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Review Article

Acute cardiac care in rheumatic mitral stenosis

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ABSTRACT

Rheumatic heart disease (RHD) is prevented, although productive-age people may develop consequences. Indonesia, as an endemic country of RHD, predominantly reported isolated mitral stenosis among RHD patients. Acute worsening of mitral stenosis can lead to acute heart failure and cardiogenic shock, arrhythmias, stroke, pulmonary hypertension, systemic embolic events, infective endocarditis, and pregnancy-related issues. The diagnosis of critical valvular emergency, especially rheumatic mitral stenosis in acute cardiac care, is often overlooked due to the clinical history potentially resembling other disease entities. This review highlights clinical emergency manifestation and comprehensive management of rheumatic mitral stenosis in acute cardiac care.

1. Introduction

Rheumatic heart disease (RHD) results from delayed group A beta-haemolytic streptococci-induced acute rheumatic fever. At contact zones of the leaflet, acute rheumatic fever generates leaflet inflammation, oedema, and small fibrin-platelet thrombi. Scarring causes valve deformity by destroying leaflet architecture via fibrosis, neovascularization, and collagen and tissue cellularity. Extra calcification causes malfunction. Rheumatic heart disease causes chronic and gradual cardiac valve deterioration.[1]

RHD is a preventable cardiovascular disease, yet its complications contribute to challenges in middle- and low-income nations due to the high incidence and potential to occur in individuals during their productive years. Indonesia is a developing country and classified as an endemic country of RHD, with an estimated 1.18 million RHD cases per year in 2015 and a mortality rate around 4.8 per 100,000 individuals at risk (CI 95%, 4.4-5.1).[2] In 2016–2019, the National Cardiovascular Centre Harapan Kita Hospital in Jakarta, Indonesia, discovered that RHD caused most valvular heart disease (42.6%, n = 2333), and solitary mitral stenosis (MS) was the predominant valve lesion in patients with rheumatic heart disease (46.5%).[3] The larger RHD multicenter study in Indonesia (Ina-RHD) in 2020-2023 reported RHD patients predominantly female (64.4%), with an average age of 44 years, mostly isolated MS (39.6%), and 25% of RHD patients presented with NYHA functional class III-IV.[4]

Mitral stenosis in RHD arises from the thickness and deformity of the valvular apparatus (commissures, cusps, and chordae tendineae). MS can exist solely or combined with mitral regurgitation (MR), a condition known as mixed mitral valve disease.[5] The majority of RHD patients look for treatment after developing severe RHD and presenting with RHD complications, such as heart failure, arrhythmias, stroke, pulmonary hypertension, systemic embolic events, infective endocarditis, and pregnancy-related issues.[5-7] Acute mitral stenosis may produce sudden heart failure and cardiogenic shock, which may not respond to treatment and need surgical or percutaneous intervention even with adequate systolic function. Due to its clinical similarities to other diseases, urgent valvular emergency, particularly rheumatic mitral stenosis, is commonly ignored in acute cardiac treatment.

2. Discussion

Due to a structural defect of the mitral valve apparatus (commissures, cusps, and chordae), mitral stenosis (MS) blocks left ventricular inflow.[5] The causes of mitral stenosis are listed in Table 1. The valve is frequently stenosed due to infection by group A hemolytic streptococci, resulting in inflammatory damage and chronically caused rheumatic heart disease (RHD). The lack of primary prevention for group A streptococcal infections and the deficiency of screening programs to identify early RHD lead to delayed disease manifestation, with the majority of patients only pursuing medical attention due to symptoms associated with disease consequences. Decompensated mitral stenosis (MS) frequently results from the gradual progression of a chronic lesion or a sudden alteration in cardiovascular hemodynamics that the stenotic valve cannot adequately adjust.[8]

As a result, patients may present to the emergency room with several cardiovascular conditions, including acute heart failure, cardiogenic shock, arrhythmia, stroke, infective endocarditis, pulmonary hypertension, and emergency in cardio-obstetrics.[7] The problem becomes even worse by lacking clinical knowledge regarding the diagnosis and management of the condition and its associated complications. Delayed presentation precludes patients from accessing early intervention in disease management, including prompt medical treatment.

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Table 1. Prevalence of Several Mitral Stenosis Etiologies

Etiologies of Mitral Stenosis	Prevalence
Rheumatic heart disease[3]	46.5% (1085 of 2333 RHD patients)
Mitral annular calcification (MAC)[9]	14% (1881 of 13,483 patients)
Prosthetic valve thrombosis[10]	0.1-5.7% (mechanical prosthetic valve) 6% (biological prosthetic valve)
Congenital mitral stenosis[11]	1.2% (17 of 1413 autopsy patients with congenital heart disease)
Radiation-associated valve disease	N/A in MS. 2.8-6.1% (valvular heart disease, including moderate stenosis or regurgitation)
Systemic rheumatic diseases such as systemic lupus erythematosus (SLE)[12] and rheumatoid arthritis (RA)	0.74% (37 of 5018 SLE patients) 0.05% (4 of 6673 RA patients)
Other diseases include Fabry, Whipple, mucopolysaccharidosis, carcinoid valve, and endomyocardial fibrosis	N/A

