Dilatation Cardiomyopathy in the Young Adult

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INTRODUCTION

Cardiomyopathy is a primary disease of the myocardium, unrelated to hypertension, congenital defect, or disorders of the valves, coronary blood flow, arteries, or pericardium.[1,2]

In developing nations, cardiomyopathy makes up 30% of all deaths due to heart disease, while in developed nations, cardiomyopathy is not the main cause of heart disease.[3]

Cardiomyopathy is classified according to etiology and clinical findings. From the etiology, cardiomyopathy is classified into two types, the primary tipe, where the myocardiac disease is unknown/idiopathic, and the secondary type, with a clear cause, or is related with a disease of other organ systems. Based on clinical findings, cardiomyopathy is classified into dilatation cardiomyopathy or congestive, restrictive, and hypertrophic cardiomyopathy.[4]

In dilatation or congestive cardiomyopathy, there is a dilatation of the left and right ventricles, reduced systolic function, congestive heart failure, arrythmia, and emboli. In restrictive cardiomyopathy, there is endomyocardial scar tissue or myocardial infiltration that results in a restriction or inhibition of the filling of the left and right ventricles. Hypertrophic cardiomyopathy is characterized by unevenly distributed hypertrophy of the left ventricle, particularly of the ventricular septum, with or without obstruction of left ventricular output, usually in the undilated left ventricular chamber.[1,2,3]

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Congestive/dilatation cardiomyopathy is a primary or idiopathic myocardiac disease characterized by dilatation of heart chambers and congestive heart failure. The etiology of congestive cardiomyopathy is unclear, but it may be related with several factors, such as autoimmune disorders, viral infection, gravidity and puerperium, alcohol ingestion, or the effect of chemicals or physical forces.[1,2,5,6]

One of the chief clinical symptoms is congestive heart failure, particularly the left congestive heart failure, with fatigue, weakness, and signs of systemic or lung emboli, such as dyspneu d'effort, orthopnea, nocturnal paroxysmal dyspneu, peripheral edema, and palpitations. Physical examination demonstrated a greatly enlarged heart, heart sounds III and IV, and signs of congestive heart failure. Blood pressure may be normal or low, with alternating pulse, cold skin, possible mitral or tricuspid regurgitation, diastolic murmur, valve calcification, leg edema, ascites, hepatomegaly. Chest x-ray demonstrated cardiomegaly, particularly of the left ventricle, lung obstruction, and pleural effusion.[1,2,7,8,9]

Electrocardiography may demonstrate sinus tacchycardia, atrial arrhythmia, ventricular arrhythmia, ST segment changes, T wave, intra-ventricular conductional disorder, and low QRS voltage or pathologic Q wave. Echocardiography may demonstrate an enlarged left ventricle, left ventricular dysfunction, diastolic mitral valve dysmotility, in cases of tricuspid insufficiency, septum motility becomes paradoxyc, end diastolic volume is increased and ventricular pump function parameter as well as ejection fraction (EF) is reduced. Mitral valve closure is delayed, and aortal valve closure may occur sooner than normal.[1,3]

Patients with cardiomyopathy are often found with cardiogenic shock due to reduced heart performance. The most common cause other than cardiomyopathy is heart infarct, mechanical disturbance, and arrhythmia.

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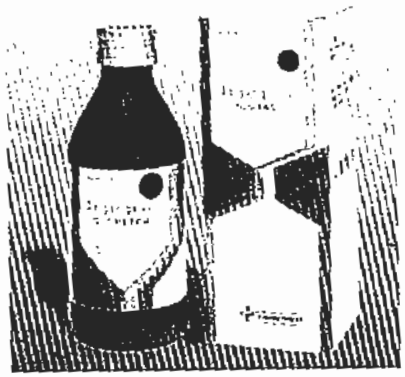

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Since the cause of primary dilatation cardiomyopathy is unclear, there is no possible specific treatment. One of which is prolonged bed rest, avoidance of severe physical activity. Treatment for cardiac failure and dilatation cardiomyopathy should be viewed as palliative and improvement of symptoms.

