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Case Series: Myxedema Crisis and Pericardial Effusion

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Abstract

Myxedema coma is a rare but highly lethal endocrine emergency. It usually presents with altered mental status, hypotension, bradycardia, hypothermia, bradypnea, hyporeflexia, hyponatremia, and hypoglycemia, all resulting from a profound deficiency of thyroid hormones. Additionally, affected individuals may present with signs of low cardiac output, generalized edema, shock, pericardial and/or pleural effusion, and altered mental status (with or without coma). We present three cases of patients with myxedema coma and pericardial effusion, all with unusual clinical features.

Keywords

Myxedema, Hypothyroidism, Pericardial, Effusion

1. Introduction

Myxedema (ME) is a serious endocrine emergency caused by decompensated hypothyroidism. Despite appropriate intensive management, the associated mortality rate can reach up to 60% of cases [1].

The term "myxedema coma" is debated because some patients do not arrive in a coma, but rather present with lethargy or confusion. Therefore, "*myxedema state*" *is proposed* as a more appropriate term [1]. Triggers include infections, trauma, cold, central nervous system depressants, and thyroid hormone deficiency [2].

Myxedema State (MS) originates from multi-organ dysfunction resulting from reduced intracellular levels of triiodothyronine. Clinically, it manifests as hypothermia, altered mental status (coma, stupor, or obtundation), hypoxemic respiratory failure and hypercapnia, decreased glomerular filtration rate, dilutional hyponatremia, adrenal insufficiency, hypoglycemia, bradycardia, QT interval prolongation, *torsades de pointes ventricular tachycardia, ventricular* fibrillation, low-voltage QRS complexes, hypotension, congestive heart failure, diastolic hypertension, pericardial effusion, and/or cardiac tamponade [3]. Its incidence is 0.22 - 1 per million people per year, being more common in those over 60 years of age, women, and during winter [4].

Pericardial effusion affects up to 80% of patients with Myxedema State. The pathophysiology is complex and involves alterations in several metabolic pathways, including the accumulation of glycosaminoglycans due to a lack of inhibition mediated by thyroid hormone (triiodothyronine). This causes myxedema, with deposits in the skin, lungs, intestines, interstitium, and muscle, including the myocardium [3]. Another explanation is transcapillary leakage into the extravascular space, which generates an imbalance in osmotic pressure between the pericardium and the myocardium, forming a protein-rich effusion [5].

Pericardial effusion associated with Myxedema State develops gradually, which prevents the frequent occurrence of cardiac tamponade. This explains the coexistence of large pericardial volumes without hemodynamic compromise, which generally improves after the initiation of hormone replacement therapy with levothyroxine and/or triiodothyronine [6].

Pericardial effusion can range from massive effusions (greater than 500 ml or an anechoic space greater than 20 mm) with echocardiographic findings of cardiac tamponade, but without hemodynamic compromise. This atypical presentation is due to the progressive formation of the effusion, making it a condition that responds to hormone replacement therapy [5]. Pericardial drainage is reserved for cardiac tamponade with clinical manifestations such as cardiogenic shock, hypotension, and pulsus paradoxus. We present three cases of severe hypothyroidism with massive pericardial effusion. Clinical and echocardiographic findings are analyzed, and the literature is reviewed.

2. Case Presentations

Case 1:

A 28-year-old man who underwent a total thyroidectomy in 2021 for Hashimoto's thyroiditis (positive anti-thyroid peroxidase antibodies and negative anti-thyroglobulin antibodies) presented to the emergency department with facial edema, accompanied by increased abdominal girth, dysphonia, dyspnea on moderate exertion, and epigastric pain. On physical examination, his vital signs were: blood pressure (BP): 90/63 mmHg; respiratory rate (RR): 20 breaths/minute; heart rate (HR): 50 beats/minute; temperature: 36.3˚C; and arterial oxygen saturation (SaO_2): 93%. He also presented with bradypsychia, bradylalia, facial edema, de-

creased breath sounds at the lung bases, muffled heart sounds without a pericardial friction rub, dullness to percussion in the flanks, and atrophic skin changes with hyperkeratosis.

