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MYOPERICARDITIS SECONDARY TO SILENT AUTOIMMUNE THYROIDITIS: A CASE OF SEVERE THYROTOXICOSIS

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ABSTRACT

Introduction: Myopericarditis is an uncommon but potentially severe complication of thyroid dysfunction. Although cardiovascular manifestations of hyperthyroidism are well documented, its association with myocardial and pericardial inflammation is rare and diagnostically challenging.

Case Presentation: A 36-year-old previously healthy male presented with precordial chest pain and a 1-month history of diarrhoea, heat intolerance, palpitations, and unintentional weight loss. Laboratory evaluation revealed elevated cardiac biomarkers, and electrocardiogram showed diffuse ST-segment elevation suggestive of acute pericarditis. Coronary angiography demonstrated no obstructive coronary lesions. Thyroid function tests revealed suppressed TSH and markedly elevated free T4, with positive anti-thyroglobulin antibodies. Thyroid scintigraphy confirmed decreased uptake consistent with thyroiditis. A diagnosis of myopericarditis secondary to silent autoimmune thyroiditis was established. The patient was treated with nonsteroidal anti-inflammatory drugs, colchicine, beta-blockers, and methimazole, achieving complete clinical and biochemical recovery.

Conclusion: This case emphasizes the importance of considering thyroid dysfunction in patients presenting with myopericarditis. Early recognition and targeted management can reverse myocardial injury and optimize patient outcomes.

KEYWORDS

Thyroiditis, autoimmune, myocarditis, pericarditis, thyrotoxicosis, chest pain, young adult

LEARNING POINTS

- Myopericarditis may be the initial and sole manifestation of silent autoimmune thyroiditis. Thyroid function testing should be routinely considered in young patients with chest pain and elevated troponins but normal coronary arteries.
- Early identification of thyroid dysfunction allows complete reversal of cardiac involvement with appropriate endocrine and anti-inflammatory treatment, preventing unnecessary invasive procedures.
- This case highlights a practical diagnostic pitfall that internists may encounter and underscores the importance of integrating endocrine evaluation in unexplained cardiac presentations.

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INTRODUCTION/BACKGROUND

Myopericarditis remains a diagnostic challenge in young adults presenting with chest pain and elevated cardiac biomarkers, particularly when coronary angiography reveals normal arteries. While viral and idiopathic aetiologies predominate, endocrine disorders such as hyperthyroidism are rarely considered in this context. Silent autoimmune thyroiditis is typically self-limiting, yet in some cases, the thyrotoxic phase may provoke systemic inflammation extending to myocardial and pericardial tissues. This case report describes a rare and reversible cause of myopericarditis secondary to severe thyrotoxicosis in a patient with no prior thyroid history, emphasizing the importance of thyroid function assessment in unexplained myocardial injury. The report highlights an uncommon diagnostic pitfall that internists may encounter and demonstrates how early recognition and targeted endocrine and anti-inflammatory therapy can prevent unnecessary invasive procedures and improve patient outcomes.

CASE DESCRIPTION

A 36-year-old previously healthy male presented with oppressive precordial chest pain while traveling in Mexico. He reported a one-month history of profuse diarrhoea, heat intolerance, palpitations, and unintentional weight loss of 5 kg. He denied recent infections, drug use, or a family history of cardiovascular or thyroid disorders.

On admission, blood pressure was 145/84 mmHg, heart rate 118 bpm, temperature 36°C, and oxygen saturation 98%. Physical examination was unremarkable, with no signs of thyrotoxicosis, goiter, or heart failure. Initial laboratory tests revealed elevated creatine phosphokinase (441 U/l), creatine kinase-MB (75.85 U/l), and troponin I (2.85 ng/ml), with high-sensitivity troponin peaking at 13,137.9 pg/ml on day 2. Erythrocyte sedimentation rate was elevated at 134 mm/h, while complete blood count, renal function, and electrolytes were within normal limits.

Electrocardiogram showed diffuse ST-segment elevation without reciprocal changes, suggestive of pericarditis *(Fig.1)*. Chest X-ray was normal. Coronary angiography revealed codominant circulation with no obstructive lesions. Transthoracic echocardiography demonstrated preserved left ventricular systolic function (ejection fraction 63%), pericardial thickening, and no pericardial effusion *(Fig.2)*.

Due to persistent tachycardia and systemic symptoms, thyroid function testing was performed. Thyroid-stimulating hormone (TSH) was suppressed (0.18 mIU/l), free thyroxine (T4) was markedly elevated (103 ng/dl), and anti-thyroglobulin antibodies were elevated (600 IU/ml). Anti-thyroid peroxidase and TSH receptor antibodies were negative *(Table 1)*. Human immunodeficiency virus and venereal disease research laboratory tests were negative. Thyroid ultrasound showed normal morphology. Technetium-99m thyroid scintigraphy demonstrated diffusely reduced uptake, consistent with thyroiditis.

