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Case Report

Dilated Cardiomyopathy Related to Hyperthyroidism With Cardiogenic Shock And Inadequate Diuretic Therapy: A Case Report

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ABSTRACT

Dilated Cardiomyopathy (DCM) is a non-ischemic heart muscle disease characterized by structural and functional myocardial abnormalities. One of its causes is hyperthyroidism. Hyperthyroidism can lead to a hyperdynamic circulatory state, increasing cardiac output and metabolic demands, which can ultimately result in heart failure. **Case Presentation:** A 35-year-old woman presented with complaints of acute shortness of breath that worsened at night, accompanied by bilateral lower limb oedema and palpitations. Physical examination showed low BP (84/60 mmHg), tachycardia 110 BPM and elevated JVP. Auscultation detected fine bilateral crackles and mitral regurgitation. Echocardiography showed consistent with DCM. **Conclusion:** Effective management of heart failure in the context of thyroid dysfunction requires a multidisciplinary approach that encompasses both the cardiac and endocrine components of the disease. This case illustrates the complexity of treating DCM with hyperthyroidism and the need for individualized therapy to optimize patient outcomes. Managing cardiomyopathy related to hyperthyroidism presents unique challenges, as it requires treatment for both hyperthyroidism and heart failure, especially when cardiogenic shock is present with an inadequate response to diuretics. This case highlights the complex interaction between thyroid dysfunction and heart failure, as well as the therapeutic strategies used to manage this condition.

Highlights:

1. This article highlights the complexity of thyroid dysfunction and how individual assessments are crucial in determining the effective management of it.

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Introduction

Thyrotoxicosis is a clinical syndrome resulting from an excess of thyroid hormones in the blood, caused by either hyperthyroidism or other mechanisms that lead to the excessive release of thyroid hormones into the systemic circulation [3]. Thyroid hormones exert effects on almost all nucleated cells and play a role in normal growth processes and energy metabolism [4]. Hyperthyroidism is a condition characterized by the overproduction of thyroid hormones, leading to a hypermetabolic state. This can have significant impacts on the cardiovascular system, including increased heart rate, increased myocardial contractility, and increased cardiac output.

Over the course of time, these effects may lead to cardiac complications such as atrial fibrillation, heart failure, and cardiomyopathy [2]. The relationship between hyperthyroidism and heart failure, particularly thyroid cardiomyopathy, is well documented but often underrecognized in clinical practice [5]. Changes in thyroid hormone concentrations directly influence abnormal changes in the structure and/or function of the heart, leading to cardiomyopathy and increasing the risk of complications such as heart failure and arrhythmias that are life-threatening.[6]

DCM is a non-ischemic heart muscle disease characterized by structural and functional myocardial abnormalities. The clinical features of DCM are defined by left or biventricular dilation and systolic dysfunction in the absence of coronary artery disease, hypertension, valvular disease, or congenital heart disease. One of the causes of cardiomyopathy is metabolic or endocrine dysfunction, such as Cushing's disease, hypothyroidism, hyperthyroidism, pheochromocytoma, chronic hypocalcemia, hypophosphatemia, and congenital metabolic disorders. Thyroid cardiomyopathy is a form of heart muscle disease that occurs due to the toxic effects of excessive thyroid hormones on the heart. Patients with thyroid cardiomyopathy may exhibit symptoms of heart failure such as dyspnea, fatigue, and peripheral oedema. Echocardiographic findings typically include left ventricular hypertrophy, chamber dilation, and reduced ejection fraction, as seen in this patient. The goal of treatment for patients with thyroid cardiomyopathy is to restore euthyroid and manage cardiovascular manifestations using oral antithyroid medications. Proper management of hyperthyroidism is crucial in preventing these cardiac complications, but adherence to treatment remains a challenge for many patients.[7]

