# MITRAL VALVE REPLACEMENT AT 11 YEARS OLD GIRL WITH RHEUMATIC MITRAL STENOSIS

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**ABSTRACT**

Rheumatic heart disease (RHD) is the permanent heart valve damage resulting from one or more attacks of acute rheumatic fever (ARF), representing the permanent lesions of the cardiac valve. Rheumatic heart disease's patient with valvar involvement usually require long term follow-up. The ultimate decision of clinical management or invasive therapy is made on an individual basis. We reported a 11-year-old girl with RHD and severe mitral stenosis whom underwent mitral valve replacement with mechanical valve. Postoperative echocardiogram showed no mitral regurgitation and we gave anticoagulant to maintain INR of 3-4 by giving warfarin 2mg / kg each day for lifelong to prevent bleeding and thrombosis. [**MEDICINA 2014;45:120-6**]

***Keywords**: rheumatic mitral stenosis, mitral valve replacement, children*

# PENGGANTIAN KATUP MITRAL PADA ANAK USIA 11 TAHUN DENGAN STENOSIS MITRAL REMATIK

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**ABSTRAK**

*Penyakit jantung rematik (PJR) adalah kerusakan katup jantung permanen yang diakibatkan oleh satu atau lebih serangan demam rematik akut (DRA). Pasien PJR dengan keterlibatan katup membutuhkan pemantauan jangka panjang. Kami melaporkan perempuan berusia 11 tahun dengan PJR dengan stenosis mitral berat dan dilakukan penggantian katup mitral dengan katup mekanik. Pada ekokardiogram setelah operasi tidak didapatkan lagi regurgitasi mitral. Pasien mendapatkan antikoagulan untuk mempertahankan INR 3-4 dengan memberikan warfarin 2mg/kg setiap hari untuk mencegah perdarahan dan trombosis.* [**MEDICINA 2014;45:120-6**]

***Kata kunci**: stenosis mitral rematik, penggantian katup mitral, anak*

## INTRODUCTION

Rheumatic heart disease (RHD) is the permanent heart valve damage resulting from one or more attacks of acute rheumatic fever (ARF), representing the permanent lesions of the cardiac valve. It has been thought that 40-60% of patients with ARF will go on developing RHD. Acute rheumatic fever follows 0.3% of cases of group A beta-hemolytic streptococcal pharyngitis in children. As many as 39% of patients with acute rheumatic fever may develop varying degrees of pancarditis with associated valve insufficiency, heart failure, pericarditis, and even death. The clinical presentation and the mortality, as well as the frequency and speed of development of an established valvar disease after the acute phase, vary considerably geographically, influenced primarily by the socioeconomic and medical backgrounds of the populations involved.[1]

Acute rheumatic fever and RHD are thought to result from an autoimmune response, but the exact pathogenesis remains unclear. Although rheumatic heart disease was the leading cause of death 100 years ago in people aged 5-20 years in the United States, incidence of this disease has decreased in developed countries, exceeds 50 per 100.000 children, and the mortality rate has dropped to just above 50% since the 1960. According to WHO, at least 15.6 million people have RHD. There are 300.000 of about 0.5 million individuals who acquire ARF every year go on to develop RHD, and 233.000 deaths annually are directly attributable to ARF.[1-3]

The healing process of rheumatic carditis results in varying degrees of fibrosis and valvar damage. In some instances, fusion and thickening of the pericardium may also occur, but this rarely affect the ultimate cardiac performance. All age groups, the mitral valve is the

most common affected, followed by a combination of mitral and aortic valves, then isolated aortic valvar disease, usually incompetence, and combined mitral, aortic, and tricuspid disease.[4,5]

The ultimate decision of clinical management or invasive therapy was made on an individual basis. The operative risks and potential post operative complications, should be balanced against the benefits to an invasive treatment and the risk produced by the valvar lesion in the absence of an invasive approach.[6-8] The purpose of the paper is to report the case of a 11-year-old girl with rheumatic heart disease and severe mitral stenosis whom underwent mitral valve replacement with prosthetic valve.

