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Dilated Cardiomyopathy Attributed to Hypothyroidism: Clinical Case and Literature Overview

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Abstract **Case Report**

Dilated cardiomyopathies generally carry a poor prognosis due to progressive and often irreversible myocardial dysfunction. In rare cases, a metabolic etiology may be identified, allowing for significant improvement in cardiac function with appropriate treatment. Prolonged hypothyroidism is known to reduce myocardial contractility; however, its direct role in the development of dilated cardiomyopathy with heart failure remains a matter of debate. We report the case of a patient with severe peripheral hypothyroidism associated with dilated cardiomyopathy and global heart failure. Hormone replacement therapy leading to the restoration of euthyroidism resulted in marked improvement of cardiac function. This case highlights the importance of screening for hypothyroidism in all patients presenting with dilated cardiomyopathy of unknown etiology.

Keywords: Hypothyroidism, Dilated cardiomyopathy.

INTRODUCTION

Dilated cardiomyopathy (DCM) is the most common form of cardiomyopathy. It typically follows a progressive course and is associated with a poor prognosis. DCM may be idiopathic, hereditary, or secondary to various acquired conditions such as pregnancy, chronic alcoholism, chronic anemia, toxicity from doxorubicin or other chemotherapeutic agents, ischemic heart disease, myocarditis, or infections. A metabolic etiology is rare and, when present, may respond to specific treatment, making DCM exceptionally reversible in such cases.

Although several studies have demonstrated that thyroid hormone deficiency can lead to a reversible decrease in cardiac contractility, it remains controversial whether hypothyroidism alone, in the absence of any underlying cardiac disease, can cause dilated cardiomyopathy associated with severe heart failure.

We report the case of a woman with longstanding, previously undiagnosed hypothyroidism who developed heart failure secondary to dilated cardiomyopathy. Her clinical condition improved significantly following appropriate management of the hypothyroidism.

CASE REPORT

We present the case of a 25-year-old female patient with no significant medical history and no cardiovascular risk factors, who presented to the emergency department with generalized weakness and progressively worsening resting dyspnea evolving over a period of six months.

The patient also reports weight gain, hair loss, constipation, and swelling in the legs. The general examination reveals pale skin and mucous membranes, the patient is conscious but sluggish, with a blood pressure of 125/70 mmHg, a heart rate of 110 bpm, tachypneic at 28 cycles per minute, orthopneic with signs of respiratory distress, and an oxygen saturation of 89% on ambient air. Bilateral lower limb edema, hepatjugular reflux, and jugular vein distension are present. The thyroid is not palpable. Cardiovascular examination revealed well-perceived, regular heart sounds, a systolic ejection murmur graded 3/6 at the apex, radiating towards the left axilla. Pulmonary examination showed bilateral crepitant rales with a syndrome of pleural effusion on the right basal thorax. Chest X-ray (figure 1) revealed cardiomegaly with a cardiothoracic ratio of 0.60, alveolar-interstitial syndrome, and right-sided pleuritis. The echocardiogram showed a regular sinus

rhythm with a ventricular rate of 110 beats per minute, a constant PR interval, narrow QRS complexes, and no repolarization abnormalities. The transthoracic echocardiogram (figure 2) revealed a globularly dilated left ventricle with global hypokinesis and a severely impaired ejection fraction of 23%, minimal functional mitral regurgitation due to annular dilation, a non-dilated atrium, elevated filling pressures, a high likelihood of pulmonary hypertension, and a minimal circumferential pericardial effusion without signs of hemodynamic or morphological impact.

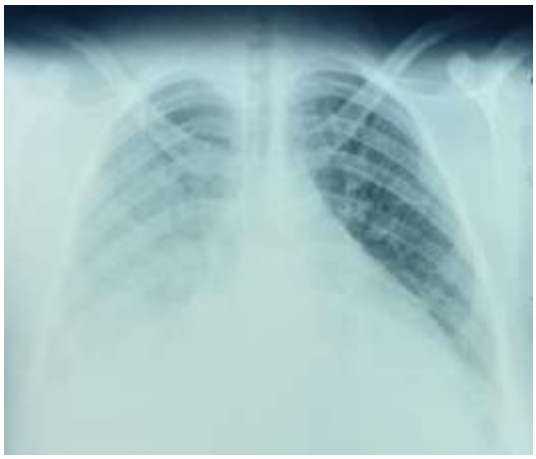

Figure 1: Chest X-ray. A posteroanterior chest X-ray view shows cardiomegaly and both pleural effusions

