



(CASE REPORT)



Large pericardial effusion as the first manifestation of severe Hashimoto's thyroiditis: Clinical and echocardiographic insights

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Abstract

Introduction: Severe hypothyroidism may lead to pericardial effusion, but the development of a large effusion is uncommon. This complication results from increased pericardial capillary permeability and reduced lymphatic clearance, though most cases remain mild and clinically silent.

Case presentation: We report the case of a 65-year-old patient who developed progressively worsening dyspnea, evolving from NYHA class II to class III prior to admission. Echocardiography revealed a large circumferential pericardial effusion exceeding 30 mm, notably without hemodynamic compromise. Laboratory investigations demonstrated profound hypothyroidism, with a TSH level $>100,000$ $\mu\text{U/mL}$ and markedly reduced free T4 levels. Thyroid hormone replacement therapy led to rapid clinical improvement and complete resolution of the effusion on follow-up. Hashimoto's thyroiditis was subsequently identified as the underlying etiology.

Conclusions: This case illustrates that even markedly large pericardial effusions in severe hypothyroidism may remain hemodynamically silent and resolve fully with adequate hormonal therapy. Early recognition of hypothyroidism as a reversible cause of massive pericardial effusion is key to preventing unnecessary invasive procedures.

Keywords: Pericardial Effusion; Hypothyroidism; Echocardiography; Hashimoto; Hormone replacement therapy

1. Introduction

Hypothyroidism is a systemic endocrine disorder with potential involvement of multiple organ systems. Pericardial effusion is a recognized but typically mild manifestation, attributed to increased capillary permeability and reduced lymphatic clearance. Large or massive effusions are uncommon and often prompt evaluation for infectious, inflammatory, or malignant causes. We describe a case of severe hypothyroidism presenting with a substantial circumferential pericardial effusion in the absence of hemodynamic compromise, underscoring the need to consider endocrine aetiologies when assessing unexplained pericardial effusions.

2. Patient presentation

We report a case of a 65-year-old patient with no significant past medical history who presented in our department with progressively worsening dyspnea, initially classified as NYHA class II and advancing to class III over several weeks. His primary concern was exertional breathlessness and pronounced fatigability without chest pain, palpitations, or syncope.

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3. Clinical Findings:

On examination, vital signs were stable, including normal blood pressure, a heart rate of 80 bpm, and oxygen saturation of 96%. Cardiac auscultation revealed muffled heart sounds, There was no peripheral oedema or other signs of heart failure. ECG demonstrated diffuse low-voltage QRS complexes and Chest radiography showed a globular cardiomegaly.

Findings on clinical examination suggested the possibility of a pericardial effusion; therefore, a Transthoracic echocardiography was performed and revealed a massive circumferential pericardial effusion, measuring 33 mm adjacent to the left ventricle and 27 mm near the right ventricle, without echocardiographic signs of tamponade or constrictive physiology (figure1 and 2).

