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Research Article

Reversible Dilated Cardiomyopathy Secondary to Severe Hypothyroidism: A Case Report from Indiana University Health

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ABSTRACT

Background: Dilated cardiomyopathy (DCM) can be a rare but treatable cause of severe hypothyroidism. Untimely recognition and thyroid hormone replacement therapy can lead to significant recovery of cardiac function, while delayed diagnosis can lead to more advanced heart failure. This is a very unusual case of severe hypothyroidism-induced DCM after prolonged levothyroxine treatment was stopped after complete thyroidectomy.

Case Presentation: A 45-year-old woman with a history of Graves' disease following total thyroidectomy developed gradually worsening dyspnea, orthopnea, bilateral lower extremity edema, fatigue, constipation, cold intolerance, and 40 lb weight gain after stopping levothyroxine for about five months. Laboratory tests showed severe hypothyroidism (TSH 24.5 mIU/L; free T4 0.09 ng/dL) and high levels of B-type natriuretic peptide (BNP) (496 pg/mL). An electrocardiogram showed Q waves in leads V1 and V2 and T-wave inversions in V3, V4, and V5. TTE revealed severe left ventricular (LV) dilatation, global hypokinesis and LV ejection fraction (LVEF) of ~10%. Obstructive coronary artery disease was ruled out by coronary angiography. The patient was treated with levothyroxine replacement therapy along with guideline-directed medical therapy of heart failure. At 2 months follow up, LV EF had improved to about 40% with significant clinical improvement.

Conclusion: This case is a good reminder of the role of severe hypothyroidism as a potentially reversible cause of non-ischemic dilated cardiomyopathy. In patients with an apparently unexplained case of heart failure or dilated cardiomyopathy, particularly those who have had thyroid surgery or have been non-compliant with thyroid hormone replacement therapy, it would be prudent to consider routine thyroid function testing. Early diagnosis and rapid restoration of euthyroidism and multidisciplinary management can lead to good myocardial recovery without the need for unnecessary advanced heart failure interventions.

1. INTRODUCTION

Dilated cardiomyopathy (DCM) is a phenotype for a varied myocardial condition that involves left ventricular dilatation with systolic dysfunction when there is little, if any, coronary artery disease or abnormal loading condition. It is one of the most common causes of heart failure, ventricular arrhythmias and heart transplantation in the world. While ischemic heart disease is the most common cause of ventricular dysfunction, non-ischemic causes such as genetic diseases, inflammatory diseases, metabolic abnormalities, toxic exposure, and endocrine dysfunction do make up a significant percentage of DCM cases and should be thoroughly evaluated as some of these diseases can be reversed [1].

Thyroid hormones are involved in the regulation of normal cardiovascular function as they alter myocardial contractility, heart rate, vascular tone, myocardial oxygen consumption, calcium cycling and gene expression regulating cardiac structure and function. The changes in thyroid hormone homeostasis thus have a significant effect on myocardial performance and systemic hemodynamics. Many patients with hypothyroidism have sinus bradycardia, impaired ventricular relaxation, increased systemic vascular resistance, diastolic hypertension, decreased cardiac output, and pericardial effusion. The cardiovascular defects tend to resolve after re-establishment of euthyroidism, highlighting the intimate relationship between thyroid function and cardiac performance [2].

Hypothyroidism is a condition that affects some 3–5% of the general population, and occurs much more commonly in women and in older people [3]. Cardiac symptoms are seen in many patients with untreated hypothyroidism, however,

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advanced left ventricular systolic dysfunction with dilated cardiomyopathy is very rare. Hypothyroidism induced DCM is, therefore, an underdiagnosed clinical condition, even though it is potentially reversible if diagnosed and treated quickly [4].

Thyroid hormone deficiency on its own has both non-genomic and genomic effects in the development of hypothyroid DCM, which are complex. A combination of decreased sarcoplasmic reticulum Ca^{2+}-ATPase (SERCA2) expression, changes in myosin heavy-chain isoform composition, decreased β-adrenergic receptor activity, impaired mitochondrial energy production and abnormal intracellular calcium handling all play a role in decreased myocardial contractility, ventricular remodeling and progressive heart failure. A chronically unmanaged hypothyroidism can thus result in severe reversible myocardial dysfunction [5].

Patients often have non-specific symptoms that can be confused with primary cardiomyopathy or ischemic heart disease such as fatigue, worsening dyspnea, weight gain, cold intolerance, constipation, peripheral oedema and general weakness. If the patient's cardiac dysfunction is underestimated as being from an endocrine cause, this may lead to a delay in definitive treatment and unnecessary tests or lengthened heart failure care. Therefore, thyroid function testing must be performed as a routine part of the evaluation of patients with newly diagnosed non-ischeamic DCM, especially if there is no obvious cause [1,3].

Increased awareness of thyroid-related cardiovascular disease has not led to high yield of published evidence of hypothyroidism-induced reversible DCM other than isolated case reports and small case series. The clinical features, diagnostic difficulty, response to treatment and recovery time periods are, therefore, poorly documented. Reporting more well described cases thus adds to the existing evidence base with the aim of raising awareness of this rare but treatable disease among emergency physicians, endocrinologists and cardiologists [3,5]. We report a patient who developed severe hypothyroidism, profound LVSD and decompensated HF without obstructive CAD. After levothyroxine replacement, the patient recovered with significant improvement in left-ventricular ejection fraction remarkably. The case highlights the need to understand that severe hypothyroidism is a rare but reversible cause of DCM and to stress the importance of early diagnosis and prompt thyroid replacement in restoring myocardial function.

2. CASE PRESENTATION

A 45-year woman presented to the ED with a 2-week history of increasing shortness of breath. The dyspnea started in the middle of physical activity but then worsened to low levels of activity, interfering with her quality of life. She also complained of orthopnea (difficulty breathing while lying flat) and swelling of both her lower legs. She also had generalized fatigue, cold intolerance, constipation and unintentional weight gain of about 40 pounds in the previous 5 months.

Her past medical history included a diagnosis of Graves' disease; she had previously had a total thyroidectomy. After surgery she had been prescribed lifelong levothyroxine replacement therapy. She does admit however that she stopped the treatment some 5 months prior to presentation due to lack of compliance. She reported no history of ischemic heart disease, chronic heart failure, hypertension, or diabetes mellitus, chronic kidney disease, or any other significant cardiovascular disease. No report of use of tobacco, alcohol or illicit drugs were reported. In addition, the family history of cardiomyopathy or early cardiovascular disease was negative.

The patient when presented was chronically ill with signs suggestive of volume overload. Physical exam showed bilateral lower extremity edema with CHF. She was hemodynamically stable, but was obviously dyspneic during minimal activity. The rest of the physical examination showed signs of untreated hypothyroidism with generalized fatigue and decreased exercise tolerance.

