

## When the Thyroid Hides: Dilated Cardiomyopathy as the First Clue to Undiagnosed Hypothyroidism

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### Abstract

#### Background:

Hypothyroidism commonly affects cardiovascular function, typically causing bradycardia, pericardial effusion, and diastolic dysfunction. Dilated cardiomyopathy (DCM) and heart failure may be the initial presentation of hypothyroidism, and failure to recognize the underlying thyroid dysfunction can delay appropriate therapy.

#### Case Presentation:

A 23-year-old man presented with progressive dyspnea and fatigue. Examination revealed icteric sclera and bilateral lower-extremity edema. Electrocardiography showed sinus rhythm with right axis deviation, and chest imaging demonstrated cardiomegaly. Laboratory tests revealed hyperbilirubinemia and elevated transaminases. Echocardiography showed dilation of all cardiac chambers, severely reduced left ventricular ejection fraction (26%), eccentric hypertrophy, diastolic dysfunction, mild mitral and tricuspid regurgitation, reduced right ventricular function, and elevated estimated right atrial pressure. Thyroid function tests revealed markedly elevated thyroid-stimulating hormone (240  $\mu$ IU/mL) and profoundly decreased free thyroxine (<0.038 ng/dL). The patient received standard heart failure therapy followed by levothyroxine replacement, resulting in clinical improvement.

#### Discussion:

Thyroid hormone deficiency can impair myocardial contractility, alter vascular resistance, and promote adverse ventricular remodeling. Although diastolic dysfunction is common, progression to DCM is uncommon but potentially reversible. Cardiovascular manifestations may precede overt hypothyroid symptoms, delaying diagnosis. Restoration of euthyroid status plays a pivotal role in myocardial recovery beyond conventional heart failure therapy.

#### Conclusion:

This case highlights severe hypothyroidism as a cause of DCM in young adults and emphasizes routine thyroid function testing in unexplained cardiomyopathy, despite the lack of typical endocrine symptoms.

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### Introduction

Thyroid hormone deficiency is known to cause a range of cardiac manifestations including bradycardia, pericardial effusion, diastolic dysfunction, and, in rare cases, left ventricular systolic dysfunction and dilated cardiomyopathy (DCM). DCM is characterized by dilation and impaired systolic function of the left or both ventricles in the absence of significant coronary artery disease or loading conditions. Although the majority of cases are idiopathic or associated with genetic, infectious, or toxic origins and are typically irreversible, endocrine disorders like hypothyroidism have been increasingly acknowledged as reversible causes of DCM, especially when cardiac contractility improves following thyroid hormone replacement.<sup>1,2</sup>

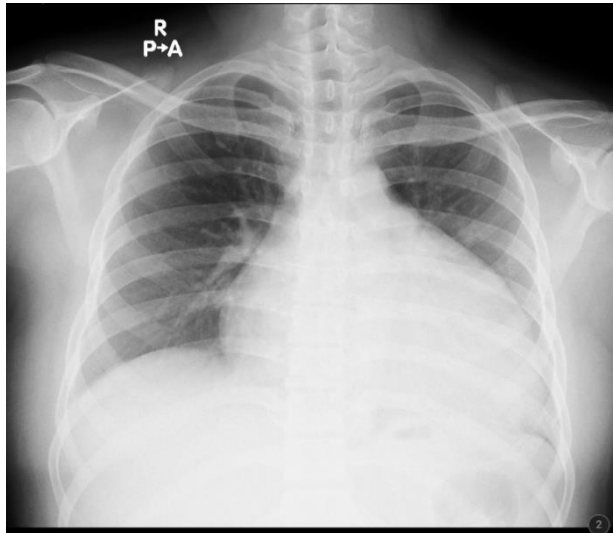
Cardiovascular manifestations may occasionally dominate the clinical picture and precede overt endocrine features, posing a diagnostic challenge. In such cases, heart failure may be the initial presentation, and failure to recognize the underlying thyroid dysfunction can delay appropriate therapy. This case highlights an unusual presentation in which dilated cardiomyopathy served as the first clinical clue to previously undiagnosed severe hypothyroidism in a young adult, underscoring the importance of routine thyroid function evaluation in unexplained cardiomyopathy.

### Case Presentation

A 23-year-old man presented to the emergency department with progressive dyspnea of two months' duration, which had worsened over time and was accompanied by marked fatigue with minimal exertion. Physical examination revealed icteric sclera and bilateral lower-extremity edema. The patient denied symptoms suggestive of hypothyroidism, such as cold intolerance, decreased sweating, constipation, depression, or irritability. His blood pressure was 103/83 mmHg, heart rate 61 beats per minute, and body mass index 28.2 kg/m<sup>2</sup>.

Electrocardiography demonstrated sinus rhythm with right axis deviation. Chest imaging showed cardiomegaly. Laboratory investigations revealed elevated total, direct, and indirect bilirubin levels, along with increased transaminase enzymes. Transthoracic echocardiography demonstrated dilation of all cardiac chambers, severely