Hyperthyroidism affects about 1-2% of the global population, with prevalence variations based on geographic, ethnic, and age factors. Women are significantly more likely to be affected by this condition compared to men, and it is most commonly

diagnosed in individuals aged 20-40 years [7]. Several epidemiological studies show varying rates of heart disease incidence in thyrotoxicosis. Although there are no publications in Indonesia reporting the incidence rate of cardiomyopathy in patients with thyrotoxicosis, global data indicates that about 1% of thyrotoxicosis patients develop DCM, leading to severe left ventricular dysfunction and resulting in cardiogenic shock.[3]

The management of hyperthyroidism in Indonesia poses unique challenges due to varying levels of access and awareness of healthcare services, highlighting the need for public health interventions which can hit the right target and improvements in diagnostic and treatment facilities. In this case, a 35-year-old woman with a history of hyperthyroidism presented with symptoms of heart failure and cardiogenic shock. Although she was diagnosed with hyperthyroidism in 2021, she had not been compliant with her treatment. This lack of compliance likely contributed to the progression of her condition, culminating in her acute symptoms. The clinical manifestations of her disease, including shortness of breath, leg swelling, and chest pain, underscore the severe impact of untreated hyperthyroidism on heart function.

This case report emphasizes the importance of recognizing and managing hyperthyroidism to prevent severe cardiac complications. This also illustrates the need for regular follow-up and patient education to ensure adherence to prescribed treatments. Through this case, we aim to raise awareness about the cardiovascular risks associated with hyperthyroidism and the importance of early and sustained intervention.

Case Presentation

A 35-year-old woman presents with acute onset shortness of breath that began one day ago. The dyspnea initially occurred during light activities and did not improve with rest, leading to significant discomfort and causing her to wake up frequently at night. Alongside the shortness of breath, the patient also reports swelling in both legs that started simultaneously with the dyspnea. Additionally, she experiences palpitations and occasional chest pain.

The patient's medical history reveals a previous hospitalization in 2021 for similar complaints, during which she was admitted to the Intensive Cardiac Care Unit (ICCU). It was then that she was diagnosed with hyperthyroidism. Despite the diagnosis, she has not been diligent in taking her prescribed medication, thiamizole, 3x10mg, and has not taken it for a significant period. The patient denies any history of diabetes mellitus, hypertension, or stroke, which are often associated with cardiovascular issues. There is no relevant family medical history of similar conditions, indicating that her current health issues may be isolated rather than hereditary. The lack of medication adherence since her last hospitalization could have contributed

to the exacerbation of her symptoms, leading to her current acute presentation. Given the combination of respiratory distress, peripheral oedema, palpitations, and chest pain, along with a history of untreated hyperthyroidism, a comprehensive evaluation and management plan is essential to address her condition and prevent further complications.

On physical examination, the patient is conscious (compos mentis) but appears to be in severe distress. Her Glasgow Coma Scale (GCS) score is E4V5M6. Vital signs reveal a blood pressure of 84/60 mmHg, a heart rate of 110 beats per minute, a respiratory rate of 26 breaths per minute, a temperature of 36.6°C, and an oxygen saturation of 99% on a Non-Rebreather Mask at 12 liters per minute.

General examination shows no signs of anemia or jaundice in the eyes, but there is an elevated JVP observed in the neck. Examination of the ear, nose, and throat reveals no abnormalities. Cardiovascular assessment reveals normal S1S2 heart sounds with the murmur regurgitation. Respiratory examination indicates vesicular breath sounds with fine crackles bilaterally, but no wheezing. The abdominal examination shows normal bowel sounds, no distension, and no tenderness on palpation. Examination of the extremities reveals bilateral leg oedema, warm extremities, and a capillary refill time of less than 2 seconds.

Overall, the physical examination indicates signs of severe distress with cardiovascular and respiratory system involvement, highlighting the critical nature of the patient's condition.

