiopulmonary bypass (CPB).

Case presentation. A 41-year-old man presented with a 14-year history of exertional syncope, progressive dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. Transthoracic echocardiography demonstrated severe rheumatic MS (mitral valve area 0.8 cm^2) and severe rheumatic AS (aortic valve area 0.8 cm^2, mean gradient 56 mmHg) with preserved left ventricular ejection fraction (67%), reduced right ventricular contractility (TAPSE 17 mm), and atrial fibrillation. He underwent double valve replacement under general anesthesia using an opioid-based, hemodynamically stable induction with full invasive monitoring. Separation from CPB was complicated by two episodes of ventricular tachycardia requiring synchronized cardioversion (30 J and 20 J) and was managed with a milrinone–dobutamine–norepinephrine strategy. The patient was transferred ventilated to intensive care on inotropic and antiarrhythmic support and stabilized.

Conclusion. Combined severe MS and AS demands an individualized plan reconciling contradictory goals: adequate preload and a controlled, unhurried heart rate for MS, against maintained afterload and coronary perfusion for AS. Meticulous invasive monitoring, a stable induction, anticipation of right ventricular dysfunction, and readiness for perioperative arrhythmia are decisive for a safe outcome.

1. Introduction

Valvular heart disease (VHD) is an increasingly prominent contributor to global cardiovascular morbidity and mortality. As populations age and as rheumatic disease persists in resource-limited regions, the absolute number of people living with clinically significant valve lesions continues to climb, and the disease now represents a substantial and rising share of the cardiovascular burden worldwide.[1,2] In high-income countries degenerative calcific disease

predominates, whereas in low- and middle-income countries rheumatic heart disease (RHD) remains the dominant etiology and characteristically affects young, economically productive adults.[3,4] The Global Burden of Disease analyses estimate tens of millions of prevalent RHD cases and hundreds of thousands of deaths each year, the majority concentrated in sub-Saharan Africa, South Asia, Oceania, and parts of Southeast Asia, including Indonesia.[3] Surgical valve replacement remains the definitive treatment for advanced rheumatic valve disease in settings where transcatheter therapies are limited, and equitable access to cardiac surgery is itself a recognized global-health priority.[5] Primary and secondary prophylaxis against streptococcal infection remain the only means of preventing the disease at its source.[6]

Rheumatic inflammation seldom respects a single valve. Mixed and multivalvular involvement is common, and the simultaneous presence of two severe lesions transforms an already demanding perioperative course into one of the most delicate exercises in cardiac anesthesia. Severe mitral stenosis (MS) imposes a fixed obstruction to left ventricular (LV) filling: diastolic time and adequate preload are precious, while tachycardia, the loss of organized atrial contraction, and abrupt increases in pulmonary blood flow precipitate pulmonary congestion.[7,8] Severe aortic stenosis (AS), conversely, imposes a fixed obstruction to LV ejection: the pressure-overloaded, concentrically hypertrophied ventricle is exquisitely dependent on adequate preload, maintained systemic vascular resistance, sinus rhythm, and an unhurried but not bradycardic heart rate to preserve the coronary perfusion of a thick, oxygen-hungry myocardium.[9,10] When these two lesions coexist at a severe grade in the same patient, their hemodynamic prescriptions collide, and every anesthetic intervention that benefits one lesion threatens the other.[11]

In Indonesia and the wider Southeast Asian region, rheumatic heart disease continues to account for a disproportionate share of valve surgery, and patients frequently present late, after years of compensated disease, with multivalvular involvement and secondary chamber remodeling already established. The combination encountered here—two valves stenosed to a critical orifice area in a man still in his fifth decade—is therefore not a curiosity but a recurring clinical pattern in tertiary referral centers serving endemic populations. Each such patient compresses into one operation the full spectrum of perioperative cardiovascular risk, and the anesthetic plan must be constructed from first principles of valvular physiology rather than from any single protocol.

Definitive management of such a patient is double valve replacement (DVR), a procedure that compounds the physiologic insult of each valve with the systemic effects of cardiopulmonary bypass (CPB), aortic cross-clamping, myocardial ischemia–reperfusion, the risk of embolism, and the frequent unmasking of right ventricular (RV) dysfunction and pulmonary hypertension during weaning.[12,13] Reports of the anesthetic conduct of DVR most often describe aortic-and-pulmonary combinations, congenital syndromes, or endocarditis-driven lesions; detailed accounts of combined severe rheumatic MS and AS in a young adult, with explicit attention to the reconciliation of their opposed hemodynamic goals, remain comparatively scarce.

The novelty of this report lies in its illustration of how two fixed obstructive lesions with mutually contradictory management targets can be balanced through a single, deliberately conservative anesthetic plan, and in its documentation of intraoperative ventricular tachycardia requiring repeated cardioversion at the moment of CPB separation—an arrhythmic event superimposed on pre-existing atrial fibrillation and right ventricular impairment. The aim of this study is to describe, in a guideline-anchored and practically reproducible manner, the perioperative anesthetic management of a 41-year-old man with severe rheumatic mitral and aortic stenosis undergoing double valve replacement, and to derive from this experience a set of transferable principles for the anesthesiologist confronting combined severe valvular obstruction.

2. Case Presentation

A 41-year-old man, an entrepreneur, was admitted for elective surgical correction of long-standing valvular heart disease. Written informed consent for anesthesia, surgery, and the publication of clinical data and images was obtained from the patient. His dominant complaint was recurrent syncope on exertion, first noted approximately 14 years before admission and recurring intermittently thereafter, with the most recent episode one week prior to admission. He described progressive effort intolerance: breathlessness after walking more than twenty meters, although he could still manage stairs slowly. He slept propped on three to four pillows and reported episodes of waking at night gasping for breath, indicating orthopnea and paroxysmal nocturnal dyspnea. He denied lower-limb swelling.

His past medical history was otherwise unremarkable: no prior surgery, hypertension, diabetes mellitus, known heart disease, stroke, or asthma, and no history of antituberculosis therapy, chemotherapy, anticoagulant use, or drug allergy. He was, however, a heavy long-term smoker, having consumed two to three packs daily for more than twenty years, stopping only three days before admission. There was no family history of hypertension, diabetes, congenital anomaly, or hereditary disease. At admission he was receiving a typical congestive regimen reflecting the chronicity and severity of his valve disease: oral furosemide 40 mg once daily, spironolactone 25 mg once daily, digoxin 0.25 mg once daily, and bisoprolol 2.5 mg once daily, together with omeprazole, ondansetron, vitamin K, N-acetylcysteine, cetirizine, low-dose alprazolam, and scheduled combivent/budesonide nebulization. His metabolic capacity was estimated at only 2–3 METs, and his Revised Cardiac Risk Index was 2.