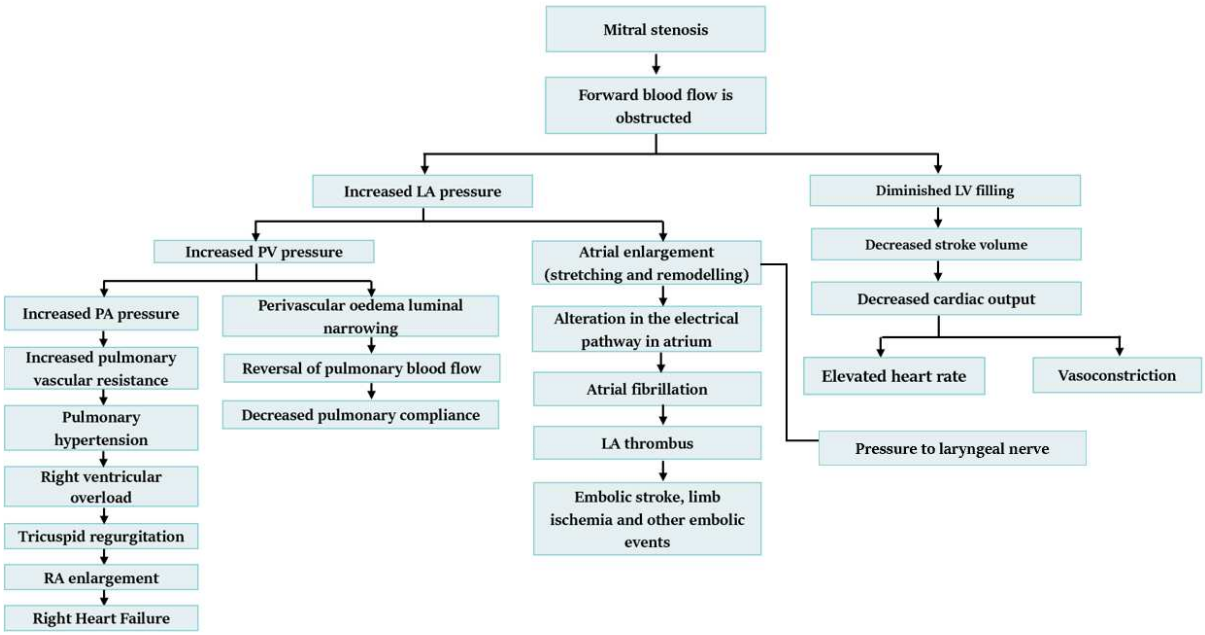

Figure 1. Pathophysiology of rheumatic mitral stenosis

Pathophysiology of Rheumatic Mitral Stenosis

Poststreptococcal rheumatic heart disease and mitral stenosis have complex mechanisms. Potential causes include genetics, antibody and T-cell-mediated molecular mimicry, and cytokine proinflammatory responses. Many interconnected systems activate innate and adaptive immune responses during infection. Inflammation produces several acute phase reactants needed for RHD development (Figure 1). RHD illness uses molecular mimicry, the sharing of epitopes between foreign and self-peptides, to cross-activate the host's immune system. Anticardiac antibodies imitate analogous amino acid sequences, homologous but nonidentical amino acid sequences, and sugar, ganglioside, and DNA epitopes to increase cross-reactivity. These approaches reveal that antistreptococcal monoclonal antibodies (mAbs) may detect and cross-react with streptococcal M protein and cardiac myosin, a key contractile protein.[13]

The average mitral valve orifice is 4-6 cm^2. Normal mitral valve opening occurs during the diastolic phase with equal left atrium and left ventricle pressure. Early diastole blood flows to the left ventricle. The "atrial kick" during late ventricular diastole pumps some blood from the left atrium into the left ventricle. Left atrium blood flow was decreased by a mitral valve area under 2 cm^2. For optimal LV filling, atrial kick is needed because of the mitral valve pressure gradient. Mitral stenosis is indicated by increased left atrial pressure when the mitral valve area is <1.5 cm^2. Normal LV diastolic pressure is 5 mmHg. At normal heart rate, severe mitral stenosis creates a 5-10 mmHg mitral valve pressure gradient. Heart rate, diastolic filling duration, and transvalvular flow rate impact mean pressure gradient. Left ventricular and left atrial compliance and stenosis severity affect mitral pressure half-time.[14,15]

Pulmonary hypertension results from left atrial pressure returning to pulmonary capillaries. Left atrium enlargement along with elevated LA pressure. During atrial fibrillation the atrial kick is diminished. During severe mitral stenosis, the atrial kick regulates left ventricular filling. The absence of atrial kick causes congestive heart failure by lowering cardiac output. Early mitral stenosis symptoms progress slowly to NYHA functional class II, atrial fibrillation, and class III or IV symptoms.[16]

How to Diagnose Rheumatic Mitral Stenosis in the Emergency Room?