The additional examinations conducted provide further insight into the patient's condition. The ECG reveals sinus tachycardia at a rate of 110 BPM (Figure 1). A chest X-ray shows cardiomegaly (Figure 2), indicating an enlarged heart which is consistent with the patient's symptoms and history of hyperthyroidism.

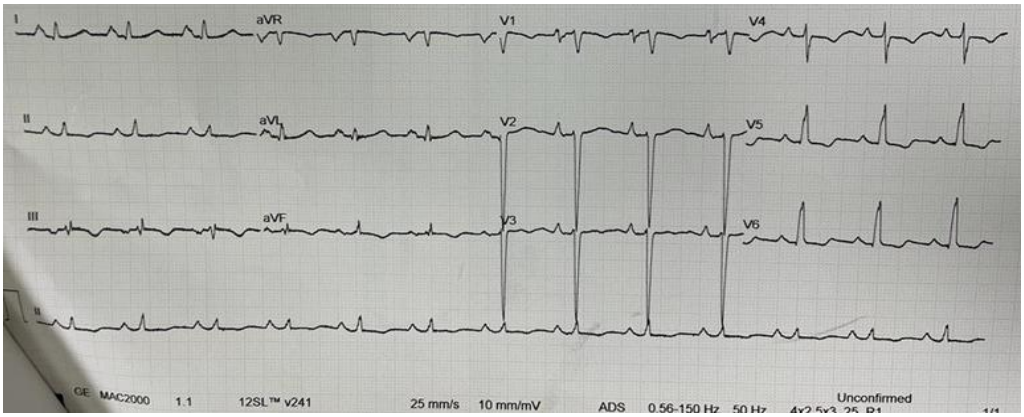

Figure 1. The electrocardiogram (ECG) of the patient reveals sinus tachycardia at a rate of 110 BPM.

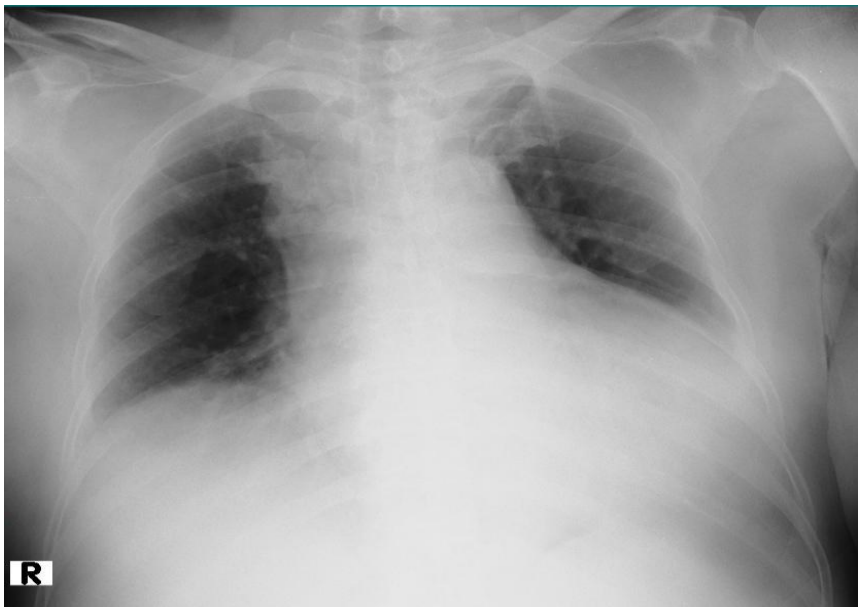

Figure 2. Chest X-Ray shows an enlarged heart which is consistent with the image of cardiomegaly.