On examination he appeared moderately ill but was fully alert (Glasgow Coma Scale E4M6V5). Blood pressure was 128/100 mmHg, heart rate 71 beats per minute, respiratory rate 19 breaths per minute, and oxygen saturation 96% on room air. He was 171 cm tall and weighed 81 kg, giving a body surface area of 1.96 m^2 and a body mass index of 27.7 kg/m^2. Airway assessment was reassuring: Mallampati class I, mouth opening of three fingerbreadths, full neck movement, and no loose or prosthetic teeth. The chest was clear to auscultation with symmetric, vesicular breath sounds and no wheeze or crackles. Cardiac examination revealed displaced apical impulse and an audible murmur without gallop; there was no pretibial edema. The remainder of the general examination was normal.

Baseline laboratory and echocardiographic findings are compiled together in Table 1; the laboratory values quoted below correspond exactly to that table. Hemoglobin (14.3 g/dL), leukocyte count, platelet count, renal function (creatinine 1.02 mg/dL, estimated glomerular filtration rate 94.7 $mL/min/1.73m^2$), electrolytes, coagulation profile (PT 15.5 s, INR 1.14, aPTT 31.9 s), and albumin were within acceptable limits; viral serologies for hepatitis B and C and HIV were non-reactive. As detailed in Table 1, two values stood out: a markedly elevated serum uric acid (10.2 mg/dL, above the 3.4–7.0 mg/dL reference range) and a high-normal C-reactive protein (4.12 mg/L), the former consistent with chronic diuretic therapy and a congested, low-output state and increasingly recognized as a marker of heart-failure severity, the latter with chronic rheumatic inflammation.

The plain chest radiograph is presented in Figure 1. The radiograph demonstrated cardiomegaly with clear lung fields, in keeping with chronic biatrial and ventricular enlargement without overt pulmonary edema at rest. The resting twelve-lead electrocardiogram was consistent with the patient's documented atrial fibrillation and additionally displayed features of right-sided strain—right-axis deviation and a right bundle branch pattern with right ventricular hypertrophy—reflecting the pulmonary hypertension and right ventricular pressure overload imposed by long-standing left-sided valve obstruction. The loss of organized atrial activity is the expected and hemodynamically hazardous consequence of chronic left atrial dilation in rheumatic mitral disease.

Table 1. Baseline laboratory and echocardiographic findings.

Parameter	Result	Reference range
Laboratory investigations		
Hemoglobin	14.3 g/dL	13.0–17.0 g/dL
Leukocytes	8,630 /µL	4,000–10,000 /µL
Platelets	174,000 /µL	150,000–450,000 /µL
Hematocrit	43.1 %	40–50 %
Fasting glucose	90 mg/dL	70–100 mg/dL
Urea	26.4 mg/dL	17–43 mg/dL
Creatinine	1.02 mg/dL	0.7–1.3 mg/dL
eGFR	94.7 mL/min/1.73m²	≥ 90
Sodium	143 mmol/L	135–145 mmol/L
Potassium	4.1 mmol/L	3.5–5.1 mmol/L
Chloride	**110 mmol/L ***	**98–107 mmol/L**
Uric acid	**10.2 mg/dL †**	**3.4–7.0 mg/dL**
Prothrombin time	15.5 s	11–15 s
INR	1.14	0.9–1.2
aPTT	31.9 s	25–35 s
C-reactive protein	4.12 mg/L	< 5 mg/L
Albumin	4.3 g/dL	3.5–5.2 g/dL
Anti-HCV / HBsAg / Anti-HIV	Non-reactive	Non-reactive
Transthoracic echocardiography		
Left ventricular ejection fraction	67% (preserved)	
Left ventricular geometry	Concentric hypertrophy	
Left atrium	Dilated	
Aortic valve	**Severe rheumatic stenosis; AVA 0.8 cm²; mean gradient 56 mmHg ***	
Aortic regurgitation	Mild–moderate (pressure half-time 442 ms)	
Mitral valve	**Severe rheumatic stenosis; MVA 0.8 cm² ***	
Mitral regurgitation	Mild	
Global wall motion	Normokinetic	
Right ventricle	**Reduced contractility (TAPSE 17 mm) †**	
Coronary angiography	No obstructive epicardial disease (small diagonal branch)	

*Notes: Laboratory: * mildly elevated chloride; † markedly elevated uric acid. Echocardiography: * values meeting guideline thresholds for severe stenosis; † reduced right ventricular systolic function. AVA, aortic valve area; MVA, mitral valve area; TAPSE, tricuspid annular plane systolic excursion; INR, international normalized ratio; aPTT, activated partial thromboplastin time; eGFR, estimated glomerular filtration rate.*

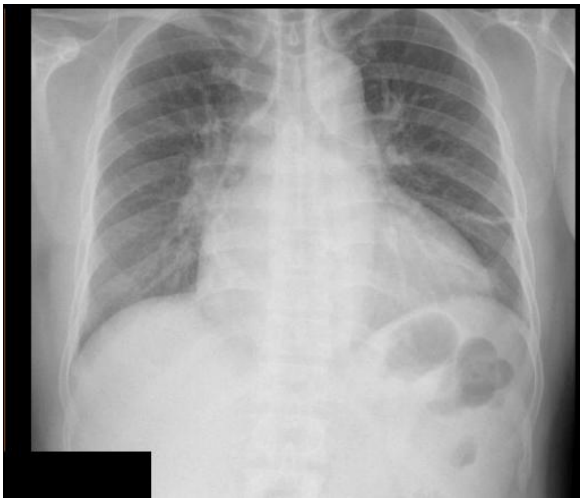

Figure 1. Preoperative posteroanterior chest radiograph demonstrating cardiomegaly (enlarged cardiac silhouette) with clear lung fields. Patient identifiers have been redacted to protect privacy.