Rheumatic mitral stenosis appears in the 2nd to 4th decade after rheumatic fever. Orthopnea and PND are the main symptoms. Palpitations, chest discomfort, hemoptysis, thrombosis, and congestion may arise from elevated left atrial volume. Physical examination in rheumatic mitral stenosis includes forceful S1 (due to higher mitral valve closure force), loud P2 (in severe pulmonary hypertension instances), and opening snap (additional sound after A2), reflecting a forceful mitral valve opening when LA pressure exceeds LV pressure, and mid-diastolic rumbling murmur with presystolic accentuation (low-frequency murmur, louder when in the left lateral decubitus position and with isometric exercise). Left atrial enlargement (double shadow cardiac silhouette) or left cardiac border straightening (owing to massive left atrial appendage), upward bronchi, conspicuous pulmonary arteries, and interstitial lung oedema

Table 2. Stages of MS[14]

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of MS	Mild valve doming during diastole	Normal transmitral flow velocity	None	None
B	Progressive MS	Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets Planimetered mitral valve area >1.5 cm2	Increased transmitral flow velocities Mitral valve area >1.5 cm2 Diastolic pressure half-time <150 ms	Mild to moderate LA enlargement Normal pulmonary pressure at rest	None
C	Asymptomatic severe MS	Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets Planimetered mitral valve area ≤1.5 cm2	Mitral valve area ≤1.5 cm2 Diastolic pressure half-time ≥150 ms	Severe LA enlargement Elevated PASP >50 mm Hg	None
D	Symptomatic severe MS	Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets Planimetered mitral valve area ≤1.5 cm2	Mitral valve area ≤1.5 cm2 Diastolic pressure half-time ≥150 ms	Severe LA enlargement Elevated PASP >50 mm Hg	Decreased exercise tolerance Exertional dyspnea

Table 3. Wilkin score: grading of mitral valve characteristics from echocardiography[22]

Grade	Mobility	Subvalvar thickening	Thickening	Calcification
1	Highly mobile vale with only leaflet tips restricted	Minimal thickening just below the mitral leaflets	Leaflets near normal in thickness (4-5 mm)	A single area of increased echo brightness
2	Leaflet mid and base portions have normal mobility	Thickening of chordal structures extending up to one third of the chordal length	Mid-leaflets normal, considerable thickening of margins (5-8 mm)	Scattered areas of brightness confined to leaflet margins
3	Valve continues to move forward in diastole, mainly from the base	Thickening extending to the distal third of the chords	Thickening extending through the entire leaflet (5-8 mm)	Brightness extending into the mid-portion of the leaflets
4	No or minimal forward movement of the leaflets in diastole	Extensive thickening and shortening of all chordal structures extending down to the papillary muscles	Considerable thickening of all leaflet tissue (>8-10 mm)	Extensive brightness throughout much of the leaflet tissue

may be seen on chest X-ray. Electrocardiography can detect left atrial enlargement (P wave duration > 120 ms, P wave axis +45^0 to -30^0, P terminal forced negative V1 1 mm wide and 1 mm deep), atrial fibrillation, and right ventricular hypertrophy (R/S ratio > 1, axis frontal > 80^0 in pulmonary hypertension due to MS).[14,16-18]

Auscultation of rheumatic mitral stenosis patients in an acute cardiac care setting might be challenging due to the requirement of some maneuvers or positioning. Echocardiography is a noninvasive imaging modality that is essential for screening and evaluating patients that are suspected of having rheumatic mitral stenosis. Bedside transthoracic echocardiography (TTE) helps clinicians in the emergency room to assess anatomy and hemodynamic consequences, the degree of severity of MS, identify related complications, and determine the suitable approach to treatment for the patient.[19] Echocardiography characteristics for rheumatic mitral stenosis include leaflet morphology (mobility, thickness, calcification, subvalvular fusion, and commissural fusion), mitral valve area, mitral pressure gradient, and left atrial size. Several grading methods have been used to evaluate mitral valve (MV) disease severity, appropriateness for balloon mitral valvuloplasty (BMV), and surgery success and consequences. Wilkins score (or "splitability score") is often used because of its simplicity and maximum value of 16. Wilkin score <8 indicates mitral valve responds effectively to balloon dilation.[20,21] Table 2 shows four MS phases and hemodynamic effects.

Patients with mitral stenosis may present to the emergency room with left-sided (pulmonary) and right-sided (systemic) heart failure symptoms. Pulmonary congestion typically presents as exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and nocturnal cough. Concomitantly, right-sided failure may produce systemic venous hypertension, resulting in peripheral edema, ascites, and congestive hepatomegaly. On physical examination, "mitral facies"—characterized by malar flushing and cyanosis—serves as a pathognomonic sign of diminished cardiac output and advanced pulmonary hypertension.[23]

The British Heart Valve Society published clinical indications and echocardiography triage for patients with heart valve disease. Red flags for mitral stenosis patients that need urgent cardiologist review include right heart assessment (SPAP >50 mmHg or RV impairment/dilatation), heart rhythm (new onset atrial fibrillation), stroke risk (confirmed TIA or stroke, LA spontaneous echo contrast (LA SEC).[24]

