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Heart Failure Due to Mitral Stenosis Complicated Atrial Fibrillation in Pregnancy

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ABSTRACT

Background. This case report aims to discuss the management of heart failure due to mitral stenosis with complications of atrial fibrillation in pregnancy. **Case presentations.** A 35-year-old pregnant woman came to the ER M. Djamil Hospital with complaints of shortness of breath 3 days before being admitted to the hospital. Complaints of shortness of breath accompanied by palpitations felt fast and irregular, appeared suddenly when the patient was resting, and lasted continuously. Physical examination showed a pulse rate of 120-130x/minute, increased jugular venous pressure, and palpable cardiac ictus 1 finger lateral to the left midclavicular at the V intercostal space. ECG examination revealed atrial fibrillation- rapid ventricular response (AF RVR). The patient was diagnosed with atrial fibrillation- rapid ventricular response et causa suspect mitral stenosis NYHA Fc III, et causa rheumatic heart disease. **Conclusion:** The management of atrial fibrillation rapid ventricular response in this patient was done by giving digoxin 0.25 mg orally. Patients are given anticoagulants to prevent thromboembolic complications. This patient was advised to undergo valve replacement surgery considering the degree of stenosis, complications, and a thorough assessment.

1. Introduction

In developing countries, the prevalence of valvular heart disease is approximately 2.5%. Because it is dominated by degenerative causes, the prevalence is high in people over 65 years of age. Rheumatic heart disease still represents 22% of valvular heart disease in Europe. In developing countries, rheumatic heart disease remains the leading cause of valvular heart disease with a high prevalence, between 20-30 cases per 1000 people.[1] Today, 0.2-4% of all pregnancies in western industrialized countries are complicated by cardiovascular disease. Valve rheumatic disease predominates in non-western countries, accounting for 56-89% of heart disease in pregnancy. Rheumatic heart disease is the most common cause of mitral stenosis. Approximately 50% of patients with symptomatic mitral stenosis have a history of acute rheumatic fever, a median of 20 years before the onset of symptoms.[2]

The most common complication of MS is atrial fibrillation (AF). The prevalence of AF in patients with mitral stenosis is related to the severity of the obstruction and the patient's age. Atrial fibrillation can worsen symptoms because of the loss of atrial contribution to ventricular filling and the short period of diastolic filling when the ventricular rate is not controlled. In addition, AF predisposes to left atrial thrombus formation and systemic thromboembolism. The incidence of AF makes the overall prognosis of MS patients worse. In MS patients with AF, the 5-year survival rate is only 64% compared with 85% in AF patients without MS.[3] This case report aims to discuss

the management of heart failure due to mitral stenosis with complications of atrial fibrillation in pregnancy.

2. Case Presentation

A 35-year-old woman came to the emergency department of M. Djamil General Hospital with complaints of shortness of breath for 3 days before being admitted to the hospital. Shortness of breath without wheezing and unrelated to weather and food. There was a history of shortness of breath before, especially when he was doing the light-moderate activity and if he was lying down for a long time, but the patient never woke up at night because of shortness of breath. Edema in both legs since 3 months ago, getting heavier this 1 month. Complaints of shortness of breath accompanied by palpitations felt fast and irregular, appeared suddenly when the patient was resting, and lasted continuously. The previous history of palpitations like this has often been felt for the past 2 weeks, and sometimes it goes away on its own.

The patient never complained of chest pain, cold sweat, nausea, or vomiting. There was no history of hypertension and diabetes. The patient had neither asthma nor thyroid. The patient admitted that he had a history of fever with intermittent joint pain and a history of recurrent cough with a fever about 15 years ago and was taken to the hospital for treatment and given antipyretics, cough, and cold medicines. Referred patients from Payakumbuh Hospital with a diagnosis of severe mitral stenosis NYHA Fc III-IV + G3P2A0H2 gravid 20-21 weeks and received therapy with furosemide 1 x 40 mg and spironolactone 1 x 25 mg.