Transthoracic echocardiography established the definitive anatomic and functional diagnosis and is detailed in Table 1. Left ventricular systolic function was preserved, with an ejection fraction of 67%, but the chamber was concentrically hypertrophied and the left atrium was dilated. As detailed in Table 1, the aortic valve showed severe rheumatic stenosis, with a planimetered valve area of 0.8 cm² and a mean transvalvular gradient of 56 mmHg, accompanied by mild-to-moderate aortic regurgitation (pressure half-time 442 ms). The mitral valve showed severe rheumatic stenosis, with a planimetered valve area of 0.8 cm² and associated mild mitral regurgitation. Global wall motion was normokinetic, but right ventricular contractility was reduced, with a tricuspid annular plane systolic excursion (TAPSE) of only 17 mm (Table 1)—an early but important marker of the right-sided strain that chronic left-sided obstruction inevitably imposes. Coronary angiography excluded significant epicardial coronary disease, demonstrating normal left main, left anterior descending, circumflex, and right coronary arteries with only a small diagonal branch.

On the basis of these findings the patient was classified as American Society of Anesthesiologists physical status II and given a working diagnosis of severe mitral stenosis and severe aortic stenosis with atrial fibrillation, of rheumatic etiology. After multidisciplinary review he was scheduled for double (mitral and aortic) valve replacement under general anesthesia with endotracheal intubation. Preoperative optimization targeted a hemoglobin above 10 g/dL with packed red cells held in reserve, with two units of packed red cells and 600 mL of fresh frozen plasma cross-matched; the patient fasted for six hours, and postoperative intensive care was arranged in advance.

Anesthetic preparation followed a deliberate, low-stress sequence. Standard monitoring—electrocardiography, noninvasive then invasive arterial pressure, pulse oximetry, capnography, central venous pressure, and temperature—was established, supplemented by intraoperative transesophageal echocardiography to guide volume status, confirm valve function, de-air the heart, and assess ventricular performance during weaning. A controlled, opioid-based induction was chosen specifically to avoid the swings in heart rate and systemic vascular resistance that combined MS and AS tolerate so poorly. The principal vasoactive and inotropic agents prepared and titrated through the perioperative period are listed in Table 2. As set out in Table 2, premedication was minimal (midazolam 1 mg). Induction used morphine 10 mg and sufentanil 500 µg for a dense, hemodynamically stable opioid base, with rocuronium 90 mg for neuromuscular blockade; the trachea was intubated with a size-8 tube. Anesthesia was maintained with sevoflurane at approximately 2 vol% supplemented by the opioid loading. Vasoactive support was prepared and titrated to need: dobutamine 5–10 µg/kg/min and milrinone 0.2 µg/kg/min for inotropy and pulmonary vasodilation, norepinephrine 0.05–0.4 µg/kg/min to defend systemic and coronary perfusion pressure, and nitroglycerin 0.5–5 µg/kg/min for venocapacitance and coronary tone (all agents and doses are listed in Table 2).

The intraoperative hemodynamic course is shown in Figure 2, which tracks systolic and diastolic arterial pressure, mean pulmonary arterial pressure, and central venous pressure across the procedure. After a stable induction and surgical exposure, the patient was established on cardiopulmonary bypass—a period spanning approximately two and a half hours as recorded on the intraoperative trend—and the aortic cross-clamp applied for excision of both diseased valves and implantation of prostheses. The most demanding phase was separation from bypass. As the heart was reperfused and rewarmed, the patient developed ventricular tachycardia at 17:13, treated immediately with synchronized cardioversion at 30 J; a second episode of ventricular tachycardia at 18:00 was again terminated by synchronized cardioversion at 20 J, both events being annotated directly on the trend in Figure 2. These arrhythmic events, arising on a background of atrial fibrillation and reduced right ventricular reserve, were managed alongside

escalation of the milrinone–dobutamine–norepinephrine regimen to support biventricular function and unload the pulmonary circulation, allowing successful separation from bypass as reflected in the recovery of arterial pressure on the right of the trend record.

Table 2. Perioperative vasoactive, anesthetic, and antiarrhythmic agents.

Phase	Agent	Dose / regimen	Rationale
Premedication	Midazolam	1 mg IV	Anxiolysis
Induction	Morphine	10 mg IV	Opioid base, sympatholysis
Induction	Sufentanil	500 µg IV	Dense, stable opioid anesthesia
Induction	Rocuronium	90 mg IV	Neuromuscular blockade
Maintenance	Sevoflurane	≈ 2 vol%	Amnesia and titratable depth
Intraoperative	Dobutamine	5–10 µg/kg/min	Inotropy
Intraoperative	Milrinone	0.2 µg/kg/min	Inodilation, pulmonary unloading
Intraoperative	Norepinephrine	0.05–0.4 µg/kg/min	Systemic and coronary perfusion
Intraoperative	Nitroglycerin	0.5–5 µg/kg/min	Venodilation, coronary tone
Intensive care	Propofol	40 mg/h	Sedation
Intensive care	Milrinone	0.4 µg/kg/min	Inodilation
Intensive care	Norepinephrine	0.2 µg/kg/min	Perfusion pressure
Intensive care	Dobutamine	3 µg/kg/min	Inotropy
Intensive care	Amiodarone	600 mg / 24 h	Rhythm control

Notes: IV, intravenous. Infusion rates expressed per kilogram of body weight per minute were titrated to hemodynamic targets.