Acute Heart Failure in Rheumatic Mitral Stenosis

There is no medication that will alleviate the mechanical restriction caused by mitral stenosis (MS) or the pulmonary vascular congestion and pulmonary hypertension that arise in severe MS. Whereas pharmacological agents cannot alleviate valve blockage, prolonging diastole by heart rate reduction can improve hemodynamic irregularities and symptoms. This can be accomplished with β-blockers or, with nondihydropyridine calcium channel blockers, but not with digoxin.[25] A randomized crossover study reported metoprolol had more benefits in symptomatic mitral stenosis with sinus rhythm compared to verapamil and digoxin. Verapamil demonstrated superiority as the best choice for symptomatic mitral stenosis with atrial fibrillation.[26] In chronic mitral stenosis, an accelerated heart rate reduces diastolic filling time and worsens hemodynamic instability. Atrial fibrillation, a common complication, reduces atrial contribution to ventricular filling. Even without atrial fibrillation, vigorous rate control to maintain a resting heart rate of 60 bpm may enhance diastolic filling time using beta-blockers and diltiazem on stable blood pressure and digoxin or intravenous amiodarone on low blood pressure.

Intravenous diuretics reduce pulmonary congestion and symptoms in mitral stenosis. Patients with acute cardiac failure who are never given a diuretic should receive an intravenous dose of furosemide ranging from 20 to 40 mg equivalent. Patients with pre-existing kidney disease should be administered a higher dose, as it correlates with a rightward shift in the dose–response curve. Patients on an ambulatory diuretic regimen should be delivered at least the previously established oral dose by intravenous route. A loading dose should precede continuous infusion to ensure the rapid attainment of a steady-state plasma concentration of loop diuretics.[27] Despite preserved left ventricular function, intravenous vasodilators and inotropes do not alleviate congestion or increase cardiac output in mitral stenosis with limited left ventricular filling. In hypotensive patients, carefully titrated intravenous norepinephrine or vasopressors devoid of tachycardic effects, such as vasopressin, enhance forward flow.[28]

Mitral stenosis patient with pulmonary insufficiency (a sign of respiratory distress or oxygen saturation <92%) can be started with oxygen therapy. In case after monitoring there is persistent respiratory distress (respiratory rate >20-25 x/minute or increased work of breathing), consider non-invasive ventilation (NIV) or immediate intubation if there is severe respiratory distress or contraindication to NIV.[29,30] Positive-pressure breathing may worsen mitral stenosis

