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PATIENT WITH RHD EC MR SEVERE, MS MODERATE AND TROMBUS IN LV, WHEN WE DO THE SURGEOM IN CUT MEUTIA REGIONAL GENERAL HOSPITAL

Yuri Savitri[1], Helma Humairah[2], Jauza Raudhatul Jannah Mendrofa[2], Nurul Afni[2], Della Vega Nisha Ayuna[2*]
[1] Department of Cardiology, Cut Meutia General Hospital, Aceh Utara, Indonesia
[2] Undergraduate Program, Faculty of Medicine, Malikussaleh University, Lhokseumawe, Indonesia
*Corresponding Author : E-mail : dellaadella@gmail.com

ABSTRACT

Rheumatic heart disease (RHD) is an acquired heart valve disorder that caused by acute rheumatic fever (ARF) which results from autoimmune response to throat infection by Streptococcus pyogenes (group A Streptococcus bacteria). In developing countries, acute rheumatic fever become one of the most important causes of cardiovascular events. A 25-years-old male patient, domiciled in Alue Dalam, Darul Aman, East Aceh, admitted to emergency department of Cut Mutia hospital, the patient was brought to the hospital by his family at 05.0o pm on January 27th, 2022. Patient referred from Graha Bunda hospital. Patient was admitted to the hospital with complaints shortness of breath. Shortness of breath is felt during activity and worsens at night. Shortness of breath since yesterday. The patient also complained of pounding. The pounding has been felt for five days before arriving at the hospital. The patient also has chest pain, and tired easily. Past medical history such hypertension and diabetes mellitus are denied. Physical examination before treatment was obtained: the patient looked restless, weakness, and akker. Echocardiography examination showed the MR Severe, MS moderate, AR moderate, TR moderate, PH mild, AML calsification, all chamber dilatation, and thrombus LV. The patient was given initial treatment in Cut Mutia General Hospital.

Keywords: *Rheumatic heart disease; rheumatic fever; Severe MR; Moderate MS; Trobus in LV*

INTRODUCTION

Rheumatic heart disease (RHD) is an acquired heart valve disorder that caused by acute rheumatic fever (ARF) which results from autoimmune response to throat infection by Streptococcus pyogenes (group A Streptococcus bacteria). In developing countries, acute rheumatic fever become one of the most important causes of cardiovascular events.[1]

Rheumatic carditis incorporates a range of lesions, counting pericarditis and valvulitis amid clinical or subclinical ARF; there is a transition from rheumatic carditis to RHD, with chronic valvular lesions that advance yearly after ≥1 ARF episodes. Progressive valvular disease usually develops in the years after ≥1 ARF episodes, in spite of ARF is recognized only in 30% to 50% of cases. [2]

There are 27% of individuals getting their first ARF episode and no RHD at that time, then developed RHD a year after. In 5 years, 44% had developed RHD. More than half (52%) had progressed to RHD within 10 years. Among RHD patients, 10% had severe RHD in the first diagnosis. By one year, the number had multiplied to 20%. By 10 years, 35% had severe RHD.[3]

In 2017, The Global Burden of Disease (GBD) estimated 39.3 million cases of RHD, 285.517 RHD deaths, and 9.39 million disability-adjusted life-years. In spite of the reductions of 47.5% in the burden between 1990 and 2015, large regional disparities persist, with ARF hyperendemic in low and middle-income countries where >80% of the global ARF cases occur.[4] Recently, RHD continues unabated in poor countries and among vulnerable groups in high income ones.[5]

This disease typically develops in childhood. The peak of ARF incidence is in the 5-14 year age group, reducing significantly with age and being rare over the age 35 years. In opposite, RHD prevalence increased with age, peaking at 35-44 years old. Above one-third of RHD cases had severe RHD, meaning that these people had also been hospitalized for heart failure or a heart valve intervention.[3]

Primary prevention through treatment of group A streptococcal pharyngitis with antibiotics (BPG) has typically been given through primary health care to make care accessible to achieve necessary coverage. Monthly BPG injection for secondary prevention is recommended for years after ARF event including carditis, with varied duration depending partly on age and severity. Heart failure management and other sequel of severe disease, assessment for heart valves surgical procedure, and postoperative anticoagulation and follow-up benefit from integrated care strategy.[6]

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CASE REPORT

A 25-years-old male patient, domiciled in Alue Dalam, Darul Aman, East Aceh, admitted to emergency department of Cut Mutia hospital, the patient was brought to the hospital by his family at 05.0o pm on January 27th, 2022. Patient referred from Graha Bunda hospital. Patient was admitted to the hospital with complaints shortness of breath. Shortness of breath is felt during activity and worsens at night. Shortness of breath since yesterday. The patient also complained of pounding. The pounding has been felt for five days before arriving at the hospital. The patient also has chest pain, and tired easily. Past medical history such hypertension and diabetes mellitus are denied.

