



## LAPORAN KASUS—CASE REPORT

# AN UNUSUAL CASE OF PATIENT WITH DILATED CARDIOMYOPATHY SECONDARY TO HYPOTHYROIDISM: A CASE REPORT

Ketut Angga Aditya Putra Pramana<sup>1\*</sup>, Yusra Pintaningrum<sup>2</sup>

<sup>1</sup> Faculty of Medicine Mataram University, Mataram, Indonesia,

<sup>2</sup> Department of Cardiology and Vascular Medicine, West Nusa Tenggara General Hospital, Mataram, Indonesia

\*Korespondensi:  
angga997453@gmail.com

### Abstrak

**Background:** Alteration in thyroid hormone may affect on the cardiovascular system because the heart is very sensitive to an alteration of thyroid hormone. Alteration in thyroid hormone has been known to cause the changes in cardiac structure and function, such as the development of dilated cardiomyopathy which is a very rare manifestation of hypothyroidism.

**Case Illustration:** A 26-year-old woman came to a cardiology clinic with a chief complaint of fatigue and breathlessness. On physical examination are unremarkable. On clinical laboratory examinations showed elevated TSH serum levels and decreased FT4 serum levels. On echocardiography, the patient had moderate mitral regurgitation, mild tricuspid regurgitation, all chamber dilatation, and abnormal left ventricular systolic function with ejection fraction 26.9%. The treatment of this patient are levothyroxine 50ug/day, furosemide 40 mg, spironolactone 25 mg, bisoprolol 2.5 mg, ramipril 1.25 mg, and acetylsalicylic acid 80 mg all in once daily.

**Discussion:** Dilated cardiomyopathy is characterized by enlargement of the ventricular chamber with impaired systolic function. Hypothyroidism is associated with decreased cardiac contractility and decreased cardiac output because of the genomic effect of thyroid hormone to the cardiovascular system is involved in the regulation of mRNA transcription of genes associated with the contractile system.

**Conclusions:** Hyperthyroidism is often cause dilated cardiomyopathy, but in this case, hypothyroidism also can cause dilated cardiomyopathy. Early diagnosis and management of hypothyroidism are very important to restore the cardiac function.

**Key words:** Hypothyroidism, Dilated Cardiomyopathy, Thyroid Hormone

## INTRODUCTION

A broad category of myocardial disorders known as cardiomyopathies are characterized by mechanical and/or electrical dysfunction and frequently show inappropriate ventricular hypertrophy or dilatation. Traditionally, structural and functional characteristics, particularly dilated, hypertrophic, and restricted phenotypes, have been used to characterize cardiomyopathies.[1] Dilated cardiomyopathy (DCM) is an idiopathic condition that results from impaired ventricular systolic function, leading to progressive cardiac remodeling and dilatation.[2] DCM are characterized by the presence of a dilated and ineffective left ventricle in the absence of aberrant loading circumstances or severe coronary artery

disease (CAD). Patients with this conditions have a bad prognosis since cardiac impairment is gradual and permanent. Nevertheless, certain uncommon curable etiologies, such as endocrine imbalance, may be discovered with a thorough clinical history and investigation.[3]

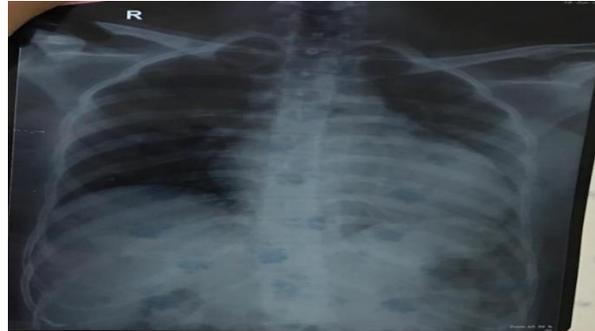
Heart structure and function have been known to change in numerous ways as a result of thyroid conditions. Both hyperthyroidism and hypothyroidism alter blood pressure, myocardial oxygen consumption, cardiac output, and systemic vascular resistance in the heart.[4,5] Hyperthyroidism-related hemodynamic alterations result in the typical hyperdynamic cardiovascular condition, which is accompanied by an increase in cardiac output and a decrease in peripheral vascular resistance. Contrarily, hypothyroidism is

linked to bradycardia, moderate diastolic hypertension, narrow pulse pressure, and a somewhat elevated mean arterial pressure.[6] The most frequent finding in individuals with hypothyroidism is diastolic dysfunction. Additionally, it is frequently seen that the left ventricular systolic function has a mildly decreased ejection fraction and slightly decreased stroke volume. DCM is an uncommon form of hypothyroidism.[7] Here, we report a case of a young female who presented with hypothyroidism-induced DCM.