Based on clinical, biochemical, and imaging findings, a diagnosis of myopericarditis secondary to silent autoimmune thyroiditis was established. The patient was treated with high-dose nonsteroidal anti-inflammatory drugs (including aspirin), colchicine, and low-dose metoprolol. His symptoms

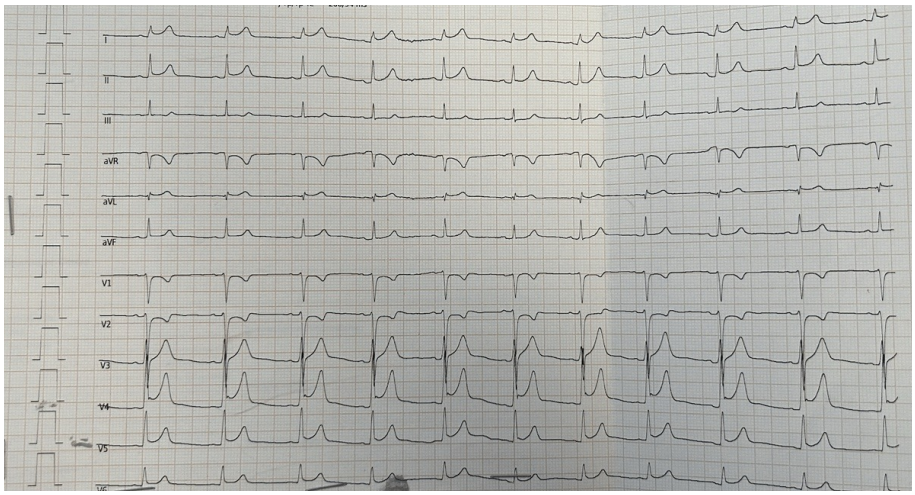


Figure 1. 12-lead electrocardiogram showing diffuse ST-segment elevation and PR-segment depression consistent with acute pericarditis.

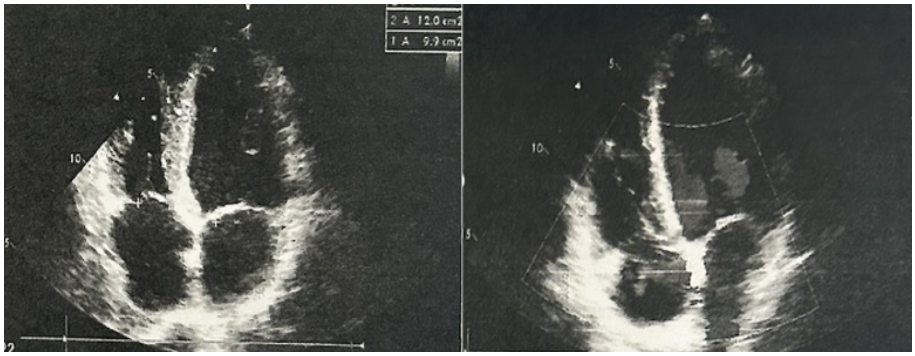


Figure 2. Transthoracic echocardiogram demonstrating preserved biventricular function increased and pericardial echogenicity suggestive of active inflammation.

Date	TSH (µIU/ml)	T4 (pmol/l)	Free T3 (pmo/l)	Anti-TPO (IU/ml)	Anti-Tg (IU/ml)	Ant TSH (IU/l)	hs-Tnl (pg/ml)
2024-06-01	0.01	35.2	18.3	355	NA	8.5	3.25
2024-06-05	0.02	29,8	15.1	120	NA	5.6	2.1
2024-06-10	0.15	22.1	NA	90	45	2.3	1.5
2024-06-15	0,.6	17	5.2	90	40	1.2	0.8
2024-06-20	1,.2	15.5	4.5	78	35	0.8	0.5

Abbreviations: TSH-R, thyroid stimulainghormonereceptor; TPO, thyroidperoxidase; Tg, thyroglobulin; T4, thiyroxine; T3, triiodothyronine; NA, not available.

Table 1. Evolution of thyroid function, thyroid autoantibodies, and cardiac biomarkers during hospitalization.

progressively improved with normalization of cardiac biomarkers. He was discharged with endocrinology and cardiology follow-up. At 2-month follow-up, the patient remained asymptomatic with normal thyroid function and resolution of antibody levels.