His laboratory results showed normocytic normochromic anemia (hemoglobin 11.7 g/dL) (normal: 13 - 16 g/dL), elevated total bilirubin 1.45 mg/dL (0 - 1 mg/dL), mild hyponatremia 133 mmol/L (normal: 136 - 145 mmol/L), and elevated TSH 38.06 μIU/mL. Free thyroxine was <0.4 ng/dL (normal: 0.7 - 1.48 ng/dL). Arterial blood gases showed no evidence of hypoxemia or hypercapnia. A full abdominal ultrasound revealed moderate free fluid in the abdominal cavity. A chest X-ray showed an enlarged cardiac silhouette (the "jug sign") (**Figure 1**). The Popoveniuc score was 60, highly suggestive of Myxedema State. We decided to transfer her to the intensive care unit (ICU) and start oral levothyroxine (300 mcg/day) due to the unavailability of intravenous levothyroxine.

We also initiated fluid resuscitation with intravenous hydrocortisone (50 mg every 8 hours). This was due to the finding of cardiomegaly on chest X-ray. Echocardiogram showed severely dilated cardiomyopathy with generalized hypokinesis and severe systolic dysfunction with an ejection fraction (LVEF) of 12%. A 33 mm pericardial effusion with mild right ventricular collapse was also present. The patient presented with Beck's triad (jugular venous distension, hypotension, and muffled heart sounds) and pulsus paradoxus within the first 24 hours of admission. An emergency pericardiotomy was performed, removing 700 milliliters of yellowish fluid from an apparently healthy pericardium.

The pathology report described dense, irregular fibrous connective tissue without fibrin, necrosis, inflammation, or neoplastic infiltration. The morphological study was considered normal (**Figure 2**). In the ICU, due to severe cardiac dysfunction, we continued with 300 mcg/day of levothyroxine until we reached free thyroxine levels of 1 ng/dL, which we achieved in two weeks.

The patient improved progressively, with improvement in bradypsychia, facial edema, ejection fraction (35%), dyspnea, and functional class. He was discharged on levothyroxine sodium at 1.6 mcg/kg/day, with a follow-up appointment in 4 weeks for TSH and free T4, a new echocardiogram, and monitoring by endocrinology and cardiology.

Initial laboratory tests revealed normocytic normochromic anemia (hemoglobin 11.7 g/dL; reference range 13 - 16 g/dL), elevated total bilirubin 1.45 mg/dL (reference range 0 - 1 mg/dL), mild hyponatremia 133 mmol/L (reference range 136 - 145 mmol/L), markedly elevated thyroid-stimulating hormone (TSH) 38.06 μIU/mL, and free T4 < 0.4 ng/dL (reference range 0.7 - 1.48 ng/dL). Arterial blood gases showed no hypoxemia or hypercapnia.

A total abdominal ultrasound revealed moderate ascites, and a chest X-ray was ordered, which showed cardiomegaly with a "water jug sign" (**Figure 1**). According to the Popoveniuc scale, a score of 60 points was obtained, which is considered highly suggestive of myxedema crisis.

The patient was transferred to the intensive care unit (ICU), and a dose of oral

levothyroxine 300 mcg/day was initiated (since intravenous levothyroxine was not available at the institution).

Treatment with hydrocortisone 50 mg every 8 hours (intravenously) and hydration with crystalloids was also started.

Given the cardiomegaly evidenced on the chest X-ray, a transthoracic echocardiogram was requested, which reported severe dilated cardiomyopathy with diffuse generalized hypokinesis, left ventricular dysfunction with an ejection fraction of 12%, and a pericardial effusion of 33 mm, with mild right ventricular collapse.