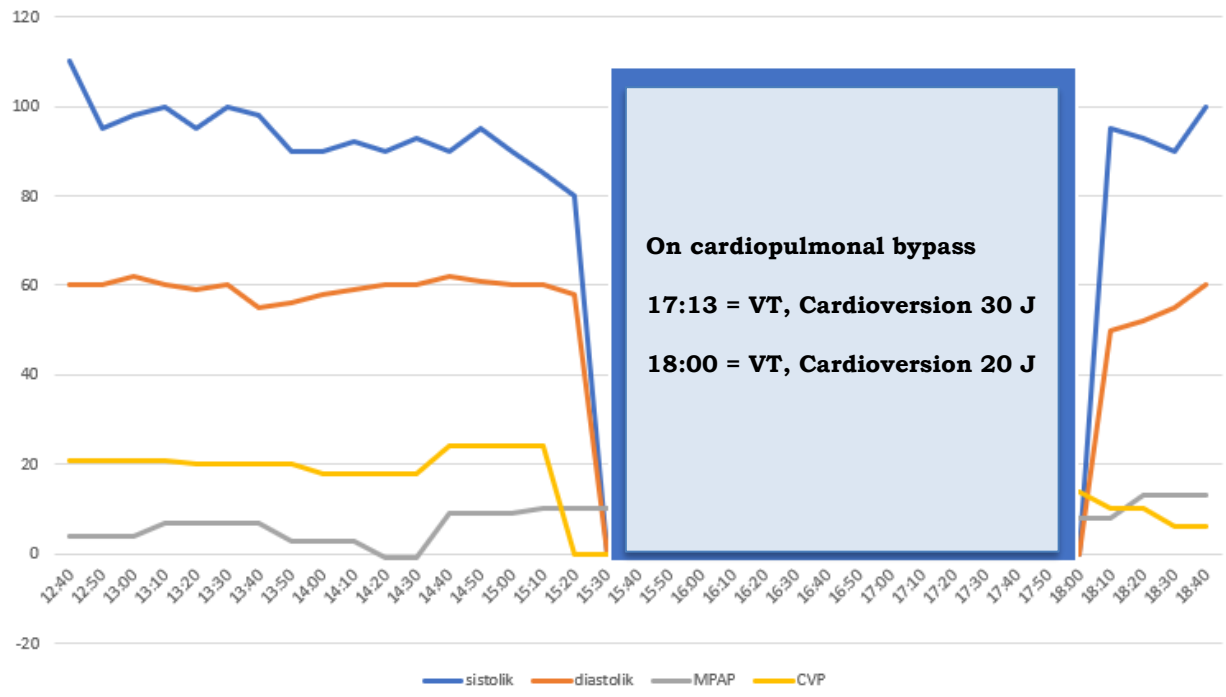

Figure 2. Intraoperative hemodynamic trends across the perioperative period. Systolic and diastolic arterial pressures, mean pulmonary arterial pressure (MPAP), and central venous pressure (CVP) are plotted against time. The shaded block marks the cardiopulmonary bypass period; annotations record two episodes of ventricular tachycardia treated with synchronized cardioversion (30 J at 17:13 and 20 J at 18:00). Original intraoperative record (Indonesian labels: sistolik = systolic, diastolik = diastolic).

At the conclusion of surgery the post-procedure diagnosis was recorded as status post double valve replacement for severe mitral stenosis, severe aortic stenosis, and atrial fibrillation. The patient was transferred, intubated and ventilated, to the intensive care unit. Immediate post-procedure vital signs were a blood pressure of 90/60 mmHg, heart rate of 126 beats per minute, and oxygen saturation of 99% on synchronized intermittent mandatory ventilation (volume control, tidal volume 400 mL, rate 12, pressure support 14 cmH_2O, PEEP 5 cmH_2O, FiO_2 50%). Intensive care orders comprised close observation of consciousness, vital signs, and bleeding; maintenance Ringer's lactate at 60 mL/h; sedation with a propofol infusion at 40 mg/h; and continued circulatory and rhythm support with milrinone 0.4 µg/kg/min, norepinephrine 0.2 µg/kg/min, dobutamine 3 µg/kg/min, and an amiodarone infusion of 600 mg over 24 hours (the complete intensive-care regimen is included in Table 2). The patient stabilized on this regimen.

3. Discussion

This case distills, in a single patient, the central challenge of contemporary rheumatic valve anesthesia: the management of two severe, fixed obstructive lesions whose physiologic requirements are diametrically opposed. The discussion that follows situates the patient's presentation, the rationale of the anesthetic plan, the management of the intraoperative arrhythmia and bypass separation, and the postoperative trajectory within the current evidence base.

Epidemiologic and etiologic context

The patient is, in many respects, an archetype of rheumatic heart disease in an endemic country. He is a young adult—41 years old—with advanced, multivalvular, calcific-rheumatic disease of a kind that is now uncommon at his age in high-income settings but remains the leading valvular pathology across much of Asia and Africa.[3,4] The high-normal C-reactive protein is consistent with the chronic inflammatory substrate of rheumatic disease, the pathogenesis of which traces to inadequately treated group A streptococcal infection and the absence of secondary prophylaxis.[6] His presentation also reflects a health-systems reality: fourteen years elapsed between his first syncopal episode and definitive surgery, a delay that allowed both valves to progress to severe stenosis, the left atrium to dilate into atrial fibrillation, and the right ventricle to begin to fail. This natural history underscores why expanded access to timely cardiac surgery is regarded as a global-health imperative rather than a purely technical concern.[5] The growing worldwide burden of valvular disease ensures that anesthesiologists everywhere will increasingly encounter such patients.[1,2]

Reconciling opposed hemodynamic goals

The intellectual core of this case is the collision of management targets. In isolation, severe mitral stenosis is managed by protecting diastolic filling time and preload: a controlled, relatively slow heart rate, avoidance of tachycardia, preservation of any residual atrial contribution, and careful avoidance of the pulmonary congestion that follows fluid overload or sudden increases in pulmonary flow.[7,8] Atrial fibrillation, present in this patient, removes the atrial "kick" and, if the ventricular response is rapid, abbreviates diastole precisely when the stenotic mitral valve most needs time—hence the long-standing pharmacologic strategy of rate control and the central role of anticoagulation in rheumatic mitral disease.[14] Severe aortic stenosis, by contrast, is managed by defending afterload and coronary perfusion: maintenance of systemic vascular resistance, avoidance of hypotension that would collapse the perfusion of a hypertrophied myocardium, avoidance of tachycardia that both shortens diastolic coronary filling and increases oxygen demand, and avoidance of bradycardia that would compromise a relatively fixed stroke volume.[9,10]