## CASE ILLUSTRATION

NM, a 11-year-old-girl, has been referred to Sanglah Hospital from Mataram Hospital at Oktober 5th 2011, with diagnosis rheumatic heart disease (RHD). The major complain was chest pain since 2 weeks before admitted (September 20th 2011) when she was at school. The chest pain spreading to the left arm and the left lower limb. It came suddenly, and made she can't move her upper and lower extremities, usually happened when patient was in active condition and diminished at rest. The history of trauma was denial. Swollen or redness of the extremities or other body parts indisputably by the patient. The patient has been admitted for six days at Mataram Hospital.

The history of an migratory or consistent pain on her joints was denial. She was never complained the presence of lumps in skin neither in the arms, a limb, the back or other part of the body. There was no history of sore throat one month prior to the sickness.

There was history easy to get fatigue and difficulty of breathing during her activity since 7 months prior to admission. Patient was cared at hospital and diagnosed with heart disease. Since then, patient had routine check-up at a pediatrician and was given medicines of furosemide, captopril and injection of Penicillin G Benzatin of 1.2 million IU every 28 days.

Patient lost her weight since 7 months ago. The patient was born full term, spontaneously, and was assisted by midwifes. No visible abnormalities were found. Her mother did not get any prior medications for specific diseases during pregnancy. The immunization history was complete according to the recommended immunization plan by government. There was no abnormality in growth and development. Now she is at 6th grade elementary school. Daily activities prior to illness were normal, in which she participated well in school and in peer activities after school time. There was no history about family with the same complaint.

Physical examination revealed an alert girl. The blood pressure was 100/70 mmHg. The pulse was 122 beat per minutes and regular, respiratory rate 30 x/min, axillary temperature 36.9°C. Her body weight was 22 kg, body height 141 cm. Thus, according to the Waterlow criteria, her nutritional status was 62.5% (malnourished).

The hair was fine and black. The conjunctiva was not anemic, the sclera was not icteric, and the pupil reflexes were normal. The ENT as well as neck examination were within normal limit. There were no palpable lymph nodes nor nuchal rigidity. The chest examination showed no precordial bulging, ictus cordis was appeared and palpable at ICS IV left MCL, RV heave and thrill was palpable. Left ventricular lifting was also noted. The heart sound was regular, with presystolic murmur at apex area (ICS IV MCL sinistra), with grade 4/6, with rumbling sound during diastolic phase, and radiating along axilla line. The movement of both sides of the chest was symmetrical. Vesicular respiratory sounds were noted, without wheezing or rales.

There were no hepatomegaly nor splenomegaly. Bowel sound was normal. The examination of upper and lower extremities showed no deformities. Physiological reflexes were normal, there was no pathologic reflex. There was no edema nor cyanosis. Based on anamnesis and physical examination, we diagnosed the patient with rheumatic heart disease, with differential diagnosed by recurrent attack of RF in a patient with established rheumatic heart disease and chronic valve lession of RHD.

From the first investigation, on of chest X-ray showed cardiomegaly with cardio thoracic ratio of 63% with prominent pulmonal segment, the enlargement left atrial appendage visible on the left cardiac border, widened left cardiac border, descent apex, and left aortic arch. The lung showed increasing pulmonary vascular markings and there was no infiltrate (**Figure 1**). The electrocardiographic features revealed heart rate 124 times/min with sinus rhythm, with right axis deviation ($90^0$-$120^0$), notched P waves are seen in the limb leads, biphasic P wave in leads V2, right and left ventrikel hypertrophy with no prolongation of PR interval.

Echocardiograph revealed atrial situs solitus, normal systemic and pulmonal veins drain, AV-VA concordant, LVH, LA enlarge, LA/Ao 1.84, no ASD/VSD/PDA/CoA, left Ao arch, Severe MS (thick A2 and P2 MV, PG 22.67 mmHg, opening MV 5.0 mm, MV annulus 38 mm), mild AR, trivial PR (PG 20.04 mmHg), mild TR (PG 21.69 mmHg), no pericardial effusion, slight decrease LV systolic function (EF 60%) with results were severe MS, Mild AR, mild TR are manifestation of RHD. The laboratory result include complete blood count, chain

reactive protein (CRP), ESR, and throat swab. The CBC revealed WBC:12.5 k/µL (Ne 56.0%; Ly 17.7%), Hgb 12.1 g/dL, Hct 35.5 %, MCV 79.9 fL, MCH 27.8 pg, PLT 285 k/µL. sodium 136.20 mmol/L, potassium 4.172 mmol/L, chloride 98.73 mmol/L, calcium 9.697 mg/dL. Blood glucose 116 mg/dL, ASTO 200 IU/ml, CRP 11 mg/L, and ESR 2 mm. We also revealed throat swab and the result was coccus gram positive. The culture of the throat swab revealed no growth. Based on the clinical manifestation, imaging finding, and laboratory results, we diagnosed the patient with rheumatic heart disease with chronic valve lesion, functional heart failure NYHA class III and malnourished state.