Treatment of heart failure through sodium restriction, administration of diuretics, digitalis, and vasodilators can only produce improved symptoms at the initial phase of the disease. Vasodilator agents may be administered if signs of refractory heart failure.[1,2,3,4]

CASE REPORT

Miss I, 15 years, unemployed, was hospitalized with a chief complaint of difficulty breathing since the 1st day prior to hospitalization.

Current history demonstrated that since 2 weeks prior to hospitalization, the patient suffered from nausea and vomiting every time the patient ate or drank, and epigastric tenderness. The patient went to a physician, was given antacids but the pain did not subside.

Five days prior to hospital admission, the patient suffered from continuous fever, without coughs, chills, and with normal urination and defecation. Two days prior to administration the patient suffered from difficulty breathing, particularly during activity. The patient had to sleep with 2 pillows. (The patient reported dyspneu d'effort, orthopneu, as well as paroxysmal nocturnal dyspneu). The patient also had difficulty sleeping due to difficulty breathing.

One day prior to hospitalization the difficulty breathing increased, and the patient consulted a private hospital, but was then referred to Cipto Mangunkusumo Hospital due to cardiogenic shock.

Based on previous history, previous breathing difficulties, bluish skin, sore throat, chest pain, asthma, diabetes mellitus, and hypertension was denied. Menstruation was regular every month.

Based on family history, the patient's mother suffered from heart disease and was hospitalized the hospital for 2 weeks with a diagnosis of post partum heart disease due to myopathy, but went home without permission. The patient's mother had 5 children, and 2 of them died. The patient is the second child.

From physical examination, the patient was found to be severely ill, somnolent, with a blood pressure of 90/60 mmHg, pulse rate of 126 times/minute, weak, and irregular, with a temperature of 36°C, a respiratory rate of 31 times per minute, there was intercostal retraction, and abdominothoracic shallow breathing. The patient's conjunctiva were anemic and the sclera were yellowish. Her jugular venous pressure was 5+2 cmH_2O. At cardiac evaluation, ictus cordis was not observable, but palpable at the left mid-clavicula spreading to the left medial axillary line, the left border of the heart at the left anterior axillary line, the right border at the right parasternal line. First and second heart sounds were irregular, with grade I-II systolic murmur at the second intercostal space at the left sternum, with no gallop. Pulmonary auscultational examination turned out sonor, vesicular, with soft rales at the base, with no wheezing. The patient's abdomen was soft, the liver and spleen were not palpable. Bowel sounds were normal. Extremities: acrals were cold, and there was pretibial edema.

Laboratory examination demonstrated a hemoglobin level of 9.6 g%, hematocryte of 30 vol%, leukocyte count of 9,200/mm^3, platelet count of 336,000 u/L, blood ureum level of 37 mg/dl, blood creatinine level of 1.0 mg/dl, random blood sugar of 76 mg/dl, sodium level of 132 mEq/l, potassium level of 5 mEq/L, chloride of 112 mEq/L, amylase of 143, and lipase of 56.

Chest x-ray demonstrated cardiomegaly (cardiothoracic ratio was 65.3%), and lungs within normal limits. Electrocardiography demonstrated sinus rhythm, normal axis, QRS rate of 120 times/minute, irregularity, no Left or Right Ventricular Hypertrophy, low voltage at I, II, III, AVR, AVL, AVF, and ventricular extra-systole. Echocardiography demonstrated an LAD of 4.9, AOD 2.4 cm, EPSS 25 cm, ESD 58, EDD 60, FS 10%, EF 20%, dilated heart, global hypokinetic at the left ventricular with severe dysfunction. Doppler color echo demonstrated moderate MR, mild TR, pericardial effusion, with a differential diagnosis of cardiomyopathy and myocarditis.

The problem in this patient was formulated as cardiogenic shock, functional heart failure class IV due to cardiomyopathy, with a differential diagnosis of myocarditis, other problems were dyspepsia and anemia.