Physical examination before treatment was obtained: the patient looked restless, weakness, and akkert. BP: 100/70 mmHg, HR: 64x/m, RR: 30x/m, T: 36,5 ^{O}C, SpO_2: 94%. Chest examination show breath sounds were rhonki in both lung. In this case on physical examination with auscultation at rest found murmur or abnormal heart sound.

Table 1. Laboratory

Test	Result	Unit	Normal
Hematology			
Hemoglobin	15,14	g/dl	12.0-16.0
Erythrocytes	5.72	juta/uL	3.8-5.8
Hematocrit	43.66	%	37.0-47.0
MCV	76.36	fL	79-99
MCH	26.48	Pg	27.0-31.2
MCHC	34.68	g/dl	33.0-37.0
Leukocyte	17.86	ribu/uL	4.0-11.0
Thrombocyte	268	ribu/uL	150-450
RDW-CV	11.02	%	11.5-14.5
Blood Type	O	-	-
Chemical Bloot Test			
Urea	27	mg/dl	<50
Creatinine	1.21	mg/dl	0.5-0.9
Uric Acid	7.8	mg/dl	2.4-5.7
Blood Glocose Level			
Blood Glucose	82.0	mg/dl	<180
Electrolyte			
Na	133	mmol/L	136-146
K	3.5	mmol/L	3.5-5.0
Cl	99	mmol/L	98-106
Ca	0.36	mmol/L	1.12-1.32
Immunology			
CRP	Positif 48 mg/L	mg/L	Negatif
ASTO	negatif	IU/mL	Negative

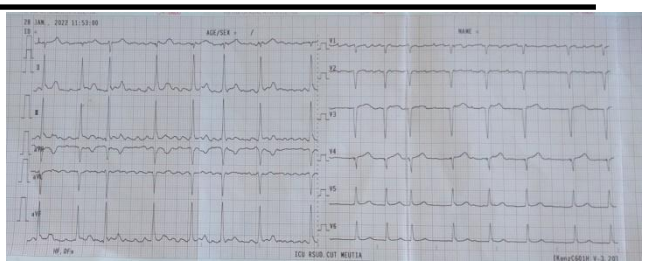

Figure 1. ECG Findings

12-lead electrocardiogram obtained at admission showed unidentify P wave, narrow QRS wave and irregular R in all leads. The conclusion was Atrial Fibrilation.

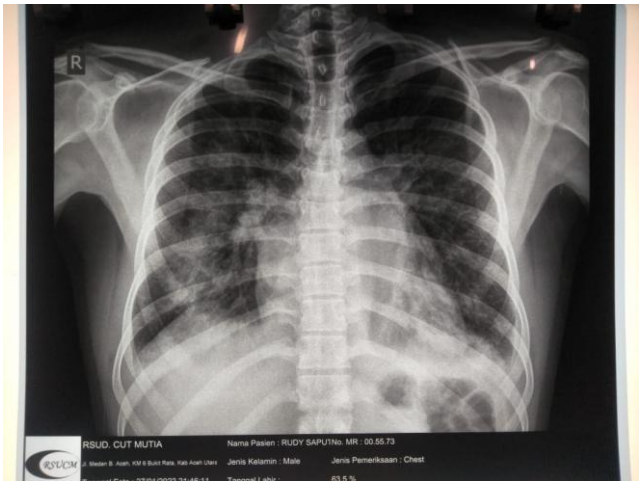

Figure 2. Chest x-ray

Chest x-ray result showed consolidation right paracardial, sinus of lancip, Slippery diaphragm, cor:CTR<0,5.The result suspect pneumonia