The treatment were activity limitation and bed rest for 3-4 weeks, then gradual mobilization for 3 months. Benzatin G Penicillin injection 1.2 million every 28 days up to 40 years old or lifelong, heart failure therapies consists of dopamine 10 mcg/kg/min, furosemide injection 20mg and spinorolakton 12,5mg for twice daily, respectively. For malnourished, patient was given according her calory daily need based on RDA consists of soft food and high density milk formula.

During 10 days hospital care, patient had good health progression, so anti-heart failure medication was shift to oral medicines. Her nutritional status was change from malnourished to undernourished. Patient underwent mitral valve replacement using mechanical mitral valve and after had procedure, the patient received warfarin 2 mg/kg/day continuously for lifelong to prevent thromboemboly, also careful counseling prior to pregnant and discussion of the risks associated with available anticoagulant options.

## DISCUSSION

Rheumatic heart disease is the only clinical manifestation of rheumatic fever (RF) which results in residual or permanent damage occurring in 14 to 99% patients. According to WHO, at least 15.6 million people have RHD, 300.000 of about 0.5 million individuals who acquire ARF every year go on to develop RHD, and 233.000 deaths annually are directly attributable to ARF. Acute rheumatic fever is a rare disease in the very young, only 5% of first episodes arise in children younger than age 5 years and the disease is almost unheard of in those younger than 2 years. Rheumatic heart disease usually results from the cumulative damage of recurrent episodes of ARF. The prevalence of RHD increases by age, with peak in adults aged 25–34 years.[2-4] In our case, patient was first diagnosed with RHD at 11 years old.

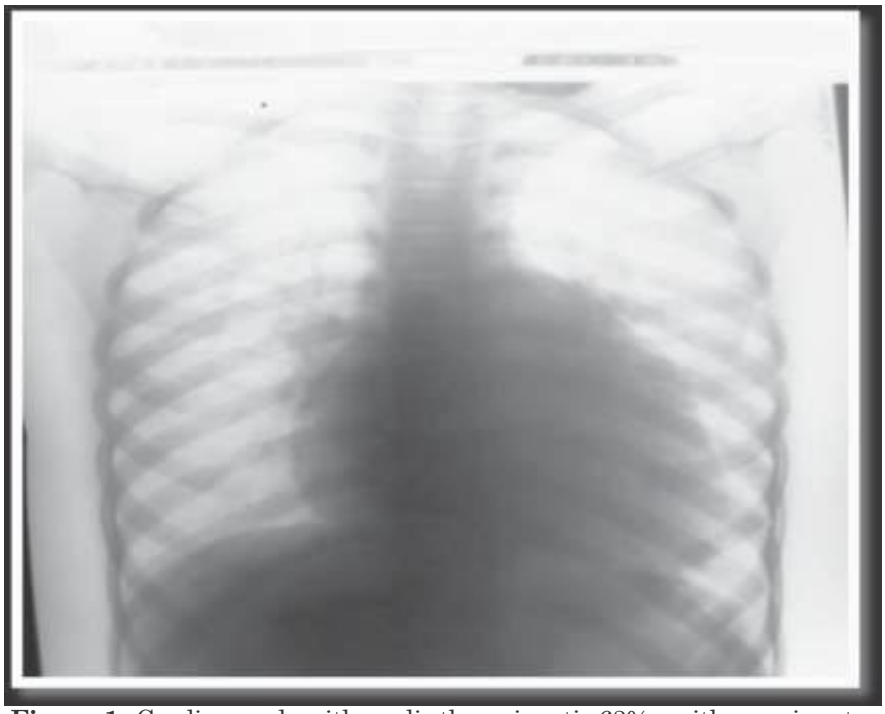

**Figure 1.** Cardiomegaly with cardio thoracic ratio 63% a with prominent pulmonum segment, the enlargement left atrial appendage visible on the left cardiac border, wide left cardiac border, descent apex. and increasing pulmonary vascular markings in chest X-ray was taken at the first admission.