When both lesions are severe, the anesthesiologist cannot fully satisfy either prescription and must instead steer a narrow middle course.[11] It must also be noted that the aortic lesion here was a mixed one—severe stenosis with mild-to-moderate regurgitation—so that a degree of volume load was superimposed on the dominant pressure load; nonetheless the fixed stenosis governed management, and the coexisting

regurgitation principally reinforced the imperative to avoid both extremes of heart rate and to preserve diastolic perfusion. Because a regurgitant volume augments the transvalvular gradient, the 56 mmHg mean aortic gradient was interpreted alongside the planimetered valve area of 0.8 cm^2, which remained the anchor for grading stenosis severity. The shared, non-negotiable principles are the maintenance of sinus-equivalent rate control—neither the tachycardia that ruins the mitral valve nor the bradycardia that starves the aortic ventricle—the vigilant preservation of systemic vascular resistance and therefore coronary perfusion pressure, and the cautious, echo-guided titration of preload to fill the stenotic valves without flooding the pulmonary bed. The opioid-based induction selected here directly serves these aims: high-dose synthetic and natural opioids (sufentanil and morphine) blunt the sympathetic response to laryngoscopy and surgery while producing minimal myocardial depression and little change in systemic vascular resistance, in deliberate preference to agents that would drop afterload or provoke reflex tachycardia. The modest dose of volatile agent and the early availability of norepinephrine to restore vascular tone reflect the same logic. This conservative, perfusion-protective philosophy is consistent with contemporary recommendations for the perioperative cardiovascular management of high-risk patients.[15]

The specific pharmacology of the induction deserves emphasis because, in combined severe MS and AS, the choice of agent is itself a hemodynamic decision. A pure propofol induction, while convenient, produces dose-dependent venodilation and a fall in systemic vascular resistance that the aortic ventricle cannot tolerate and that can collapse coronary perfusion pressure in a heartbeat; if used at all it must be given in small, titrated increments with a vasopressor immediately at hand. Agents that provoke tachycardia, such as ketamine in some patients or the reflex response to a light plane, are equally hazardous because tachycardia simultaneously shortens mitral diastolic filling and aortic diastolic coronary filling. The high-dose opioid technique employed here—sufentanil combined with morphine—was therefore chosen deliberately: synthetic opioids attenuate the sympathetic surge of laryngoscopy and sternotomy while leaving contractility and vascular tone largely intact, producing the hemodynamic "stillness" that two fixed obstructions require. Rocuronium provided rapid, reliable relaxation without histamine release, and sevoflurane at a low concentration supplied amnesia and a measure of titratable depth without the profound vasodilation of higher volatile doses. The overarching aim throughout induction and maintenance was to keep heart rate, rhythm, preload, and afterload within the narrow corridor that both valves can survive, intervening early and in small steps rather than chasing large deviations after they occur.

Monitoring and the role of intraoperative echocardiography

The decision to deploy full invasive monitoring and intraoperative transesophageal echocardiography (TEE) was central to safe conduct. In a patient whose ventricle tolerates only a narrow band of loading conditions, beat-to-beat arterial pressure, central venous pressure, and real-time imaging of chamber filling and contractility convert an otherwise blind titration into a guided one. TEE additionally confirms the adequacy of valve excision and prosthesis seating, detects paravalvular leak, guides de-airing, and—critically for this case—provides a continuous window on right ventricular performance and the response to inotropic and vasodilator therapy during the vulnerable period of bypass separation.[16] The patient's preoperative echocardiographic profile (Table 1) defined the battlefield in advance: two valve areas of 0.8 cm^2, a 56 mmHg mean aortic gradient, preserved left ventricular ejection fraction with concentric hypertrophy, and—most ominously—a reduced TAPSE of 17 mm signaling early right ventricular impairment.

The right ventricle, pulmonary hypertension, and separation from bypass

Chronic severe left-sided valve obstruction transmits elevated filling pressures backward into the pulmonary circulation, producing pulmonary hypertension and, in time, right ventricular dysfunction—the very combination heralded by this patient's reduced TAPSE.[17-19] It is during separation from cardiopulmonary bypass that this right-sided

pathology exacts its price, as the reperfused, potentially stunned right ventricle is suddenly asked to eject against a stiff, hypertensive pulmonary bed. The anesthetic strategy anticipated this. Milrinone, an inodilator, was selected precisely because it augments contractility while lowering pulmonary vascular resistance, an attractive profile when the failing chamber is the right ventricle facing pulmonary hypertension; it was combined with dobutamine for additional inotropy.[20] Norepinephrine was reserved to defend systemic and coronary perfusion pressure without unduly aggravating pulmonary pressures. This graded approach mirrors the principle, increasingly emphasized in cardiac surgical practice, that in the setting of right ventricular dysfunction and pulmonary hypertension the choice and sequence of vasoactive agents matter as much as their dose, and that escalating a pure vasoconstrictor can be counterproductive when a vasopressin-type agent or an inodilator would better preserve the delicate balance between systemic and pulmonary pressures.[21,22] The recovery of arterial pressure documented at the right of the intraoperative trend (Figure 2) attests to the success of this combined regimen in supporting biventricular function through weaning.

The pharmacologic logic of pulmonary unloading merits elaboration. The failing right ventricle is afterload-sensitive in a way the left ventricle is not; small reductions in pulmonary vascular resistance can translate into disproportionate improvements in right ventricular stroke work. Milrinone, a phosphodiesterase-3 inhibitor, is attractive precisely because it raises contractility and dilates the pulmonary vasculature simultaneously, and randomized data in adult cardiac surgery support inodilator therapy for perioperative pulmonary hypertension, whether delivered intravenously or by inhalation to concentrate the pulmonary vasodilatory effect while limiting systemic hypotension.[20] Its principal liability—systemic vasodilation—was offset here by norepinephrine, titrated to hold coronary and systemic perfusion pressure. Nitroglycerin contributed venous capacitance control and coronary vasodilation, useful when preload must be trimmed without provoking reflex tachycardia. Where a pure vasoconstrictor strategy fails or worsens pulmonary pressures, the literature increasingly favors vasopressin, which restores systemic pressure with comparatively little effect on the pulmonary bed, a distinction demonstrated in randomized comparison with norepinephrine after cardiac surgery and illustrated in reports of bypass separation complicated by pulmonary hypertension.[21,22] The graded, agent-specific approach used here reflects this evidence: support the right ventricle, unload the lungs, and defend systemic perfusion, each with the agent best suited to the task.