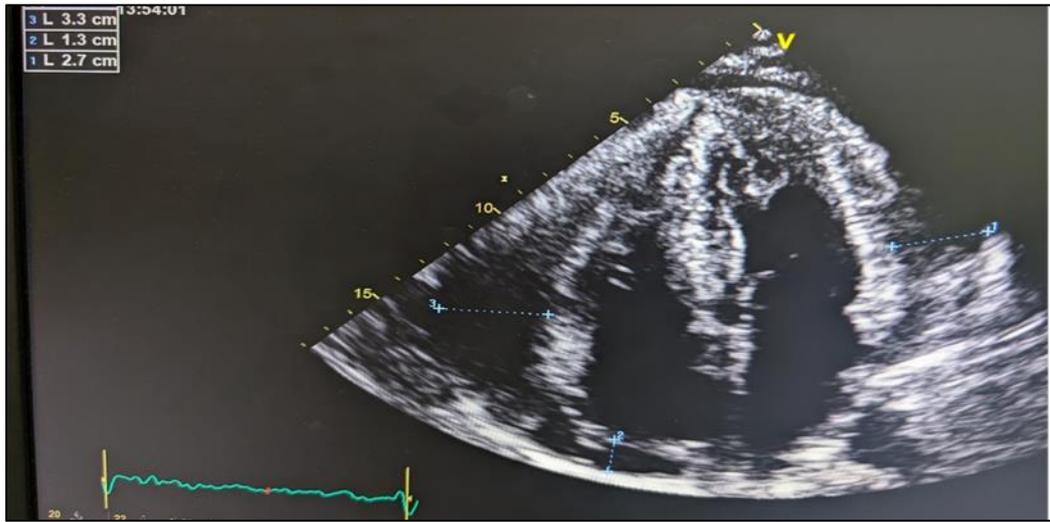


Figure 1 Baseline transthoracic echocardiography showing a large circumferential pericardial effusion (>30 mm) without signs of hemodynamic compromise. Cardiac chambers appear preserved

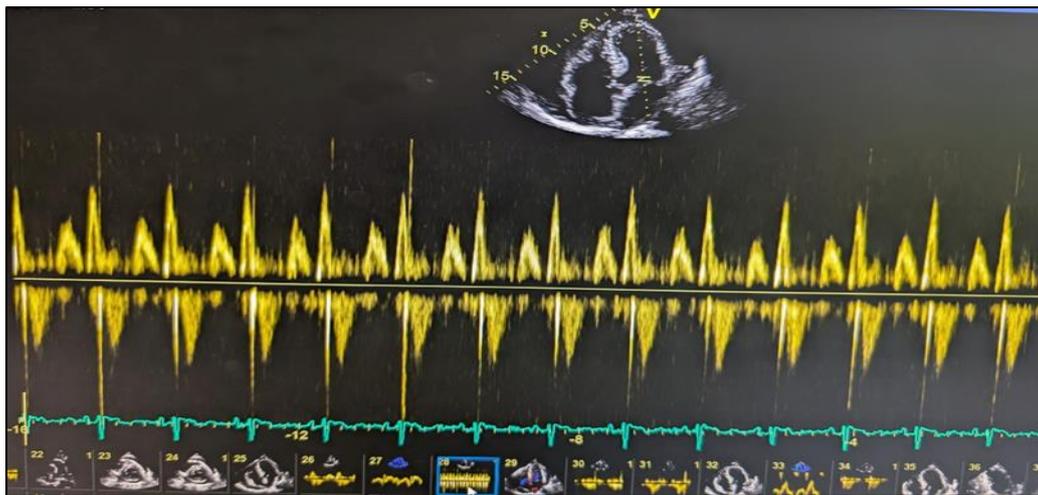


Figure 2 Pulsed-wave Doppler of the mitral inflow demonstrating the absence of significant respiratory variation (<25%)

4. Diagnostic Assessment

A comprehensive diagnostic workup was performed to identify potential malignant, infectious, or inflammatory etiologies for this pericardial effusion. Thoraco-abdominal CT imaging was unremarkable. Tumor markers were within normal limits, excluding neoplastic disease. Infectious investigations including bacterial cultures, viral serologies, and mycobacterial screening were negative. Laboratory evaluation revealed profound hypothyroidism, with TSH levels exceeding 100,000 $\mu\text{U}/\text{mL}$ and markedly reduced free T4. Cervical ultrasound and immunologic testing confirmed

autoimmune Hashimoto thyroiditis. The diagnosis of pericardial effusion secondary to severe hypothyroidism due to Hashimoto thyroiditis was established.

4.1. Timeline

Over the two months preceding admission, the patient experienced progressively worsening dyspnea. Upon presentation, transthoracic echocardiography revealed a large pericardial effusion. Etiological investigations were initiated, and severe hypothyroidism was identified the following day, leading to the initiation of thyroid hormone replacement therapy. Additional biological tests and a thoraco-abdominopelvic CT scan were performed within the first week, allowing exclusion of alternative causes. Regular follow-up showed marked improvement at one month and complete resolution of the pericardial effusion by the third month.

