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24-year-old obese woman with dilated cardiomyopathy: a case report of obese cardiomyopathy



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ABSTRACT

Background: Dilated cardiomyopathy is a disease characterized by enlargement of the heart ventricles with impaired left ventricular or biventricular systolic function. Obesity is a risk factor for changes in the structure and function of the heart, known as obese cardiomyopathy. The purpose of writing this case report is to holistically discuss obesity as a risk factor for dilated cardiomyopathy based on a literature review.

Case presentation: A 24-year-old woman complained of shortness of breath 2 months ago, appeared during activities and while she slept at night, and improved when sitting and resting. Other complaints are palpitations, swollen feet, nausea, and vomiting. Childhood history of obesity. Blood pressure 160/95 mmHg, respiratory rate 22 times/minute, and body mass index 33.8 kg/m² (obesity II). The jugular venous pressure is increased. Auscultation revealed a systolic murmur at ICS 4 grade 3/6 and pulmonary crackles, with pitting edema in the legs. Electrocardiography revealed sinus tachycardia. Laboratory tests showed high triglycerides and total cholesterol, and other laboratories were within

normal limits. Echocardiographic examination showed decreased left ventricular systolic function, severe left ventricular dilatation, and an ejection fraction of 27.9%. No significant lesions were found during the angiographic examination. A magnetic resonance imaging examination revealed non-ischemic cardiomyopathy, moderate left ventricular dysfunction, and severe right ventricular dysfunction. Patients were given heart failure therapy according to guidelines. On routine control, the patient's condition was stable.

Conclusion: Obesity is believed to be a risk factor for dilated cardiomyopathy through an increased hemodynamic load, neurohormonal dysregulation, inflammation, and lipotoxicity. Modalities such as the electrocardiogram, echocardiography, and magnetic resonance imaging can support the results of examinations for diagnosing dilated cardiomyopathy due to obesity. The management of obese cardiomyopathy involves changing lifestyles and administering drugs according to the clinical manifestations of heart failure.

Keywords: Dilated cardiomyopathy, obesity, risk factors.

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INTRODUCTION

Cardiomyopathy is a bunch of heart diseases with structural problems limited to the structure of the myocardium, this condition can cause symptoms of heart failure, although the basic cause of abnormal myocardial function due to cardiomyopathy can be identified but often the etiology is unknown (idiopathic). Cardiomyopathy itself is classified into several types such as dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy, restrictive cardiomyopathy, and arrhythmogenic

right ventricle (ARVC).^{1,2}

DCM is a heart disease characterized by enlargement of the ventricular chambers with impaired left ventricular or biventricular systolic function contractions.¹ DCM is caused by various things such as genetic (familial) problems and environmental problems such as inflammatory, infectious, toxic, autoimmune, metabolic, neuromuscular, peripartum, and idiopathic.^{1,2} Studies in America report that there are 5 and 8 cases per 100,000 residents who experience DCM.³ Studies conducted in Asia found

DCM cases to be the third highest case after coronary heart disease and hypertensive heart disease. Based on gender, men are three times more at risk of experiencing DCM than women. Nearly 50 percent of cases of heart failure are idiopathic dilated cardiomyopathy, patients with this condition themselves are relatively young, between 20 and 60 years.^{3,4}

One of the risk factors that can cause the heart to develop into cardiomyopathy is obesity, the condition of obesity in the long term is known to cause changes in the structure and function of the heart so

that it can experience heart failure. Heart failure due to the risk factor of obesity itself is called “Obesity Cardiomyopathy”. In this case, we report a young woman with longstanding risk factors for obesity who developed dilated cardiomyopathy, a relatively rare case of a young obese person. The symptoms of this disease are quite diverse, from having no symptoms to experiencing progressive heart failure, depending on the diagnostic criteria used.^{4,5} Treatment can be carried out by making lifestyle and pharmacological changes. Improvement in symptoms has also been associated with increased use of drugs such as ACE inhibitors, beta-blockers, the use of implantable cardioverter-defibrillators (ICDs), and heart transplants.⁶ This case report will discuss obesity as a risk factor for dilated cardiomyopathy holistically based on a literature review.