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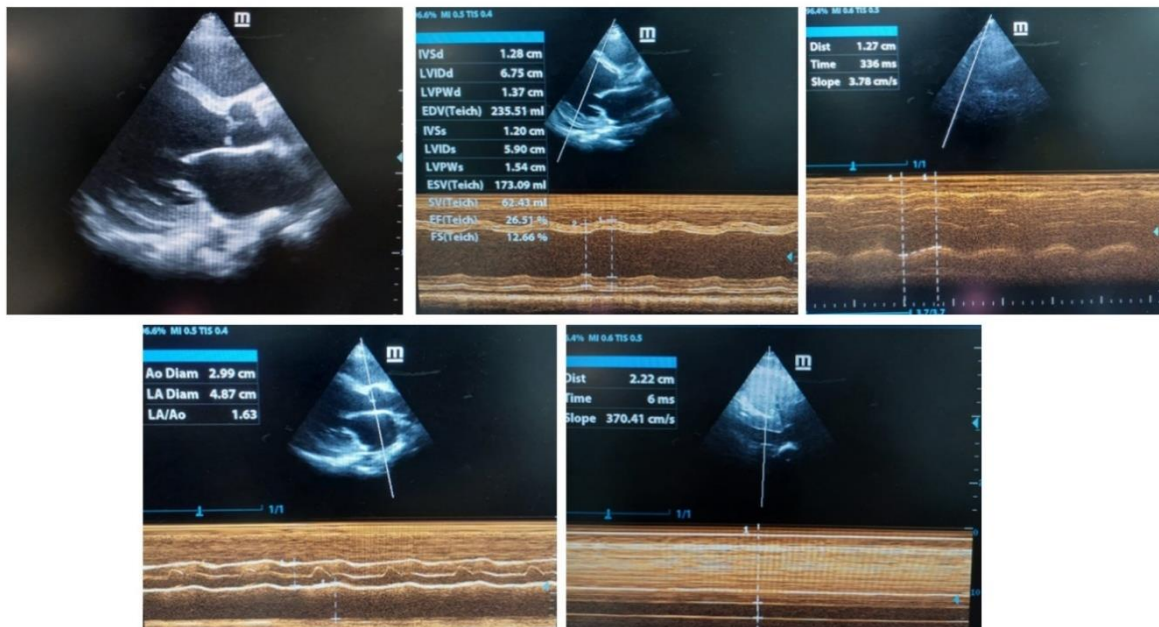
mmHg. Figure 1. Thoracic X-ray revealed

detected. Estimated right atrial pressure (eRAP) was 15 mmHg

The patient was treated with furosemide, digoxin, spironolactone, beraprost, and supportive therapy. Thyroid function tests subsequently showed a markedly elevated thyroid-stimulating hormone (TSH) level of 240  $\mu$ IU/mL and free thyroxine (FT4) < 0.038 ng/dL. The patient was started on levothyroxine and continues to receive standard heart failure management. Following five days of treatment, the patient's symptoms ameliorated, and the patient was discharged.

Discussion

This case demonstrates a rare presentation of severe hypothyroidism manifesting as dilated cardiomyopathy (DCM) with significant systolic dysfunction in a young adult. Overt hypothyroidism typically causes diastolic dysfunction, bradycardia, and pericardial effusion. However, its progression to markedly reduced left ventricular systolic function with



**Figure 2. Echocardiography showed dilation of all cardiac chambers, severely reduced global left ventricular (LV) systolic function with a left ventricular ejection fraction (LVEF) of 26% (Teichholz method), eccentric LV hypertrophy, and diastolic dysfunction with impaired relaxation. Mild mitral regurgitation due to anterior mitral leaflet prolapse and mild tricuspid regurgitation were observed. Right ventricular contractility was reduced. No spontaneous echo contrast or LV thrombus was detected. Estimated right atrial pressure (eRAP) was 15 mmHg**

reduced global left ventricular (LV) systolic function with a left ventricular ejection fraction (LVEF) of 26% (Teichholz method), eccentric LV hypertrophy, and diastolic dysfunction with impaired relaxation. Mild mitral regurgitation due to anterior mitral leaflet prolapse and mild tricuspid regurgitation were observed. Right ventricular contractility was reduced. No spontaneous echo contrast or LV thrombus was

chamber dilation is uncommon and often underrecognized in clinical settings. Thyroid hormones are pivotal in modulating myocardial contractility, systemic vascular resistance, and cardiac output, primarily through genomic effects on myocardial structural proteins and calcium-handling mechanisms. Severely lowered thyroid hormone levels affect regulatory mechanisms, resulting in reduced

contractility and adverse ventricular remodeling that manifest as DCM.<sup>3,4</sup>

The improvement of heart failure symptoms following standard heart failure therapy and subsequent institution of levothyroxine underscores two important therapeutic aspects. First, optimization of hemodynamics through diuretics and guideline-directed medical therapy provides immediate symptomatic relief. Second, correction of the underlying endocrine disturbance is crucial for long-term myocardial recovery in hypothyroidism-associated DCM.<sup>5</sup> Evidence suggests that restoration of euthyroid status can result in improvement or normalization of cardiac contractile function, reflecting the reversible nature of thyroid hormone-mediated myocardial impairment. This combined approach is particularly important in hypothyroidism-associated dilated cardiomyopathy, where restoration of euthyroid status plays a central role in facilitating myocardial functional recovery beyond the benefits of conventional heart failure therapy alone.<sup>6,7</sup>

Furthermore, the absence of classic hypothyroid systemic symptoms in this patient underscores an important clinical caveat: cardiovascular manifestations of severe thyroid hormone deficiency

may precede or predominate over typical endocrine symptoms, particularly in cases of hypothyroidism-induced dilated cardiomyopathy. In several reported cases, young adults presented primarily with symptoms of heart failure due to DCM, with minimal overt hypothyroid features, underscoring the atypical clinical course and the potential for misdiagnosis in the absence of thyroid function evaluation. These observations support maintaining a heightened suspicion of thyroid dysfunction in patients with unexplained cardiomyopathy, even in the absence of classic systemic signs of hypothyroidism.<sup>8–10</sup>

### Conclusion

This case exemplifies the complex interplay between endocrine dysfunction and cardiac pathology, highlighting the broader clinical lesson that cardiac dysfunction may predate overt endocrine manifestations. Comprehensive thyroid screening should be considered in all patients with idiopathic cardiomyopathy, given the potential for myocardial recovery with timely and appropriate hormone replacement

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