## CASE REPORT

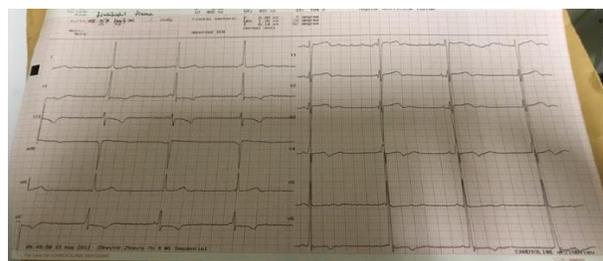
A 26 year old woman came to the cardiac polyclinic with the chief complaint of fatigue and shortness of breath. Complaints are felt to get worse, especially when the patient is doing moderate activity and gets better when the patient is resting. The patient's shortness of breath is also felt to be worse when the patient sleeps and the complaint improves when the patient sleeps with his head elevated using two pillows. Complaints of choking or loud coughing when the patient was sound asleep were denied by the patient. The patient also complained of swelling in both lower limbs and hands for the past year. Complaints of swelling in the four limbs of the patient felt intermittent. The patient denied any history of chest pain during activity, diabetes mellitus and hypertension. There is no family history of similar complaints.

On physical examination, examination of vital signs found blood pressure 100/60 mmHg pulse 51 beats/minute, respiratory rate 22 times/minute, and 98% O<sub>2</sub> saturation in room air. On physical examination of the thorax, a palpable ictus cordis was found in the 6th intercostal space, left anterior axillary line, left ventricular third heart sound (S<sub>3</sub>), and vesicular breath sounds throughout the lung fields. On clinical laboratory examination, there was an increase in serum TSH level (65.75 uIU/mL) and a decrease in serum FT<sub>4</sub> level (4.42 Pmol/L) and there was an appearance of cardiomegaly on the patient's plain chest x-ray (Figure 1).



**Figure 1.** Chest x-ray examination showed cardiomegaly.

Electrocardiographic (ECG) examination revealed sinus bradycardia with a pulse rate of 44 beats per minute, inversion T-wave in inferior leads and precordial leads, and left ventricular hypertrophy with strain (Figure 2). On echocardiographic examination, there was moderate mitral regurgitation, mild tricuspid regurgitation, dilatation of all ventricles, and abnormal left ventricular (LV) systolic function with an ejection fraction of 26.9%. From these examinations, the patient was diagnosed with hypothyroidism with dilated cardiomyopathy and the patient's complaints improved slightly after being given levothyroxine 50ug/day, furosemide 40 mg, spironolactone 25 mg, bisoprolol 2.5 mg, ramipril 1.25 mg, and acetylsalicylic acid 80 mg all of them once daily.



**Figure 2.** Patient's electrocardiography examination showed sinus bradycardia, inversion T-wave on inferior and precordial leads, and left ventricular hypertrophy with strain.



## DISCUSSION

Hypothyroidism is associated with decreased cardiac contractility, increased systemic vascular resistance and decreased cardiac output. Its manifestations are insidious and subtle in its progression and clinical behavior.[4] The most prevalent type of cardiomyopathy, DCM, can be caused by a number of things, including toxins, immune system problems, inflammation, infections, metabolic/endocrine issues, and electrolyte abnormalities.[8] The cardiac myocyte and peripheral vasculature are affected by thyroid hormones. The cardiac function and cardiovascular hemodynamics are affected by the genetic and non-genomic actions of thyroid hormone.[4]

The direct cellular effects of thyroid hormones on practically all bodily tissues, including the heart, have been known for many years. The information that is now available indicates that the main way that thyroid hormone affects the heart is via altering protein synthesis. Thyroid hormone's actions on calcium-activated ATPase and phospholamban, which are principally involved in the control of systodiastolic calcium concentrations in cardiomyocytes, might cause certain cardiac function impairments in people with thyroid dysfunction. Sarcoplasmic reticulum calcium-activated ATPase is responsible for the rate of calcium reuptake into the lumen of the sarcoplasmic reticulum during diastole that, in turn, is a major determinant of the velocity of myocardial relaxation after contraction. Additionally, the expression of additional ion channels including Na<sup>+</sup>/K<sup>+</sup>-activated ATPase and Na<sup>+</sup>/Ca<sup>++</sup> exchanger is altered by thyroid hormone, which helps to balance the myocardium's electrochemical and mechanical responses.[5,6]

In this patient we found she complaint of exercise impairment and on physical examination is bradycardia. The most prevalent symptoms in people with overt hypothyroidism are bradycardia, systemic hypertension, narrow pulse pressure, slightly elevated mean arterial pressure, and some degree of activity impairment. A typical ECG such as QT prolongation and flattening/inversion of the

T wave, which represent extended cardiac action potential, are common in individuals with overt hypothyroidism.[7,9] The results of the ECG examination in this patient also obtained similar results, that there was a T wave inversion in inferior and precordial leads.

