JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : https://doi.org/10.14710/dmj.v11i6.35990
JKD (DMJ), Volume 12, Number 1, January 2023 :

CASE REPORT : A 52-YEARS OLD MALE PATIENT WITH AORTA REGURGITATION ET CAUSA RHEUMATIC HEART DESEASE IN CUT MEUTIA REGIONAL GENERAL HOSPITAL

Yuri Savitri[1], Jauza Raudhatul Jannah Mendrofa[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 : jauzaraudhatuljannah@gmail.com

ABSTRACT

Rheumatic heart disease (RHD) is an acquired heart disease with 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 valves lungs. A 52-year-old male patient, domiciled in Mutiara (Alue Awe) Lhokseumawe City, Indonesia. Come for outpatient treatment at Polyclinic of Cut Meutia Hospital. Patients are who routinely go to the polyclinic every month to take medicine. The patient complains of left chest pain, shortness of breath, heart palpitations, cough, tired easily during activities, especially during strenuous activities. History of Diabetes Mellitus is denied, history of hypertension (+). The patient admitted that he was diagnosed with rheumatic heart disease at the age of 19 years. The patient has been undergoing treatment for 33 years by routinely complete checks related to his heart every year and regularly taking medication at the polyclinic every month. The patient admitted at junior high school age he often experienced attacks of fever that went up and down accompanied by pain in the throat. Physical examination found: Compos Mentis (E4V5M6), BP:140/40 mmHg, HR : 92x/i, RR: 23x/i, T; 37.1°C, SpO2 : 95%. Chest examination showed vesicular breath sounds, Rhonki (-), Wheezing (-), Diastolic murmur (+).Echocardiography showed an EF of 70%. AR Severe, MR Moderate. AML Prolapse, Calcification (+), LV Dilatation, LVH Eccentric

Keywords: *rheumatic heart disease; Aorta regurgitation*

INTRODUCTION

Rheumatic heart disease (RHD) is an acquired heart disease which 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 valves lungs. Rheumatic fever is a multisystem collagen vascular disease that occurs after *group A Streptococcus* infection in individuals with predisposing factors. Cardiovascular involvement in this disease is characterized by inflammation of the endocardium and myocardium through an 'autoimmune' process that causes tissue damage. Autoimmune is aberration in the body's normal development such that the immune system mounts an attack against its own cells.[1]

Acute rheumatic fever is caused by an immunologic response that occurs as a sequel to group A streptococcal infection of the pharynx but not of the skin. Autoimmune reactions to streptococcal infection will hypothetically cause tissue damage or manifestations of rheumatic fever. The damage to this tissue will cause inflammation of the lining of the heart, especially the endothelial valve.[2]

Rheumatic heart disease can lead to stenosis or insufficiency or both. Rheumatic heart disease (RHD) causes damage to the heart valves that occurs after an episode of acute rheumatic fever (ARF). Every year an average of 55 cases are estimated with acute rheumatic fever (ARF) and RHD. It is estimated that the prevalence of RHD in Indonesia is 0.3-0.8%. Worldwide ARF is estimated to occur in 5-30 million. 90,000 of them will die every year. The mortality of this disease in the world is 1-10%. Fever is rare before the age of 5 years and after the age of 25 years, most commonly found in children and young adults. The highest incidence is in children aged 5-15 years and in undeveloped or developing countries where antibiotics are not routinely used for the treatment of pharyngitis.[3]

More than 60% of rheumatic fever will progress to rheumatic heart disease. As the basis of rheumatic heart disease, rheumatic fever in the pathogenesis of the disease is influenced by several factors. There are several factors that play a role in the pathogenesis of rheumatic fever , including organism factors, host factors and immune system factors. *Group A beta haemolyticus Streptococcus* bacteria as an infectious organism has an important

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role in the pathogenesis of rheumatic fever . These bacteria often colonize and proliferate in the throat area, where these bacteria have supra-antigens that can bind to major histocompatibility complex (MHC) class 2 which will bind to T cell receptors which when activated will release cytokines and become cytotoxic. The Supra-antigen of *group A beta-hemolytic Streptococcus* bacteria involved in the pathogenesis of rheumatic fever is M protein, which is a *Streptococcus pyogenic exotoxin*. In addition, group *A beta-hemolytic Streptococcus* bacteria also produce extracellular products such as streptolysin, streptokinase, DNA-ase, and hyaluronidase that activate the production of autoreactive antibody numbers. The most common antibody is antistreptolysin-O (ASTO) whose purpose is to neutralize these bacterial toxins. However, simultaneously the body's protective efforts also cause pathological damage to the body's own tissues. The body has a structure that is similar to the *group A beta haemolyticus Streptococcus* bacteria antigen so that cross-reactivity occurs between the epitope of the organism and the host which will lead to tissue damage.[4]

