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Case Report

Rheumatic Heart Disease Causes Heart Failure: How is It Treated?

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ABSTRACT

Rheumatic heart disease is the leading cause of heart failure in school-age children, affects 30 million people worldwide, and is still prevalent, especially in developing countries. Rheumatic heart disease was the result of untreated acute rheumatic fever. Knowledge of the proper management should be encouraged in those with rheumatic fever to halt the progression of cardiac damage that can lead to heart failure. This article discusses one such case. **Case Summary.** A 13-year-old girl complained of breathlessness during the activity, which improved with rest. She had a history of multiple joint pain and recurrent upper respiratory infection, which was not treated with antibiotics. On physical examination, the blood pressure was 90/60 mmHg, heart rate 128 bpm, facial and palpebral swelling, and the jugular vein pressure increased 5+3 cm H2O. The heart sound was S1>S2, regular with gallop and murmur in mitral, aortic, pulmonary, and tricuspid valves. Hepatomegaly and swelling of both lower extremities were discovered. Laboratories tests found ASTO 400 IU/ml. Chest X-Ray showed cardiomegaly. Echocardiography showed the regurgitation of mitral, aortic, tricuspid, and pulmonary valves. **Discussion.** The patient was diagnosed with heart failure fc. NYHA II *ec* rheumatic heart disease and treated with penicillin benzathine 1.2 units, furosemide injection 30 mg b.i.d, spironolactone 25 mg b.i.d, tenace 5 mg q.d, and prednisone 5-4-4 mg t.i.d. Adequate management of acute rheumatic fever can reduce the recurrence, prevent rheumatic heart disease and cardiac deterioration, and improve quality of life.

Highlights:

1. Rheumatic heart disease is among a leading cause of heart failure in school-age children.
2. It discusses how to proper manage rheumatic heart damage.

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Introduction

Heart failure is a complex clinical syndrome caused by disruption of the structure and function of the heart; it affects the ability of the heart to pump blood according to the body's needs. Heart failure results from ventricular dysfunction and volume or pressure overload, alone or in combination, and involves circulatory, neurohormonal, and molecular abnormalities[1]. Heart failure in children can be caused by congenital heart disease and acquired heart disease. It is estimated that 15-25% of children with congenital heart disease will experience heart failure later in life. The incidence of heart failure in school-age children is mainly caused by rheumatic heart disease, which is one example of acquired heart disease.[2,3]

Rheumatic heart disease is a sequelae of rheumatic fever characterized by abnormalities or disabilities in the heart valves. Every year, ± 300,000 cases of rheumatic heart disease are obtained. The high incidence rate in developing countries is related to predisposing factors in the form of a family history of having suffered from rheumatic fever, low socioeconomic status, throat infections that are not treated or slow handling, inadequate health services, poor sanitation, crowded environments, and industrialization and urbanization[4]. Rheumatic heart disease impacts 30 million individuals worldwide. It was estimated in 2015 to be responsible for 305 000 deaths and 11.5 million disability-adjusted life years lost. 60% of these deaths occurred prematurely (before 70), though these figures are highly uncertain due to incomplete data in many countries. Despite the availability of effective prevention and treatment measures, the contribution of rheumatic heart disease to overall global mortality has remained stable between 2000 and 2015. The African, South-East Asia, and Western Pacific regions are the worst affected, accounting for 84% of all prevalent cases and 80% of all estimated deaths from rheumatic heart disease in 2015.[5] In Indonesia, the prevalence of rheumatic heart disease was 0.3-0.8 per 1,000 population between 1981 and 1990. Indonesia was classified as endemic based on data from the Global, Regional, and National Burden of Rheumatic Heart Disease from 1990 to 2015, but the prevalence data was unclear. Rheumatic heart disease primarily affects children between the ages of 5-15 years, with a peak of incidence at the age of 8 years.[2,6]

Sequelae and progressive heart valve deformities of rheumatic fever can lead to chronic manifestations or even death. Rheumatic heart disease was the result of untreated acute rheumatic fever. Due to the persisting world burden of this disease, the author wants to declare one such case with the proper management in this manuscript.[7]

Case Presentation

A 13-year-old girl (weight 32 Kg) complained of breathlessness during activity, such as walking ±10 meters, which improved with rest. The

breathlessness has been accompanied by a feeling of fatigue and a dry cough for the last 1 month. The patient should sleep using 2 pillows to alleviate breathlessness. The patient also complained of swollen face and legs and palpitations in the last 1 week ago. There were no complaints of chest pain. The patient complained of a low-grade fever that is relieved by an antipyretic. The patient had a history of joint pain ± 1 year ago; the pain was felt moving around, starting with the shoulder joint and then the knee and ankle joints. In addition, the patient had a recurring upper respiratory infection in the last 4 years, which was not treated with antibiotics.