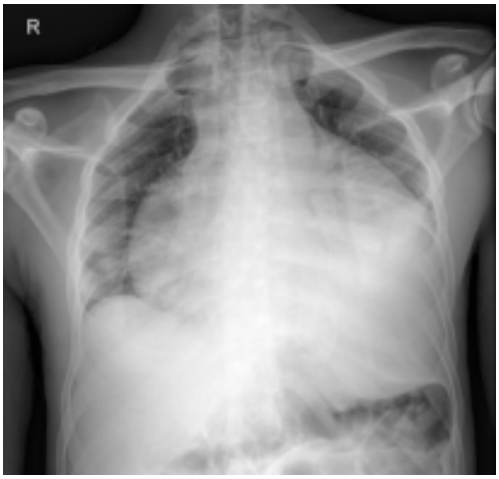

Figure 1. Case 1: Chest X-ray showing the jug sign secondary to massive pericardial effusion.

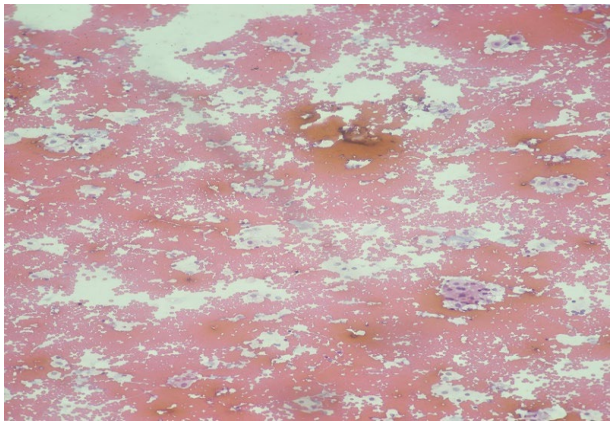

Figure 2. Pericardial biopsy: Morphologically normal connective tissue, with the presence of a hemorrhagic background.

Case 2:

A 25-year-old woman with systemic lupus erythematosus (SLE) for 4 years and hypothyroidism, who was non-adherent to treatment with levothyroxine 100 mcg/day, presented with seizures, Raynaud's phenomenon, and lupus nephritis. She is currently receiving outpatient treatment with mycophenolate mofetil 1.5 g/day, hydroxychloroquine 200 mg/day, and levetiracetam 500 mg every 12 hours.

The patient arrived at the emergency department with seven days of facial edema, unquantified fever, asthenia, adynamia, and intermittent dry cough. Vital signs were: BP: 90/48 mmHg, HR: 110 bpm, RR: 22 rpm, T: 36.2˚C, SpO_2: 95% requiring low-flow oxygen via nasal cannula. Oral ulcers and bibasilar crackles were present. Blood tests showed normocytic normochromic anemia, thrombocytosis, elevated acute phase reactants, low serum C3 and C4, reactive anti-DNA, TSH greater than 100 μIU/mL, and free thyroxine < 0.4 ng/dL. The risk of lupus activity, calculated using SLEDAI-2K, was 8 points (active), and the risk of Myxedema State, according to the Popoveniuc score, was 45 points. Treatment was initiated with prednisolone 1 mg/kg, chloroquine 150 mg/day, and levothyroxine 300 mcg on the first day, followed by 1.6 mcg/kg/day. On admission, **a Chest computed tomography demonstrated bilateral pleural effusions and a moderate circumferential pericardial effusion (Figure 3)**. A transthoracic echocardiogram revealed severe pericardial effusion with signs of cardiac tamponade (right heart chamber collapse) and clinical evidence of pulsus paradoxus. A pericardiotomy was performed to drain the fluid and obtain a biopsy. **Histopathological examination of the pericardial biopsy showed normal fibroconnective tissue lined by mesothelial cells without inflammatory infiltrates (Figure 4)**.

Pericardial tuberculosis was ruled out in the pericardial fluid; PCR for Mycobacterium tuberculosis and culture for tuberculosis were negative. She continued corticosteroid and levothyroxine treatment for 10 days, achieving free thyroxine levels above 1 ng/dL, with clinical improvement. She was discharged with levothyroxine 200 mcg/day, management for her systemic lupus erythematosus (SLE), and follow-up by rheumatology, endocrinology, and cardiology.

