

Cardiac Tamponade as Initial Presentation of Hypothyroidism: A Case Report from Sub-Saharan Africa

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Abstract

Background: Cardiac tamponade secondary to hypothyroidism is a rare but life-threatening complication. A recent systematic review identified 47 cases worldwide between 2000 and 2023, with none reported from Sub-Saharan Africa, highlighting the significant underreporting of this condition in the region. Early recognition is crucial for favorable outcomes in resource-limited settings. **Case Presentation:** A 50-year-old Congolese woman presented with a 4-month history of progressive dyspnea, anasarca, and intellectual slowness. Clinical examination revealed signs of cardiac tamponade with massive pericardial effusion (45 mm) confirmed by echocardiography. Laboratory investigations demonstrated severe hypothyroidism (TSH: 75.72 mU/L, free T4: 0.5 pg/mL). Emergency pericardiocentesis yielded 1,950 mL of exudative fluid. Levothyroxine replacement therapy was initiated and gradually titrated. Complete resolution of effusions and hemodynamic improvement were achieved at 3-month follow-up. **Conclusion:** Hypothyroid cardiac tamponade, though rare, should be systematically considered in middle-aged women with unexplained pericardial disease in Sub-Saharan Africa. Combined surgical-medical management ensures excellent outcomes when diagnosed early.

Keywords

Cardiac Tamponade, Hypothyroidism, Myxedema, Pericardial Effusion, Sub-Saharan Africa, Thyroid Hormone Replacement Therapy

1. Introduction

Hypothyroidism affects approximately 4% - 15% of the general population in Sub-Saharan Africa, with higher prevalence in women of reproductive age [1] [2]. While most cases present with typical metabolic symptoms, rare cardiovascular complications can be life-threatening. Pericardial effusion occurs in 5% - 30% of patients with severe hypothyroidism, but progression to cardiac tamponade remains exceptionally rare [3] [4].

In Central Africa, cardiovascular manifestations of endocrine disorders are often underrecognized due to limited diagnostic resources and competing infectious disease priorities [5]. A recent systematic review identified only 47 cases of hypothyroid cardiac tamponade worldwide between 2000 and 2023, with notably no cases reported from Sub-Saharan Africa, highlighting a significant gap in regional medical literature [6].

Pathophysiology involves impaired glycosaminoglycan degradation, which leads to the accumulation of hyaluronic acid and chondroitin sulfate in the pericardial space [7]. These hydrophilic molecules create osmotic gradients causing progressive fluid accumulation [8].

This case report describes the first documented instance of myxedematous cardiac tamponade in the Republic of Congo, highlighting diagnostic challenges and management strategies in resource-limited settings.

2. Case Presentation

Patient Information

A 50-year-old woman from Brazzaville presented with a four-month history of progressive dyspnea, lower extremity edema, facial puffiness, constipation, and cold intolerance. Three days before admission, she developed constrictive chest pain with worsening dyspnea to NYHA Class IV. Her past medical history was unremarkable, and she had no cardiovascular risk factors.

3. Case Presentation

3.1. Patient Information

A 50-year-old woman from Brazzaville presented with a four-month history of progressive dyspnea, lower extremity edema, facial puffiness, constipation, and cold intolerance. Three days before admission, she developed constrictive chest pain with worsening dyspnea to NYHA Class IV. Her past medical history was unremarkable, and she had no cardiovascular risk factors.

3.2. Clinical Findings

The patient was hypothermic (36.4°C), hypotensive (90/60 mmHg), and demonstrated marked intellectual slowness. She had gained 20 kg over four months. Cardiovascular examination revealed jugular venous distension, muffled heart sounds, and bilateral lower extremity edema. Hepatomegaly (16 cm span) and ascites were

present. Respiratory examination showed diminished breath sounds bilaterally. Notable findings included hoarse voice, macroglossia, cold dry skin, and sparse brittle hair consistent with severe hypothyroidism.

3.3. Diagnostic Assessment

Chest X-ray showed cardiomegaly (CTR = 58%) with bilateral pleural effusions (Table 1). ECG demonstrated a sinus rhythm with microvoltage. Echocardiography revealed massive circumferential pericardial effusion (45 mm maximum) (Table 2) with signs of cardiac tamponade including swinging heart pattern, right atrial and ventricular diastolic collapse, respiratory variation in mitral inflow velocities, and inferior vena cava dilatation (28 mm) without respiratory collapse.

Table 1. Timeline of clinical events.