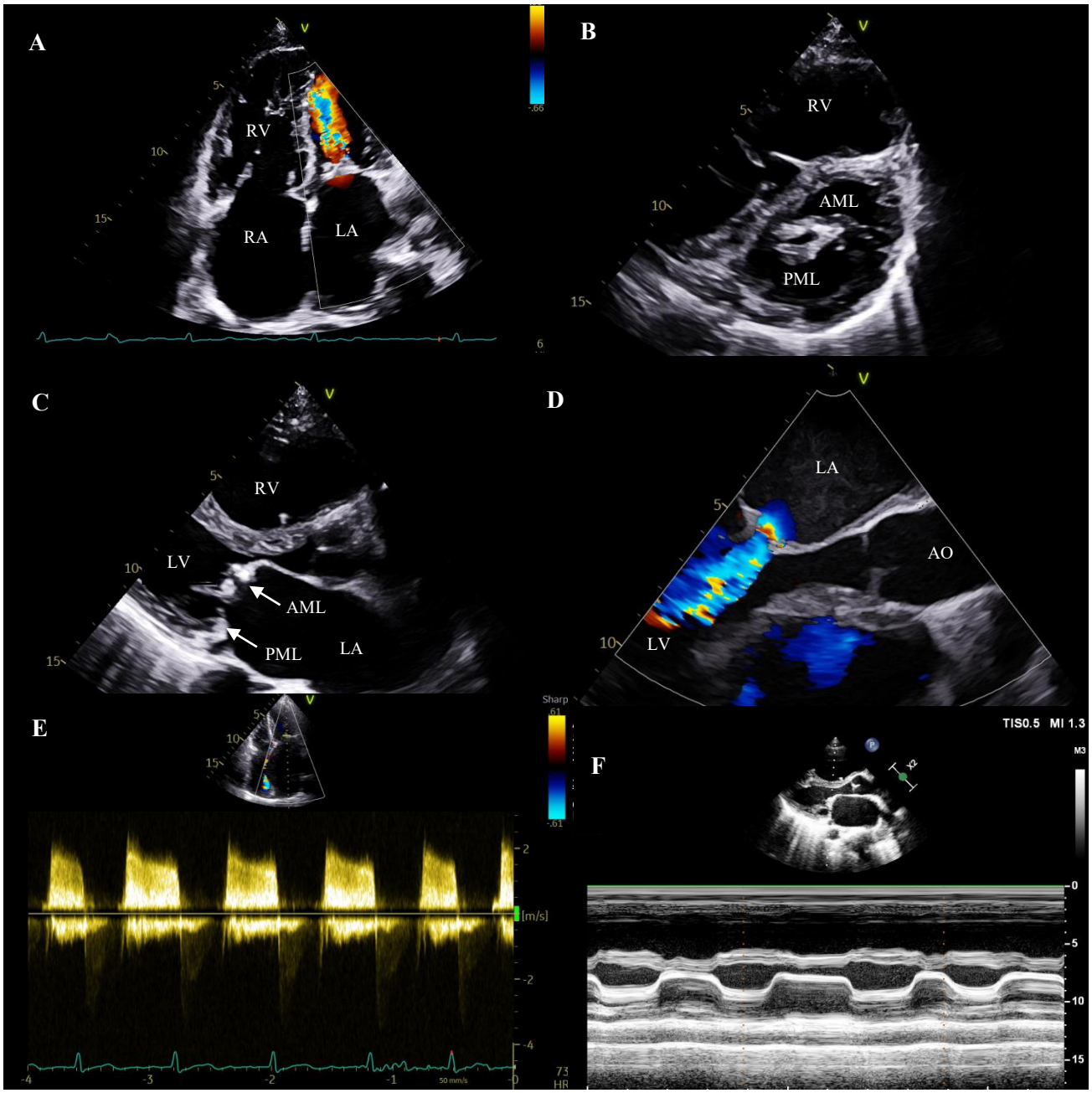

Figure 1. (A) TTE Apical-four-chamber view showing restricted leaflet motion during diastolic phase, (B) TTE Parasternal short-axis view showing commissural fusion with calcification, (C) TTE Parasternal long-axis view showing "hockey-stick or doming" appearance during diastolic phase and also thickening and shortening of chordae tendinae, (D) TEE parasternal long-axis view showing mitral stenosis, LA dilation, and spontaneous echo contrast (SEC) at the left atrial. (E) Continuous wave doppler in the severe MS, (F) M-mode showed thickened and calcified leaflet with decreased E-F slope. TTE = Transthoracic Echocardiography; TEE = Transesophageal Echocardiography; LA = Left Atrium; RA = Right Atrium; RV = Right Ventricle; AML = Anterior Mitral Leaflet; PML = Posterior Mitral Leaflet.

hypotension by lowering left ventricular preload, which prevents the left ventricle from increasing forward flow.[31-34]

Cardiogenic Shock in Rheumatic Mitral Stenosis

Cardiogenic shock, a serious condition in which the heart cannot pump enough blood to fulfill the body's needs, requires immediate diagnosis, etiological assessment, and treatment.[35] Diagnosis of cardiogenic shock based on SHOCK Trial (1999)[36] SBP < 90 mmHg for 30 minutes, and/or need pharmacological or mechanical care to maintain SBP at least 90 mmHg followed by end-organ damage (urine output < 30 ml/hour or chilly extremities, heart rate > 60 beats/minute), and hemodynamic criteria (CI ≤ 2.2 L/min per m2 and PCWP ≥ 15 mmHg). SCAI divided cardiogenic shock into 5 stages (table 4).[37] Nair et al. reported the prevalence of mitral stenosis as the cause of valvular cardiogenic shock was 4% (18 of 442 patients with valvular cardiogenic shock) with 30-day all-cause mortality rates of 50% and 1-year all-cause mortality of 56%.[38]

Mitral stenosis (MS) blocks blood flow from the left atrium to the left ventricle, reducing left ventricular filling and cardiac output and causing cardiogenic shock. Mitral stenosis typically does not affect cardiac contraction, but reflex vasoconstriction, elevated afterload, reduced left ventricular filling, and left atrial dilation can cause pulmonary congestion and hypoxia, which are the main causes of congestive symptoms.[39] Despite mitral valve stenosis may not developing rapidly, concurrent conditions such as arrhythmias, ischemia, and sepsis can negatively affect the hemodynamics of individuals with mitral stenosis, resulting in a decrease in cardiac output and circulatory stability. Consequently, medical management becomes complicated, necessitating the immediate use of circulatory and respiratory support, treatment of the underlying etiology, and control of the precipitating factors.[39]

Due to its distinct pathophysiology, managing cardiogenic shock complicated by rheumatic mitral stenosis might be challenging. Tachycardia reduces ventricular filling time and may impair