The differential diagnosis of pericardial effusion included lupus serositis and infective pericarditis; therefore, molecular tests and culture for Mycobacterium tuberculosis were requested, both of which were negative. Uremic pericarditis and effusion associated with hypothyroidism were also considered.

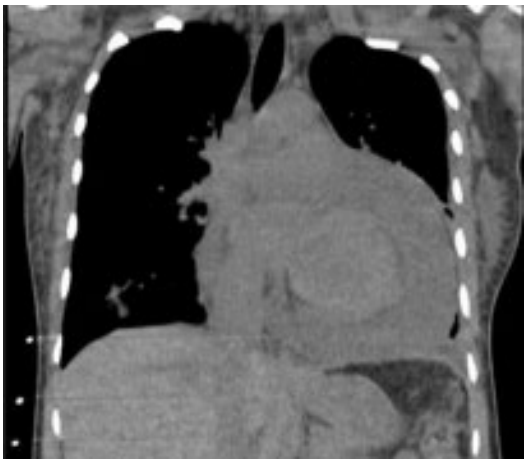

Figure 3. Coronal section of computed tomography of the chest with evidence of bilateral pleural effusion and moderate amount of pericardial fluid.

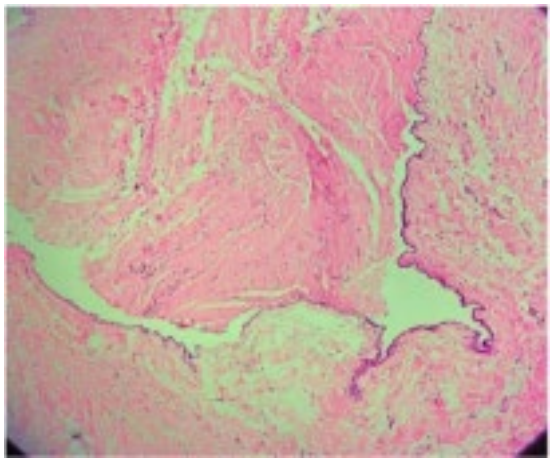

Figure 4. Pericardial biopsy: Fragment of fibroconnective tissue lined by mesothelial cells without atypia. No associated inflammatory process is recognized.

Although the patient had active systemic lupus erythematosus (SLE), several findings supported severe hypothyroidism as the primary cause, given the markedly elevated TSH, profoundly suppressed free T4 concentration, other myxedematous symptoms, low-voltage electrocardiographic findings, absence of inflammatory infiltrates in the pericardial biopsy, and rapid improvement following levothyroxine replacement therapy. Furthermore, the pericardial effusion improved without requiring an increase in the steroid dose.

Case 3:

A 48-year-old woman with hypertension and type 2 non-insulin-dependent diabetes mellitus, without prior treatment, presented to the emergency department with a 15-day history of bradypsychia, progressive edema in the lower extremities, increased abdominal girth, and mild to moderate dyspnea. Vital signs: BP 160/76 mmHg, HR: 88 bpm, RR: 18 rpm, T: 36.5˚C, SaO_2: 95%. She had a puffy face, a positive Hertoghe sign, bradylalia, goiter without indurated nodules, crackles in bilateral lung bases, negative pulsus paradoxus, a distended abdomen without swelling, a positive ascites wave, dry skin, and bilateral non-pitting edema in the legs.

On admission, the electrocardiogram showed low-voltage QRS complexes. Due to goiter, TSH and free T4 were ordered, resulting in a TSH of 150 μIU/mL and a free T4 of <0.4 ng/dL. **Chest radiography demonstrated marked cardiomegaly with the classic "water bottle" (jug) sign caused by massive pericardial effusion (Figure 5)**. The chest CT scan revealed moderate bilateral pleural effusion and severe pericardial effusion, confirmed by echocardiography with an LVEF of 60% and right heart chamber collapse. Clinically, there was no pulsus paradoxus or Kussmaul's sign. Treatment with levothyroxine was initiated, 400 mcg /day for 4 days and then 200 mcg/day. The patient's hemodynamic status remained stable, and symptoms improved. After 15 days, an echocardiogram showed less pericardial effusion. She was discharged and re-evaluated at 6 weeks, with an echocardiogram showing no effusion and a TSH of 1.8 μIU/mL.