Rheumatic heart disease criteria is used for patient whom first came in only mitral stenosis or with combination on of mitral stenosis and mitral insufficiency and or aortic valve disease. Special for chorea, we don't need other mayor criteria or evidence of previous of group A streptococcus infection (**Table 1**).

In this case, patient presenting for the first time with severe mitral stenosis with mild aorta regurgitation. Based on 2002-2003 WHO criteria for RF/RHD, we can diagnose the patient with RHD.

In all age group, the mitral valve is commonly affected, followed by a combination of mitral and aortic valves, then isolated aortic valvar disease, usually incompetence, and combined mitral, aortic, and tricuspid disease.[5,6] In this case, based on physical examination and echocardiography, we found severe mitral stenosis with mild aorta insufficiency.

Mitral stenosis (MS) is a common chronic rheumatic valvar lesion, becoming established earlier than aortic stenosis. Normal mitral valve area: 4-6 $cm^2$, mild mitral stenosis: 1.5-2.5 $cm^2$,

**Table 1**. 2002-2003 WHO criteria for the diagnosis of rheumatic heart disease (based on the revised Jones criteria).[4]

| Diagnostic categories | Criteria |
|---|---|
| Primary episode of RF | Two major or one major and two minor manifestations plus evidence of preceding group A streptococcal infection |
| Recurrent attack of RF in a patient without established rheumatic heart disease | Two major or one major and two minor manifestations plus evidence of preceding group A streptococcal infection |
| Recurrent attack of RF in a patient with established rheumatic heart disease | Two minor manifestation plus evidence of preceding group A streptococcal infection |
| Rheumatic choreaInsidicus onset rheumatic carditis | Other major manifestations or evidence of group A streptococcal infection not required |
| Chronic valve lesions of RHD (patients presenting for the first time with pure mitral stenosis or mixed mitral valve disease and/or aortic valve disease) | Do not required any other criteria to be diagnosed as having rheumatic heart disease |

moderate mitral stenosis: 1.0-1.5 $cm^2$ and severe mitral stenosis : < 1.0 $cm^2$. [6-8] In this patient, we found opening mitral valve 0.5 $cm^2$ (severe stenosis) with annulus valve was 38 mm and slight decrease left ventrical systolic function with ejection fraction 60%.

The symptoms, physical signs, and amount of clinical disability are parallel to the severity of mitral stenosis. The characteristic findings of MS on auscultation are an accentuated first heart sound, an opening snap, and a mid-diastolic rumble. The duration of the murmur increases and the opening snap occurs earlier during diastole as a result of higher left atrial pressure while severe stenosis is worsen.[8] In our case, we heard single first sound, presystolic murmur at apex area (ICS IV MCL sinistra), grade 4/6, with rumbling sound during diastolic phase, and radiating along axilla line.

The chest cardiography shows various degrees of left atrial enlargement, which displaces the esophagus backward, with elevation of the left main bronchus and widening of the carina.[8] In this case, we found cardiomegaly with cardio thoracic ratio 63%, prominent pulmonum segment, enlargement of left atrial appendage that visible on the left cardiac border, widened left cardiac border, and descent apex.

The electrocardiogram shows evidence of left atrial enlargment. Broad, notched P waves are seen in the limb leads, or biphasic P wave in leads V1 dan V2, with a marked negative component. Right atrial enlargement, right axis deviation, and right ventrivuler hyperthropy in various degrees reflect the severity of the pulmonary hypertension.[8] In this case, right axis deviation ($90^0$-$120^0$), notched P waves are seen in the limb leads, biphasic P wave in leads V2, right and left ventrikel hypertrophy with no prolongation of PR interval.

Echocardiography has become the most useful tool for both diagnosis and follow up mitral stenosis. In this case, Echocardiograph revealed atrial situs solitus, normal systemic and pulmonal veins drainage, AV-VA concordant, LVH, LA enlarge, LA/Ao 1.84, no ASD/VSD/PDA/CoA, Left Ao Arch, Severe MS (thick A2 and P2 MV, PG 22.67 mmHg, opening MV 5.0 mm, MV annulus 38 mm), mild AR, trivial PR (PG 20.04 mmHg), mild TR (PG 21.69 mmmHg), no pericardial effusion, slight decrease of LV systolic function (EF 60%), with results were severe MS, mild AR, mild TR are manifestation of RHD.