Perioperative arrhythmia: from atrial fibrillation to ventricular tachycardia

The patient entered the operating room in atrial fibrillation, a rhythm intrinsic to his chronic mitral disease and one whose loss of atrial systole and tendency to rapid ventricular response are independently deleterious in both of his stenotic lesions.[14,23] The intraoperative course, however, was punctuated by a more acute threat: two discrete episodes of ventricular tachycardia at the moment of reperfusion and bypass separation, each terminated promptly by synchronized cardioversion (30 J and 20 J). Ventricular arrhythmia in this context is multifactorial—ischemia–reperfusion injury, mechanical irritation, electrolyte and acid–base shifts, and the heightened irritability of a hypertrophied, recently arrested myocardium all contribute. The decisive response was immediate electrical cardioversion, the most reliable means of restoring an organized rhythm in a hemodynamically significant tachyarrhythmia, followed by an amiodarone infusion for rhythm stabilization in the intensive care unit.[24] The episode is a reminder that, in valvular cardiac surgery, the anesthesiologist must remain perpetually prepared for sudden rhythm collapse precisely when the patient can least afford it, with a defibrillator charged and antiarrhythmic therapy immediately available.

Prevention is as important as treatment. Because reperfusion arrhythmia is provoked by metabolic derangement, deliberate optimization of the internal

milieu during rewarming—correction of potassium toward the high-normal range, generous magnesium supplementation, normalization of pH and ionized calcium, and avoidance of hypothermia at the moment of separation—reduces the substrate for ventricular tachycardia before it arises. The patient's preoperative rhythm strategy was equally considered: chronic atrial fibrillation in rheumatic mitral disease is typically managed by rate control with agents such as the digoxin and bisoprolol this patient was already receiving, together with anticoagulation to mitigate the high thromboembolic risk that left atrial dilatation and stasis confer.[14,23] The intraoperative shift from a supraventricular to a ventricular arrhythmia, and the immediate move from rate control to electrical cardioversion, illustrates how rapidly the rhythm priorities of such a patient can change and why both pharmacologic and electrical tools must be ready throughout.

Surgical decision and prosthesis considerations

Double valve replacement was the appropriate definitive treatment for a patient meeting severe-disease thresholds at both the mitral and aortic positions, in line with current valvular heart disease guidelines that recommend intervention for symptomatic severe MS and severe AS.[12,13] DVR is technically and physiologically more demanding than single-valve surgery, entailing longer bypass and cross-clamp times and a correspondingly greater risk of myocardial stunning, low cardiac output, and the arrhythmias this patient experienced; the published DVR literature, though dominated by congenital and syndromic cases, consistently emphasizes the multidisciplinary planning and individualized perioperative strategy that such procedures require.[25,26,27] In a 41-year-old, mechanical prostheses are commonly preferred for their durability, with the understanding that they commit the patient to lifelong vitamin-K-antagonist anticoagulation and its attendant, lifelong risk of prosthetic valve thrombosis and bleeding—a trade-off that must be explicitly discussed and carefully managed thereafter.[28] His pre-existing atrial fibrillation independently mandates anticoagulation, somewhat simplifying that calculus. After mechanical double valve replacement, lifelong vitamin-K-antagonist therapy targeted to an appropriate international normalized ratio is required, with the early postoperative period demanding careful initiation balanced against surgical bleeding risk; meticulous long-term monitoring is essential, since both subtherapeutic anticoagulation, with its risk of valve thrombosis, and supratherapeutic anticoagulation, with its risk of hemorrhage, carry serious consequences for these patients.[28]

To situate the present case within the existing literature, Table 3 compares it with previously reported double valve replacement experiences managed by anesthesiologists.[22,26,27] The comparison makes the distinctive features of this case explicit: whereas the prior reports center on degenerative or syndromic disease and are dominated by difficult-airway or post-bypass pulmonary-hypertension challenges, the present case is defined by two opposed fixed obstructions of rheumatic origin in a young adult, compounded by atrial fibrillation and by intraoperative ventricular tachycardia requiring cardioversion during separation from cardiopulmonary bypass. As shown in Table 3, the unifying lesson across all of these reports is that double valve replacement is survived not by a single recipe but by an individualized, anticipatory hemodynamic plan matched to the specific lesions and comorbidities at hand.

Fluid, transfusion, and blood-conservation strategy

Volume management in combined severe stenotic disease is a problem of precision rather than abundance. The stenotic mitral and aortic valves both depend on adequate preload, yet the congested pulmonary circulation and the impaired right ventricle punish even modest over-resuscitation. The preoperative plan reflected this tension: a hemoglobin target above 10 g/dL to preserve oxygen-carrying capacity in a patient with limited cardiac reserve, with two units of packed red cells and fresh frozen plasma cross-matched in advance, against a deliberately restrained crystalloid approach to avoid pulmonary overload. With an estimated blood volume of approximately 6,075 mL, the allowable blood loss

corresponding to a 10%, 20%, and 30% volume reduction was calculated in advance so that transfusion thresholds were defined before, rather than during, hemorrhage. Cardiopulmonary bypass itself imposes hemodilution, a coagulopathic insult, and an inflammatory response, all of which argue for a proactive transfusion and hemostasis strategy and for the judicious use of blood products guided by point-of-care assessment where available. Throughout, the guiding principle was to fill the stenotic valves enough to maintain forward flow while never exceeding the narrow ceiling that the pulmonary bed and right ventricle would tolerate—an echocardiographically guided, continuously re-evaluated balance rather than a fixed fluid prescription.

Table 3. Comparison of the present case with previously reported double valve replacement experiences in the anesthesia literature.

Study (year)	Population and valve lesions (etiology)	Distinctive challenge	Key anesthetic–hemodynamic strategy	Outcome
Present case (2026)	41-year-old man; severe mitral + aortic stenosis (rheumatic); atrial fibrillation; reduced RV	Two opposed fixed obstructions; intraoperative VT during separation from CPB	Opioid-based stable induction; invasive + TEE monitoring; milrinone–dobutamine–norepinephrine; synchronized cardioversion	Stabilized in ICU on inotropic and antiarrhythmic support
Sato et al. (2025)[22]	71-year-old woman; aortic + pulmonary stenosis (Ozaki reconstruction) with CABG	Severe pulmonary hypertension on separation from CPB	Vasopressin preferred over norepinephrine to avoid worsening pulmonary pressures	Stabilized; first reported Ozaki double-valve anesthetic
Keshavamurthy et al. (2022)[27]	Hunter syndrome (MPS II); mitral + aortic valve replacement	Difficult airway (macroglossia, supraglottic narrowing) and multisystem disease	Multidisciplinary airway and anesthetic planning; mechanical prostheses	Successful valve replacement
Demis et al. (2021)[26]	Maroteaux–Lamy syndrome (MPS VI); double valve replacement	Multisystem storage disease (“ultimate team challenge”)	Multidisciplinary perioperative planning	Successful double valve replacement

Notes: AS, aortic stenosis; CABG, coronary artery bypass grafting; CPB, cardiopulmonary bypass; ICU, intensive care unit; MPS, mucopolysaccharidosis; MS, mitral stenosis; RV, right ventricle; TEE, transesophageal echocardiography; VT, ventricular tachycardia.