CASE PRESENTATION

A woman, 24 years old, of Balinese ethnicity, came to the heart clinic at the Hospital complaining of shortness of breath about 2 months before going to the hospital, felt tightness in her chest all day, appearing especially during physical activity and sleeping position, when the patient sleeps at night always uncomfortable and wakes up because of shortness of breath, shortness of breath was decrease when sitting and resting, during rest at night the patient felt comfortable sleeping with 2 pillows. The patient also complained of palpitations, especially during activity, and got better at rest, the patient has had swollen feet since about 2 months ago, had been vomiting for about a month, the patient felt tired easily at work. Other complaints such as coughing, runny nose, decreased appetite, chest pain, fever, cold sweats, and syncope were denied. The patient had never experienced anything similar. history of high blood pressure, high cholesterol, surgery, or hospitalization were denied. None of the patient’s family experienced anything similar, the patient’s mother and grandmother had a history of hypertension, the patient was a freelance massage worker and felt her work was disrupted due to complaints experienced by the patient, and the patient had been obese since childhood. history of smoking,

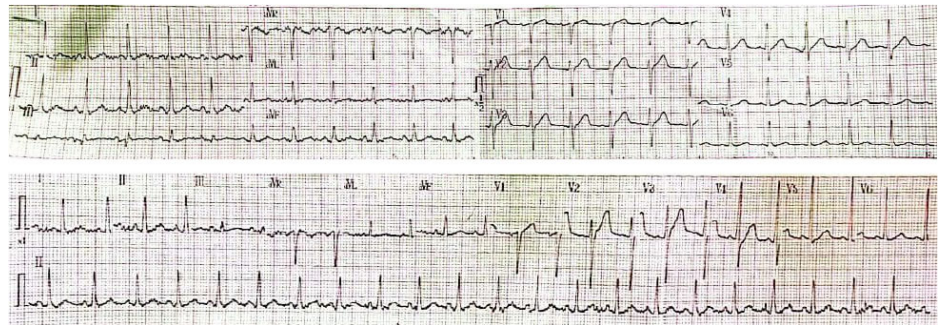


Figure 1. Electrocardiography: sinus tachycardia 114 x/min regular.

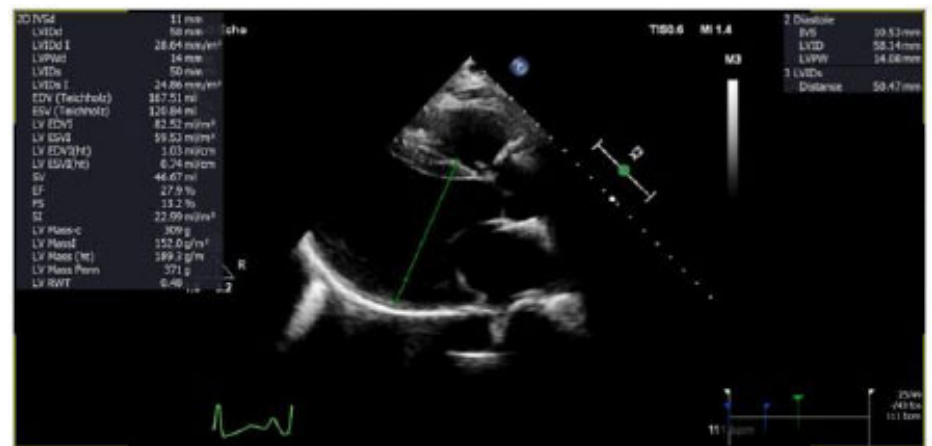


Figure 2. Echocardiography. severely dilated left ventricle, with an EF of 27.9%.