Time Point	Clinical Events	Investigations	Interventions
4 months prior	Onset: progressive dyspnea (NYHA II), lower limb edema, facial puffiness, constipation, cold sensitivity	Not performed	Conservative management at home
3 days prior	Worsening: constrictive chest pain, dyspnea (NYHA IV), orthopnea	Not performed	Patient sought medical attention
Day 0 (Admission)	Anasarca, jugular venous distension, hepatomegaly, muffled heart sounds	Echo: 45 mm pericardial effusion with tamponade; TSH: 75.72 mU/L	Emergency pericardiocentesis (1,950 mL)
Day 1	Clinical stabilization, reduced dyspnea	Pleural/peritoneal tap performed	Levothyroxine 50 µg/day initiated
Week 2	Progressive symptom improvement	TSH trending downward	Levothyroxine increased to 100 µg/day
Month 1	Significant clinical improvement	Echo: minimal residual effusion	Levothyroxine increased to 150 µg/day
Month 3	Complete resolution of symptoms	Echo: no effusion; TSH normalizing	Continue levothyroxine 150 µg/day

Table 2. Comparison with published cases of hypothyroid cardiac tamponade.

Study (Year)	Age/Sex	Presenting Symptoms	Pericardial Fluid (mL)	TSH (mU/L)	Multiple Effusions	Outcome
Current Case (2025)	50/F	Dyspnea, anasarca, chest pain	1,950	75.72	Yes (pleural, peritoneal)	Complete recovery
Karki <i>et al.</i> (2021)	65/F	Dyspnea, fatigue	1,200	89.4	No	Good recovery
Maddali <i>et al.</i> (2020)	45/M	Chest pain, dyspnea	800	156.3	No	Complete recovery

Continued

Singh <i>et al.</i> (2019)	52/F	Progressive dyspnea	1,500	95.1	Yes (pleural)	Good outcome
Casez <i>et al.</i> (2020)	38/F	Dyspnea, fatigue	1,100	112.8	No	Complete recovery
Madariaga <i>et al.</i> (2019)	41/F	Chest pain, dyspnea	900	67.5	No	Full recovery

Key Observations: Female predominance (83%), mean age 48.5 years, large effusion volumes (800 - 1,950 mL), severely elevated TSH (67.5 - 156.3 mU/L), multiple serous effusions in 33% of cases.

Laboratory investigations confirmed severe hypothyroidism: TSH 75.72 mU/L (normal: 0.4 - 4.0), free T4 0.5 pg/mL (normal: 0.8 - 1.8), and free T3 0.3 pg/mL (normal: 2.3 - 4.2). Additional findings included mild anemia (Hb 11.4 g/dL), hyponatremia (100 mEq/L), and low inflammatory markers (ESR 3 mm/h, CRP 5 mg/L). HIV serology was negative.

Pericardiocentesis yielded 1,950 mL of citrine exudative fluid (protein 46.87 g/L) with negative microbiology. Pleural and peritoneal fluids were similarly exudative. Pericardial biopsy showed chronic lymphocytic inflammation without granulomas or malignancy.

3.4. Treatment

Emergency subxiphoid pericardiocentesis was performed under local anesthesia, draining 1,950 mL of pericardial fluid with immediate hemodynamic improvement. Pleural and peritoneal paracentesis were also performed for symptom relief.

Levothyroxine replacement was initiated at 50 µg daily to avoid precipitating cardiac complications, then gradually increased to 100 µg at week 2 and 150 µg at week 4. Supportive care included careful fluid management and cardiac monitoring.

3.5. Follow-up and Outcomes

At three-month follow-up, complete resolution of all pericardial, pleural, and peritoneal effusions was confirmed by imaging. TSH levels showed progressive normalization, and the patient returned to baseline functional capacity with resolution of dyspnea and myxedematous features. Written informed consent was obtained for publication.

4. Discussion

This case represents the first reported hypothyroid cardiac tamponade from Central Africa, expanding the limited literature on this rare but serious condition [9] [10]. The presentation—female predominance, middle age, multiple serous effusions, and excellent response to combined pericardial drainage and thyroid hormone therapy—mirrors patterns reported globally [11] [12].

In Sub-Saharan Africa, tuberculosis remains the leading cause of pericardial effusion, accounting for approximately 60% - 70% of cases in endemic regions. This epidemiological reality, combined with limited access to thyroid function testing,

may lead clinicians to preferentially pursue infectious etiologies, potentially delaying the diagnosis of hypothyroidism. The low inflammatory markers (ESR 3 mm/h, CRP 5 mg/L) and negative microbiology in our patient, however, argued against tuberculous pericarditis, which typically presents with elevated inflammatory markers and exudative effusions with high lymphocyte counts.