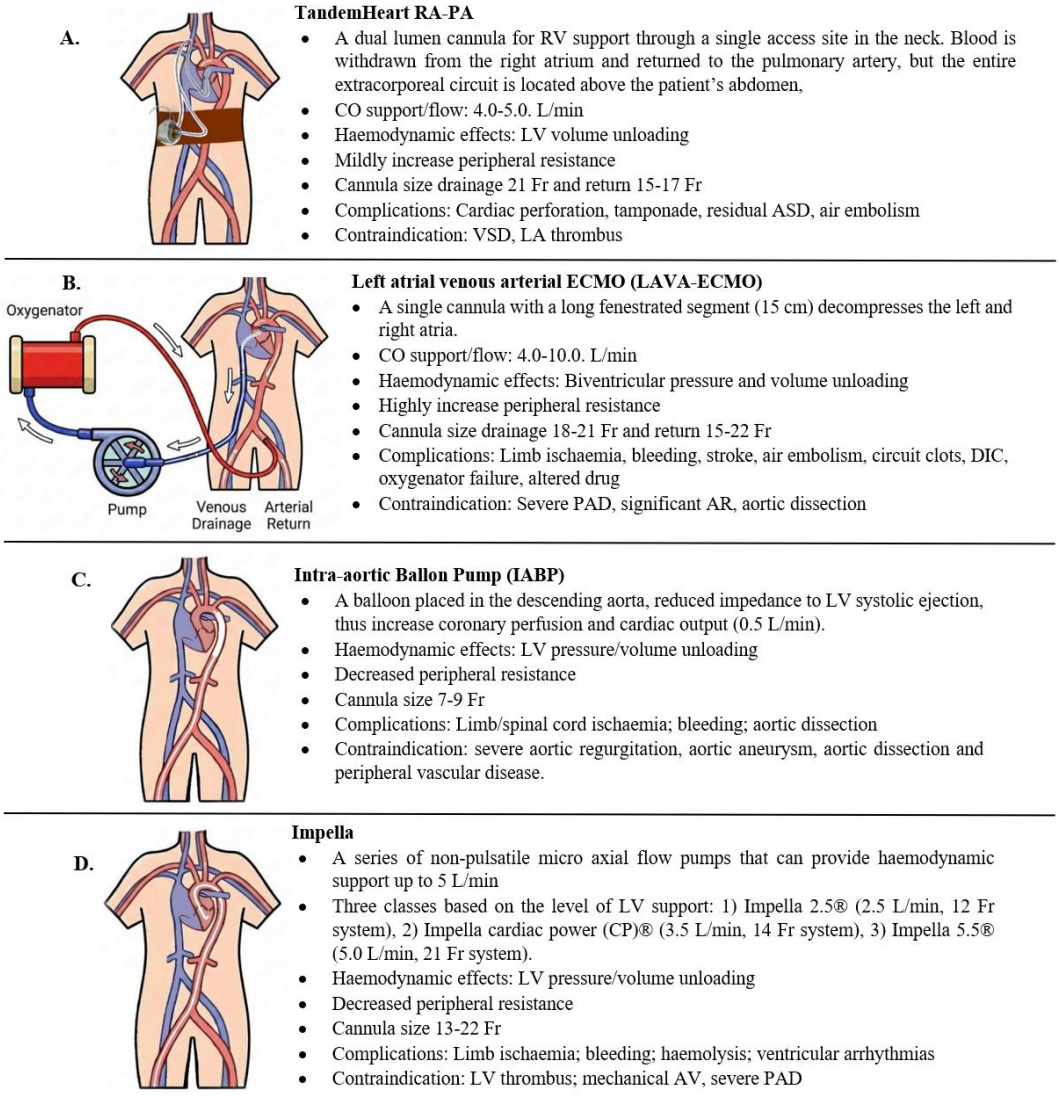

Figure 2. Mechanical circulatory support: (A) TandemHeart RA-PA, (B) Left Atrial Venoarterial Extracorporeal Membrane Oxygenation (LAVA-ECMO), (C) IABP, (D) Impella.

hemodynamics; therefore vasopressor selection should account for heart rate. Norepinephrine is typically administered as a first-line treatment for cardiogenic shock, although its chronotropic effects may impair hemodynamic in mitral stenosis patients, requiring cautious heart rate monitoring. For severe mitral stenosis, phenylephrine with or without vasopressin is a good alternative. Dopamine and inotropes should not be used initially unless necessary. Whereas, decompensated rheumatic mitral stenosis patients may also have secondary pulmonary hypertension and/or right ventricular failure, necessitating inotropic support. In these circumstances, meticulous titration of inotropic support may be required, preferably under the guidance of invasive hemodynamic measures.[8,40]

Selection of mechanical circulatory support in rheumatic mitral stenosis can be tricky. The increased left atrial/left ventricular gradient, which impacts the pulmonary circulation, is the primary hemodynamic characteristic of mitral stenosis. TandemHeart RA-PA or LAVA-ECMO would therefore be the best MCS devices because they immediately empty the left atrium into the systemic arterial circulation, making up for the inadequate flow and output caused by MS.[41,42] TandemHeart RA-PA device is a centrifugal pump with an inflow cannula placed in the RA and an outflow cannula in the PA, directly bypassing the RV as the RV unloads with cardiac output/flow 4.0 L/min. Because left atrial emptying into the left ventricle is frequently insufficient in MS patients, IABP and Impella are less likely to be beneficial due to the mechanism of cardiogenic shock in MS are pre ventricular and low LV end-diastolic pressure.[43,44] A bridge to definitive valve repair, such as emergency catheter-based or surgical intervention, should be provided by both mechanical and pharmaceutical circulatory support.[45,46]