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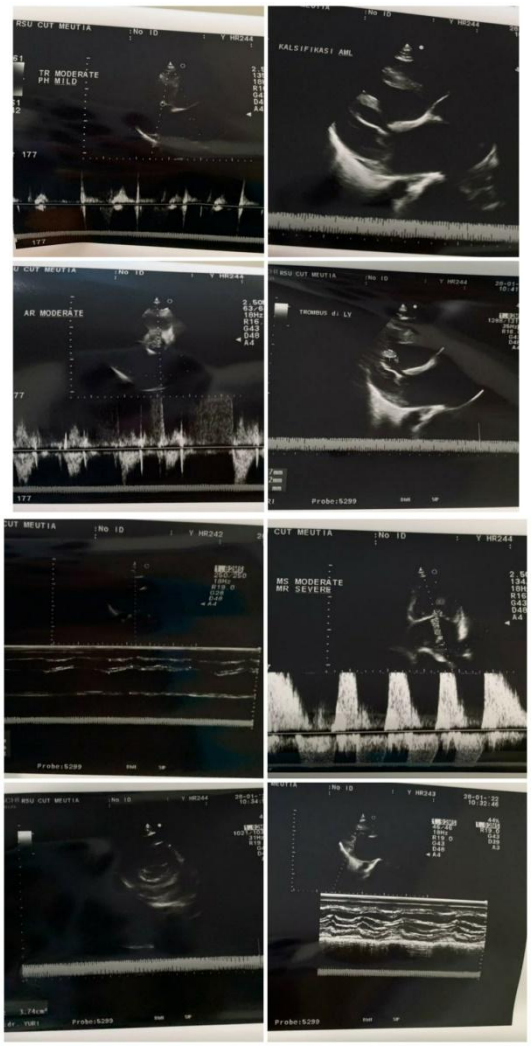

Figure 3. Transthoracic echocardiogram

Echocardiography examination showed the MR Severe, MS moderate, AR moderate, TR moderate, PH mild, AML calsification, all chamber dilatation, and thrombus LV.

The patient was given initial treatment in Cut Mutia General Hospital. We administrate O_2 3-4 lpm, injection of ceftriaxone 2 gr/12h, injection of furosemide 1cc/hour, simarc 1x2 mg, concor 1x 5 mg, spironolakton 1x25 mg, KSR 2 x 600 mg, eritromisin 2x250 mg, and sucralfate syrup 2xCI.

DISCUSSION

Rheumatic fever is a continuation of pharyngeal infection caused by group A beta hemolytic Streptococci. An autoimmune reaction to Streptococcal infection hypothetically will cause tissue damage or manifestations of rheumatic fever, as follows (1) Group A Streptococci will cause infection of the pharynx, (2) Streptococcal antigens will cause the formation of antibodies in hyperimmune hosts, (3) antibodies will react with Streptococcal antigens, and with host tissues that are antigenically the same as Streptococci (in other words, antibodies cannot distinguish between Streptococcal antigens and heart tissue antigens), (4) autoantibodies These cells react with host tissues, causing tissue damage. [7]

The damage to this tissue will cause endocardial lining inflammation, especially regarding the valve endothelium, which results in swelling of the valve leaflets and erosion of the edge of the valve leaf. This results in incomplete closure of the mitral valve during systole, causing a decrease in blood supply to the aorta and blood regurgitation from the left ventricle to the left atrium, this results in a decrease in ventricular stroke output so that the heart compensates by dilatation of the left ventricle, increased contraction of the myocardium, hypertrophy ventricular walls and atrial walls resulting in a decrease in the ability of the left atrium to pump blood this results in pulmonary venous congestion and blood returning to the lungs resulting in pulmonary interstitial edema, pulmonary arterial hypertension.[7]

Patient was admitted to the hospital with complaints shortness of breath. Shortness of breath is felt during activity. Shortness of breath since yesterday. The patient also complained of pounding. The pounding has been felt for five days before arriving at the hospital. The patient also has chest pain, and tired easily.

In this case, mitral regurgitation occurs due to the formation of fibrotic tissue caused by inflammation of the mitral valve, so that the blood flowing into the left ventricle returns to the left atrium. At the same time, the left atrium also receives blood from the pulmonary veins so that a lot of blood from the atria will enter the left ventricle which causes the left ventricle to work more and left ventricular hypertrophy occurs.[7] Shortness of breath occurs due to increased pressure in the left atrium resulting in backward failure which causes high hydrostatic pressure in the pulmonary veins (pulmonary hypertension) resulting in transudation

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into the alveoli which eventually causes pulmonary edema.[7]