Given that her heart failure symptoms were progressive, as well as the fact that she had been off thyroid hormone replacement therapy for a long time after her thyroidectomy, a thorough investigation was launched to help identify the cause of her heart failure and to help determine the extent of cardiac involvement. Table I summarizes the patient's demographic data, pertinent medical history, and clinical presentation.

TABLE I. BASELINE DEMOGRAPHIC AND CLINICAL CHARACTERISTICS AT PRESENTATION

Variable	**Findings**
Age	45 years
Sex	Female
Past medical history	Graves' disease
Previous surgery	Total thyroidectomy
Long-term medication	Levothyroxine replacement therapy
Medication adherence	Discontinued levothyroxine for approximately 5 months
Chief complaint	Progressive shortness of breath
Duration of symptoms	Two weeks
Associated symptoms	Orthopnea, bilateral lower extremity edema, fatigue, cold intolerance, constipation, approximately 40-lb weight gain
Previous cardiovascular disease	None reported
Family history	No known inherited cardiomyopathy or premature cardiovascular disease
Social history	No reported tobacco, alcohol, or illicit drug use

3. INVESTIGATIONS

A detailed diagnostic workup was performed after initial clinical evaluation for the underlying cause of the acute decompensated heart failure and to evaluate cardiac involvement. Laboratory tests, chest X-rays, electrocardiography (ECG), and transthoracic echocardiography (TTE) were performed, as were coronary angiography. Biochemical evidence of severe Primary hypothyroidism was found in the lab. Thyroid function tests showed a very high TSH level of 24.5 mIU/L (normal range 0.40–4.50 mIU/L) and a very low level of free thyroxine (Free T4) of 0.09 ng/dL (normal range 0.8–1.8 ng/dL). The serum B-type natriuretic peptide (BNP) level was 496 pg/mL (normal <100 pg/mL), indicative of acute heart failure of myocardial stress. Chest radiography revealed only mild cardiomegaly and no focal pulmonary consolidation, nor was there evidence of pulmonary edema (Figure 1).

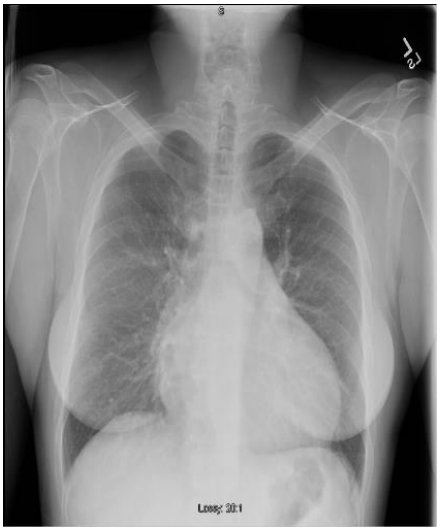

Fig. 1. Posteroanterior chest radiograph obtained on admission demonstrating mild cardiomegaly without focal pulmonary consolidation, pleural effusion, or radiographic evidence of pulmonary edema.

The 12-lead electrocardiogram revealed normal Q waves in leads V1 and V2 and T-wave inversions in leads V3 through V5 but no evidence of acute ST-segment elevation myocardial infarction. Transthoracic echocardiogram revealed severe left ventricular systolic dysfunction, with an estimated left ventricular ejection fraction (LVEF) of about 10%, and diffuse global hypokinesis (Figure 2).

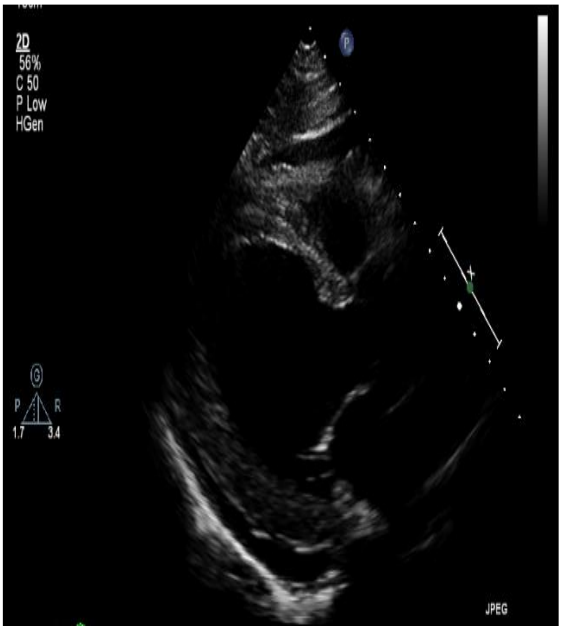

Fig. 2. Transthoracic echocardiography (parasternal long-axis view) obtained at presentation demonstrating marked left ventricular dilatation with diffuse global hypokinesis and severe left ventricular systolic dysfunction. The estimated left ventricular ejection fraction was approximately 10%.

Ischemic cardiomyopathy was the most frequent etiology of new onset severe LVDS and therefore, an invasive coronary angiography was then performed in order to assess whether there was an ischemic cause. The examination showed an angiographically normal coronary arteries with no sign of obstructive coronary artery disease, effectively excluding ischemic cardiomyopathy. The principal laboratory, radiographic, electrocardiographic, echocardiographic and angiographic findings are summarized in Table II.

TABLE II. SUMMARY OF LABORATORY AND CARDIOVASCULAR INVESTIGATIONS

Investigation	Result	Reference Range / Interpretation
Thyroid-Stimulating Hormone (TSH)	24.5 mIU/L	0.40–4.50 mIU/L (Markedly elevated)
Free Thyroxine (Free T4)	0.09 ng/dL	0.8–1.8 ng/dL (Markedly decreased)
B-type Natriuretic Peptide (BNP)	496 pg/mL	<100 pg/mL (Elevated)
Chest radiography	Mild cardiomegaly	No focal pulmonary consolidation, pleural effusion, or pulmonary edema
Electrocardiography (ECG)	Q waves in V1–V2; T-wave inversions in V3–V5	Abnormal ventricular repolarization
Transthoracic echocardiography	Marked left ventricular dilatation, diffuse global hypokinesis, LVEF ≈10%	Severe left ventricular systolic dysfunction
Coronary angiography	Angiographically normal coronary arteries	No obstructive coronary artery disease

4. DIAGNOSIS

The patient's symptoms of progressive dyspnea, orthopnea, peripheral edema, and severe LV systolic dysfunction led to a thorough differential diagnosis for newly diagnosed DCM. The ischemic cardiomyopathy was the main differential diagnosis since coronary artery disease is the leading cause of marked left ventricular dysfunction. But the invasive coronary angiography was negative and angiographically did not reveal any coronary artery obstruction, thus ruling out an ischemic cause. Another non-ischemic dilated cardiomyopathy were then explored. The possibility of viral myocarditis was raised but the patient had no history of any previous viral infection, fever, chest pain to suggest myocarditis, and other clinical features to support this diagnosis. Similarly, IDCM was ruled out as it was less likely given the presence of a clear reversible endocrine defect. The patient's medical, social, and family history also suggested that toxic, alcohol-related, and inherited cardiomyopathies were unlikely.