The New York Heart Association (NYHA) Functional classification provides a simple way of classifying the extent of congestive heart failure (CHF). It places patients in one of four categories based on how much they are limited during physical activity, the limitations/symptoms are in regards to normal breathing and varying degrees in shortness of breath and or angina pain. In this case, patient had limitation of physical activity, comfortable at rest, less than ordinary activity might cause fatigue, palpitation, and dyspnea, so we classified patient on CHF class III based on NYHA Functional classification.

Patient rheumatic heart disease with valvar involvement usually require long term follow-up. This is based on serial clinical examinations, supplemented by periodic laboratorial evaluations, including electrocardiography, chest radiography, and echocardiography. Medical treatment essentially consists of adequate prevention of recurrences and infective endocarditis, and treatment of congestive cardiac failure. These patient require close observation to detect progression

of the disease, besides control of medical therapy, supervision of physical activities, and recognition of complications.[7-9] In this case, the patient received anti-heart failure therapy, adequate prevention of recurrence, BPG 1.2 million IU, and was planned to undergo a mitral valve replacement procedure.

The ultimate decision of clinical management or invasive therapy is made on an individual basis. The operative risks and potential post-operative complications, should be balance against the benefits of an invasive treatment and the risks produced by the valvar lesion in the absence of an invasive approach. The timing and indication for invasive approach are mostly determined by the severity of the clinical condition, degree of valvar dysfunction, and overall cardiac function.[9] Most authors use the Wilkins score to general assessment of valve anatomy, with cut off point[3]10 to developed replacement mitral valve (**Table 3**).[10] In this case, we found mitral stenosis with mitral valve area of 0.5 $cm^2$, with annulus size of 38 mm, thickened fused cordae, so we put patient under invasive treatment.

Replacement was indicated for any of the following: 1) Extensive calcification or degeneration of a leaflet or annulus, 2) Prolapse of more than one third of the leaflet tissue, 3) Active endocarditis, 4) Extensive chordal fusion, calcification, or papillary muscle. Mitral valve repair was indicated if the following conditions existed: 1) Circumscript calcium of leaflets or annulus, 2) Prolapse of less than one third of either leaflet, 3) Pure annular dilatation, 4) Valvular perforations, 5) Incomplete papillary muscle rupture.[9]

In this case, intraoperative, we found minimal forward movement of the leaflets in diastole, thickening of all leaflet tissue, extensive calcification or degeneration of a leaflet or annulus and thickening of chordal fusion up to one third of the chordal length. The Wilkin score is 14, so we proceed with valve replacement procedure.

The main determinants of valve selection to replacement are gender, individual patient life expectancy, tolerance to the need for repeat valve replacement or re-operation, and the use of oral anticoagulants for lifelong. Some studies have claimed that bioprosthetic valve with porcine valve maybe beneficial because of their low rate of thrombogenesis. Mechanical prostheses, although the requirement of anticoagulation, have certain advantages. Their implantation is simple, they have good hemodynamic performance and durability, and they can be remarkably resistant to infection. In this case, the patient wish to minimize the risk of the re-operation and will take or has existing indication or anticoagulation. Therefore, we choose to use mechanical valve.

Valve replacement in children is different from adult patients because of the physiological characteristics of children. Pediatric patients who require valve replacement have several problems, including the small size

**Table 3**. Anatomic classification of the mitral valve[10]

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

of the annulus, frequent congenital heart malformations, limited valve selection, and obstructive hemodynamics of small prostheses. Long term anticoagulation therapy following valve replacement is another problem in children because of its negative effect on mental health and quality of life.[11,12]

Mechanical prostheses appear superior to other substitutes in children despite the requirement of anticoagulation, which exposes active, growing children to the risks of bleeding or thrombosis. The technique of their implantation is simple, they have good hemodynamic performance and durability, they can be used in a variety of complex congenital rheumatic or degenerative conditions, and they can be remarkably resistant to infection.[13]