From the physical examination, the general condition was moderately ill with co-operative awareness of compos mentis, blood pressure 120/70 mmHg, pulse rate 120-130 x/minute, irregular, respiratory rate 26 x/minute, body temperature 37^{o} C. Jugular venous pressure was obtained. 5+3 cmH_2O. The conjunctiva was not anemic, and the sclera was not icteric. On cardiac examination, cardiac ictus was palpated 1 finger lateral to the left midclavicular at intercostal V. On auscultation, irregular S1 and S2 were found, and murmurs and gallops were difficult to assess. A lung examination revealed vesicular breath sounds and fine wet crackles in both lung fields. On abdominal examination, the liver and spleen were not palpable. The extremities are warm, and there is minimal leg edema.

Electrocardiography (ECG) has been performed twice. The first ECG examination at Payakumbuh Hospital showed an atrial fibrillation rhythm QRS rate of 100-110 x/minute (figure 1). A repeat ECG examination at the emergency unit of M. Djamil General Hospital still found atrial fibrillation-rapid ventricular response (AF RVR) (Figure 2).

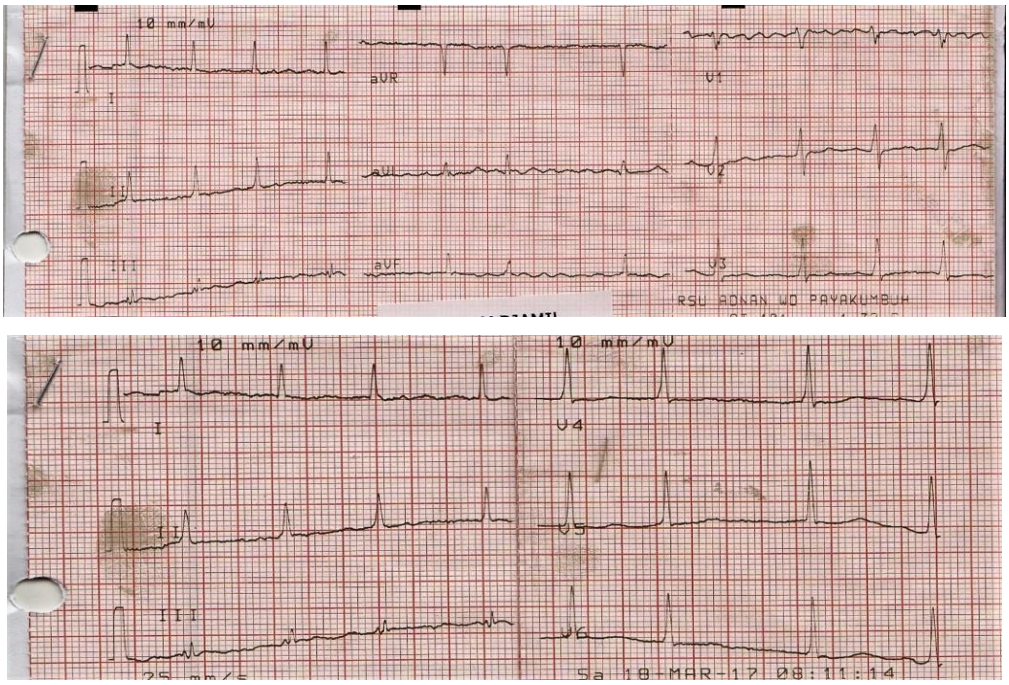


Figure 1. ECG in Payakumbuh Hospital, AF RVR, QRS rate 100-110x/minute

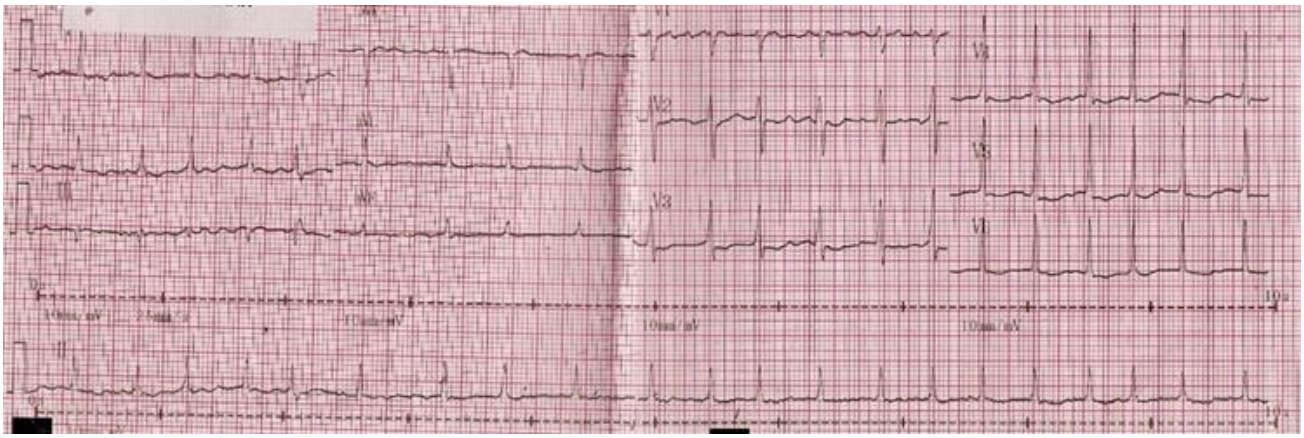


Figure 2. ECG in ER M. Djamil General Hospital shows AF RVR.

In this patient, no chest x-ray imaging was performed because she was pregnant. On laboratory examination found hemoglobin levels of 14.5 g/dl, leukocytes 13830/mm^3, hematocrit 41%, platelets 315,000/mm^3, urea 15 mg/dl, creatinine 0.6 mg/dl with creatinine clearance test (CCT) 79 ,25. Electrolyte sodium 136 meq/dl, potassium 3.1 meq/dl, chloride 100 meq/dl, and calcium 8.6 meq/dl. Blood gas analysis showed pH: 7.46, pCO2: 33, pO2:136, HCO3: 23.5, BE -0.3, SaO2: 99% with the effect of respiratory alkalosis.