One important diagnostic finding was that the patient had a history of Graves' disease treated with total thyroidectomy and then without taking levothyroxine for about five months. This history, along with significantly elevated serum TSH level, and a strikingly low free T4 level, confirmed the diagnosis of severe primary hypothyroidism. The combination of these findings with the lack of significant coronary artery disease, and the echocardiographic findings of severe left ventricular systolic dysfunction, strongly supported an endocrine etiology for this patient's cardiomyopathy. Therefore, the diagnosis was made as reversible dilated cardiomyopathy due to hypothyroidism with acute decompensated heart failure. A summary of the diagnostic reasoning, the principal differential diagnoses, and the reason for excluding them from the differential diagnosis, is listed in Table III.

TABLE III. DIFFERENTIAL DIAGNOSIS AND BASIS FOR EXCLUSION

Differential Diagnosis	Basis for Exclusion
Ischemic cardiomyopathy	Coronary angiography demonstrated normal coronary arteries without obstructive coronary artery disease.
Viral myocarditis	No preceding viral illness, fever, chest pain, or clinical evidence suggestive of myocardial inflammation.
Idiopathic dilated cardiomyopathy	A reversible endocrine cause was identified through clinical history and thyroid function testing.
Toxic or alcohol-induced cardiomyopathy	No history of alcohol abuse, cardiotoxic drug exposure, or other toxic insults.
Inherited (familial) cardiomyopathy	No known family history of cardiomyopathy or premature cardiovascular disease.
Hypothyroidism-induced dilated cardiomyopathy	Supported by previous thyroidectomy, prolonged discontinuation of levothyroxine, severe biochemical hypothyroidism, severe left ventricular systolic dysfunction, and exclusion of alternative etiologies.

5. TREATMENT AND CLINICAL MANAGEMENT

After diagnosis, the patient was admitted for the care of acute decompensated heart failure due to severe underlying hypothyroidism. The treatment goal was directed toward two main targets, namely, stabilizing the heart failure and correcting the underlying thyroid hormone deficiency. Treatment started aimed at alleviating symptoms of volume overload. Intravenous diuretics were given, and dyspnea and peripheral edema improved with time. The patient was closely monitored throughout the hospital stay, with regular checks on vital signs, fluid status, kidney function, electrolyte levels and haemodynamic status, to optimize the effectiveness and safety of treatment.

Because levothyroxine therapy was discontinued for a long period of time only to be confirmed to be a case of severe hypothyroidism, treatment was immediately resumed with levothyroxine. Thyroid replacement was maintained during the

hospital stay and biochemical monitoring was performed periodically for therapeutic response and for appropriate dose adjustment. At the same time, the patient was prescribed heart failure with reduced ejection fraction (HFrEF) guideline directed medical therapy (GDMT). Pharmacological therapy was begun and optimized, based on current guidelines for heart failure treatment and patient's clinical condition and hemodynamic tolerance. Treatment was modified while in hospital depending on how well the patient responds and tolerates the treatment.

After evaluation with coronary angiography, the coronary arteries were normal and there was no evidence of obstructive coronary artery disease, and no coronary revascularization nor other invasive cardiac procedures were warranted. Therefore, the management was entirely medical, aimed at the reversible endocrine disorder, as well as the myocardial dysfunction.

During hospitalisation there was gradual improvement in the clinical condition with resolution of congestion and improvement in functional status. She was discharged in a stable condition on optimal heart failure medication, where she will also be followed-up by cardiology and endocrinology to continue to monitor for cardiac recovery and thyroid function, respectively. A summary of the therapeutic interventions is presented in Table IV.

TABLE IV. SUMMARY OF THERAPEUTIC INTERVENTIONS

Therapeutic Intervention	Purpose	Clinical Outcome / Rationale
Hospital admission	Initial stabilization and continuous monitoring	Allowed comprehensive management of acute decompensated heart failure and severe hypothyroidism
Intravenous diuretic therapy	Relief of volume overload and congestive symptoms	Improved dyspnea, peripheral edema, and overall clinical status
Levothyroxine replacement therapy	Correction of severe hypothyroidism	Addressed the underlying reversible endocrine cause of cardiomyopathy
Guideline-directed medical therapy (GDMT) for HFrEF	Improve cardiac function, reduce symptoms, and prevent disease progression	Initiated and optimized according to the patient's clinical condition
Continuous clinical and laboratory monitoring	Evaluate therapeutic response and detect complications	Included serial assessment of vital signs, renal function, electrolytes, and thyroid function
Coronary angiography	Exclusion of ischemic heart disease	Demonstrated normal coronary arteries; no coronary intervention was required
Discharge planning and multidisciplinary follow-up	Long-term management and monitoring of recovery	Continued levothyroxine therapy and heart failure medications with cardiology and endocrinology follow-up

6. OUTCOME AND FOLLOW-UP

After starting thyroid hormone replacement therapy and heart failure guideline directed medical therapy, the patient showed progressive clinical improvement. During the period of hospitalization, the symptoms of dyspnea, orthopnea and peripheral edema gradually disappeared and functional capacity and clinical condition improved. Treatment-related complications and adverse events were not significant during the inpatient course. The patient was discharged in stable condition on levothyroxine replacement and optimized heart failure medications. Outpatient follow-up care was arranged through both the cardiology clinic and endocrinology clinic to assure cardiac function, thyroid function and clinical recovery.

Following 1 month, the patient reported an improved exercise capacity and alleviation of orthopnea, plus a dramatic decrease in fatigue and edema of the lower extremities. Thyroid replacement was maintained and serial lab investigations showed progressive normalization of thyroid function. The left ventricular ejection fraction (LVEF) at 2-month follow-up of transthoracic echocardiography was approximately 40% compared with approximately 10% at presentation. There were no signs of heart failure, and he was clinically stable, which confirmed the diagnosis of reversible dilated cardiomyopathy due to severe hypothyroidism. This is a summary of the patient's clinical course in chronological order (Table V).