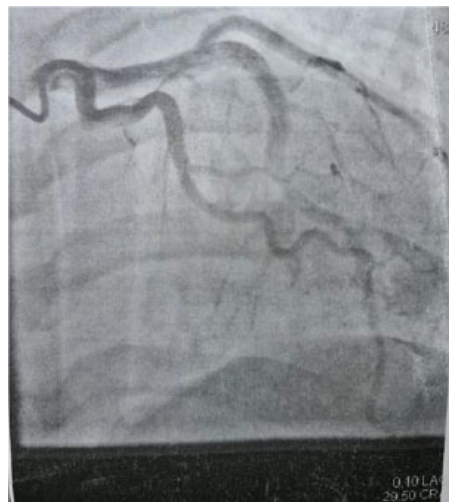


Figure 3. Coronary angiography: LAO Cranial LM, LAD, LCx found no significant lesions.



Figure 4. Coronary angiography: RAO Caudal LM, LAD, LCx found no significant lesions.

drinking alcohol, consumption of certain drugs or use of illegal drugs was denied.

On physical examination the patient looked moderately ill, Compos mentis (GCS E4V5M6), the patient’s height was 165 cm, weight 92 kg, with a BMI of 33.8 (obese level II). Examination of vital signs, blood pressure 160/95 mmHg, pulse

114 x/minute regular, respiration 22 x/minute, temperature 36 degrees Celsius and SpO2 96%. JVP obtained 5 + 4 cmH2O (increased), on cardiac examination he got regular S1 S2 there was a left 4 parasternal ICS systolic murmur grade 3/6, on lung examination he got symmetrical vesicular sounds in both lung fields accompanied

by crackles at the lung bases, in both lower extremities there were pitting edema. On ECG examination, sinus tachycardia was found 114 x/minute, echocardiography examination obtained RWMA (+), Mild AR, Mild TR with low probability of PH, severely decreased LV systolic function, left ventricle severely dilated normal RV systolic function, Grade III diastolic dysfunction, EF 27.9%.

The patient was diagnosed with CHF FC II et Causa Dilated Cardiomyopathy with Hypertension stage II. The initial therapy was given furosemide 2 x 40 mg, spironolactone 1 x 50 mg, CPG 1 x 75 mg, simvastatin 1 x 20 mg, coralan 2 x 2.5 mg, digoxin 1 x 0.25 mg. At the time of the next control, the patient felt that his complaints of shortness of breath had decreased, he never felt pounding and tired easily, and

the swelling in his legs had decreased. Then other laboratory tests were carried out such as lipid profile and blood sugar. The Lipid profile revealed an increase in triglyceride and total cholesterol. Then added therapy with fenofibrate 1 x 300 mg and ramipril 1 x 2.5 mg.

The patient was examined for coronary angiography, magnetic resonance imaging (MRI), and other laboratories at the Sanglah General Hospital referral hospital to find out the cause of the disease. Other supporting examinations of the patient, such as kidney function, liver function, thyroid function, complete blood count, hemostasis physiology, inflammatory factors, and electrolytes were within normal limits.

Coronary angiography did not find any significant lesions on the left main artery (LM), left artery descending (LAD), left circumflex (LCx), and right coronary ascending (RCA). on MRI examination Suggests non-ischemic cardiomyopathy accompanied by non-viable myocardium and hypokinesia, Moderate LV, and severe RV dysfunction. The last control patient in stable condition with continued therapy was given Candesartan 1x 32mg, Spironolactone 1x50mg, Bisoprolol 1x10mg, Furosemide 1x40, and Coralan 2 x 2.5 mg.