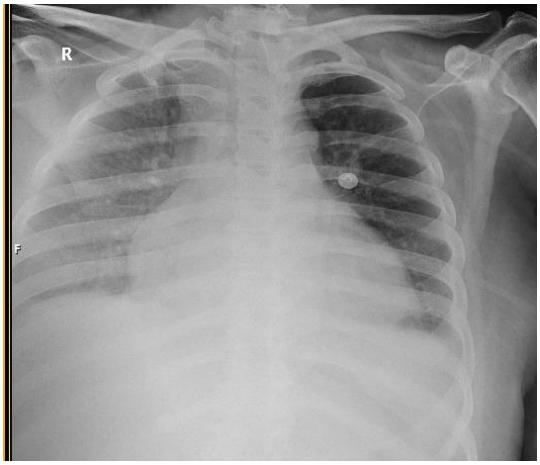

Figure 5. Case 3: Chest X-ray showing the jug sign secondary to massive pericardial effusion.

3. Discussion

We present three cases of severe hypothyroidism with clinical and laboratory findings. In two cases, the Popoveniuc score was 45 or higher, indicating a high risk of Myxedema State. All three cases presented with severe pericardial effusion, requiring different approaches.

The first patient presented with a massive pericardial effusion, signs of cardiac tamponade, and pulsus paradoxus, requiring emergency pericardiotomy. He exhibited severe systolic dysfunction with an ejection fraction of 12% and reversible dilated cardiomyopathy. **Similar cases of hypothryoidism induced dilated cardiomyophaty with recovery of left ventricular function after thyroid hormone replacement have been previously reported** [7]. Left ventricular function improved with hormone replacement therapy, demonstrating the direct effect of T3 deficiency on myocardial contractility, intracellular calcium regulation, and β-adrenergic sensitivity. This case demonstrates that early hormone replacement therapy, even without intravenous administration, can restore myocardial function and reverse hemodynamic compromise in conjunction with pericardial drainage.

The second patient, with SLE and untreated hypothyroidism, presented with simultaneous decompensation of both diseases. Due to the presence of severe pericardial effusion and signs of cardiac tamponade with hemodynamic instability, an emergency pericardiotomy was performed for diagnostic and therapeutic purposes. Subsequently, decompensated hypothyroidism was confirmed as the primary cause.

In the third case, the patient presented with Myxedema State for the first time, with no prior history but with risk factors such as type 2 diabetes and hypertension. Despite severe pericardial effusion and echocardiographic collapse of the right heart chambers, she showed no signs of hemodynamic instability. She opted for conservative management and, with hormone replacement therapy, had a good clinical course. This demonstrates that, in Myxedema State, the correlation

between echocardiography and clinical signs can be inconsistent. Therefore, treatment, including invasive procedures such as pericardiocentesis or pericardiotomy, should be based on clinical and hemodynamic compromise and not solely on imaging findings.

In our institution, intravenous levothyroxine is not available as it is in many hospitals across the country; therefore, oral administration was used. Evidence comparing oral versus intravenous levothyroxine remains limited and is still based on small sample groups.

Recent reviews have indicated no significant difference in mortality between the two routes of administration. Historically, high-dose levothyroxine regimens have been reported to replenish depleted extrathyroidal hormone stores. However, excessively high doses, especially in elderly patients, can increase the risk of arrhythmias, myocardial ischemia, and fatal outcomes.

According to the 2014 American Thyroid Association guidelines, patients with myxedema crisis should receive an initial loading dose of 200 - 400 mcg of intravenous levothyroxine, followed by daily hormone replacement therapy, while lower doses are recommended for patients with established cardiovascular disease. In our cases, dosing strategies were individualized based on age and hemodynamic status, with close clinical and hemodynamic monitoring during hospitalization.