TABLE V. TIMELINE OF THE CLINICAL COURSE

Time Point	Clinical Events
Approximately 5 months before admission	Discontinuation of levothyroxine therapy following total thyroidectomy.
Weeks before admission	Progressive fatigue, cold intolerance, constipation, weight gain, dyspnea, orthopnea, and bilateral lower-extremity edema developed.
Hospital admission (Day 0)	Presented with acute decompensated heart failure. Laboratory investigations confirmed severe hypothyroidism. Echocardiography demonstrated severe left ventricular systolic dysfunction (LVEF ≈10%).
Hospitalization	Intravenous diuretics, levothyroxine replacement therapy, and guideline-directed medical therapy for heart failure were initiated. Coronary angiography demonstrated normal coronary arteries.
Hospital discharge	Clinical symptoms improved. Patient discharged in stable condition with optimized medical therapy and scheduled cardiology and endocrinology follow-up.
1-month follow-up	Marked symptomatic improvement with resolution of congestion and improved functional capacity. Thyroid hormone replacement therapy continued with improving thyroid function.
2-month follow-up	Repeat echocardiography demonstrated recovery of left ventricular systolic function, with LVEF improving from approximately **10%** to **40%**. The patient remained free of recurrent heart failure symptoms.

7. DISCUSSION

7.1 Hypothyroidism and Cardiac Dysfunction

Thyroid hormones are crucial regulators of normal cardiac physiology and play a role in both genomic and non-genomic mechanisms to regulate myocardial contractility, heart rate, vascular tone and cardiac energy metabolism. The expression of several cardiac genes related to calcium homeostasis, β-adrenergic signaling, myocardial contraction and vascular function is regulated by the biologically active thyroid hormone triiodothyronine (T3). Thus, long-term thyroid hormone deficiency affects the performance of the heart both systemically and locally, with decreases in systolic and diastolic function, cardiac output, systemic vascular resistance, and ultimately heart failure. Recent evidence also indicates that many of these cardiovascular abnormalities may be functional and are likely to improve significantly after re-establishment of euthyroidism [6].

While the cardiovascular consequences of hypothyroidism such as sinus bradycardia, pericardial effusion, and mild left ventricular dysfunction are well known, reversible dilated cardiomyopathy (DCM) is a rare clinical syndrome. However, hypothyroidism is a rare but important reversible cause of non-ischemic cardiomyopathy and only a few well-documented cases have been reported in the literature. Failure to recognize this condition could delay the timely initiation of endocrine treatment, as well as delay patients from undergoing unnecessary advanced heart failure therapies or diagnostic tests [7].

The current case presents some interesting points. Firstly, the patient had severe LVEF (≈10%) following long-term cessation of levothyroxine replacement after total thyroidectomy. Secondly, extensive testing including coronary angiography revealed no obstructive coronary artery disease, lending credence to an endocrine cause. Last but not least, the significant improvement of left ventricular systolic function following the re-initiation of levothyroxine, combined with the administration of guideline-directed medical therapy strongly supports the diagnosis of reversible hypothyroidism-induced dilated cardiomyopathy.

Clinically, this case highlights the need to periodically monitor thyroid function in heart failure patients with unknown etiology or in patients with a newly diagnosed dilated cardiomyopathy, especially in those without a clear history of ischemic heart disease. Severe hypothyroidism can be identified early and thyroid hormone replacement therapy can be commenced which may prevent unnecessary invasive procedures or advanced heart failure intervention and reverse myocardial dysfunction. These observations correspond to recent systematic reviews that showed that in "appropriate" patient's levothyroxine replacement produced significant improvements in cardiac function [8].

Taken as a whole, this case underscores the importance of hypothyroidism as a reversible and possible cause of non-ischemic dilated cardiomyopathy. The pathophysiological mechanisms by which thyroid hormone deficiency results in changes in myocardial structure and function are of critical importance for early diagnosis, proper treatment and best clinical outcomes. These mechanisms are discussed in the following section.

7.2 Pathophysiological Mechanisms

Cardiovascular effects of hypothyroidism are mainly caused by the lack of the biologically active thyroid hormone, triiodothyronine (T3), which controls myocardial structure, contractility, electrophysiology and vascular function. The actions of T3 are mediated both by genomic actions (by binding to nuclear thyroid hormone receptors and by the modulation of cardiac gene transcription) and non-genomic actions (by rapid modulation of membrane ion channels, intracellular calcium handling, mitochondrial activity and function of vascular smooth muscle). Thus, long-term T3 deficiency leads to chronic molecular and hemodynamic changes progressively affecting cardiac performance [9].

Thyroid hormone deficiency results in a decrease in the levels of multiple proteins required for normal myocardial contraction at the genomic level. This is particularly with a decrease in sarcoplasmic reticulum Ca^{2+}-ATPase (SERCA2), which pumps calcium back in to the sarcoplasmic reticulum during diastole. At the same time the activity of phospholamban, which inhibits SERCA2, is relatively increased, thus worsening intracellular calcium cycling. These changes slow down relaxation of the myocardium and impair its function for the next contraction. Concomitantly, hypothyroidism causes a down-regulation of beta-adrenergic receptors, with reduced sensitivity to sympathetic stimulation, decreased chronotropic and inotropic reserve and impaired cardiac adaptation to physiological stress [10].

The progressive decline in myocardial contractility is a result of the interplay between disturbed calcium balance and decreased β-adrenergic responsiveness. The heart rate is often reduced as a result of decreased sinoatrial node activity, and stroke volume decreases as the efficacy and strength of the heart's contractions are decreased. All of these changes combine to cause a great decrease in cardiac output, which decreases the amount of oxygen available in the body, and help cause exercise intolerance, fatigue, and heart failure symptoms. Recent imaging studies also indicate that these abnormalities are mostly functional, and might be reversible after restoration of euthyroidism, thus echoing the notion of reversible myocardial remodeling during hypothyroidism [11].

Hypothyroidism also significantly affects systemic vascular physiology as well as myocardial function. This results in a decrease in the production of endothelial nitric oxide (NO), which leads to peripheral vasoconstriction and increased vascular smooth muscle tone and therefore increased systemic vascular resistance (SVR). This raised afterload further impairs left ventricular systolic function, adding to the already poor left ventricular function. Renal hypoperfusion also

stimulates neurohormonal mechanisms that will lead to sodium and water retention, causing peripheral edema and exacerbation of congestive heart failure [12].

If these molecular, cellular and hemodynamic changes are unresolved due to chronic untreated hypothyroidism, chronic ventricular remodeling can develop. Ultimately, in the presence of progressive ventricular dilatation, decreased systolic function, and global hypokinesis, a dilated cardiomyopathy (DCM) may develop. Of note, hypothyroid-induced DCM is reversible if thyroid hormone replacement is started before irreversible myocardial fibrosis occurs, unlike many other forms of non-ischemic cardiomyopathy. This pathophysiological sequence closely resembles that of the present patient, who experienced a dramatic recovery of LV systolic function after reintroduction of levothyroxine replacement after about five months of its discontinuation, and a dramatic decline of function after interruption of levothyroxine therapy.