Arrhythmia in Rheumatic Mitral Stenosis

The most common arrhythmia complication of MS is atrial fibrillation (AF). Atrial fibrillation in rheumatic valvular disease (RVD) and nonrheumatic valvular disease (NRVD) differ in a number of ways, including younger age, lesser comorbidities, larger LA volume, and not applying the $CHAD_2DS_2$-VA Score.[47] Ina-RHD multicenter study reported 62-72% of patients with rheumatic mitral stenosis having atrial fibrillation rhythm.[3,4,48] Rheumatic carditis causes mitral stenosis, atrial inflammation, left atrial dilatation, atrial wall fibrosis, and atrial muscle bundle instability. These changes cause different conduction velocities and refractory periods. A premature atrial activation from an automatic focus or re-entry may stimulate the left atrium during the susceptible period, causing atrial fibrillation. Mitral stenosis severity, left atrial dilatation, and left atrial pressure independently predict this arrhythmia. Maintaining sinus rhythm in MS patients reduces the risk of cerebral embolism, preserves cardiac output and exercise capacity, and relieves symptoms. AF in MS patients is treated similarly to AF in other aetiologies. The three main goals of managing AF in RHD are to prevent tachycardiomyopathy and control symptoms through rate or rhythm control, prevent stroke and peripheral embolism through therapeutic anticoagulation, and increase survival through timely valve interventions.[47] Restoring and maintaining sinus rhythm is often more challenging due to left atrial pressure overload combined with the impact of the rheumatic process on atrial tissue and the conduction system.[1,49]

Heparin anticoagulation and heart rate control should treat rapid atrial fibrillation in the ER. To modify ventricular response, intravenous digoxin, nondihydropyridine calcium channel antagonists,

Table 5. Medication commonly used to control atrial fibrillation rapid ventricular response[50]

Drug	Intravenous		Per oral		Side effects
	Dose	Onset of action	Dose	Onset of action	
Diltiazem	Loading 0.25 mg/kg over 2 min, maintenance 5-15 mg/h	2-7 min	120-360 mg/day	2-4 h	Hypotension, heart failure
Verapamil	Loading 0.075-0.15mg/kg over 2 min	3-5 min	120-360 mg/day	1-2 h	Hypotension, heart failure, interaction with digoxin
Esmolol	Loading 0.5 mg/kg over 1 min, maintenance 0.05-0.2 mg/kg/min	5 min	-	-	Hypotension, dizziness, infusion site reaction
Metoprolol	Loading 2.5-5 mg/kg over 2 min Up to a max. of 3 doses	5 min	25-100 mg/12 h	4-6 h	Hypotension, dizziness, blurred vision
Propranolol	Loading 0.15 mg/kg	5 min	80-240 mg/day	60-90 min	Hypotension, Dizziness, fatigue, nausea, cold extremities
Amiodarone	Loading 5-7 mg/kg over 30 min followed by 1200 mg/day (continuous infusion)	2 min	Loading 400 mg/8 h, maintenance 200 mg/day	2 days-3 weeks	Hypo/hyperthyroidism, pulmonary toxicity, liver toxicity, photosensitivity, corneal deposits, skin discoloration, polyneuropathy, optical neuropathy, interaction with acenocoumarol
Digoxin	Loading 0.25 mg /2 h to a max. of 15 mg, maintenance 0.125-0.25 mg/day	2 h	0.125-0.325 mg/day	2 h	Digitalic intoxication (digestive, ocular, neurological, pro-arrhythmic)

or beta blockers limit atrioventricular node conduction. When beta blockers or nondihydropyridine calcium channel antagonists are contraindicated, intravenous or oral amiodarone may be utilized. In hemodynamic instability, immediate electrical cardioversion with intravenous heparin before, during, and after is advised. Atrial fibrillation without anticoagulation for more than 24–48 hours increases the risk of embolic events post-cardioversion, however, embolization may begin before 24 hours.[1,15]

Based on nonrheumatic atrial fibrillation data, one of two elective cardioversion procedures is recommended for individuals with established atrial fibrillation lasting more than 24 to 48 hours and no long-term anticoagulation. Elective cardioversion is recommended after three weeks of warfarin. If a transoesophageal echocardiography indicates no atrial thrombus, urgent cardioversion may be performed if the patient is anticoagulated with intravenous heparin before and during the procedure and warfarin for one month thereafter. Multiple conversions and paroxysmal atrial fibrillation increase embolization risk. Individuals lacking sinus rhythm should take digitalis to lower resting ventricular rate to 60 beats per minute. If necessary, low-dose beta-blockers such as atenolol (25 mg daily) or metoprolol (50–100 mg daily) may be given. Beta blockers effectively slow exercise-induced cardiac responses. After receiving adequate antiarrhythmics, further cardioversions are unnecessary if the patient cannot maintain sinus rhythm.[1,50]

Rheumatic Mitral Stenosis in Pregnancy

One of the leading causes of pregnancy deaths is cardiac disease. ROPAC identified 273 rheumatic MS patients among 390 pregnant women with cardiac illness, and 23.1% were hospitalized for heart failure[51] Cardiovascular circulation alterations during pregnancy make rheumatic heart diseases poorly tolerated and worsen throughout delivery and postpartum. Heart decompensation and pulmonary hypertension result from mitral stenosis expanding blood volume and pregnancy-induced tachycardia decreasing ventricular filling time. Other causes include discomfort, infection, hypertension, and anemia. Stenosed mitral valves worsen pregnancy-related cardiac symptoms in women. The mother's and baby's metabolic demands drive heart and blood changes throughout pregnancy. Plasma volume peaks before delivery from fertilization. Left ventricular diastolic filling is hindered by the stenotic mitral valve and pregnancy-induced tachycardia. As the heart tries to handle circumstances that demand higher metabolic activity or blood volume, trans-mitral gradient and left atrial filling pressure rise, increasing the risk of pulmonary congestion and oedema. Pain and catecholamine release increase heart rate during childbirth. Right ventricular dysfunction may aggravate secondary pulmonary arterial hypertension (SPAH). The additional volume burden and tachyarrhythmia may worsen the patient's cardiac condition, causing a progression through NYHA classes or, in severe cases, a rapid decline from stable to congestive heart failure and cardiogenic shock.[52]