The patient was diagnosed with atrial fibrillation-rapid ventricular response et causa suspect mitral stenosis NYHA Fc III, et causa rheumatic heart disease. The patient was treated with nasal oxygen 4 liters/minute, IVFD RL 500 cc/24 hours, furosemide 1 x 40 mg, spironolactone 1 x 25 mg, digoxin 1 x 0.25 mg, simarc according to the nomogram, and transferred to a cardiac ward. The patient was planned for a complete clinical chemistry examination and echocardiography. In this patient, the target fluid requirement is 1500 ml, given the DJ III diet of 1500 kcal, and the target fluid balance is 0 to -200 cc.

On the second day of treatment, complaints of shortness of breath and palpitations decreased. Blood pressure 98/61 mmHg, pulse rate 90-100x/minute irregular. On physical examination, heart sounds were found to be a mid-diastolic murmur grade III/IV and an opening snap. At the apex. Fluid balance in 24 hours – 200 ccs with diuresis 1.44 cc/kg BW/hour.

On echocardiographic examination found moderate-severe grade mitral stenosis, moderate MR et causa rheumatic heart disease (WS 8), AR mild ec RHD, TR mild-moderate, high probability PH, good global left ventricular systolic function, EF 55%, global neurokinetic, good right ventricular contractility, sec, thrombus not seen on transthoracic echocardiography (TTE). Current therapy is furosemide 1 x 40 mg, spironolactone 1 x 25 mg, digoxin 1 x 0.25 mg, KSR 2 x 600 mg, discontinued simarc, given UFH 1150 U/hour, and corrected Mg 1 gr.