In (Figure 3), the proposed pathophysiological pathway between PTHD and reversible dilated cardiomyopathy is shown. Continued low levels of T3 result in a sequence of genomic and non-genomic changes which affect the function of intracellular calcium handling, impair myocardial contractility, raise systemic vascular resistance, and ultimately trigger ventricular remodeling and heart failure.

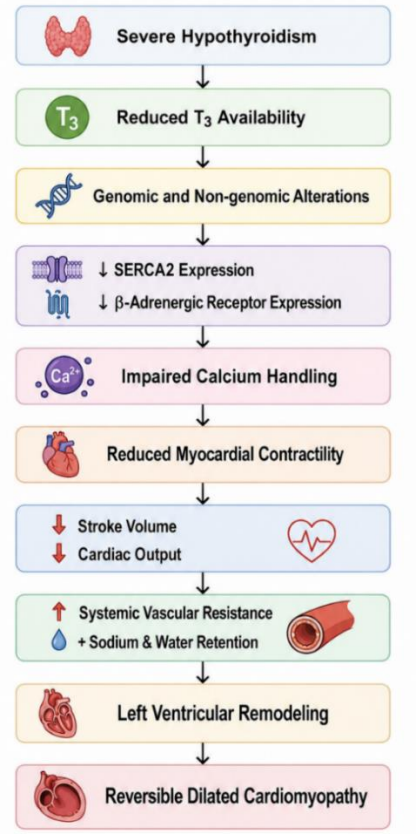

Fig. 3. Proposed Pathophysiological Mechanisms of Hypothyroidism-Induced Dilated Cardiomyopathy.

7.3 Review of Recent Literature (2022–2025)

Hypothyroidism-induced dilated cardiomyopathy (DCM) is a relatively rare but clinically important potentially reversible myocardial dysfunction. A literature search revealed only a few relevant case reports, and most of the evidence published was since 2022. This is not indicative of a lack of clinical relevance of the association of severe systolic cardiomyopathy and hypothyroidism, but rather a lack of its occurrence. The recent literature, however, does show a fairly uniform presentation of affected patients which includes a history of symptomatic heart failure, LV dilatation, global hypokinesis, and severely depressed EF, with the thyroid evaluation often finding overt or occasionally subclinical hypothyroidism [13].

In 2023, a pediatric report detailed a hypothyroidism related DCM in an adolescent boy that was severe enough to consider for a heart transplant. It was found that, while the initial treatment with levothyroxine alone was not sufficient to obtain clinical and cardiac recovery, the introduction of liothyronine was linked to clinical and cardiac recovery. Though not applicable to all patients, this treatment approach would seem to be clinically important in some who continue to have myocardial dysfunction despite optimal conventional T4 replacement therapy, and the report suggests that inadequate peripheral T3 availability may be a factor in these patients. This should not, however, be taken as proof that combination therapy is consistently needed for hypothyroidism-induced cardiomyopathy [13].

In 2023, Mennani et al were able to report two paediatric cases where subclinical hypothyroidism occurred in the setting of severe DCM. LVEF was 15% at the time of admission for the first patient (an 11-year-old female), and 10% for the second patient (a 9-year-old child); TSH was 11.2 mIU/L at admission for the first patient and 7.7 mIU/L for the second patient. These are interesting cases because the biochemical thyroid changes were not that severe in comparison to the degree of ventricular dysfunction. Thus, they show an association but not that subclinical hypothyroidism was the only reason for the development of DCM. They also are quite different from the current adult case, where severe hypothyroidism occurred following documented discontinuation of levothyroxine after thyroidectomy [14].

In 2024 Tabatabai and Barzargani presented a case of a 37-year-old man with heart failure, global left ventricular hypokinesis, severely impaired left ventricular systolic function, severe hypothyroidism and normocytic normochromic anemia. Severe anemia is a significant contributing factor that could be responsible for myocardial dysfunction and preclude the ability to assign the cardiomyopathy solely to hypothyroidism. The report, however, reiterates the importance of systematic thyroid-function testing if a case of otherwise unexplained DCM is detected [15].

Sani et al. published a more directly comparable case of an adult in 2025. A 25-year-old woman with no previous diagnosis of hypothyroidism complained of increasing dyspnea, peripheral edema, and heart failure in general. Echocardiography showed dilated left ventricle, global hypokinesis and an LVEF of 23%. Gradually introduced levothyroxine, diuretics, angiotensin-converting enzyme inhibitor, beta blocker, aldosterone antagonist and dapagliflozin were used. Follow-up revealed resolution of heart-failure symptoms and almost normalization of left ventricular function, without specifying the exact LVEF or the exact follow-up period [16].

In another report of congenital hypothyroidism in an adult, heart failure and DCM were reported after stopping long-term thyroxine replacement in 2002. This report is pertinent to the current situation as both incidents were caused by a lack of essential thyroid replacement as opposed to a new diagnosis of primary spontaneous hypothyroidism. All of these findings suggest that, in individual patients who are totally or fully dependent on exogenous thyroid hormone, discontinuation of thyroid medications might lead to a clinically significant myocardial deterioration [17].

There is an agreement across all of the recent reports that heart failure with severe myocardial dysfunction is often improved on correction of hypothyroidism, especially with the initiation of guideline directed medical therapy for heart failure. But recovery is not always equal in quantity or quality, and can take many forms. This variability is probably related to the duration and intensity of the thyroid hormone deficiency, to the ventricular impairment at the beginning of the treatment, to the age of the patients, to the compliance with the treatment, to the coexistence of myocardial stressors, and to the presence or not of irreversible fibrosis. Evidence published is so far limited to isolated case reports and few of the cases received thyroid replacement drugs in addition to heart-failure drugs. Thus, the effect of levothyroxine could not be specifically attributed to its sole effect on the heart.

The present patient had a few unique characteristics when compared with the recent literature summarized in Table VI. First, she had a well-defined precipitating mechanism – withdrawal of levothyroxine for about 5 months following total thyroidectomy for Graves' disease. Second, her initial LVEF of about 10% is an extremely high level of systolic dysfunction, and lower than that reported by Sani et al. in the recent adult case (23%). Thirdly, coronary angiography excluded ischemic cardiomyopathy due to normal coronary arteries. In addition, a relatively rapid reverse remodeling despite severely depressed LVEF at presentation (improving to about 40% within two months) supports relatively rapid reverse remodeling after presentation. This is especially so given that levothyroxine and guideline-directed medical therapy were begun at the same time, and thus it is difficult to determine whether recovery was due to the thyroid replacement therapy or to the other components of the heart-failure treatment plan.