Laboratory findings reveal several abnormalities: a complete blood count shows thrombocytopenia with platelets at 127 x 10^3/µL, leukocytosis with white blood cells at 16.04 x 10^3/µL, and a slightly reduced hemoglobin level at 11.6 g/dL. Hematocrit is 36.0%, and red blood cells count is 4.62 x 10^6/µL. Electrolyte tests indicate hyponatremia with sodium at 127 mmol/L, while potassium and chloride levels are within normal ranges at 4.1 mmol/L and 101 mmol/L, respectively. Renal function tests show elevated urea at 89.3 mg/dL and creatinine at 1.22 mg/dL, suggesting some degree of renal impairment. Liver function tests reveal significantly elevated SGPT at 1130 U/L and a moderately increased SGOT at 43 U/L. Total protein is 6.39 g/dL, with albumin at 3.29 g/dL. Procalcitonin is significantly elevated at 19.54 ng/mL, and calcium is low at 7.5 mg/dL. Thyroid function tests show a low Thyroid-Stimulating Hormone (TSH) level at 0.22 µIU/mL and elevated Free T4 (FT4) at 26.29 pmol/L, consistent with hyperthyroidism. Echocardiography (Figure 3) performed reveals dilatation of all cardiac chambers, eccentric Left Ventricular Hypertrophy (LVH), severely reduced left ventricular systolic function with an EF of 30%, grade III diastolic dysfunction, normal right ventricular contractility, global hypokinesis, mild mitral regurgitation, an Estimated Right Atrial Pressure (eRAP) of 15 mmHg, and a normal pericardium. The findings are consistent with cardiomyopathy. Overall, these additional examinations suggest a complex interplay of cardiovascular, respiratory, renal, and metabolic dysfunctions, likely exacerbated by the patient's untreated hyperthyroidism, contributing to her acute presentation.