DISCUSSION

This report presents a rare case of myopericarditis secondary to silent autoimmune thyroiditis with severe thyrotoxicosis in a patient with no prior thyroid history. The predominance of cardiac symptoms highlights the importance of considering thyroid dysfunction in young patients presenting with chest pain and absence of coronary artery disease to ensure early diagnosis and treatment.

The actual incidence of myopericarditis associated with hyperthyroidism or thyrotoxicosis is uncertain and likely underestimated. Although the literature addressing this association is limited, available evidence suggests that pericardial and myocardial inflammation may occur as an infrequent but clinically relevant manifestation of thyroid dysfunction[1]. The lack of clear pathophysiological mechanisms complicates frequency estimates, and absence of clinical suspicion and thyroid screening may contribute to underreporting. Case reports such as this one are valuable for raising awareness and stimulating further research.

The association between thyrotoxicosis and cardiac involvement has been recognized for decades. In 1981, a case of perimyocarditis associated with hyperthyroidism was described, with resolution only after thyroid function normalization[2]. Since then, multiple cardiac manifestations have been described in hyperthyroidism, including tachyarrhythmias, high-output heart failure, transient systolic dysfunction, and, rarely, autoimmune pericarditis or myocarditis in many cases, treating thyrotoxicosis results in clinical improvement and normalization of cardiac biomarkers. Our case contributes to this body of evidence, documenting myopericarditis in the setting of silent autoimmune thyrotoxicosis with a favourable response to targeted therapy.

Subacute thyroiditis has been reported in young patients without typical triggers, as in our case, thus thyroid function evaluation and scintigraphy confirmation are recommended[3]. Non-infectious myocarditis may occur either in isolation or as part of autoimmune diseases. Unlike viral myocarditis, it frequently lacks an identifiable trigger. Medications, toxins, heavy metals, and spontaneous immune activation have been implicated. In autoimmune thyroid disease, inflammation may extend beyond the thyroid gland and involve the myocardium[4]. In this patient, absence of infection, negative serologies, and lack of drug exposure support an autoimmune cause. Thyrotoxicosis may induce myocardial injury via hemodynamic stress, increased oxygen consumption, sustained tachycardia, and immune activation. Retrospective studies report myocarditis in 9–16% of patients with non-ischemic cardiomyopathy, suggesting under recognition in autoimmune thyroid disease[4].

Thyroid storm can lead to acute heart failure and dilated cardiomyopathy through direct thyroid hormone effects on myocardial ion channels and heightened arrhythmic risk[5]. Even without thyroid storm, hyperthyroidism creates a hyperdynamic circulation with increased stroke volume, heart rate, and arrhythmias that may precipitate autoimmune myocarditis and ventricular dysfunction[6].

Mavrogeni et al. studied euthyroid patients' post-treatment for hyperthyroidism who presented with persistent cardiac symptoms. Cardiac magnetic resonance imaging (MRI) showed active myocarditis in 30% of cases, and endomyocardial biopsy revealed lymphocytic infiltration without viral genomes, supporting an immune-mediated, non-viral mechanism[7]. Our case reinforces the hypothesis of myopericarditis as the initial manifestation of silent thyroiditis with predominant cardiac involvement.

Ischaemic and infectious causes were excluded based on normal coronary angiography, negative viral serologies, and absence of constitutional symptoms[8]. Thyroid tests revealed suppressed TSH, elevated free T4, absent uptake on scintigraphy, and negative TSH receptor antibodies, consistent with autoimmune thyroiditis. Fan and Sha-yi reported a similar case of myocardial injury with reduced thyroid uptake that improved after thyroid-directed treatment, further supporting endocrine aetiology[8].

The integration of clinical, imaging, and biochemical

findings, combined with exclusion of alternative causes, confirmed the diagnosis of myopericarditis secondary to thyroid dysfunction. This case emphasizes the importance of multidisciplinary management between cardiology and endocrinology. The patient improved with beta-blockers and nonsteroidal anti-inflammatory drugs without requiring antithyroid agents. Thyroid evaluation is recommended in myopericarditis of unclear aetiology[9].

A limitation in this case was the inability to perform cardiac MRI or endomyocardial biopsy, which would have allowed direct documentation of inflammation. Cardiac MRI has demonstrated utility in autoimmune thyroiditis[7]. Diagnosis in this case was based on clinical presentation and favourable therapeutic response. Although viral causes cannot be entirely excluded, the clinical course strongly supports autoimmune thyroid dysfunction as the underlying mechanism.

In conclusion, this case underscores the importance of considering thyroid dysfunction as a cause of myopericarditis in cases where there is no clear aetiology. Interdisciplinary evaluation and systematic thyroid function testing may optimize diagnosis and improve prognosis.

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