TABLE VI. COMPARISON OF RECENTLY PUBLISHED CASES OF HYPOTHYROIDISM-ASSOCIATED DILATED CARDIOMYOPATHY

Author	**Year**	**Age/Sex**	**Thyroid disorder or precipitating factor**	**TSH**	**Initial cardiac findings**	**Treatment**	**Reported cardiac outcome**
Choi et al. [13]	2022	Adolescent/M	Hypothyroidism; specific etiology NR in accessible report	NR	Severe DCM; heart transplantation was considered	Levothyroxine followed by combined levothyroxine and liothyronine therapy	Cardiac improvement reported after combination therapy
Mennani et al. (Case 1) [14]	2023	11/F	Subclinical hypothyroidism	11.2 mIU/L	LV dilatation, global hypokinesis, LVEF 15%	Thyroid and heart-failure management reported	Exact final LVEF and recovery interval NR
Mennani et al. (Case 2) [14]	2023	9/NR	Subclinical hypothyroidism	7.7 mIU/L	Severe DCM, LVEF 10%	Thyroid and heart-failure management reported	Exact final LVEF and recovery interval NR
Tabatabai & Barzargani [15]	2024	37/M	Severe hypothyroidism with severe normocytic anemia	NR	Global LV hypokinesis and severely depressed systolic function	Thyroid replacement and clinical management	Follow-up values NR in the accessible report
Sani et al. [16]	2025	25/F	Previously undiagnosed severe	461 mIU/L	Dilated LV, global hypokinesis,	Gradual levothyroxine	Resolution of clinical

			hypothyroidism with atrophic thyroid		LVEF 23%, mild pericardial effusion	replacement, diuretics, ACE inhibitor, beta-blocker, aldosterone antagonist, and dapagliflozin	manifestations and near-normalization of LV function; exact final LVEF NR
Njoku et al. [17]	2025	Adult/NR	Congenital hypothyroidism; withdrawal of thyroxine supplementation	NR	Heart failure associated with DCM	Reinstitution of thyroid replacement with heart-failure management	Improvement reported; exact serial LVEF data NR in accessible indexing record
Present case	Current study	45/F	Post-thyroidectomy hypothyroidism after approximately five months of levothyroxine discontinuation	24.5 mIU/L; free T4 0.09	Dilated LV, severe global hypokinesis, LVEF ≈10%; normal coronary arteries	Levothyroxine plus guideline-directed medical therapy	LVEF improved to ≈40% after two months

Abbreviations: ACE, angiotensin-converting enzyme; DCM, dilated cardiomyopathy; F, female; LVEF, left ventricular ejection fraction; LV, left ventricle; M, male; NR, not reported or not available in the accessible publication; TSH, thyroid-stimulating hormone.

The recent literature as a whole allows us to draw four major conclusions. First, hypothyroidism must be included in the workup of an unknown etiology of non-ischemic DCM. Second, recovery can be seen even in the setting of severely decreased baseline systolic function. Third, the length of time required for improvement is not predictable by the initial TSH concentration and varies from weeks to several months. Lastly, the interpretation of causal relationships is limited because of the observational nature of case reports, inadequate reporting of serial echocardiographic measurements, and concurrent use of thyroid replacement and other agents for heart failure. The present case contributes clinically relevant data, as it demonstrates a clear temporal association between levothyroxine discontinuation, severe thyroid hormone deficiency, severe non ischemic ventricular dysfunction and significant improvement following thyroid hormone replacement therapy.

7.4 Comparison with the Present Case

In the present case, the authors offer additional rare clinical and therapeutic features which add to the growing body of literature supporting hypothyroidism as a cause of reversible myocardial failure. Previous reports have uniformly demonstrated improvement in cardiac function with thyroid hormone replacement therapy, but the present patient had severe ventricular dysfunction, a definite precipitating event, a thorough exclusion of other causes, and a swift recovery of cardiac function after treatment.

The most striking aspect of this case was the acute onset of LVSD at presentation. Transthoracic echocardiography revealed a grossly dilated left ventricle with severe global hypokinesis and an LVEF of ~10%, which was one of the lowest baseline LVEFs reported in recent adult cases of hypothyroidism-associated DCM. Even though myocardial dysfunction was relatively severe, significant recovery of ventricular function was seen within 2 months of treatment, suggesting that ventricular dysfunction may be reversible if therapy is provided before irreversible structural changes occur.

Another factor that distinguished was that there was a clearly identified precipitating cause. The current patient was a case of hypothyroidism, which was not the first symptom of an unknown thyroid condition, as many of the reported cases were; instead, she was a patient who had received total thyroidectomy for Graves disease, and had stopped taking levothyroxine for about five months. As thyroid hormone production had been stopped altogether, the interruption of replacement treatment for a considerable period led to severe thyroid hormone deficiency and an obvious temporal relationship between hormone deficiency and the onset of severe cardiomyopathy. This is a clear chronology that greatly enhances the clinical inference of causality.

The present report differs from several previously reported cases with the diagnostic work-up. Electrocardiography showed anterior Q wave and T-wave inversion, which may indicate ischemia, but there was no evidence of it on the invasive coronary angiogram, which revealed normal coronary arteries. The observation effectively ruled out obstructive coronary artery disease as the key problem in the development of ventricular dysfunction, and greatly boosted the assurance that the cardiomyopathy observed was a secondary complication of severe hypothyroidism rather than ischemic myocardial injury. An echocardiogram was performed which revealed diffuse global hypokinesia instead of regional wall-motion abnormalities, which further supported a non-ischemic mechanism.

Favorable therapeutic response was also an important feature. Reinstated with levothyroxine in combination with guideline directed medical therapy (GDMT) for heart failure, the patient has had a significant improvement in symptoms accompanied by a rise in LVEF from ~10% to 40% in just 2 months. This is a level of reverse ventricular remodeling that is comparable to what was recently reported in the literature and further supports the notion that hypothyroidism-induced DCM is a potentially reversible non-ischemic cardiomyopathy. It should be noted, however, that myocardial recovery

occurred during concurrent initiation of thyroid hormone replacement and GDMT, and thus must be viewed as the outcome of all aspects of multidisciplinary treatment, not just thyroid hormone replacement alone.

This case adds a number of clinically relevant observations to the current literature. It outlines one of the most dramatic decline in baseline LVEF, shows a clear time lag between total thyroidectomy and cardiac decompensation in the absence of coronary angiography for confirmation of the absence of ischemic heart disease, and demonstrates remarkable recovery of ventricular function over a relatively brief follow-up period. The results of the investigation highlight the need for comprehensive evaluation of the thyroid gland in patients with otherwise idiopathic dilated cardiomyopathy (DCM) and the need to routinely monitor thyroid function in these patients, as well as to test for non-compliance to treatment.