4.2. Therapeutic Intervention

Following multidisciplinary discussion with endocrinology specialists, progressive thyroid hormone replacement therapy was initiated, in combination with pericarditis management using aspirin and colchicine, and the patient was placed under regular follow-up. No invasive procedures were performed, and pericardiocentesis was not required.

4.3. Follow-up and Outcomes

Serial transthoracic echocardiographic evaluations demonstrated a gradual and continuous reduction in the pericardial effusion, corresponding with the improvement in thyroid function. The patient became asymptomatic within six weeks, with near-normalization of thyroid hormone levels. Complete resolution of the pericardial effusion was confirmed at the three-month follow-up (Figure 3).

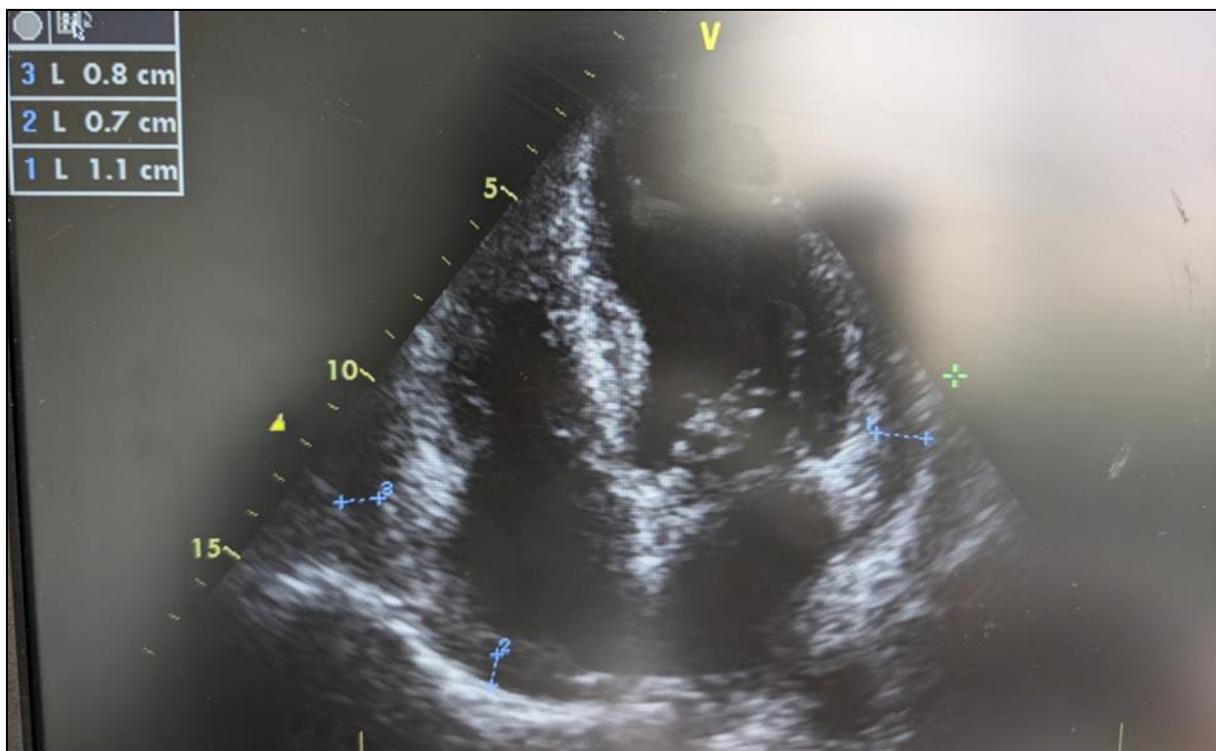


Figure 3 Follow-up echocardiography 6 weeks after initiation of levothyroxine therapy showing a marked reduction of the pericardial effusion thickness

5. Discussion

Hypothyroidism represents a systemic endocrine disorder characterized by a global reduction in metabolic activity, with potential involvement of multiple organ systems. Pericardial effusion is a well-documented cardiovascular manifestation, reported with variable frequency ranging from 3% to 37%, although the occurrence of large or massive effusions remains distinctly uncommon (1,2). Recent reports continue to confirm this rarity, with several cases

describing massive effusions mimicking malignant or infectious aetiologies before hypothyroidism was identified as the underlying cause (3,4).