The similarities between *group A beta haemolyticus Streptococcus* bacteria antigens with body tissues recognized by antibodies are: 1) identical amino acid sequences, 2) homologous but not identical amino acid sequences, 3) epitopes on different molecules such as peptides and carbohyARFtes or between DNA and peptides. The affinity of cross-reacting antibodies can be different and strong enough to cause cytotoxicity and induce cell surface receptor antibodies. Epitopes located in cell walls, cell membranes, and M protein of group A beta haemolyticus streptococci have the same immunological structure as myosin, tropomyosin, keratin, actin, laminin, vimentin, and N-acetylglucosamine proteins in the human body. This similar molecule forms the basis of the autoimmune reaction that leads to rheumatic fever . Another relationship of laminin which is a protein similar to myosin and M protein found in the cardiac endothelium and recognized by anti-myosin T cells and anti-M protein, causes an autoimmune reaction to the heart valves because the body misrecognizes and rheumatic heart disease occurs.[5]

Rheumatic heart disease often causes stenosis or regurgitation. One of the second highest incidences besides the mitral valve is the incidence of stenosis or regurgitation of the aortic valve. Aortic valve regurgitation is a leak in the aortic valve that occurs whenever the ventricles relax. Regurgitation is determined by the presence of aortic valve incompetence where a portion of the heart volume from the left ventricle flows back into the ventricular space during diastole. Aortic regurgitation is the flow of blood in the diastolic phase from the aorta back to the left ventricle. Diastolic reflux across the aortic valve can cause left ventricular volume overload.[6]

Acute aortic regurgitation results in an increase in the end-diastolic volume of the left ventricle. Because of the increase, the left ventricle does not have time to dilate in response to the increase in volume. As a result, the final left ventricular volume increases rapidly, resulting in increased pulmonary venous pressure and impaired coronary blood flow. Increased pressure in the pulmonary veins causes an increase in pressure in the pulmonary circulation, this then develops into complaints of shortness of breath and pulmonary edema. In severe cases, heart failure may occur, followed by cardiogenic shock. Decreased myocardial perfusion can lead to myocardial ischemia.[7]

Chronic aortic regurgitation by increased left ventricular volume and left ventricle compensate for the increased volume with use and hypertrophy. Left ventricular dilatation will changes in to sarcomere of the heart muscle to become longer due to the left ventricle being larger and able to accommodate the excess end-diastolic volume due to aortic regurgitation. Hypertrophy is also important to support the increase in ventricular wall pressure as a result of ventricular dilatation itself.[6]

CASE REPORT

A 52-year-old male patient, domiciled in Mutiara (Alue Awe) Lhokseumawe City, Indonesia. Come to Polyclinic of Cut Meutia Hospital. Patient was routinely go to the polyclinic every month to take medicine. The main complains of the patient is left chest pain, shortness of breath, heart palpitations, cough, tired easily during activities, especially during strenuous activities. History of Diabetes Mellitus is denied, the patient have a history of hypertension. The patient admitted that he was diagnosed with rheumatic heart disease at the age of 19 years. The

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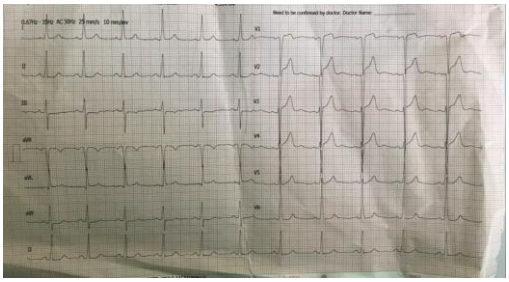

Figure 1. ECG Findings

Electrocardiogram obtained at admission showed, sinus rhythm, heart rate 75 times pe minute , regular, Normal P wave axis: P waves are perpendicular in leads I and II, inverted in aVR, PR interval remains constant, QRS complex is normal, ST elevation was found in V1-V4 which indicates OMI (Old Myocardial Infarction), and S in V3 + R AVL >28 (men), R in V5 + S in V1 >35, Cornell criteria and Sokollow lion criteria indicate LVH (Left Ventricular Hypertrophy).