The primary disadvantage of mechanical valves is their thrombogenicity. Because of the low pressure and more turbulent flow across the mitral valve, a higher target international normalized ratio (INR) of 3-4 is recommended for patients with mechanical mitral prostheses (compared with 2-3 for aortic valve replacement). The incidence of major hemorrhage (internal or external bleeding resulting in death, permanent injury, hospitalization or transfusion) while on therapeutic levels of anticoagulation for mechanical mitral valve prosthesis was approximately 21% over an 11-year period in one study cited by the AHA guidelines.[13,14] In this case, the annulus size of 38mm, suitable to mechanical prosthese. After we done the mitral valve replacement using mechanical prosthese, postoperative echocardiogram showed no mitral regurgitation and we gave anticoagulant to maintain INR of 3-4 by giving warfarin 2mg / kg each day for lifelong to prevent bleeding and thrombosis **(Figure 2).**

Women with prosthetic heart valves exhibit a heightened risk of thromboembolic events during pregnancy. Anticoagulation with warfarin provides protection against these complications, but the use of this drug increases the risk of embryopathy when dose is more than 5 mg/kg and the other side, subcutaneous administration of heparin sodium has been reported to be less effective anticoagulant resulting in more maternal complications, but it is more protective of the fetus.[15,16] In this case, the gender of this patient is female adolescent and received warfarin 2 mg/kg/day continuously for lifelong to prevent thromboemboly. The choice of anticoagulant regimens for mechanical heart valve thromboprophylaxis when she get pregnant is very difficult. So, we recommend for her parents to careful counseling prior or shortly after the diagnosis of pregnancy and discussion of the risks associated with available anticoagulant options.

## SUMMARY

A-11 year old female with chest pain since 2 weeks, tachycardia, presystolic murmur at apex area (ICS IV MCL sinistra), grade 4/6, with rumbling sound during diastolic phase,and from echocardiography we found severe MS, mild AR, and mild TR, was reported. The diagnose was RHD. The patient received anti-failure therapy, adequate prevention of recurrence, BPG 1.2 million IU, and the mitral valve replacement using mechanical prosthese. The postoperative echocardiogram showed no mitral regurgitation after we had done the mitral valve replacement using mechanical prosthese. We gave anticoagulant to maintain INR of 3-4 by giving warfarin 2mg / kg each day for lifelong to prevent bleeding and thrombosis.

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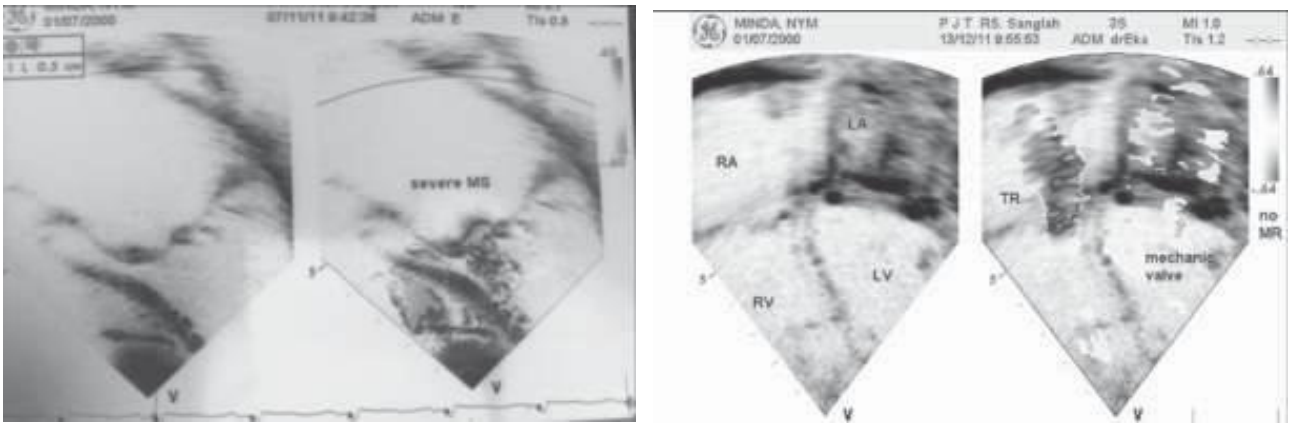


**Figure 2.** (A) Preoperative echocardiogram showing severe mitral stenosis; (B) Postoperative echocardiogram showing no mitral regurgitation.

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