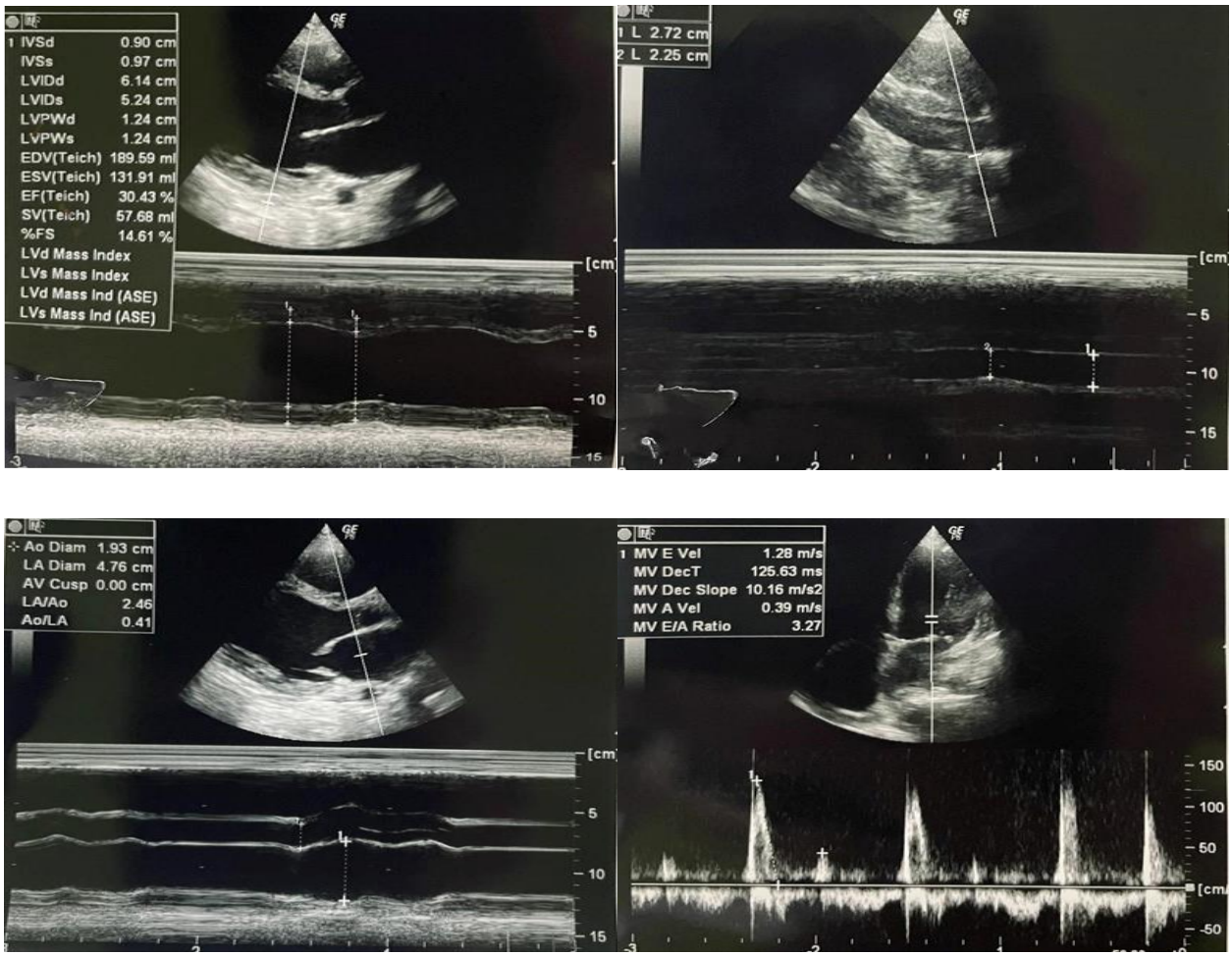

Figure 3. Echocardiography shows dilatation of all cardiac chambers, eccentric LVH, severely reduced left ventricular systolic function with an EF of 30%, grade III diastolic dysfunction, normal right ventricular contractility, global hypokinesis, and mild mitra

Upon admission to the hospital, the patient with a diagnosis DCM related to hyperthyroidism was started on a comprehensive treatment regimen. The therapeutic approach aimed to stabilize her hemodynamic status, manage her heart failure symptoms, address her thyroid condition, and prevent thromboembolic complications. The patient was initiated on intravenous fluid therapy with normal saline (IVFD NS) at a rate of 8 drops per minute to maintain adequate hydration and support her intravascular volume status. Additionally, to improve her cardiac output and address the cardiogenic shock, a continuous infusion of dobutamine was administered at a dose of 5 µg/kg body weight per minute. Dobutamine, a beta-adrenergic agonist, helps enhance myocardial contractility and increase cardiac output, which is crucial in managing cardiogenic shock. Given the patient's significant fluid overload and oedema, a continuous infusion of furosemide at 20 mg per hour (the maximal dose) was initiated. Furosemide, a potent loop diuretic, is essential for promoting diuresis and reducing fluid

retention. However, due to furosemide in maximal dose, acetazolamide was added at a dose of 250 mg once daily. Acetazolamide, a carbonic anhydrase inhibitor, enhances the diuretic effect by promoting the excretion of bicarbonate, sodium, and water, thus aiding in effective fluid management.

The patient was also started on spironolactone at a dose of 50 mg once daily and ramipril at a dose of 2.5 mg once daily. Spironolactone, an aldosterone antagonist, helps in reducing fluid retention and preventing cardiac remodeling, while ramipril, an Angiotensin-Converting Enzyme (ACE) inhibitor, aids in lowering blood pressure and reducing afterload, thereby improving cardiac function. To correct hypocalcemia, calcium carbonate (CaCo3) was administered at a dose of one tablet twice daily. This supplementation is crucial for maintaining normal cardiac and neuromuscular function. Enoxaparin, a low molecular weight heparin, was given at a dose of 0.4 cc subcutaneously once daily to prevent thromboembolic events, which are a significant risk in patients with heart failure and reduced mobility. This comprehensive therapeutic approach was aimed at stabilizing the patient's condition, optimizing cardiac function, managing symptoms, and preventing complications. The patient's response to this treatment regimen was closely monitored, with adjustments made as necessary to ensure the best possible outcomes.