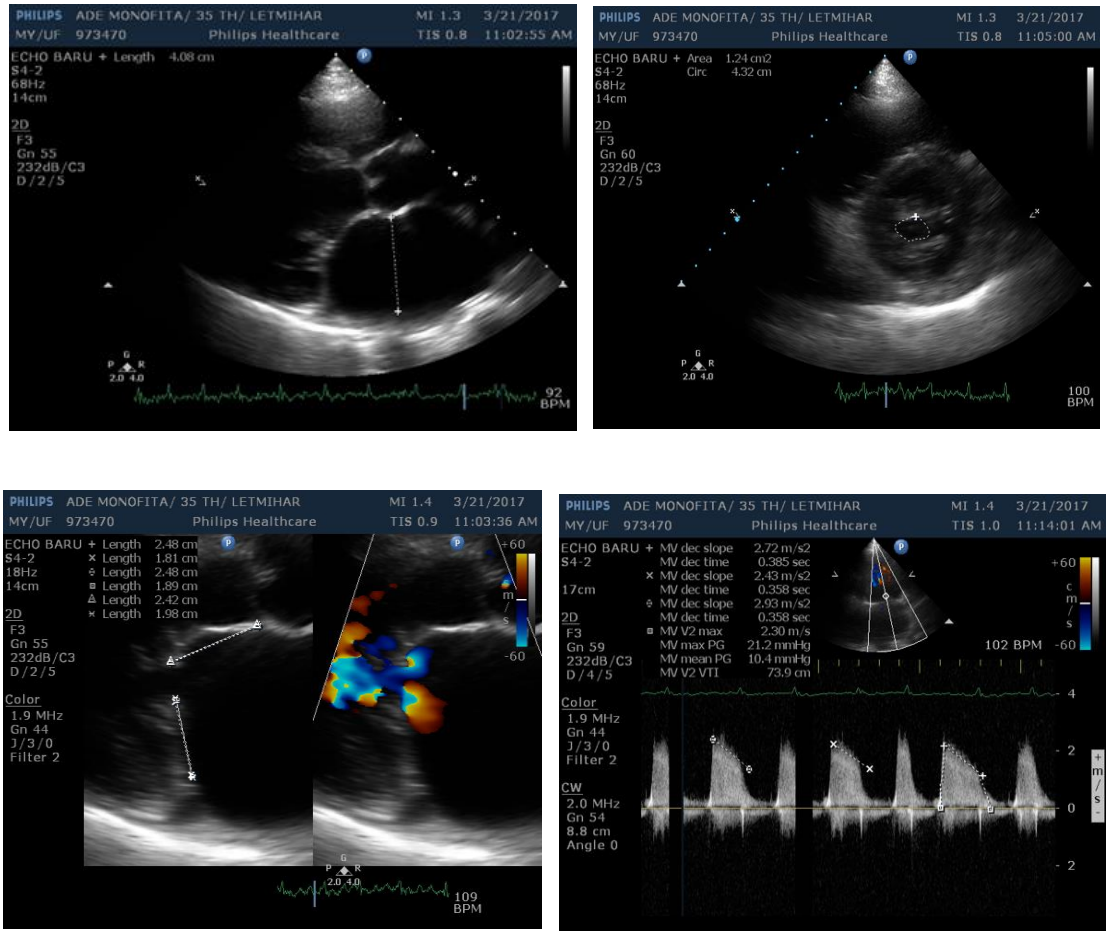


Figure 3. Echocardiography

Third-day treatment, clinical condition improvement. Blood pressure 116/70 mmHg with pulse still irregular 80-90x/minute. ECG impression of Atrial fibrillation with the normal ventricular response. Treatment with furosemide 1 x 40 mg, spironolactone 1 x 25 mg, digoxin 1 x 0.25, KSR 2 x 600 mg, and UFH 1150U/hour. The patient is planning to go home and is recommended for control to the cardiac polyclinic and Obgyn at Payakumbuh Hospital.