Tachycardia occurs due to abnormal electrical signals in the atria. As a result, the contractions of the atrium are rapid, irregular and weak.[7] The chest pain and feeling of fatigue that the patient feels are caused by problems with the mitral valve which causes disruption of blood flow so that the blood supply to the coronary arteries and throughout the body is disrupted.[3] Symtoms depend upon the type and severity of diseases like breathlessness with exertion, waking at night feeling breathless, tiredness, pheriperal oedema and palitations if atrial fibrillation or other rhytme problem develops.[3]

Physical examination before treatment was obtained: the patient looked restless, weakness, and allert. BP: 100/70 mmHg, HR: 64x/i, RR: 30x/i, T: 36,5 ^{O}C, SpO_2: 94%. Chest examination show breath sounds were rhonki in both lung. In this case on physical examination with auscultation at rest found murmur or abnormal heart sound. The systolic murmur obtained in this patient indicates a mitral valve abnormality such as in rheumatic heart disease. In rheumatic heart disease, heart valve abnormalities persist due to previous acute rheumatic fever, mainly affecting the mitral valve (75%), the aorta (25%).[8,9]

In this case on laboratory examination on January 27th,2022 results didn't show significant abnormalities. We only found abnormality of leukocyte, creatinin, uric acid, all of that higher from normal range and laboratory examination on January 28th,2022 results didn't show significant abnormalities. We only found abnormality of Na, and Ca under from normal range and at the same time we found abnormality CRP(+48mg/L) and ASTO exemination is negativ the result, it's mean no streptococcal infection.

CRP is a protein produced by the liver in response to inflammation in the body. Healthy people generally have low CRP levels. On the other hand, high CRP levels can be a sign of disease or infection in the body. CRP or C-reactive protein levels in the blood can be checked by CRP examination. This examination has been widely used to diagnose diseases associated with inflammation.[8,10]

The Result of ECG Show Atrial Fibrillation, The electrocardiogram examination showed unidentify P wave, narrow QRS wave and irregular R in all leads. The conclusion was Atrial Fibrilation. Atrial fibrillation is divided into two, namely atrial fibrillation with valve abnormalities (valvular) and without heart valve abnormalities (nonvalvular) Causes of atrial fibrillation are divided into two, namely atrial fibrillation with valve abnormalities (valvular) and without heart valve abnormalities (nonvalvular). Atrial fibrillation with heart valve abnormalities (valvular) is atrial fibrillation that occurs due to valve abnormalities, especially mitral stenosis, mitral regurgitation and the use of prosthetic valves. Mitral stenosis is associated with atrial fibrillation through an increase in left atrial diameter. The mitral valve area is normally 4-6 m2. This size can be reduced due to infection, birth defects, degenerative, tumors and thrombus. This situation results in narrowing of the mitral valve area resulting in dam due to an increase in blood volume in the left atrium. Blood dams directly cause the left atrium to stretch to compensate for the overloadblood to be pumped during the diastolic phase. Chronic elevation results in maximal stretching and dilatation of the left atrium. Stretching of myocytes and myocardium muscle cells results in disturbances in conduction which will have an impact on atrial fibrillation. Mitral valve area is less than 1 cm². The new patient will be hospitalized due to complaining of activity disturbances at rest.[11,12]

Chest x-ray showed consolidation in the right paracardial, sharp costophrenic angles, slippery diaphragm, cor:CTR<0,5.The result suspect pneumonia. Echocardiography examination showed the MR Severe, MS moderate, AR moderate, TR moderate, PH mild, AML calsification, all chamber dilatation, and thrombus LV. Echocardiography is essential for diagnosing RHD, assessing the severity of the valve afflictions, determining the chamber enlargement and function, looking for pulmonary artery hypertension, and also planning for surgical intervention. The most of the patients have either isolated MR, MR and MS (mixed), or mitral and aortic valve disease. The minimum criteria using echocardiographic to diagnose RHD in this context are described in the 2012 World Heart Federation (WHF) guidelines.[13]

MR quantifying includes several parameters described in the American Society of Echocardiography and American College of Cardiology/American Heart Association guidelines.