Discussion

Hyperthyroidism can cause heart failure through several mechanisms. Excess thyroid hormones increase the metabolic rate and oxygen consumption, thereby placing greater demands on the cardiovascular system. They also increase beta-adrenergic receptor sensitivity, resulting in increased heart rate, contractility, and cardiac output. Chronic exposure to high levels of thyroid hormones can lead to structural changes in the heart, such as left ventricular hypertrophy and dilation, as well as functional impairments such as diastolic dysfunction. These changes can culminate in thyroid cardiomyopathy, a condition characterized by heart failure symptoms in the context of hyperthyroidism. The patient's echocardiographic findings of enlarged heart chambers and reduced ejection fraction are consistent with this pathophysiology.[8]

The goal of treatment for patients with thyroid cardiomyopathy is to restore euthyroid and manage cardiovascular manifestations using oral antithyroid medications. The treatment of cardiomyopathy follows the standard heart failure therapy, initially involving salt restriction with diuretics if there is excessive fluid volume, vasodilator therapy such as ACE Inhibitors or *Angiotensin Receptor Blockers* (ARBs), beta-blockers in hemodynamically stable patients, and adding aldosterone antagonists for patients with persistent symptoms. Thyroid cardiomyopathy poses unique therapeutic

challenges. Hyperthyroidism can lead to a hyperdynamic circulatory state, increasing cardiac output and metabolic demands, which over time can result in heart failure. In such cases, the addition of acetazolamide, a carbonic anhydrase inhibitor, can be beneficial. Acetazolamide enhances diuresis by inhibiting bicarbonate reabsorption in the proximal tubules, thereby increasing sodium and water excretion.[9]

Acetazolamide is used as an adjunctive diuretic therapy in cases of furosemide resistance in heart failure. It works by inhibiting carbonic anhydrase in the proximal tubules, leading to increased excretion of bicarbonate, sodium, and water. This mechanism is complementary to the action of loop diuretics like furosemide, which act on the ascending limb of the loop of Henle. The combined use of these diuretics can enhance overall diuretic efficacy, particularly in patients who have developed resistance to loop diuretics alone. In this case, the addition of acetazolamide helped to achieve better fluid balance and symptom control, illustrating its role in the management of diuretic-resistant heart failure [10]. In the presented case, the patient's furosemide inadequate is indicated by the persistence of oedema in both legs and the severity of her symptoms despite receiving high doses of furosemide. Specifically, the patient exhibited significant bilateral lower limb oedema and signs of fluid overload, which are typical indicators of inadequate diuretic response. The continued presence of these symptoms suggests that the furosemide, despite being administered at a maximal dose of 20 mg/hour, was not sufficient to achieve the desired diuretic effect and relieve the patient's fluid retention.

Conclusion

Acetazolamide was introduced as an adjunctive therapy. Acetazolamide's mechanism of action differs from that of furosemide, as it inhibits carbonic anhydrase in the proximal tubules, thereby promoting the excretion of bicarbonate, sodium, and water. This complementary approach helps overcome diuretic resistance and improve overall fluid management in patients with DCM who do not respond adequately to loop diuretics alone. Cardiogenic shock is a severe form of heart failure where the heart is unable to pump sufficient blood to meet the body's needs, resulting in inadequate tissue perfusion and oxygenation [11]. This condition is characterized by hypotension, elevated central venous pressure, and signs of end-organ dysfunction. In this case, the patient's severe low blood pressure, along with symptoms of severe dyspnea and peripheral oedema, indicated cardiogenic shock. The management of cardiogenic shock involves hemodynamic support with inotropes like dobutamine, which increases cardiac contractility and output. Additionally, diuretics and vasodilators are used to reduce preload and

afterload, thereby improving cardiac efficiency. The use of comprehensive supportive measures, including fluid management and inotropic support, was crucial in stabilizing the patient's condition and addressing the underlying causes of her heart failure.[12]

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