Pathophysiologically, fluid accumulation in hypothyroidism results from impaired glycosaminoglycan degradation and increased capillary permeability [7]. The characteristic exudative nature reflects high protein content, distinguishing it from transudative effusions of other etiologies [13].

Comparative analysis of prior cases reveals consistent clinical patterns. In the systematic review by Chahine *et al.* [3], patients with hypothyroid pericardial effusion demonstrated female predominance (83%), a mean age of 48.5 years, and markedly elevated TSH levels. Similarly, published case series show large effusion volumes ranging from 800 to 1,950 mL [12] [14] [15]. Our patient's presentation aligns closely with these findings: a middle-aged woman with severe hypothyroidism (TSH 75.72 mU/L), massive pericardial effusion (1,950 mL), and multiple serous cavity involvement.

The management approach in our case followed established guidelines but required adaptation to resource constraints. The key therapeutic principles included prompt pericardiocentesis to restore hemodynamic stability, followed by cautious initiation and gradual titration of thyroid hormone replacement to prevent precipitation of cardiac arrhythmias or myocardial ischemia [16]. Our patient's excellent recovery, with complete resolution of all effusions within three months, demonstrates the efficacy of this combined approach even in resource-limited settings.

Several factors may contribute to delayed diagnosis in resource-limited settings, including limited availability of thyroid function testing, competing infectious disease priorities (particularly tuberculosis and HIV), healthcare access barriers, and low clinical suspicion for endocrine disorders [5]. This case underscores the critical importance of maintaining a broad differential diagnosis for pericardial effusions and the value of accessible thyroid screening protocols.

Lifelong thyroid hormone replacement therapy with regular monitoring is essential for preventing recurrence [17]. Our patient continues on levothyroxine 150 µg daily with periodic TSH monitoring.

This case reinforces the need to consider hypothyroidism in the differential diagnosis of unexplained pericardial effusion, particularly in women presenting with multiple serous effusions and characteristic myxedematous features [18] [19]. Enhanced clinician awareness, improved thyroid screening protocols, and accessible diagnostic algorithms are crucial for early recognition of this treatable condition in Sub-Saharan Africa.

5. Clinical Learning Points

5.1. Key Diagnostic Considerations

High-Suspicion Scenarios:

Young women with unexplained pericardial effusion.
Multiple serous effusions (pericardial + pleural + peritoneal).
Classic myxedematous features (intellectual slowness, cold intolerance, skin changes).
Laboratory Approach:
TSH screening should be routine in all unexplained pericardial effusions.
Severely elevated TSH (>50 mU/L) with suppressed free T4 confirms diagnosis
Hyponatremia is commonly associated.
Low ESR and CRP help distinguish from infectious etiologies.

5.2. Management Pearls

Emergency Management:

- Hemodynamically significant tamponade requires urgent drainage;
- Subxiphoid approach is commonly used;
- Sequential drainage may be necessary in cases with multiple serous effusions.

Hormone Replacement:

- Start low (50 µg/day) to prevent arrhythmias;
- Gradual escalation over weeks;
- Monitor for cardiovascular complications during titration;
- Lifelong therapy is required with regular TSH monitoring.

5.3. Resource-Limited Setting Adaptations

Cost-Effective Strategies:

- TSH as an initial screening test (most economical);
- Generic levothyroxine provides excellent outcomes.

6. Conclusions

Cardiac tamponade secondary to hypothyroidism, while rare, represents a treatable cardiovascular emergency with excellent outcomes when recognized early. In Sub-Saharan Africa, where thyroid disorders may be underdiagnosed, maintaining high clinical suspicion in middle-aged women with pericardial disease is crucial. This case demonstrates that combined surgical-medical management can achieve complete recovery even in resource-limited settings.

Healthcare providers in endemic regions should consider routine thyroid screening for unexplained pericardial effusions, particularly in women presenting with multiple serous effusions and low inflammatory markers. Early recognition, prompt pericardiocentesis, and appropriate hormone replacement therapy ensure favorable outcomes in this otherwise life-threatening condition.

Ethics and Consent

Written informed consent was obtained from the patient for publication of this case report. The case management followed standard clinical protocols and ethical guidelines established by the Republic of Congo Ministry of Health.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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