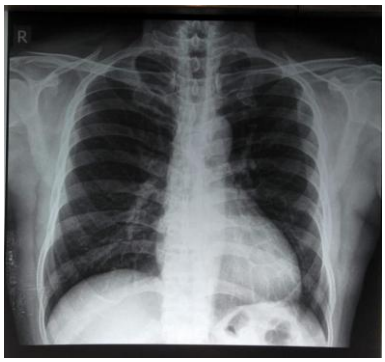

Figure 2. Chest x-ray

Chest X-ray showed cardiomegaly, normal pulmonary, negative heart waist, negative congestion, negative infiltrate. Conclusion: Cardiomegaly and aortic dilatation

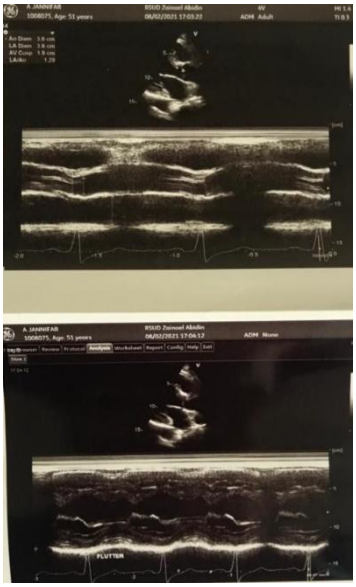

Figure 3. Transthoracic echocardiogram

Echocardiography showed an EF of 70%. AR Severe, MR Moderate. AML Prolapse, Calcification (+), LV Dilatation, LVH Eccentric. Patients are given follow-up treatmentRoutine at the Cut Meutia Hospital Candesartan 1x8mg tab, Bisoprolol Fumarate 1x2,5mg tab, Omeprazole 2x20mg caps.

DISCUSSION

In a case report, a 52-year-old male patient is a routine outpatient diagnosed with rheumatic heart disease for the first time at the age of 19 years. Rheumatic fever is an autoimmune disease that attacks multisystems due to infection with group A beta hemolytic streptococci in the pharynx (pharyngitis) which usually attacks children and young adults. The most important etiology of rheumatic heart disease is rheumatic fever. The peak incidence of rheumatism is in the 5-25 year group. Although fever can affect anyone, however, fever follows attacking children and young adults. Rarely affects children less than 4 years of age and more than 25 years of age. In the past, heart disease often affect the female sex, and along with many studies which state that there is no significant difference

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between the sexes of women and men. RHD is often also found in the male sex.[8]

The patient complains of left chest pain, shortness of breath, heart palpitations, cough, tired easily during activities, especially during strenuous activities. Rheumatic heart disease is an acute or chronic inflammatory disease which is an autoimmune reaction known to Group A Beta *Streptococcus Haemolyticus* infection whose mechanism of travel is not yet known. The damage to this tissue will cause inflammation of the lining of the heart, 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 aortic valve leaflets and will experience aortic regurgitation. This condition forces the heart to work harder so that the leaked blood flows back and passes through the left ventricle. Over time, the walls of the ventricles will thicken (hypertrophy). The blood pumping process to be ineffective. This will widen the heart cavity which in its natural course will cause congestive heart failure.[9]

Shortness of breath is caused by pulmonary congestion or fluid in the interstitial spaces and alveoli of the lungs. This fluid will inhibit the expansion of the lungs so that it becomes difficult to breathe. The cause is the supply of oxygen to the myocardium which has decreased which results in the death of heart cells. In addition, blood will collect and accumulate in the lungs (congestion). congestion causing shortness of breath and coughing. As a result, the air sacs as a place for the exchange of oxygen and carbon dioxide can be filled with fluid, thus interfering with lung function. Fatigue is caused by reduced oxygen and blood flow to the brain and muscles. The heart can't have enough blood to meet the body's needs for tissues.[10]

The patient admitted that when he was in junior high school he often experienced attacks of fever that went up and down accompanied by pain in the throat. Group A beta haemolyticus *Streptococcus* infection in the throat always prepares for rheumatic fever, both in the first attack and in repeated attacks. Group A streptococci must cause pharyngeal infection, not just superficial colonization. Rheumatic fever is an auto immune response to group A hemolytic streptococcal infection in the throat. The response of clinical manifestations and the degree of disease that occurs is determined by the sensitivity of the genetic host, the malignancy of the organism and the conducive environment.[11]