Impairment of LV diastolic function, which is characterized by delayed myocardial relaxation and impaired early ventricular filling, is the most often seen cardiac abnormality in individuals with overt hypothyroidism. However, it is unusual for hypothyroidism to manifest as cardiomyopathy and reduced LV systolic function.[9,10,11] In this case we found that this patient had LV systolic dysfunction with reduced ejection fraction. This indicates that there is a possible relationship between the hypothyroidism and the development of the left ventricular dysfunction in the current case. The patient's complaints improved slightly after being given levothyroxine 50ug/day, furosemide 40 mg, spironolactone 25 mg, bisoprolol 2.5 mg, ramipril 1.25 mg, and acetylsalicylic acid 80 mg all of them once daily. After receiving replacement hormone therapy and having their thyroid function corrected, some DCM cases have been reported to have a regression of heart failure (HF) symptoms and a reversal of the dilated pattern. In fact, hypothyroidism is listed as a precipitating factor of acute HF in the ESC recommendations. In this instance, the management of HF and the therapy of the thyroid abnormalities with L-thyroxin led to an improvement in cardiac systolic function as well as a decrease in the patient's complaint.12,13

## CONCLUSION

DCM is generally an idiopathic illness with a progressive and irreversible course. Early diagnosis and management of hypothyroidism are very important to restore the cardiac function. In DCM secondary to hypothyroidism, L-thyroxin hormonal replacement therapy may improves the prognosis. This case showed the importance of thyroid function tests in the assessment of DCM.



### Declaration of patient consent

The authors certify that appropriate patient consent was obtained.

### Financial support and sponsorship

None.

### Conflicts of interest

There are no conflicts of interest.

W., Jaarsma, T., Jankowska, E. A., ... Skibelund, A. K. (2021). 2021 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure. *European Heart Journal*, 42(36), 3599–3726.

## REFERENCES

1. I. Maron BJ, Towbin JA, Thiene G, Antzelevitch C, Corrado D, Arnett D, et al. Contemporary definitions and classification of the cardiomyopathies: An American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. *Circulation*. 2006;113:1807–16
2. Elliott P. Cardiomyopathy. Diagnosis and management of dilated cardiomyopathy. *Heart*. 2000;84:106–12
3. Elliott P, Andersson B, Arbustini E, Bilinska Z, Cecchi F, Charron F, et al. Classification of the cardiomyopathies: a position statement from the European Society Of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J*. 2008;29:270–6.
4. Klein I, Ojamaa K. Thyroid hormone and the cardiovascular system. *N Engl J Med* 2001;344:501-9.
5. Klein I, Danzi S. Thyroid disease and the heart. *Circulation* 2007; 116:1725-35
6. Fazio S, Palmieri EA, Lombardi G, Biondi B. Effects of thyroid hormone on the cardiovascular system. *Recent Prog Horm Res* 2004;59:31-50.
7. Kumar Kota S, Tripathy PR, Krishna Kota S, Jammula S, Kumar Meher L, Modi KD. Primary hypothyroidism: uncommon presentation with reversible dilated cardiomyopathy in a young subject. *Int J Endocrinol Metab* 2012;10:440-3.
8. Kuethe F, Sigusch HH, Bornstein SR, Hilbig K, Kamvissi V, Figulla HR. Apoptosis in patients with dilated cardiomyopathy and diabetes: a feature of diabetic cardiomyopathy? *Horm Metab Res* 2007;39:672-6.
9. Bezdah L, Slimène H, Kammoun M, Haddad A, Belhani A. Hypothyroid dilated cardiomyopathy. *Ann Cardiol Angeiol (Paris)* 2004;53:217–220.
10. François M, Delemer B. [What's new in the couple thyroid and heart in 2008?]. *Ann Endocrinol (Paris)* 2008;69 Suppl 1:S37-43.
11. Stańescu C, Branidou K, Ranetti EA. Heart failure and dilated cardiomyopathy associated with severe longstanding untreated hypothyroidism. *Rom J Intern Med* 2007;45:77-83.
12. Duntas LH. Thyroid disease and lipids. *Thyroid*. 2002;12:287–93.
13. McDonagh, T. A., Metra, M., Adamo, M., Gardner, R. S., Baumbach, A., Böhm, M., Burri, H., Butler, J., Čelutkienė, J., Chioncel, O., Cleland, J. G., Coats, A. J., Crespo-Leiro, M. G., Farmakis, D., Gilard, M., Heymans, S., Hoes, A.