Comparing the present case with the recent literature highlights the unique features of the case. Despite the fact that hypothyroidism-induced dilated cardiomyopathy has been reported in several cases and has been termed a potentially reversible disease, the present case had a number of unusual clinical and laboratory characteristics. These include an extremely low LVEF at presentation, a known history of prolonged cessation of levothyroxine therapy after total thyroidectomy, angiographic exclusion of obstructive CAD and rapid recovery of LVEF from thorough treatment. Together, these features make the present report unique when compared with past reports, contributing to its educational and clinical value. A summary of the main distinguishing characteristics is given in Table VII.

TABLE VII. DISTINCTIVE CLINICAL FEATURES OF THE PRESENT CASE COMPARED WITH RECENTLY PUBLISHED REPORTS

Clinical Feature	**Recently Published Cases (2022–2025)**	**Present Case**	**Clinical Importance**
Underlying thyroid disorder	Newly diagnosed primary or autoimmune hypothyroidism in most reports	Severe hypothyroidism following total thyroidectomy with approximately 5 months of levothyroxine discontinuation	Provides a clear temporal cause-and-effect relationship between hormone withdrawal and DCM.
Initial LVEF	Generally severe impairment (approximately 15–30% in most adult reports)	≈10%	Represents one of the most severe reported reductions in left ventricular systolic function.
Coronary artery assessment	Frequently not fully documented	Normal coronary angiography	Confidently excludes ischemic cardiomyopathy.
Ventricular dysfunction	Dilated LV with global hypokinesis	Dilated LV with severe global hypokinesis	Supports a diffuse non-ischemic myocardial process.
Therapeutic approach	Levothyroxine with standard heart failure therapy	Levothyroxine plus guideline-directed medical therapy (GDMT)	Reflects comprehensive multidisciplinary management.
Cardiac recovery	Improvement generally reported over weeks to months	LVEF improved from ≈10% to ≈40% within 2 months	Demonstrates rapid reverse ventricular remodeling despite profound baseline dysfunction.
Educational contribution	Highlights reversibility of hypothyroidism-induced DCM	Emphasizes lifelong adherence to levothyroxine after thyroidectomy and the importance of early recognition of reversible cardiomyopathy	Provides a practical message for both endocrinologists and cardiologists.

Abbreviations: DCM, dilated cardiomyopathy; GDMT, guideline-directed medical therapy; LVEF, left ventricular ejection fraction; LV, left ventricle.

7.5 Clinical Implications

The present case has several clinically relevant implications that go beyond the current case and may be useful for diagnosis and therapy of unexplained non-ischemic dilated cardiomyopathy. Hypothyroidism is a well described endocrine disorder but a very rare complication of this disease is the development of acute or new onset left ventricular systolic dysfunction which may not be a first thought condition when the patient presents with acute or new onset heart failure. Therefore, it is important to recognize this reversible cause in order to wait for the proper treatment and to impact the clinical outcome negatively.

This case highlights the need for thyroid function tests as a regular part of the evaluation of patients with dilated cardiomyopathy of unknown etiology or newly diagnosed heart failure, not least because of the rarity of the condition. Determination of the levels of serum thyroid-stimulating hormone (TSH) and free thyroxine (free T4) is cheap, widely available, and can detect a disorder of the thyroid gland that is easily treatable and can profoundly change patient management. A timely inclusion of thyroid function tests in the diagnostic process can help in the early initiation of the HRT treatment and enhance the chances of myocardial recovery before the irreversible process of ventricular remodeling.

In the present report, the critical importance of lifelong levothyroxine replacement therapy after total thyroidectomy is highlighted. Complete removal of the thyroid gland puts the patient totally at the mercy of exogenous thyroid hormone for proper metabolic and cardiac regulation. In this patient, the withdrawal of treatment for about 5 months led to a significant worsening of left ventricular systolic function, severe heart failure and marked hypothyroidism. Medication non-adherence can have life-threatening effects and a multidisciplinary education program, routine endocrine follow-up, and reinforcement of treatment adherence are vital.

The other implication in the clinical setting is that of avoiding unnecessary advanced cardiovascular interventions. If severe hypothyroidism is not recognized as the underlying cause of cardiomyopathy, patients may undergo significant testing, lengthy hospitalization, undergo evaluation for implantable cardioverter-defibrillator (ICD) and cardiac resynchronization

therapy (CRT), cardio-mechanical support, or early referral for heart transplantation. Early recognition of hypothyroidism as a reversible cause of myocardial dysfunction, therefore, could limit or prevent unnecessary utilization of healthcare resources, invasive procedures, and treatment of hypothyroidism alongside guideline-directed medical therapy for heart failure.

In the present case, it is important to emphasize the need for good multidisciplinary cooperation between cardiologists and endocrinologists. Thyroid hormone replacement and heart failure therapy should be optimized simultaneously and thyroid function monitored serially, as should be the heart failure status and ventricular performance. These synchronized care plans allow for titration of levothyroxine, optimization of cardiac medications and prompt identification of ventricular recovery or ongoing dysfunction, requiring further evaluation. This patient's good results demonstrate the importance of a comprehensive, multidisciplinary approach.

Lastly, this case further highlights the importance of always considering potentially reversible systemic disorders before irreversible or more advanced therapeutic options are implemented in cardiovascular medicine. This marked improvement in left ventricular ejection fraction from around 10% to 40% over the two month period is a testament to the fact that even very marked myocardial dysfunction can recover if the underlying endocrine disorder is recognised early and treated appropriately. Therefore, it is advisable to consider routine evaluation of thyroid function, thorough evaluation of drug adherence and prompt referral for a team approach as an integral part of the diagnostic process in patients with non-ischemic dilated cardiomyopathy of unknown etiology.

7.6 Strengths and Limitations

Present case adds meaningful clinical data about the reversible nature of dilated cardiomyopathy, as well as several methodological limitations inherent of single patient case reports. The strengths and limitations of the study should be evaluated so the results can be properly interpreted, the clinical applicability determined, and the role of this report in the body of literature identified. Table VIII gives a summary of the key strengths and limitations, which are discussed below.

TABLE VIII. STRENGTHS AND LIMITATIONS OF THE PRESENT CASE

Strengths	Limitations
Rare but clinically significant presentation of reversible hypothyroidism-induced dilated cardiomyopathy.	Single-patient case report, limiting generalizability.
Comprehensive endocrine, laboratory, and cardiovascular evaluation.	Relatively short follow-up period.
Obstructive coronary artery disease definitively excluded by coronary angiography.	Cardiac magnetic resonance imaging (CMR) was not performed for myocardial tissue characterization.
Objective documentation of ventricular recovery using serial transthoracic echocardiography.	Endomyocardial biopsy was not performed because it was not clinically indicated.
Well-defined temporal relationship between prolonged levothyroxine discontinuation, severe hypothyroidism, treatment initiation, and myocardial recovery.	Concurrent initiation of levothyroxine and guideline-directed medical therapy prevents assessment of the independent contribution of each intervention.