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Assessment includes the size of the chamber, vena contracta width on color Doppler, continuous- and pulsed-wave Doppler characteristics of mitral inflow and regurgitation, and use of proximal isovelocity surface area for calculating effective regurgitant orifice, regurgitant fraction and volume. Additional findings of severe MR, include left atrial enlargement, pulmonary hypertension, and LV systolic dysfunction.[13,14]

MS in RHD results from valve apparatus thickening and deformity. MS can occur alone or with MR, which is mixed mitral valve disease. The degree of mitral valve narrowing in MS is determined on echocardiography using direct planimetry in the parasternal short-axis view or using pressure half-time in the apical 4-chamber view. The morphological features of the mitral valve mobility, thickening, or calcification can also be graded to assess the BMV suitability.[13]

Less than 1.5 cm^2 mitral valve opening will cause hemodynamic disturbances. In mitral valve stenosis, in addition to hemodynamic changes, rheological changes also occur, namely changes in the nature of blood flow and interactions between blood components. This rheological change is caused by a change in flow from laminar or straight to turbulent or rotating. The magnitude of the blood flow turbulence depends on the mitral valve stenosis severity. The more severe the mitral valve stenosis, the higher the trans mitral gradient, and the greater the hemodynamic force, so the greater the turbulence of blood flow, the greater the mechanical effect on the endothelium that causes endothelial dysfunction. The presence of endothelial dysfunction in the left atrium, which causes the loss of endothelial non-thrombogenic properties and the occurrence of stasis and activation of the blood clotting system, is thought to be a triggering factor for the formation of thrombus. [15,16]

All-chamber dilation occurs due to a disturbance in the valve which causes interference with the backflow of blood to the heart, resulting in an increase in pressure in the heart chambers which causes the heart to need stronger energy in order to pump blood throughout the body. So the dilation that occurs is due to compensation from the heart in maintainence.[15]

Assessing using Jones criteria can determine Rheumatic fever. The major criteria consist of carditis, polyarthritis, subcutaneous nodules, erythema marginatum and Chorea Sydenham . While the minor criteria consisted of fever (>38 °C), erythrocyte sedimentation, arthralgia, leukocytosis, ECG showing prolonged PR. In this case, the major manifestations found based on the Jones criteria were carditis, because the echochrdiogram exemination showed all chamber dilation, and the patient showed clinical heart failure. In this case the signs of minor manifestations found are; increased acute phase reactants (C- reactive protein), leukositosis. Rheumatic heart disease is a heart valve disorder that persists due to previous acute rheumatic fever, mainly affecting the mitral valve (75%), the aorta (25%), rarely affecting the tricuspid valve, and never affecting the pulmonary valve.[3,17]

According to WHO criteria in 2002-2003 for the diagnosis of Rheumatic Fever & Rheumatic Heart Disease (based on Jones criteria), the patient is included in rheumatic heart disease where 2 major criteria and 2 minor criteria are found + Streptococcus B hemolyticus group A evidence of previous infection with valve abnormalities.Based on this, the diagnosis made was RHD e.c valve abnormalities (MR severe,MS moderate + trombus in LV).[3]

The patient was given initial treatment in Cut Mutia General Hospital. We administrate O_2 3-4 lpm, injection of ceftriaxone 2 gr/12h, injection of furosemide 1cc/hour, simarc 1x2 mg, concor 1x 5 mg, spironolakton 1x25 mg, KSR 2 x 600 mg, eritromisin 2x250 mg, and sucralfate syrup 2xCI. Management of RHD includes several steps. The first is the prevention of GM in GABHS pharyngitis patients. If a person has been detected as suffering from GABHS pharyngitis, the first thing to do is to prevent the progression of the disease to RF by giving antibiotics. The antibiotics given are generally penicillin, but ampicillin and amoxicillin have also been shown to be quite effective for this preventive measure.[10]

The next step is to suppress the inflammatory response from the autoimmune response, especially for patients with early indications of RF. At this stage the treatment given is aspirin and steroids. What must be considered is that aspirin can cause liver and digestive disorders. So doctors must do close monitoring of both functions. [10] The next step is to provide therapy for the heart in

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patients who have reached the RHD stage. At this stage the treatment given is prednisone (as a substitute for aspirin), digoxin, diuretics, and conventional therapies such as regulation of fluid intake, oxygen, and bed rest. If heart failure gets worse, doctors will usually recommend surgery on the heart which generally aims to correct heart valve disorders.[10] in this case, the patient was given ceftriaxone which is one of the cephalosporin antibiotics of choice in the treatment of RHD. The patient was also given antibiotics in the form of azythromycin which is an option in the treatment of RHD.[3]