The pathophysiology is thought to result from increased permeability of pericardial capillaries combined with diminished lymphatic resorption of albumin-rich fluid, ultimately leading to progressive accumulation within the pericardial cavity. Although autoimmunity is central to the aetiology of primary hypothyroidism, it appears to have only a limited influence on the development of pericardial effusion (5). Most pericardial effusions remain clinically silent because the slow accumulation of fluid allows the pericardium to adapt without triggering hemodynamic compromise. Consequently, the absence of overt symptoms may lead to underestimation of disease severity and delay the diagnosis of both the effusion and the underlying condition (5).

This case highlights the necessity of considering hypothyroidism in the differential diagnosis of massive pericardial effusion. In our patient, the effusion accumulated gradually, ultimately causing notable dyspnea despite the absence of classical echocardiographic or clinical signs of cardiac tamponade. Comparable atypical or minimally symptomatic presentations have been reported in recent literature, in which large effusions were identified incidentally or in the absence of overt features of hypothyroidism, particularly among individuals with poor adherence to thyroid hormone replacement therapy (6). In the present case, the diagnosis of severe hypothyroidism was supported by profoundly elevated thyroid-stimulating hormone (TSH) levels and markedly reduced free T4 concentrations, findings that were subsequently reinforced by imaging and immunologic assessments.

Management of pericardial effusion in the setting of hypothyroidism is centered on thyroid hormone replacement, which directly targets the underlying endocrine disturbance. In the present case, initiation of oral levothyroxine resulted in marked clinical improvement, accompanied by substantial echocardiographic regression of the effusion within three months (2). Recent case reports and small series consistently indicate that hormone replacement alone is sufficient for resolution in most patients, with even large effusions gradually regressing without the need for pericardiocentesis, provided that no features of tamponade are present (3,7). These observations underscore the therapeutic efficacy of restoring euthyroidism, not only in correcting hormonal abnormalities but also in resolving associated cardiovascular manifestations.

In summary, this case highlights the necessity of considering hypothyroidism in the differential diagnosis of massive pericardial effusion. It also illustrates that pericardial effusion can be the sole and initial manifestation of hypothyroidism, a presentation that should not be overlooked. Early recognition and appropriate treatment can lead to favorable outcomes, emphasizing the critical interplay between endocrine disorders and cardiovascular health. Pericardial effusion associated with hypothyroidism remains a reversible condition when adequately treated, even in its most severe presentations (3,7).

List of Abbreviations

- **NYHA** : New York Heart Association (*classification de la sévérité de l'insuffisance cardiaque*)
- **TSH** : Thyroid Stimulating Hormone.
- **T4** : Thyroxine.
- **Bpm** : Beats per minute.
- **ECG** : Electrocardiogram.

6. Conclusion

Severe hypothyroidism is an important and reversible cause of massive pericardial effusion. Early recognition and appropriate thyroid hormone replacement therapy can lead to full clinical and echocardiographic recovery, preventing unnecessary invasive interventions such as pericardiocentesis.

6.1. Patient perspective

The patient described several weeks of progressively worsening dyspnea, profound fatigue, and excessive daytime sleepiness, which significantly limited his daily activities and led him to seek medical evaluation. He expressed that the diagnosis of severe hypothyroidism with associated pericardial effusion was unexpected, and reported relief at the subsequent clinical improvement following appropriate thyroid hormone replacement therapy. He appreciated the clarity of explanation provided and the effectiveness of the non-invasive management approach.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

Ethical approval for this case report was obtained from the Ethics Committee of the Military Hospital, Mohamed V University, Rabat, Morocco. The committee does not issue reference numbers. Written informed consent to participate was obtained from the patient

Statement of informed consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. The patient was informed that all personal identifiers would be removed to ensure anonymity.

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