Physical examination revealed compos mentis, blood pressure 90/60 mmHg, heart rate 128 bpm, respiratory rate 24 times per minute, and axillary temperature 37.6 ºC. The head is normocephalic, with facial and palpebral swelling, jugular vein pressure increased 5+3 cm H2O. Examination of the lungs found no abnormalities. On cardiac examination, there was visible ictus cordis, palpable ictus cordis on ICS V of the left anterior-axillary line, dim percussion and dilated heart margin, auscultation for heart sound S1>S2, regular, gallop and murmur in the mitral, aortic, tricuspid, and pulmonary valve. The abdominal examination looked flat and symmetrical, with palpable liver 4 cm below the right costal margin and 3 cm of the *xypoideus processus* with the tender consistency, blunt edge, and tenderness; the spleen was not palpable tympanic percussion and bowel sound auscultation within normal limits. Examination of the upper and lower extremities found swelling in both lower extremities without being accompanied by cyanosis. According to WHO Chart Standard 2006, she was malnourished.

Laboratories tests found hemoglobin 10,6 g/dL (normal range: 12,0-15,0 g/dL), hematocrit 33% (normal range: 37-47%), erythrocyte 4,1x10^6/mm^3 (normal range: 4,2-5,4 x10^6/mm^3), WBC 8,7x10^3/mm^3 (normal range: 4,5-10,5 x10^6/mm^3), platelet count 117x10^3/mm^3 (normal range: 150-450 x10^6/mm^3), the electrolyte serum was within the normal limits except potassium 3,3 mmol/L (normal range: 3,7-5,4 mmol/L), and ASTO 400 IU/ml (normal range: < 200 IU/ml). Chest X-Ray showed a cardiothoracic ratio of >50%, electrocardiography (ECG) revealed normal sinus rhythm, and echocardiography showed *situs solitus*, concordant AV-VA, normal systemic and pulmonary vein's orifice, mitral valve regurgitation, aortic valve regurgitation, tricuspid valve regurgitation, and pulmonary valve regurgitation. The left ventricle's systolic and diastolic function was within normal limits (Figure 1).

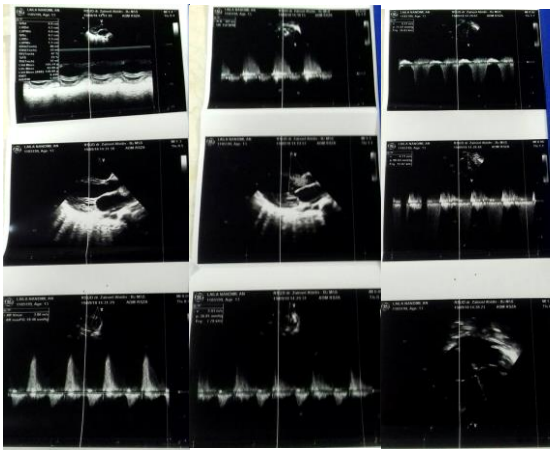

Figure 1. Echocardiography

The patient was diagnosed with congestive heart failure fc. NYHA II *ec* rheumatic heart disease treated with stopper, 1.700 kcal diet with 35 grams of protein divided into three times meals and two times snacks a day, penicillin benzathine intramuscular injection 1.2 units, furosemide intravenous injection 30 mg b.i.d, spironolactone 25 mg b.i.d, tenace 5 mg q.d, paracetamol 500 mg t.i.d, prednisone 5-4-4 mg t.i.d, and laxative syrup CI t.i.d. In this case, the author did not follow up evaluation regarding the patient's clinical outcome following the medication provided by clinicians due to a short time being a co-assistant in the cardiology department.