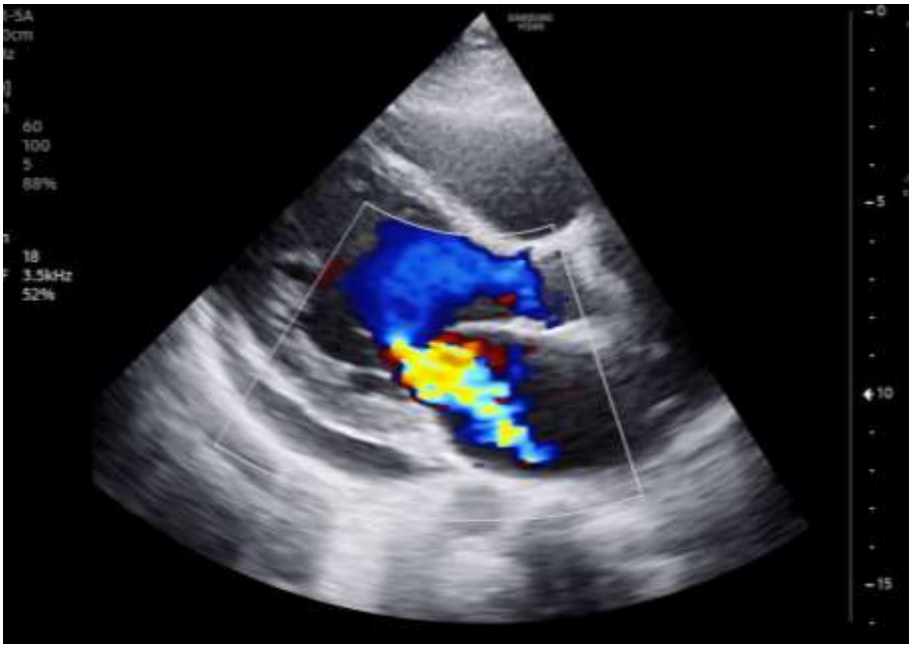

Figure 2: Parasternal long axis view showing a dilated left ventricle with mitral regurgitation and pericardial effusion

The laboratory results revealed a normochromic normocytic anemia with a hemoglobin level of 9 g/dL, white blood cells at 8370/m³, platelet count at 300,000/m³, hepatic cytolysis with ALT (13 times the normal value) and AST (4 times the normal value), cholesterol level at 2.4 g/L, and triglyceride level at 2 g/L. Thyroid hormone testing confirmed hypothyroidism, showing a decrease in free thyroxine (FT4) to 3 pmol/L and an elevation in thyroid-stimulating hormone (TSH) to 461 mUI/L. The search for antithyroid antibodies was negative. The cervical ultrasound revealed an atrophic thyroid.

The patient was started on injectable diuretics, heart failure treatment with reduced ejection fraction, including an ACE inhibitor, beta-blocker, aldosterone antagonist, and dapagliflozin, as well as hormone replacement therapy with L-thyroxine, initiated with a gradual dose escalation.

The progression was favorable, clinically marked by the regression of congestive signs and signs

of hypothyroidism. The follow-up thyroid function test performed 6 months later showed euthyroidism (table 1).

Table 1: Thyroide function tests

	Day 0	Day 60	6 months lather	12 months later
TSH	461	150	4	2
FREE T4	3	9	12	14
T3	0		120	118
TSH,thyroide-stimulating hormone.				

The follow-up echocardiogram performed after 8 months revealed near normalization of the left ventricular parameters (table 2).

Table 2: Transthoracic echocardiography

	Day 0	8 months later
LVIDd (mm)	58	50
LVIDs (mm)	54	34
LVEF (%) by method of disks	23	55
MR	Moderate to moderate-severe	Mild
LVEF, left ventricular systolic ejection fraction; LVIDs, left ventricular internal diameter end systole; LVIDd, left ventricular internal diameter end diastole.		

DISCUSSION

Thyroid hormones exert positive chronotropic, dromotropic, inotropic, and lusitropic effects on cardiac function. They induce an acceleration of heart rate, an improvement in atrioventricular conduction, an increase in myocardial contractility, and an optimization of diastolic relaxation. Furthermore, they lead to a reduction in peripheral vascular resistance through a direct vasodilatory effect [1].