This present report has several salient features that contribute to its scientific and clinical relevance. It reports a rare, but potentially curable, severe non-ischemic dilated cardiomyopathy complicating profound hypothyroidism, thereby adding to the sparse data on endocrine causes of advanced heart failure. Second, the diagnostic evaluation was comprehensive, and included detailed biochemical assessment of thyroid function, electrocardiography, transthoracic echocardiogram, laboratory investigations and invasive coronary angiography. This systematic method allowed for the elimination of the other possibilities for myocardial dysfunction and provided a further support to the diagnosis of severe thyroid hormone deficiency.

Another great advantage is the objective documentation of cardiac recovery by serial echocardiographic examinations, which successfully showed improved left ventricular ejection fraction from about 10% to 40% after treatment. In addition, the well-documented temporal sequence of events (cessation of levothyroxine following TT for an extended period, followed by severe hypothyroidism and heart failure, with eventual marked clinical and echocardiographic improvement with the reinstitution of thyroid hormone replacement and guideline-directed medical therapy) provides strong clinical evidence for a causal relationship between thyroid hormone deficiency and reversible myocardial dysfunction.

The results presented in this report are subject to several limitations. Observations from this single patient cannot be extrapolated to other patients with hypothyroidism-associated dilated cardiomyopathy. Further multicenter studies and prospective investigations are needed to determine the prevalence, predictors, and the long-term clinical outcomes of this rare condition.

The follow-up period was relatively short, and thus, long-term ventricular remodeling or normalization of systolic function or risk of recurrence could not be assessed. Also, cardiac magnetic resonance imaging (CMR) was not done. While coronary angiography ruled out obstructive coronary artery disease and the clinical improvement was encouraging, that patient's clinical improvement would have been further strengthened by the possibility of CMR to give additional tissue characterization to help rule out myocardial fibrosis, edema, or occult inflammatory disease.

Finally, endomyocardial biopsy was not warranted because the patient improved clinically quickly after starting levothyroxine replacement and guideline directed heart failure therapy but there was no clinical indication for infiltrative

or inflammatory myocardial disease or other disease that needed confirmation by biopsy. Furthermore, thyroid hormone replacement and drugs used for heart failure were started at the same time, making it difficult to know exactly which therapy contributed to the recovery of the ventricles. However, the temporal relationship of the marked improvement in cardiac function and correction of severe hypothyroidism strongly suggests the diagnosis of reversible hypothyroidism-induced dilated cardiomyopathy.

7.7 Key Clinical Learning Points

The present case provides several important lessons that can help the clinician with the early recognition and management of hypothyroidism associated dilated cardiomyopathy. It is rare, but its timely recognition of the reversible endocrine causes can have significant impact on the diagnostic process, therapeutic approach and outcome of the patient. The different learning points from the clinical case are summarized in the following Table IX.

TABLE IX. KEY CLINICAL LEARNING POINTS FROM THE PRESENT CASE

Clinical Learning Point	Clinical Relevance
Severe hypothyroidism should be considered in patients presenting with unexplained non-ischemic dilated cardiomyopathy or new-onset heart failure.	Early thyroid function testing may identify a reversible cause of myocardial dysfunction and prevent delayed diagnosis.
Long-term adherence to levothyroxine replacement is essential after total thyroidectomy.	Prolonged treatment interruption may result in profound hypothyroidism, severe heart failure, and reversible dilated cardiomyopathy.
Timely initiation of thyroid hormone replacement therapy, together with guideline-directed medical therapy for heart failure, can result in substantial recovery of left ventricular systolic function.	Early treatment increases the likelihood of reverse ventricular remodeling before irreversible myocardial damage develops.
Exclusion of ischemic heart disease is essential before attributing severe ventricular dysfunction solely to hypothyroidism.	Coronary angiography or other appropriate imaging strengthens diagnostic confidence and supports accurate etiological classification.
Multidisciplinary collaboration between cardiologists and endocrinologists is fundamental for optimal patient care.	Coordinated management facilitates safe thyroid hormone replacement, optimization of heart failure therapy, and long-term monitoring of ventricular recovery.
Regular follow-up using thyroid function tests and serial echocardiography is recommended.	Objective assessment of endocrine control and ventricular recovery guides treatment adjustment and long-term prognosis.

This case illustrates the need to always keep the diagnosis of profound hypothyroidism as a possible diagnosis in patients who present with non-ischemic dilated cardiomyopathy of unknown etiology. In patients with severely depressed LV function, comprehensive endocrine evaluation, confirmation of non-ischemic myocardial dysfunction, and timely initiation of thyroid hormone therapy may lead to significant improvement in cardiac function, and careful multidisciplinary follow-up. If identified early, this reversible condition can be avoided from unnecessary invasive procedures and can help to achieve a better clinical outcome.

8. CONCLUSION

This rare case report signals the need to be vigilant for severe reversible dilated cardiomyopathy due to prolonged discontinuation of levothyroxine therapy following thyroidectomy. Advanced heart failure and a markedly reduced left ventricular ejection fraction were initially concerning for primary cardiomyopathy; however, in-depth endocrine and cardiovascular assessment, including coronary angiography which ruled out an obstructive coronary artery disease, confirmed the diagnosis of severe hypothyroidism. Prompt diagnosis and treatment of hypothyroidism are essential for reversing myocardial dysfunction that is caused by hypothyroidism, as evidenced by this remarkable improvement in LV systolic function following the treatment of hypothyroidism with thyroid hormone replacement plus the guideline-directed medical therapy. This case highlights the need to check thyroid function in the differential diagnosis of non-ischemic dilated cardiomyopathy and the newly diagnosed heart failure, especially in patients with a history of thyroidectomy and inadequate thyroid hormone replacement therapy. This report highlights the need for long-term medication compliance, early biochemical evaluation of thyroid function and careful multidisciplinary management between the cardiologist and endocrinologist. The early diagnosis of this rare curable disorder can avert unnecessary invasive procedures, help start treatment quickly and properly, and enhance heart recovery and outcomes. Considering the limited number of studies of hypothyroidism-induced dilated cardiomyopathy, future multicenter studies and long-term follow-up investigations are needed to further characterize the epidemiology, ventricular recovery predictors, optimal management, and long-term prognosis. However, the present case contributes valuable evidence to the scarce literature in the field and supports the rule that potentially reversible endocrine causes of heart failure should always be ruled out before irreversible or advanced heart failure treatments.

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