Discussion

Congestive heart failure diagnosis is based on anamnesis, physical examination, and supporting analysis. Based on anamnesis, patient's complaints of dyspnea are the most common manifestation of heart failure. Dyspnea is caused by increased respiratory work caused by pulmonary vascular congestion, which reduces lung flexibility. This results in O2 and CO2 exchange disorders in the alveolus. The patient complained of breathlessness during activity (dyspnea d'effort) and lying down (orthopnea), so the patient should sleep with 2-3 pillows. This is due to the redistribution of blood flow from the body's peripheral areas to the central circulation. Reabsorption of interstitial fluid from the peripheral will also cause further vascular congestion of the lungs, while the onset of interstitial pulmonary edema triggers paroxysmal nocturnal dyspnea. Dyspnea, orthopnea, and paroxysmal nocturnal dyspnea are manifestations of left heart failure. In addition to breathlessness, patients also complain of a nonproductive cough due to pulmonary congestion. The onset of rales caused by transudation of pulmonary fluid is a characteristic symptom of heart failure. Rales was initially heard in the basal lungs due to the influence of gravitational forces. However, this patient's thorax physical examination results are no longer found in the presence of rales in auscultation due to the administration of diuretics in such patients.[8]

Left heart failure can trigger right heart failure because a dam in the vascular lung causes resistance to the blood flow from the right ventricle, so the right heart cannot adjust to increased venous backflow to the right atrium. Right heart failure gives rise to symptoms and signs of systemic venous congestion. This can be observed through the results of physical examination in this patient, namely an

increase in jugular venous pressure and a dam in the central vein that causes hepatomegaly. Positive tenderness is caused by stretching of the liver capsule. If manual compression is performed on the right abdomen, there will be an increase in jugular venous pressure which indicates positive hepatojugular reflux. Systemic venous congestion leads to fluid build-up in the interstitial space, then peripheral edema appears, characterized by swelling of the legs and face.[8]

Clinical manifestations of heart failure should be considered by looking at the degree of activity that can give rise to symptoms. At first, symptoms appear only during physical exertion. Nevertheless, with the increase in weight of heart failure, tolerance to exercise decreases, and symptoms appear earlier with less activity. According to the New York Heart Association (NYHA), heart failure can be divided into four classifications. Based on the anamnesis, this patient had NYHA degree II heart failure because the patient complained of breathlessness while performing regular activities (Table 1).[9]

Table 1. New york heart association (NYHA) classification of heart failure[9]

Functional capacity	Limitation
Class I Ordinary physical activity does not cause undue fatigue, dyspnea, or palpitations	None
Class II Ordinary physical activity causes fatigue, dyspnea, palpitations, or angina	Mild
Class III Comfortable at rest; less than ordinary physical activity causes fatigue, dyspnea, palpitations, or angina	Moderate
Class IV Symptoms occur at rest; any physical activity increases discomfort	Severe

Examination of the arterial pulse during heart failure shows a rapid and weak rate. Rapid pulsation (tachycardia) reflects the response to sympathetic nerve stimuli. The severe decrease in stroke volume and the presence of peripheral vasoconstriction lower the pulse pressure resulting in a weak pulsation. Systolic hypotension is found in more severe heart failure. In addition, in severe left ventricular failure, alternans pulses can arise, namely changes in the strength of the arterial pulse. Alternans pulses show severe mechanical dysfunction with repeated variations in pulsation to pulsation at a stroke volume. A ventricular gallop or a third heart sound (S3) is often obtained in auscultation. The sound of S3 in auscultation is

characteristic of left ventricular failure. Ventricular gallop occurs during the initial diastolic and is caused by the rapid filling of the ventricle that is not pliable (distension).[8]

Supporting analysis to establish a diagnosis of heart failure includes routine blood tests, chest X-Ray, electrocardiography (ECG), and echocardiography. The results of routine blood examination in this patient showed anemia characterized by a decrease in hemoglobin from normal values of 12.0-15.0 gr/dl to 10.6 gr/dl. Anemia is one of the causes or complications of heart failure. Anemia can occur due to excessive production of cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which can reduce the secretion of erythropoietin (EPO). Anemia can also worsen heart function due to increased stroke volume, reduced blood flow to the kidneys, and water retention resulting in increased heart workload[2]. Changes in electrolyte levels can also occur in patients with heart failure, namely dilution hyponatremia and potassium levels that can be normal or decreased due to diuretic therapy[8]. Potassium values in this patient were only 3.3 mmol/L compared to normal values of 3.7-5.4 mmol/L, indicating hypokalemia's presence.