Postoperative course and intensive care

The immediate postoperative profile—a blood pressure of 90/60 mmHg with a heart rate of 126 beats per minute on multi-agent vasoactive support—is characteristic of the low-output, vasodilated, tachycardic state that frequently follows prolonged bypass for severe biventricular-loading disease. The intensive care regimen was rational and goal-directed: propofol sedation for ventilator synchrony, milrinone and dobutamine for inotropy and pulmonary unloading, norepinephrine for perfusion pressure, amiodarone for rhythm control, and conservative maintenance fluids to avoid pulmonary overload in a patient with newly operated stenotic physiology and a vulnerable right ventricle. These choices align with established principles for the management of postoperative low cardiac output and heart failure.[29] The persistence of tachycardia underscores the importance of ongoing rhythm surveillance and judicious rate control in the early postoperative window, when the balance achieved intraoperatively must be actively maintained.

The patient's respiratory status warranted particular attention. As a long-term heavy smoker of more than twenty pack-years who had stopped only three days before surgery, he carried an elevated risk of perioperative bronchospasm, secretion retention, and impaired gas exchange, compounded by the pulmonary congestion intrinsic to his mitral disease and the predictable pulmonary insult of cardiopulmonary bypass. The preoperative regimen of

bronchodilator and corticosteroid nebulization and N-acetylcysteine addressed this risk directly, and the postoperative ventilator settings—a modest tidal volume with moderate positive end-expiratory pressure—were selected to recruit atelectatic lung while avoiding the increases in pulmonary vascular resistance that excessive airway pressure would impose on an already strained right ventricle. Lung-protective ventilation in this setting is thus not merely a respiratory measure but a hemodynamic one, because the right ventricle and the pulmonary circulation are tightly coupled to airway mechanics.

Lessons and transferable principles

Several practical principles emerge. First, in combined severe MS and AS, plan for the *intersection* of the two lesions' requirements rather than either in isolation: a controlled heart rate that is neither fast nor slow, religiously defended systemic vascular resistance, and echo-guided preload. Second, anticipate right ventricular failure—foreshadowed here by a reduced TAPSE—and prepare an inodilator-based weaning strategy before, not after, the chamber declares itself. Third, treat the operating room as an electrophysiologic high-wire: pre-existing atrial fibrillation can be joined at any moment by ventricular tachycardia, and immediate synchronized cardioversion with subsequent antiarrhythmic infusion must be rehearsed and ready. Fourth, regard monitoring—invasive pressures and intraoperative TEE—not as a luxury but as the instrument that makes a narrow-margin anesthetic survivable. These principles are not novel in their components, but their simultaneous application in a young rheumatic patient with two severe fixed obstructions, captured here with an intraoperative hemodynamic record, offers a concrete template for a situation that anesthesiologists in endemic regions will continue to face.

Limitations

This is a single-patient report describing perioperative and immediate postoperative management; it cannot establish the superiority of any one strategy and reflects the resources and practices of a single tertiary center. Certain quantitative data customarily reported in such cases—exact bypass and cross-clamp durations, serial blood-gas and electrolyte values, and detailed prosthesis specifications—were beyond the scope of the available perioperative record and are therefore not asserted here rather than estimated. A baseline estimated pulmonary artery systolic pressure was likewise not recorded, so the pulmonary-hypertension discussion rests on the reduced TAPSE, the intraoperative mean pulmonary arterial pressure trend, and the known natural history of the lesions rather than on a single preoperative number. The atrial fibrillation diagnosis rests on the clinical and operative record and serial assessment rather than on a single reproduced tracing, and beat-to-beat variability in atrial fibrillation imposes a recognized limitation on echocardiographic gradient and valve-area estimation; the rheumatic etiology was inferred from the patient's age and geography together with the characteristic morphologic findings. Long-term follow-up, including extubation time, length of stay, discharge rhythm, anticoagulation stability, functional recovery, and valve performance, was not available at the time of writing. Despite these constraints, the case provides a coherent, evidence-anchored account of a demanding and clinically instructive scenario.

4. Conclusion

Combined severe rheumatic mitral and aortic stenosis presents the anesthesiologist with two fixed obstructive lesions whose hemodynamic prescriptions directly oppose one another, leaving an exceptionally narrow margin for error. Safe conduct of double valve replacement in such a patient rests on an individualized plan that steers between the competing imperatives—adequate preload and an unhurried, controlled heart rate for the mitral lesion, set against preserved afterload and coronary perfusion for the aortic lesion—supported by a stable opioid-based induction, comprehensive invasive monitoring with intraoperative transesophageal echocardiography, and the early anticipation of right ventricular dysfunction and pulmonary hypertension during separation from cardiopulmonary bypass. The intraoperative emergence of ventricular tachycardia on a background

of atrial fibrillation, managed by prompt synchronized cardioversion and an inodilator-based circulatory strategy, exemplifies the perpetual readiness that valvular cardiac surgery demands. With deliberate planning and vigilant execution, even this most contradictory of valvular combinations can be brought safely through definitive surgical correction.

Declarations

Ethics and consent. Written informed consent for anesthesia, surgery, and the publication of clinical details and accompanying images was obtained from the patient. Identifying information has been removed from all figures.

Reporting guideline. This case report was prepared in accordance with the CARE guidelines.

Conflict of interest. The authors declare no conflict of interest.