affecting younger adults.⁷ The condition of obesity can cause changes in cardiac morphology and ventricular function that contribute to the development of heart failure, even in the absence of other comorbidities such as coronary artery disease or hypertension.⁸ Obesity is defined as an excess nutritional status based on body mass index (BMI), with class I obesity having a BMI of 25.0-29.9 kg/m² and class II obesity having a BMI of ≥ 30 kg/m² according to Asia-Pacific criteria.⁹ A study in Sweden mentioned that increasing BMI in young women is associated with an increased risk of cardiomyopathy, especially dilated cardiomyopathy. The lowest risk of cardiomyopathy was at a BMI of 21 kg/m², and the risk increased as BMI increased.¹⁰

Heart failure due to obesity is referred to as “obese cardiomyopathy” which refers to morphological, functional, and metabolic abnormalities of the heart with only one risk factor, namely obesity, without the involvement of other risk factors.¹¹ The relationship found between BMI and cardiomyopathy was seen in the presence of myocardial remodeling in obese subjects. Weight gain requires increased metabolic demands that require hemodynamic changes, including increased blood volume and cardiac output. Over time, this results in ventricular dilation and hypertrophy along with impaired cardiac function. Another contributing mechanism is the increased secretion of adipocyte-derived molecules, e.g., leptin, neprilysin, and aldosterone. This triggers a proinflammatory state, which can lead to cardiac muscle fibrosis and microvascular abnormalities. In addition, other processes that link adiposity with cardiac remodeling and dysfunction are high levels of oxidative stress as well as myocardial lipotoxicity, in which the accumulation of excess intracellular fatty acids and triglycerides causes the apoptosis of cardiac myocytes. Overall, the pathophysiology of obesity-related cardiomyopathy involves multiple mechanisms, including increased hemodynamic burden, neurohormonal dysregulation, inflammation, and lipotoxicity.^{10,12-14}

Obese cardiomyopathy has some of the same clinical features as other causes of heart failure, but it is unique.



Figure 5. Coronary angiography: RAO Caudal RCA found no significant lesions.

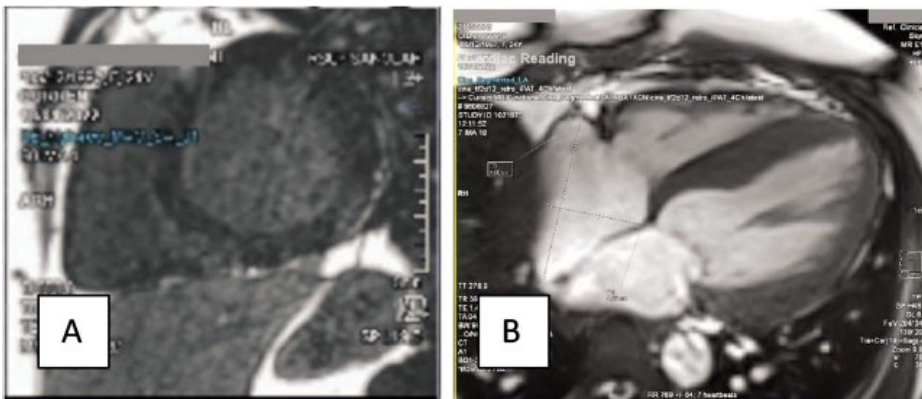


Figure 6. MRI images (A) Short Axis View and (B) 4 Chamber view. Suggests non ischemic cardiomyopathy, Moderate LV and severe RV dysfunction.

Symptoms of obese cardiomyopathy include exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea (PND), recent weight gain, increase in abdominal girth, oedema of the lower extremities, decreased mental status, and in some cases coma and sudden death. Physical signs of obese cardiomyopathy include jugular venous distention and hepatojugular reflux, pulmonary crackles, and fourth heart sound or summation gallop, heart murmurs occur relatively rarely. These symptoms and signs are the result of the combination of LV and RV failure and the sequelae of sleep apnea/obesity hypoventilation syndrome.¹⁵ Electrocardiographic abnormalities in severe obesity include low QRS voltage and right axis deviation. Electrocardiographic evidence of LVH occurs at a lower frequency than echocardiographic LVH. Several studies have demonstrated QTc interval prolongation and QTc dispersion in obese subjects.¹⁵