3. Discussion

The patient was diagnosed with mitral stenosis based on anamnesis, physical examination, and investigations. From the anamnesis, it was found that the complaint of shortness of breath was accompanied by irregular palpitations 3 days before admission to the hospital when the patient was resting and lasted continuously. A history of palpitations like this had previously been felt 2 weeks ago. Complaints are accompanied by shortness of breath that increases when the patient has light-moderate activity. In the literature, it is said that the most common complaints of mitral stenosis patients are shortness of breath and reduced exercise capacity. In mild mitral stenosis, shortness of breath usually disappears at rest and occurs with an activity where activity increases blood flow through the heart and increases heart rate while reducing diastolic filling time. Other conditions and activities that can precipitate and exacerbate symptoms of mitral stenosis include fever, anemia, hyperthyroidism, pregnancy, rapid arrhythmias such as atrial fibrillation, and emotional stress. As mitral stenosis increases, the symptoms also become more severe, in the form of shortness of breath at rest, fatigue, and symptoms of pulmonary congestion such as orthopnea and paroxysmal nocturnal dyspnea. If the progress of mitral stenosis continues, pulmonary hypertension will occur with symptoms of right heart

a failure such as jugular venous distention, hepatomegaly, and peripheral edema.[4,6]

The most common cause of mitral stenosis is rheumatic fever, especially in developing countries. This patient had a history of fever accompanied by intermittent joint pain and cough with a fever that recurred about 15 years ago, but the patient was only given medicine for fever, cough, and cold. However, it is said that more than 50% of patients with mitral stenosis do not remember an episode of rheumatic fever.[2,5,6]

On physical examination found a pulse rate of 120-130 x/minute, irregular, and noisy opening snaps. In mitral stenosis, there may be a murmur preceded by an Opening Snap, which is heard after S2. The opening snap is one of the key diagnostic features of the physical examination in mitral stenosis, which is thought to be due to a sudden increase in pressure in the chordate tendineae and a narrowing at the time of valve opening.[4] The opening snap is usually followed by a low-frequency crescendo murmur called a diastolic rumble. It is caused by turbulence in blood flow through the valve that narrows during diastole.[4]

On the ECG, rapid ventricular response to atrial fibrillation was found. This patient did not undergo a chest X-ray because the patient was pregnant. In the literature, it is said that on chest radiographs of MS patients, left atrial enlargement and redistribution of pulmonary vasculature are usually seen, and right ventricular hypertrophy is seen.[2,6]

On echocardiographic examination, moderate-severe MS was found, MR moderate ec RHD (WS 8), AR mild ec RHD, TR mild-moderate, high probability PH, good global LV systolic function, EF 55%, global neurokinetic, RV contractility well, sec, no visible thrombus from TTE. Echocardiography is a very important diagnostic tool for MS. In MS, there is usually thickening of the mitral valve and abnormal union of the commissures with limited separation during diastole. Left atrial enlargement is often seen, and an intra-atrial thrombus may be seen. Stratification of the patient's severity can be done by assessing the mitral valve area.[7,8,9]

Initial therapy in these patients is the management of rapid ventricular response atrial fibrillation because it is a potentially dangerous arrhythmia. Atrial fibrillation is a dangerous arrhythmia because 1. The rapid ventricular rate can reduce cardiac output, causing hypotension and pulmonary congestion, and 2. The absence of organized atrial contractions can cause stasis of blood in the atria, increasing the risk of thrombus formation, especially in left atrial appendages. (LAA). Embolization of the left atrium is the most common cause of stroke. Therefore, the management of AF is carried out by considering 3 aspects, namely controlling the ventricular rate, considering ways to restore heart rhythm to sinus rhythm, and using anticoagulants to prevent thromboembolism.[10,11,12]