Chest X-Ray was performed to see pulmonary congestion that could develop into pulmonary edema in more severe heart failure, vascular redistribution of the upper lobes of the lungs, and cardiomegaly. Chest X-Ray in this patient shows cardiomegaly, but there is no congestion or pulmonary edema. This can be influenced by the administration of diuretics so that pulmonary congestion can be reduced. Electrocardiography can help determine the type of defect, tachycardia sinuses, atrial enlargement, and ventricular hypertrophy. ECGs often show asymptomatic ventricular premature pulsation and become non-sustained ventricular tachycardia. Bradycardia is usually associated with a progressive worsening of heart failure.[8] Echocardiography can describe the structure of the heart, pressure data, and functional status of the heart so that it can know the enlargement of the heart chambers and the etiology of heart failure. Echocardiography examination in this patient shows the presence of regurgitation of all heart valves. Aortic regurgitation, pulmonary regurgitation, tricuspid regurgitation, and mitral regurgitation cause congestive heart failure in this patient. Valvular heart disease in children is most often caused by rheumatic fever, where valve dysfunction will improve the work of the heart. Valve regurgitation will force the heart to pump more blood to replace the amount of blood flowing back, thereby increasing the volume of work of the heart. A typical myocardial response to an increase in the volume of work is space dilatation and muscle hypertrophy. Dilatation of the myocardium and hypertrophy are compensatory mechanisms to increase the heart's pumping ability.[8]

Mitral regurgitation reverses blood flow from the left ventricle to the left atrium due to imperfect valve closure. During systolic, the ventricle pumps blood to the aorta. However, some of the blood returns to the left atrium, so the work of the left ventricle and the left atrium must be improved to maintain cardiac output. The additional volume load generated by the valve undergoing regurgitation will result in ventricular dilatation. According to Starling's law on the heart, dilatation of the ventricular wall will increase the contraction of the myocardium, which eventually makes the ventricular wall hypertrophy. Regurgitation creates a volume load not only for the left ventricle but also for the left atrium. The left atrium, dilated to accommodate the increased volume due to the blood pumped by the ventricle, will partially return to the atrium due to valve regurgitation. Therefore, in auscultation, there will be murmurs along the systolic phase called the pansystolic murmur or holosystolic murmur. Aortic regurgitation causes the return of blood from the aorta into the left ventricle during ventricular relaxation. This obviously puts a fairly heavy volume load on the left ventricle. At each contraction, the ventricle should be able to expel a certain amount of blood equal to the normal volume coupled with the regurgitation volume. The filling and emptying of the arterial pulse will take place quickly, and the pulse pressure may dilate due to increased systolic pressure and decreased diastolic pressure. On the auscultation will be heard diastolic murmurs, typical Austin Flint noise, and systolic ejection clicks caused by an increase in ejection volume. Tricuspid regurgitation is usually caused by advanced left heart failure or severe pulmonary hypertension resulting in a deterioration in the function of the right ventricle. As the right ventricle fails and enlarges, a functional regurgitation of the tricuspid valve occurs. Findings in tricuspid regurgitation were related to noise along systole in auscultation, on the ECG found pulmonary P waves when sinus rhythms were normal, atrial fibrillation, and right ventricular hypertrophy. Functional pulmonary regurgitation can occur as sequelae of left-sided valve dysfunction with chronic pulmonary hypertension and pulmonary valve oriphysium dilatation; however, lesions of the pulmonary valve are rare.[8]

The Framingham Criteria can be used to diagnose congestive heart failure by identifying two major criteria or one major criterion plus two minor criteria. In this patient found four major criteria (Table 2): cardiomegaly, hepatojugular reflux, gallop S3, and increased jugular venous pressure, as well as four minor criteria: edema of the extremities, cough, dyspnea d'effort, and hepatomegaly.[10]

Table 2. Framingham diagnostic criteria[10]

Major criteria	Minor criteria
Acute pulmonary edema	Ankle edema
Cardiomegaly	Dysnea on exertion
Hepatojugular reflux	Hepatomegaly
Neck vein distention	Nocturnal cough
Paroxysmal nocturnal dyspnea or orthopnea	Pleural effusion
Pulmonary rales	Tachycardia (heart rate greater than 120 bpm)
Third heart sound (S3 gallop)	A decrease in vital capacity by one third maximal value recorded
Weight loss of 4.5 kg or more in 5 days in response to treatment	
Central venous pressure greater than 16 cm of water	
Radiographic cardiomegaly	

Congestive heart failure in the patient is caused by rheumatic heart disease resulting from untreated acute rheumatic fever. Rheumatic fever is established based on evidence of previous Streptococcus infection and the presence of 2 major manifestations or one major manifestation coupled with two minor manifestations of JONES Criteria. In this patient, two major are present, namely carditis and polyarthritis migrans (Table 3)[11]. Carditis is distinguished by the development of a murmur as a result of valve abnormalities, cardiomegaly, and congestive heart failure. Polyarthritis migrans is characterized by a history of pain in two or more joints as well as the presence of Streptococcus Group A infection in the previous 45 days as evidenced by an increase in *Anti-Streptolysin O*(ASTO) titers of up to 400 IU / ml.[12]