In the case of a 24-year-old young adult woman with a BMI of 33.3 kg/m², including class II obesity, she came to the polyclinic in a stable condition, complaining of shortness of breath several times when she was active, shortness of breath at night rest, improved when sitting. Shortness of breath has been getting worse since 2 months ago. The patient denied a previous history of heart disease or other chronic diseases. Examination found a fast respiratory rate, jugular vein distension, crackles in both lung bases, systolic heart murmurs on the left ICS 4 parasternal line, and pitting edema on both legs. An ECG examination only found sinus tachycardia. From the results of this examination, we can suspect the patient is in a condition of heart failure. Then the patient is scheduled to undergo cardiac angiography, echocardiography, and an MRI. Angiography results showed no significant lesions, thus the possibility of ischemic cardiomyopathy can be excluded. The echocardiography and MRI results confirmed that dilated cardiomyopathy was the underlying mechanism for this patient's complaint.

Obesity causes unfavorable conditions for the structure and function of the heart. With the use of echocardiography and magnetic resonance imaging

(MRI), disturbances in left ventricular structure and function can be seen in obese individuals, such as remodeling and impaired diastolic and systolic function.¹⁶ In this case, there was severe left ventricular systolic dysfunction (EF 27.9%) and diastolic dysfunction (Grade III (E/A ratio 4.43)), which were sufficient to explain the patient's condition during examination. The MRI results suggest non-ischemic cardiomyopathy, moderate LV dysfunction, and severe RV dysfunction. This strengthens the results of the previous echocardiography.

We recommend lifestyle changes to patients, such as exercise and adjusting her diet by reducing salt and calorie intake. Adjusting the diet menu and losing weight is the simplest thing that can be done by individuals with dilated cardiomyopathy accompanied by obesity.¹⁷ At the next polyclinic control, the patient's condition improved after administering outpatient therapy such as furosemide, spirinolactone, clopidogrel, coralan, digoxin, simvastatin, fenofibrate, antihypertensives such as candesartan, and bisoprolol. Administration of pharmacological therapy based on guidelines for the management of heart failure. Other drugs that have passed clinical trials can improve quality of life and reduce the risk of hospitalization, such as administration of ACE-inhibitor drugs or angiotensin receptor antagonists (ARBs). Beta-blocker class drugs with negative inotropic properties have proven to be beneficial. Other drugs, such as the mineralocorticoid antagonist, ivabradine have been shown to reduce the risk of hospitalization and improve the quality of life of patients with cardiomyopathy.¹⁷

CONCLUSION

Dilated cardiomyopathy is defined as cardiac remodeling in the form of dilated cardiac chambers, especially the left ventricle, and systolic dysfunction to the point of causing symptoms of heart failure, in the absence of evidence of involvement of coronary artery disease, valvular heart disease, or the pericardium, especially in young adults. Obesity is believed to be a risk factor for dilated cardiomyopathy through increased hemodynamic load, neurohormonal dysregulation,

inflammation, and lipotoxicity. Echocardiographic examination to establish the diagnosis of dilated cardiomyopathy. Magnetic resonance imaging (MRI) and angiography are used to support echocardiographic readings and exclude the possibility of other organic heart disease in cases of dilated cardiomyopathy due to obesity. Prompt diagnosis and appropriate management help individuals with dilated cardiomyopathy reduce the risk of hospitalization and improve quality of life. There are still few reports regarding cases of dilated cardiomyopathy due to obesity, so it is hoped that this case report can be developed in the writing of other subsequent papers.

CONFLICT OF INTEREST

The author declares that there is no personal or financial conflict of interest in writing the case report.

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THE AUTHOR'S CONTRIBUTION

All authors contributed equally in writing and revising the case report.

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