The patient was treated with 5 u/bodyweight of dopamine, 10 u/kg bodyweight of dobutamine in 5% dextrose 10 drops per minute, and 3 x 1 tablespoon of antacids. The patient was scheduled for the following evaluations: CK, CKMB, and erythrocyte morphology.

On the 7th day of treatment, the patient's breathing difficulty improved. The pain on the upper abdomen fluctuated. Her blood pressure was 90/70 mmHg, her pulse rate 100 times/minute, and her respiratory rate 20 times/minute. There was no fever, her conjuctiva were no longer pale, yet her sclera was still jaundiced. Her jugular venous pressure was 5-2 cmH_2O. Cardiac and pul-

monary findings were within normal limits. There was ascites in the abdomen, her liver was palpable at 1 finger below the costal arc. There was no abdominal tenderness. There was no edema.

The following problem was defined: cardiogenic shock, jaundice suspected to be due to hepatitis, with a differential diagnosis of congestive heart failure (obstruction), mild anemia, functional heart failure class IV due to cardiomyopathy. The patient was scheduled for vital sign monitoring, fluid balancing, serial electrocardiography, as well as evaluation for HbsAg seromarker, IgM anti-HAV, liver function test, and erythrocyte morphology.

The patient was treated with 1 x 60 mg furosemide, 1 x 0.25 mg of digoxin, 3 x 1 Hp pro®, 3 x 1 tablespoon of antacid.

On the 18th day of treatment, the difficulty breathing lessened, the patient no longer had difficulty breathing when walking, the patient's general condition was moderate, she was fully conscious, with a blood pressure of 90/60 mmHg, pulse rate of 100 times/minute, respiratory rate of 24 times/minute, no fever, pale conjunctiva, and jugular venous pressure of 5-2 cmH_2O. Her first and second heart sounds were regular, there was no murmur, no gallop, her liver 1 finger below the costal arc. There was no jaundice. Her hemoglobin level was 9.5 g%.

The problem in this patient: functional heart failure class II-III due to cardiomyopathy, and mild anemia. The patient was scheduled for evaluation of SI and TiBC, complete stool examination, and repeat peripheral blood check. The patient was treated with 1 x 0,375 mg of digoxin, 1 x 40 mg furosemide, 2 x 12,5 mg of captopril, 3 x 1 tablet of ferrosulphate, and 3 x 2 tablets of B complex.

DISCUSSION

Cardiomyopathy in this patient was established based on symptoms classified in the primary type cardiomyopathy, consisting of cardiomyopathy due to unknown origin. The history of heart disease, diagnosed as cardiomyopathy in the patient's mother, could signify a familial correlation in this case. Cardiomyopathy in this case occurred at a young age with an unknown cause.[1,2]

Chief clinical symptoms in this patient was those of left and right congestive heart failure, in the form of difficulty breathing, particularly during activity, orthopneu, nocturnal paroxysmal dyspneu, and dyspnoe d'effort. Chest x-ray demonstrated cardiomegaly and electrocardiography demonstrated low voltage, while echocardiography demonstrated an ejection fraction of 20% and heart dilatation. [1,3]

Even though there was no clear etiology for many cardiomyopathy cases, dilatation cardiomyopathy may be the final result of damage on the heart muscle cells due to toxic or metabolic substances, or infection. Some cases of dilatation cardiomyopathy may be residual symptoms of acute viral myocarditis, possibly through an immunological mechanism. In this case, drug-related and viral cardiomyopathy have been excluded. Even though cardiomyopathy is generally a disease of the mid-age group, it can also be found in every age group. [1,2,5]

Treatment in this case is by dealing with the condition of cardiogenic shock first, prior to treating congestive heart failure. Even though treatment of congestive heart failure is palliative in nature, in this case, treatment for congestive heart failure is still administered to improve symptoms, through the use of diuretics and digitalis. The main form of treatment in patients with cardiomyopathy is heart transplantation. [1,2,3]

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