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5. References

1. Coffey S, Roberts-Thomson R, Brown A, et al. Global epidemiology of valvular heart disease. Nat Rev Cardiol. 2021;18(12):853-864. doi:10.1038/s41569-021-00570-z
2. Chen QF, Shi S, Wang YF, et al. Global, Regional, and National Burden of Valvular Heart Disease, 1990 to 2021. J Am Heart Assoc. 2024;13(24):e037991. doi:10.1161/JAHA.124.037991
3. Watkins DA, Johnson CO, Colquhoun SM, et al. Global, Regional, and National Burden of Rheumatic Heart Disease, 1990-2015. N Engl J Med. 2017;377(8):713-722. doi:10.1056/NEJMoa1603693
4. Rwebembera J, Beaton AZ, de Loizaga SR, et al. The Global Impact of Rheumatic Heart Disease. Curr Cardiol Rep. 2021;23(11):160. doi:10.1007/s11886-021-01592-2
5. Vervoort D, Antunes MJ, Pezzella AT. Rheumatic heart disease: The role of global cardiac surgery. J Card Surg. 2021;36(8):2857-2864. doi:10.1111/jocs.15597
6. Gerber MA, Baltimore RS, Eaton CB, et al. Prevention of rheumatic fever and diagnosis and treatment of acute Streptococcal pharyngitis: a scientific statement from the American Heart Association. Circulation. 2009;119(11):1541-1551. doi:10.1161/CIRCULATIONAHA.109.191959
7. Harb SC, Griffin BP. Mitral Valve Disease: a Comprehensive Review. Curr Cardiol Rep. 2017;19(8):73. doi:10.1007/s11886-017-0883-5
8. Nishimura RA, Vahanian A, Eleid MF, et al. Mitral valve disease--current management and future challenges. Lancet. 2016;387(10025):1324-1334. doi:10.1016/S0140-6736(16)00558-4
9. Lindman BR, Clavel MA, Mathieu P, et al. Calcific aortic stenosis. Nat Rev Dis Primers. 2016;2:16006. doi:10.1038/nrdp.2016.6
10. de Oliveira Sá MPB, Cavalcanti LRP, Perazzo ÁM, et al. Calcific Aortic Valve Stenosis and Atherosclerotic Calcification. Curr Atheroscler Rep. 2020;22(2):2. doi:10.1007/s11883-020-0821-7
11. Unger P, Tribouilloy C. Aortic Stenosis with Other Concomitant Valvular Disease: Aortic Regurgitation, Mitral Regurgitation, Mitral Stenosis, or Tricuspid Regurgitation. Cardiol Clin. 2020;38(1):33-46. doi:10.1016/j.ccl.2019.09.002
12. Otto CM, Nishimura RA, Bonow RO, et al C. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: Executive Summary. Circulation. 2021;143(5):e35-e71. doi:10.1161/CIR.0000000000000932
13. Vahanian A, Beyersdorf F, Praz F, et al. 2021 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J.

2022;43(7):561-632.
doi:10.1093/eurheartj/ehab395

14. Iung B, Leenhardt A, Extramiana F. Management of atrial fibrillation in patients with rheumatic mitral stenosis. Heart. 2018;104(13):1062-1068. doi:10.1136/heartjnl-2017-311425
15. Smilowitz NR, Berger JS. Perioperative Cardiovascular Risk Assessment and Management for Noncardiac Surgery: A Review. JAMA. 2020;324(3):279-290. doi:10.1001/jama.2020.7840
16. Dumps C, Umrath V, Rupprecht B, et al. Intraoperative transesophageal echocardiography for emergency diagnostics in noncardiac surgery patients. Anaesthesist. 2022;71(1):65-82. doi:10.1007/s00101-021-01034-2
17. Konstam MA, Kiernan MS, Bernstein D, et al. Evaluation and Management of Right-Sided Heart Failure: A Scientific Statement From the American Heart Association. Circulation. 2018;137(20):e578-e622. doi:10.1161/CIR.0000000000000560
18. Patel B, D'Souza S, Sahni T, et al. Pulmonary hypertension secondary to valvular heart disease: a state-of-the-art review. Heart Fail Rev. 2024;29(1):277-286. doi:10.1007/s10741-023-10372-9
19. Tichelbäcker T, Dumitrescu D, Gerhardt F, et al. Pulmonary hypertension and valvular heart disease. Herz. 2019;44(6):491-501. doi:10.1007/s00059-019-4823-6
20. Ftikos P, Gkantinas G, Karageorgos V, et al. Intravenous Levosimendan versus Inhalational Milrinone in the Management of Pulmonary Hypertension during Adult Cardiac Surgery: A Randomized Clinical Trial. Life (Basel). 2024;14(9):1164. doi:10.3390/life14091164
21. Hajjar LA, Vincent JL, Barbosa Gomes Galas FR, et al. Vasopressin versus Norepinephrine in Patients with Vasoplegic Shock after Cardiac Surgery: The VANCS Randomized Controlled Trial. Anesthesiology. 2017;126(1):85-93. doi:10.1097/ALN.0000000000001434
22. Sato J, Hino H, Watanabe R, et al. Severe pulmonary hypertension in weaning from cardiopulmonary bypass following double Ozaki procedure: a case report. JA Clin Rep. 2025;11(1):23. doi:10.1186/s40981-025-00785-w
23. Hindricks G, Potpara T, Dagres N, et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the EACTS. Eur Heart J. 2021;42(5):373-498. doi:10.1093/eurheartj/ehaa612
24. Kukendrarajah K, Ahmad M, Carrington M, et al. External electrical and pharmacological cardioversion for atrial fibrillation, atrial flutter or atrial tachycardias: a network meta-analysis. Cochrane Database Syst Rev. 2024;6(6):CD013255. doi:10.1002/14651858.CD013255.pub2
25. Szabo TA, Toole JM, Payne KJ, et al. Management of aortic valve bypass surgery. Semin Cardiothorac Vasc Anesth. 2012;16(1):52-58. doi:10.1177/1089253211434565
26. Demis AA, Oikonomidou S, Daglis F, et al. Double valve replacement in a patient with Maroteaux-Lamy syndrome as an ultimate team challenge. J Cardiothorac Surg. 2021;16(1):141. doi:10.1186/s13019-021-01530-x
27. Keshavamurthy S, Duncan A, Kumar A, et al. Double Valve Replacement in a Patient With Hunter Syndrome. Cureus. 2022;14(9):e28961. doi:10.7759/cureus.28961
28. Soria Jiménez CE, Papolos AI, Kenigsberg BB, et al. Management of Mechanical Prosthetic Heart Valve Thrombosis: JACC Review Topic of the Week. J Am Coll Cardiol. 2023;81(21):2115-2127. doi:10.1016/j.jacc.2023.03.412
29. Heidenreich PA, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure. Circulation. 2022;145(18):e895-e1032. doi:10.1161/CIR.0000000000001063