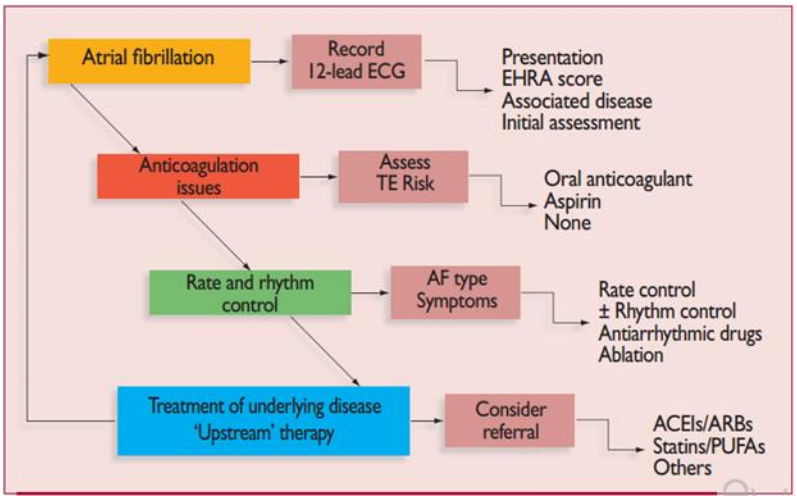


Figure 4. Management of patients with AF

According to the 2012 ESC guidelines for the management of atrial fibrillation, symptomatic AF therapy can be rate control or rhythm control. In this patient with complaints of palpitations with symptoms of heart failure, rate control with digoxin is the main choice.[10]. The target rate control in patients with AF according to the RACE 2 trial resting pulse rate of fewer than 80 beats/minute and less than 110 beats/minute during light activities.[11] Therefore, to maintain a fast pulse in this patient, 0.25 mg of oral digoxin was administered.

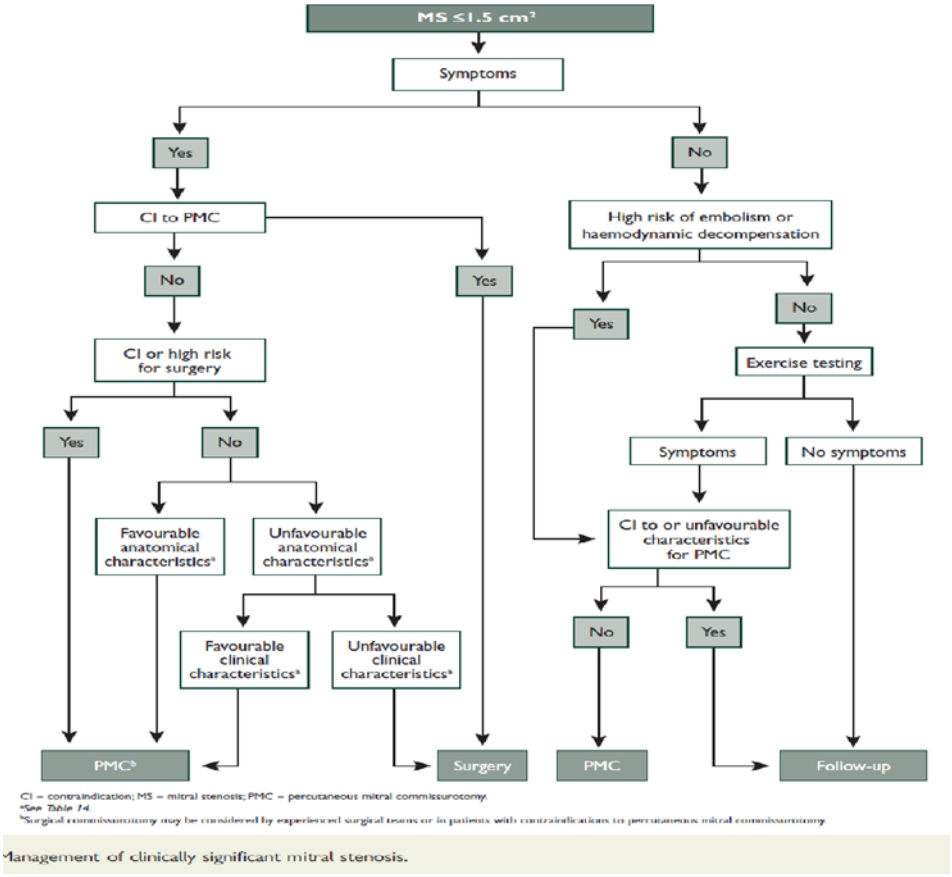
In patients with AF caused by MS, vitamin K antagonist anticoagulant therapy is given with a target INR of 2.0-3.0.[12]. In this patient, warfarin was given at a dose adjusted to the INR level at a dose of 1 x 5 mg. INR is re-examined every week in the initiation phase of therapy and every month if the anticoagulant is stable.[12]