Physical examination obtained: Compos Mentis (E4V5M6), BP: 140/40 mmHg, HR: 92 times pe minute, RR: 23 times pe minute, T; 37.1°C, SpO2 : 95%. Chest examination showed vesicular breath sounds, diastolic murmur, there are no rhonki and wheezing. A brief mechanism of rheumatic heart disease is damage to the heart valves. Because there is no aortic valve during diastole, the blood in the aorta, which is normally high pressure, will flow into the left ventricle, so the left ventricle must overcome both of which is sending blood that is normally received from the left atrium and blood returning from the aorta. The left ventricle then dilates and hypertrophies to accommodate this increased volume, as well as a more than normal urge to control blood, causing the systolic pressure to rise. The cardiovascular system tries to compensate by reflex dilating blood vessels and the peripheral arteries relax so that peripheral resistance decreases and diastolic pressure drops ARFmatically.[12]

On auscultation of a patient with mild or moderate chronic aortic regurgitation, a high-frequency, decrescendo diastolic murmur is present, usually audible in the third or fourth interspace at the left sternal border [12]. Sudden cardiac death occurs less frequently (<0.2% annually) in asymptomatic patients with relatively good left ventricular function.[13]

Based on the diagnosis, the patient included rheumatic heart disease in which 1 major and 3 minor criteria were found *Group A hemolytic Streptococcus B haemolyticus* evidence of previous infection with valvular abnormalities. Aortic regurgitation found by echocardiography as a result of previous Streptococcal infection.

Rheumatic heart disease in this patient experienced complications in the form of Cardiac Heart Failure (CHF). CHF Criteria based on the Framingham Criteria diagnosis. Major criteria: Paroxysmal nocturnal dyspnea, Neck vein distention, Increased jugular vein, Cardiomegaly rhonchi, Acute pulmonary edema, Gallop III heart sound, Positive hepatojugular reflux, Weight loss >4.5 kg in 5 days of therapy. Minor criteria: Edema of extremities, Night cough, Shortness of breath on activity, Hepatomegaly, Pleural effusion Vital capacity

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reduced by 1/3 of normal Tachycardia (>120 beats per minute).[14]

Electrocardiogram examination showed sinus rhythm, heart rate 75 times pe minute, regular, Normal P wave axis: P waves perpendicular in leads I and II, inverted in aVR. PR interval remains constant. Normal QRS complex. There was ST elevation in V1-V4 indicating Anteroseptal STEMI. And S in V3 + R AVL >28 (male), R in V5 + S in V1 >35. Cornell's Criteria and Sokollow's Lion Criteria show LVH (Left Ventricular Hypertrophy). Aortic regurgitation can cause left ventricular hypertrophy. Hypertensive states cause cardiac hypertrophy which is an independent risk factor for myocardial infarction and can lead to major cardiovascular events. ST segment elevation is an abnormality detected on a 12-lead ECG. STEMI (ST Elevation Myocardial Infarction) is one type of heart attack in the form of coronary arteries in total so that the heart muscles do not get an oxygen supply. This heart muscle damage has implications for the electrical function of the heart not working properly, so that typical signs of heart disease are obtained on the ECG picture. The backflow of blood caused by aortic regurgitation can also cause vascular conditions. The left ventricle is severely dilated and eventually hypertrophied, resulting in a spherical shape. The increased tensile strength of the ventricular wall allows an increase in diastolic volume without an abnormal increase in pressure.[15]

Chest X-ray showed cardiomegaly, normal lungs, negative heart waist, negative congestion, negative infiltration. Conclusion: Cardiomegaly and aortic dilatation. Group A streptococci will cause pharyngeal infections. Streptococcal antigen will cause the formation of antibodies in the host. These autoantibodies react with tissue tubes, causing tissue damage. In aortic regurgitation, the aortic valve cannot close tightly, resulting in an increase in volume load. Blood that will be ejected throughout the body returns to the left ventricle. Incomplete closure of the aortic valve can result from cusp abnormalities, aortic abnormalities, or trauma. Diastolic reflux through the aortic valve can cause left ventricular volume overload, giving the appearance of cardiomegaly. This valve defect also causes dilatation or tearing of the aorta.[16]

Echocardiography showed an EF of 70%. Severe AR, Moderate MR. AML Prolapse, Calcification (+), LV Dilation, Eccentric LVH. Rheumatic fever is a continuation of pharyngeal infection caused by group A beta hemolytic streptococci. Autoimmune reactions to Streptococcal infection hypothetically will cause tissue damage or manifestations of rheumatic fever. The damage to this tissue will cause inflammation of the lining of the heart, especially the endothelial valve. RHD conditions often cause valve stenosis. At the same time, mitral stenosis can cause mitral regurgitation which will increase the left ventricular volume load and cause left ventricular dilatation and hypertrophy. In other places still on the aortic valve will occur fibrosis of the aortic valve leaflets. There will be aortic regurgitation.[17]