Furosemide is a diuretic that is useful in reducing edema but does not reduce the myocardium. Furosemide is given at a dose of 1 cc/h. Diuretics cause potassium excretion to increase so that in large doses or long-term administration additional potassium is needed. The combination of furosemide and spironolactone can be additive, increasing the diuresis effect, and because spironolactone is potassium-retaining, potassium administration is not necessary.[8]

In this case, the patient was given concor® (beta blocker group) which aims to reduce the risk of thromboembolism, control heart rhythm, and control heart rate.The patient was given simarc® to prophylaxis and treatment of venous thrombosis. And also patient was given KSR to electolite imbalance.[8] Not all patients with heart valve abnormalities undergo surgery, there are several indications for surgery including when congestive heart failure can no longer be treated, progressive cardiomegaly with symptoms, and pulmonary hypertension. The development of residual heart disease can be influenced by cardiac conditions at initial management, recurrence of rheumatic fever, and regression of heart disease.[8]

The prognosis worsens when the carditis symptoms are more severe, and it turns out that acute rheumatic fever with heart failure resolves 30% in the first 5 years and 40% after 10 years. The first attack may disappear in 10-25% of patients after 10 years from the initial attack.[18] Surgery is indicated in symptomatic patient that has severe valvular dysfunction. This not includes isolated severe MS, for which BMV may be prioritized. Both for the mitral and the aortic valves, the indications for surgical intervention are similar to nonrheumatic pathologies. Coincidence of tricuspid regurgitation is frequent in chronic rheumatic mitral valve disease, and often requires simultaneous surgery. There is evidence to support left atrial appendage removal during surgery in patients with atrial fibrillation, but indications are less established in patients with sinus rhythm, although the left atrial appendage is a potential substrate for thromboembolism.[19]

Dilemma for surgical option—repair versus replacement—arises and is especially relevant in young patients from low-income regions of the globe, who face significant challenges to be compliant with any form of medical therapy, such as ARF secondary prophylaxis and anticoagulation therapy. Valve replacement with either mechanical or biological prostheses has lower global survival and survival free from prosthetic valve complications because of higher rates of thromboembolic event in the former and a faster degenerative process years after. The greater difficulty with aortic valve repair and lower complications with aortic prostheses makes aortic valve replacement be more acceptable. [8]

Since the first reports by Carpentier, Duran, and others in the 1970s, rheumatic mitral valve repair has transformed significantly. Variable aspects of the technique have been perfected, and new procedures, such as artificial chordal implantation, have made this procedure more reproducible and standardized. The most of experienced surgeons have recently reported possiility of the repair in 75% of the patients and long-term survival superior to those after replacement of the valve. However, the repair of the rheumatic mitral valve is less durable than in nonrheumatic valves. Additionally, in RHD-endemic regions, the considerations may be the risk of needing reoperation given limited resources (and low probability of being able to get a second operation) and the expertise of surgical team for valve replacement than repair. In conclusion, valve replacement becomes the practice of choice in many places, especially for double-valve surgery, regardless of the need for lifetime anticoagulation.[16]

If the aortic valve repair is not feasible, considerate essentially between mechanical prostheses and bioprostheses. Both types of prostheses have a higher incidence of complications and there is no clear evidence of superiority comparation. One of the important factor in the decision against a mechanical valve is poor patient's

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compliance with anticoagulation, but bioprostheses degeneration may quickly progress that death may occur even before reoperation can be under taken. [13]

In LMICs where RHD is endemic, access to surgery remains one of the most important problems. Except in South Africa, cardiac surgery is independently performed, without visiting mission teams, in only a few countries. Continued efforts for care cost reduction are essential. The TTK Chitra prosthetic heart valve is one of example of a low-cost solution for those in LMIC. Low-cost valve rings and open heart surgery disposables need to be developed urgently to reduce the costs.[2]

CONCLUSION

A 25-years-old male patient, domiciled in Alue Dalam, Darul Aman, East Aceh, admitted to emergency department of Cut Mutia hospital, the patient was brought to the hospital by his family at 05.0o pm on January 27th, 2022. Patient referred from Graha Bunda hospital. Patient was admitted to the hospital with complaints shortness of breath. Shortness of breath is felt during activity and worsens at night. Shortness of breath since yesterday. The patient also complained of pounding. The pounding has been felt for five days before arriving at the hospital. The patient also has chest pain, and tired easily. Past medical history such hypertension and diabetes mellitus are denied.

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