Table 1. Treatment of rheumatic according to AHA guidelines[9]

Class I	Anticoagulation (vitamin K antagonist [VKA] or heparin) is indicated in patients with 1) MS and AF (paroxysmal, persistent, or permanent), or 2) MS and a prior embolic event, or 3) MS and a left atrial thrombus (309-315). (Level of Evidence: B)
Class IIa	Heart rate control can benefit patients with MS and AF and fast ventricular response. (level of evidence: C)
Class IIb	Heart rate control may be considered for patients with MS in normal sinus rhythm and symptoms associated with exercise (317, 318). (level of evidence: B)

The choice of intervention method for this patient also needs to be considered. The choice of method and timing of intervention is decided based on clinical characteristics (including the patient's functional status, risk prediction, and results of percutaneous mitral commissurotomy (PMC), valve anatomy, and local expertise. Indications for intervention are interventions performed only on MS patients with clinically significant symptoms (valve area 1.5 cm^2) and performed on symptomatic patients.

Management of severe mitral stenosis was carried out according to the 2012 guidelines (Figure 5). An anatomical assessment of the mitral valve was performed to assess the safety and likelihood of PMC. The most widely used echocardiographic parameters were the Wilkins Score shown in table 2 and the Cormier score in table 23. Patients with mitral stenosis scores of 8-9, which was not more than moderate, were the best for PMC. Patients with a score of 9-10, especially those with more than moderate mitral regurgitation, should be referred for surgery immediately, except in cases with serious or severe comorbidities.[13] In patients with high scores, the anterior valve, and flexible cord *are* recommended for mitral valve repair. Conversely, if the valve damage is severe, with severe calcification, or the papillary muscles fuse at the valve margins, the mitral valve should be replaced.[8]



Management of clinically significant mitral stenosis.

Figure 5. Management algorithm severe mitral stenosis

Table 2. Wilkins Score

Grade	Mobility	Thickening	Calcification Subvalvular	Thickening
1	Highly mobile valve with only leaflet tips restricted	Leaflets are near normal in thickness (4-5 mm)	A single area of increased echo brightness	Minimal thickening just below the mitral leaflets
2	Leaflet mid and base portions have normal mobility	Mid Leaflets normal, with considerable thickening of margins (5-8 mm)	Scattered areas of brightness confined to leaflet margins	Thickening of chordal structures extending to one-third of the chordal length
3	Valve continues to move forward in diastole, mainly from the base	Thickening extending through the entire leaflet (5-8mm)	Brightness extends into the mid-portions of the leaflets	Thickening extended to the distal third of the chords
4	No or minimal forward movement of the leaflets in diastole	Considerable thickening of all leaflet tissue (>8-10mm)	Extensive brightness throughout much of the leaflet tissue	Extensive thickening and shortening of all chordal structures extending down to the papillary muscles
• The total score is the sum of the four items and ranges between 4 and 16.				