A prolonged deficiency in thyroid hormones leads to various cardiovascular alterations, including:

- A decrease in cardiac output of approximately 40% compared to normal, accompanied by a proportional reduction in oxygen consumption. This decrease results from bradycardia, reduced total blood volume, and impaired myocardial contractility, which is itself linked to thyroid hormone deficiency [2].
- A normal or reduced heart rate: approximately one-third of hypothyroid patients experience bradycardia ranging from 40 to 60 beats per minute, while the remaining two-thirds maintain a normal heart rate [3].
- Increased capillary permeability leading to diffuse serous effusions, particularly at the pericardial level [4].
- An increase in peripheral vascular resistance, causing a slight rise in mean and diastolic blood pressure in 20% of cases.
- Finally, hypothyroidism is associated with hypercholesterolemia due to a reduction in total cholesterol and LDL cholesterol clearance [5], which constitutes an additional coronary risk factor, in addition to hypertension.

The exact link between hypothyroidism and its cardiovascular effects has not yet been fully elucidated. Several mechanisms have been suggested, including genomic and non-genomic actions. Thyroid hormones act at the genomic level by modulating the transcription of several cardiac genes, such as those coding for the α-myosin heavy chain, the sarcoplasmic reticulum calcium ATPase (SERCA), the Na-K-ATPase, β-adrenergic receptors, and atrial natriuretic peptide. In animals, it has been observed that in hypothyroidism, the decreased expression of myosin and SERCA, along with the increased phospholamban, are associated with impaired cardiac contractility and relaxation capacity (lusitropic properties) [7].

Heart failure, although rare, most often occurs as decompensation of a pre-existing cardiomyopathy due to a prolonged deficiency in thyroid hormones. This deficiency simultaneously leads to a decrease in cardiac output and an increase in peripheral vascular resistance [8].

Although the link between hypothyroidism and coronary artery disease is well established, the occurrence of angina or myocardial infarction remains an uncommon complication. However, the initiation of hormone replacement therapy may promote their onset due to the increase in heart rate and myocardial contractility, leading to an elevation in myocardial oxygen demands, which were previously reduced [9].

In our patient, we strictly followed the recommendations for the prescription of hormone replacement therapy [10], adding beta-blockers as a precaution to avoid revealing any underlying coronary artery disease that could be responsible for the cardiomyopathy. However, the favorable clinical

progression observed under hormone treatment suggests a myxedematous rather than ischemic origin of the cardiomyopathy.

This dilated cardiomyopathy of purely myxedematous origin has been reported in the literature; it can be severe enough to lead to heart failure [17,18]. Indeed, Myung Do Seol and Young Soo Lee [18] described the clinical case of a 36-year-old man, with no cardiovascular history, who was hospitalized with a picture of heart failure that had been progressing for several months. Echocardiography showed biventricular dilation with global wall hypokinesis, resulting in a reduction in the ejection fraction to 16%, with the LVEDd and RVEDd measured at 66 mm and 62 mm, respectively. The entire etiological workup was negative, except for the presence of clinical signs of profound hypothyroidism, which was confirmed by hormonal testing. Treatment with L-thyroxine resulted in a regression of ventricular dilation with a significant improvement in the left ventricular ejection fraction, which increased to 37% after 1 year of substitution therapy [11].

This is also the case for our patient, who presented neither known heart disease nor coronary risk factors, and in whom substitution therapy with L-thyroxine led to almost normalization of left ventricular function, alongside the disappearance of clinical signs of heart failure and hypothyroidism.

Hypothyroidism is a recognized etiology of dilated cardiomyopathy, classified among the endocrine disorders capable of inducing myocardial dysfunction [12]. Identifying this thyroidal origin is of major diagnostic importance, as hypothyroid-induced cardiomyopathy does not exhibit distinct morphofunctional features compared to other etiologies, aside from the clinical symptoms and biological abnormalities suggestive of hypothyroidism, as well as the reversibility of the cardiological condition under substitution therapy with thyroid hormone treatment.

CONCLUSION

Dilated cardiomyopathy is generally a condition with an unfavorable prognosis, progressing in a progressive and irreversible manner. However, in some cases, it may be secondary to reversible causes, such as hypothyroidism. In this context, hormone treatment with L-thyroxine, combined with angiotensin-converting enzyme (ACE) inhibitors and beta-blockers, can lead to significant improvement in myocardial function.

It is therefore essential to routinely measure thyroid hormones in all patients with dilated cardiomyopathy in order to exclude possible hypothyroidism.

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