Table 3. Revised jones criteria (2015)[11]

Major criteria	
Low risk popultion	High risk population
Carditis (clinical or subclinical)	Carditis (clinical or subclinical)
Arthritis – only polyarthritis	Arthritis – monoarthritis or polyarthritis
Chorea	Polyarthralgia
Erythema marginatum	Chorea
Subcutaneous nodules	Erythema marginatum
	Subcutaneous nodules
Minor criteria	
Low risk population	High risk population
Polyathralgia	Monoarthralgia
Hyperpyrexia (≥38.5º)	Hyperpyrexia (≥ 38.0ºC)
ESR ≥ 60 mm/h and/or CRP ≥ 3.0 mg/dl	ESR ≥ 30 mm/h and/or CRP ≥ 3.0 mg/dl
Prolonged PR interval (after taking into account the differences related to age; if there is no carditis as a major criterion)	Prolonged PR interval (after taking into account the differences related to age; if there is no carditis as a major criterion)

ESR, erythrocyte sedimentation rate; CRP, C-reactive protein

The management given to this patient includes once 1.2 million units of penicillin benzathine intramuscular injection, furosemide intravenous

injection 30 mg b.i.d, spironolactone 25 mg b.i.d, tenace 5 mg q.d, paracetamol 500 mg t.i.d, prednisone 5-4-4 mg t.i.d, and laxative syrup CI t.i.d. The management carried out on this patient is appropriate where the antibiotic penicillin benzathine given to children weighing > 27 kg is 1.2 million units by intramuscular. Secondary prophylactic administration of penicillin benzathine intramuscular injection should be done regularly every 3-4 weeks for 10 or up to 40 years because this patient has carditis with sequelae in the heart valves.[12]. Anti-inflammatory options in rheumatic heart disease can be aspirin or prednisone. Aspirin is used in patients with moderate-to-severe arthritis and carditis without heart failure. Treatment is administered for at least 8 weeks.

In contrast, prednisone is administered to patients with severe carditis characterized by cardiomegaly and congestive heart failure. High-dose prednisone is administered for 2-3 week, then tapered over 3 weeks. IV corticosteroids are reserved for fulminant cases. The administration of prednisone, in this case, is indicative of severe carditis experienced by the patient. In this patient, also given tenace (enalapril) 5 mg to reduce the workload of the heart. The ACE inhibitor group should be administered in low doses to avoid the risk of hypotension and decreased kidney function. Renal and potassium function should be supervised within 1-2 weeks after therapy, especially after the dose increase. In this case, furosemide is given as a diuretic for reducing edema. Furosemide causes potassium excretion, so prolonged use of furosemide requires the administration of potassium. The combination of furosemide and spironolactone can add to the effect of diuresis, but spironolactone is potassium-resistant, so potassium administration is unnecessary. Paracetamol is given as an antipyretic if the patient has a fever, and laxative syrup is given as a laxative to stimulate the colon's peristalsis and inhibit excess water absorption from the feces. Furthermore, laxatives are used in patients who should not be allowed to push while defecating, such as those with heart disease.[7]

Children suffering from heart defects often have growth disorders, and their weight does not want to rise. This state is caused by several factors, such as attacks of breathlessness, impaired absorption of food due to decreased blood perfusion to the intestines, and infections accompanying heart failure. This patient is malnourished, so it is necessary to be educated about nutrition and feeding at home. The diet in this patient is to provide enough food without burdening the work of the heart, preventing, or eliminating the accumulation of salt and water by giving calories as much as 1700 kcal/day plus 35 grams of protein which is divided into large meals three times and snacks two times a day. Both the patient and the patient's family must be educated about medication adherence so that the

patient's symptoms do not worsen, and cooperation among family members is required to improve the patient's quality of life.[2]

The limitation of this manuscript is that the authors did not mention the interventions and factors that contributed to the decrease in the prevalence of this disease. Therefore, future studies should test interventions, several factors, and challenges to improve adherence and outcomes among patients with this disease at risk of morbidity and mortality.

Conclusion

In this case, the diagnosis is based on anamnesis, physical examination, and supporting analysis. Management of this patient is according to evidence-based medicine. Rheumatic heart disease is a heart valve disorder caused by a previous acute rheumatic fever that can lead to heart failure complications. Treatment depends on the severity of the disease, and close monitoring is required to improve the prognosis.

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