Table 3. Cormier scores [14]

Cormier Score	
Echocardiographic group	**Mitral valve anatomy**
Group 1	Pliable noncalcified anterior mitral leaflet and mild subvalvular disease [i.e., thin chordae ≥ 10 mm long]
Group 2	Pliable noncalcified anterior mitral leaflet and severe subvalvular disease [i.e., thickened chordae <10 mm long]
Group 3	Calcification of the mitral valve of any extent, as assessed by fluoroscopy, whatever the state of the subvalvular apparatus

Cormier et al. Arch Mal Coeur 1989; 82: 185-91

Based on table 4, the patient characteristics do not support PMBC. Clinical features include old age, history of commissurotomy, NYHA class IV, permanent atrial fibrillation, and severe pulmonary hypertension. Anatomical criteria that did not support PMBC were Wilkins score > 8, Cormier scores 3, very narrow mitral valve area, and severe tricuspid regurgitation.

Table 4. Contraindications for percutaneous mitral balloon commissurotomy[14]

• Mitral valve area > 1,5 cm^2
• Left atrial thrombus
• More than mild mitral regurgitation
• Severe or bicommissural fusion
• Absence of commissural fusion
• Severe concomitant aortic valve disease, or severe combined tricuspid stenosis and regurgitation
• Concomitant coronary artery disease requiring bypass surgery

Based on the above characteristics, this patient is advised not to perform percutaneous mitral commissurotomy because of a contraindication, namely moderate mitral regurgitation. The patient was advised to undergo valve replacement surgery. In valve replacement interventions, consideration should be given to whether the patient will receive prosthesis mechanical valve bioprosthetic. Each has advantages and disadvantages. All mechanical valves require lifelong use of anticoagulants. Biological valves do not require long-term anticoagulation unless AF or other indications arise unless they are susceptible to structural valve deterioration (SVD).[15]

The choice between a mechanical valve and a biologic valve is primarily determined by the anticoagulant risk associated with bleeding events and the risk of thromboembolism in the mechanical valve, as compared to the risk of SVD in the biologic valve. The risk of bleeding is controlled by monitoring INR, controlling anticoagulants, taking aspirin, and risk factors for bleeding. The risk of SVD decreases with increasing age and is higher for the mitral valve than for the aortic valve.[15]

Moderate-severe mitral stenosis is tolerated poorlyMS is the leading cause of death in rheumatic heart disease in pregnancy. The diagnosis is made on the basis of echocardiography. Heart failure frequently occurs in pregnant women with moderate-severe MS in the 2nd and 3rd trimesters. Heart failure is often progressive. Pulmonary edema may occur, especially if MS is not known or if atrial fibrillation is present.

All patients with moderate-severe MS (even those

without symptoms) should not be pregnant, and intervention should be performed before pregnancy, with the option of percutaneous intervention. Follow-up echocardiography is indicated monthly or 2 months, depending on the hemodynamics of the patient. In mild MS, evaluation is done every semester and before birth.

Activity should be limited, and B blockers are given if symptoms or pulmonary hypertension are present (echocardiography estimates systolic PAP 0.50 mmHg. Diuretics are used if symptoms persist, avoiding high doses. Anticoagulant therapy is recommended in paroxysmal or permanent AF and contra-spontaneous echocardiography in the left atrium (≥) 40mL/m^2), low cardiac output, and congestive heart failure, as these women are at high risk of thromboembolism.

PMC is better done at 20 weeks gestation. It should only be done in women with NYHA class III / IV and or systolic PAP 0.50 mmHg on echocardiography despite having received optimal drug therapy.

Vaginal delivery should be performed in patients with mild MS and in patients with moderate or severe MS with NYHA class I/II without pulmonary hypertension. Cesarean section should be performed in patients with moderate or severe MS with NYHA class III/IV or who have pulmonary hypertension despite medication. Optimal drugs that PMC cannot do or PMC fails to do.

4. Conclusion

Management of atrial fibrillation rapid ventricular response in this patient was carried out by administering digoxin 0.25 mg orally. Patients are given anticoagulants to prevent thromboembolic complications. This patient was advised to undergo valve replacement surgery considering the degree of stenosis, complications, and a thorough assessment.

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