There are specific criteria for Doppler findings in rheumatic valvulitis: 1) mitral regurgitation (four): seen on 2 views, jet length 2 cm, peak velocity >3 m/s, pansystolic; and 2) Aortic regurgitation (fourth): seen on 2 views, beam length 1 cm, peak velocity >3 m/s, pandiastolic.[2]

The patient was given regular follow-up treatment at the Cut Meutia Hospital Candesartan 1x8mg tab, Bisoprolol Fumarate 1x2,5mg tab, Omeprazole 2x20mg caps. The primary goal of medical therapy is to reduce the systolic hypertension associated with chronic AR, thereby reducing stress on the ventricular wall and improving left ventricular function. Therapeutic options that can be used include ACE inhibitors, ARBs, or Ca inhibitors. ACE inhibitors are also an option where there is left ventricular dysfunction. ACE inhibitors are heart disease medications that can widen blood vessels. The PPI group is given because of complaints of nausea that are often experienced by patients, either due to the use of other drugs or not. However, there are indeed several classes of antihypertensives that more often cause dyspepsia/ulcer due to intolerance, especially the ACEI group (captopril, lisinopril) and ARb (valsartan, losartan).[18]

Rheumatic fever without carditis At least 5 years after the last attack or until 18 years of age Rheumatic fever with carditis without evidence of residual heart disease/valvular disease. At least up to 10 years after the last attack or up to 25 years, whichever is the longest, acute rheumatic fever with carditis and residual heart disease (persistent valvular disease) At least 10 years since the last episode or at least up to age 40, and sometimes life after valve

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operation lifetime [2]. Indications for surgery for aorta regurgitation et causa RHD. Patients with symptoms of severe aortic regurgitation (AR) should be referred for surgery, Asymptomatic patients with decreased systolic function (LVEF <50%)

For patients with normal asymptomatic LV systolic function, surgery should be delayed as long as possible. Surgery should be considered in asymptomatic patients with severe AR and maintaining left ventricular systolic function with severe left ventricular dilatation (Adults: LVEDD > 70 mm, LVESD > 50 mm. Operation options is mechanical valve replacement, bioprosthetic valve replacement, aortic valve repair, homograft valve replacement, ross procedure.[2]

CONCLUSION

Reported male, 52 years old. Aortic Regurgitation et causa Rheumatic Heart Disease. The patient complains of left chest pain, shortness of breath, heart palpitations, cough, tired easily during activities, especially during strenuous activities. History of DM denied, history of hypertension (+). The patient admitted that he was diagnosed with rheumatic heart disease at the age of 19 years. The patient has been undergoing treatment for 33 years by routinely undergoing complete checks related to his heart every year and regularly taking medication at the polyclinic every month. The patient admitted that at school age (SMP) he often experienced attacks of fever that went up and down accompanied by pain in the throat.

Physical examination found: Compos Mentis (E4V5M6), BP: 140/40 mmHg, HR: 92 timer per minute, RR: 23 time per minute,, T; 37.1°C, SpO2 : 95%. Chest examination showed vesicular breath sounds, diastolic murmur, no rhonki and wheezing. Electrocardiogram examination showed sinus rhythm, heart rate 75 time per minute, regular, Normal P wave axis: P waves perpendicular in leads I and II, inverted in aVR. PR interval remains constant. QRS complex is normal. ST elevation was found in V1-V4 which indicates OMI (Old Myocardial Infarction). And S in V3 + R AVL >28 (men), R in V5 + S in V1 >35. Cornell criteria and Sokollow lion criteria indicate LVH (Left Ventricular Hypertrophy).

Chest X-ray showed cardiomegaly, normal pulmonary, negative heart waist, negative congestion, negative infiltrate. Conclusion: Cardiomegaly effect and aortic dilatation. Echocardiography showed an EF of 70%. AR Severe, MR Moderate. AML Prolapse, Calcification (+), LV Dilatation, LVH Eccentric. The patient was given routine follow-up treatment at the Cut Meutia Hospital 8 mg candesartan, 2,5mg bisoprolol fumarate, and 20 mg omeprazole twice a day.

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