If symptoms of pulmonary hypertension occur, B1-selective blockers should enhance left ventricular filling time to control heart rate. Propranolol lowers pulmonary edema risk without harming the fetus. Diuretics are recommended if symptoms continue. Percutaneous mitral commissurotomy works best at 20 weeks. Even with pharmacological treatment, NYHA II/IV patients or those with systolic pulmonary artery pressure > 50 mmHg may consider it. Once all other treatments fail, women with life-threatening diseases have open-heart surgery.[52]

Women with mitral stenosis should have surgery or percutaneous correction before pregnancy. Since pregnancy-related physiological vasodilation and tachycardia are less tolerated, left-sided stenotic valves are more likely to harm the mother. NICE advises against antibiotic prophylaxis for infective endocarditis during pregnancy or delivery. A multidisciplinary team determines delivery based on heart failure or PAH, functional capacity, exercise tolerance, therapeutic anticoagulation, and obstetric history. Tools should shorten labor's second stage. Mild and moderate MS without pulmonary hypertension allow vaginal delivery. Elective cesarean section is rarely recommended, however, it may be necessary to prevent decompensation during vaginal birth or provide a multidisciplinary team for high-risk women. In moderate to severe MS with NYHA class III/IV or increasing pulmonary artery pressure on medicinal therapy and PMBC failure, Caesarean section is recommended.[53,54]

Emergency Percutaneous Mitral Balloon Commissurotomy

Percutaneous mitral balloon commissurotomy (PMBC) effectively improves severe MS symptoms (MVA < 1.5 cm^2), particularly when subvalvular and valvular anatomies are desired for calcifications and increasing the mitral valve area.[14] Lokhandwala et al., reported 40 patients underwent emergent PMBC in a tertiary centre Mumbai, India, 65% (26 of 40) were in cardiogenic shock condition, 27.5% of patients had refractory pulmonary oedema, and 7.5% were cardiac arrest survivors. There were 26 patients who survived and improved functional capacity after a mean follow-up of 8 months (15 patients NYHA I, 4 patients NYHA II, and 1 patient NYHA III). Non-survivor included 7 patients related to the PMBC (grade 3 MR or failed PMBC), and 7 patients were not related to PMBC procedure (septic shock or multiple organ failure).[55]

Another 12-year cohort study was conducted by Pillai et al., including 96 pregnant women with severe MS and in a condition of severe heart failure who underwent PBMC. The gestational ages were 16-37 weeks (23.4 ± 10.9), 17% were in cardiogenic shock, and 33.3% were on mechanical ventilation. The outcome was 92% successful PBMC, 5.2% complicated with acute severe MR, 19 patients failed PBMC, and mortality in 11 patients. A five-year follow-up showed 7 patients with previously successful PBMC had mitral valve restenosis requiring re-intervention.[56]

3. Conclusion

Rheumatic mitral stenosis is a preventable cardiovascular disease that causes potentially serious complications in the productive-age population, with endemic countries like Indonesia reporting isolated MS as the predominant valve lesion. The acute exacerbation of rheumatic MS can precipitate cardiac emergencies such as acute heart failure and cardiogenic shock, which are often challenging to diagnose in the emergency setting. The pathophysiology of MS involves *molecular*

mimicry leading to progressive valve damage and flow obstruction, resulting in increased left atrial pressure, pulmonary hypertension, and reduced cardiac output. Management in acute cardiac care focuses on rate control (to prolong diastolic filling time), diuretics for congestion, and vasopressors without significant chronotropic effects for shock. Crucially, for cardiogenic shock caused by MS, mechanical circulatory support (MCS) devices like TandemHeart RA-PA or LAVA-ECMO are considered superior as they directly unload the left atrium, addressing the pre-ventricular shock mechanism, whereas IABP and Impella are less likely to be beneficial. Therefore, prompt diagnosis via echocardiography and early definitive interventions, such as percutaneous mitral balloon commissurotomy (PMBC), are vital for improving outcomes, even in critical scenarios like cardiogenic shock and high-risk pregnancies.

4 . Declaration

4.1 Ethics Approval and Consent to participate
Not applicable.

4.2. Consent for publication
Not applicable.

4.3 Availibility of data and materials
Data used in our study were presented in the main text.

4.4 Competing interests
Not applicable.

4.5 Funding Source
Not applicable.

4.6 Authors contributions
Idea/concept: NAN. Design: NAN. Control/supervision: IP. Data collection/processing: NAN, IP. Analysis/interpretation: NAN, IP. Literature review: NAN, IP. Writing the article: NAN. Critical review: NAN, IP.

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