Hypothyroid pericardial effusion:

Pericardial effusion occurs in 3% of cases of hypothyroidism and in up to 80% of Myxedema State. It affects women more (5% - 8%), especially those over 60 years of age [8] [9]. Causes include neoplasms, infections, heart failure, radiation, trauma, connective tissue disorders, autoimmune diseases, iatrogenic causes, idiopathic pericarditis and, rarely, viral origin associated with hypothyroidism [10].

The pathophysiology involves increased permeability of pericardial vessels, decreased lymphatic drainage of albumin, changes in oncotic pressure, accumulation of mucopolysaccharides, and disruption of protein homeostasis. This leads to gradual accumulation and an increase in pericardial diameter, allowing for adaptation. Mucinous infiltration reduces contractility and impairs diastolic filling, causing low cardiac output and, sometimes, reversible pseudoheart failure. See Table 1. Therefore, close hemodynamic monitoring and support with vasopressors or transient inotropes are required in the ICU [11].

Table 1. Pathophysiological mechanisms of pericardial effusion in hypothyroidism.

Pathophysiological mechanism		Clinical manifestation
Increase in the capillary permeability	Thyroid hormone deficiency impairs endothelial function and increases the leakage of plasma proteins into the extravascular space.	Liquid formation protein-rich pericardial.

Continued

Accumulation of mucopolysaccharides (glycosaminoglycans)	Lack of thyroid inhibition It increases the deposits of hyaluronic acid and chondroitin sulfate in tissues, including the pericardium.	Water and protein retention, causing edema and pericardial thickening.
Decreased lymphatic drainage	Metabolic hypofunction and low cardiac output reduce the reabsorption of pericardial fluid.	Progressive accumulation of fluid, generally of a certain course slow and adaptive.
Alteration of plasma oncotic pressure	Hypoalbuminemia and reduced hepatic synthesis facilitate the movement of fluid into the pericardial space.	Increased volume of pericardial effusion.
Myocardial infiltration and contractile dysfunction	T3 deficiency affects the contractility, calcium transport and β-adrenergic sensitivity.	Decreased cardiac output, low lymphatic flow and p pseudo-plugging.

Diagnosis and classification:

The diagnosis of Myxedema State remains clinical, but it must be based on clinical and biochemical criteria. There is no clear cutoff point, as laboratory results often do not correlate with the clinical status. However, in the three cases presented, TSH was greater than 30 μIU/mL and free T4 was less than 0.4 ng/dL. For a more accurate diagnostic suspicion, we propose using the scoring system of Popoveniuc *et al.* [2]. Clinical suspicion is essential to initiate timely treatment.

None of the three cases presented with coma, but rather neurological alterations such as bradypsychia and bradylalia, associated with elevated TSH and suppressed free T4. Other physical signs included bradycardia, myxedema, hypotension, and dyspnea with pericardial effusion. Echocardiography was key to documenting the effusion and evaluating ventricular function, confirming recovery after hormonal normalization [12].

In previous case series, pericardial effusions associated with Myxedema State have been documented, leading to an approach algorithm based on signs of hemodynamic instability and cardiac tamponade, such as hypotension, tachycardia, jugular venous distension, and pulsus paradoxus [5].

Given the variability in clinical presentation, a comprehensive clinical approach model is proposed with three components: diagnosis, pathophysiological classification, and a therapeutic approach. This model allows for an individualized approach adapted to the patient's clinical context, providing a useful conceptual tool to guide clinicians in diagnosis and treatment.

Diagnostic and therapeutic approach:

Look for signs of low cardiac output during clinical evaluation: hypoperfusion, bradycardia, hypothermia, hypotension, low pulse pressure, high diastolic blood

pressure, and altered mental status (bradypsychia, bradylalia, lethargy, obtundation, and coma). Biochemically confirm this with elevated TSH, low free T4, hyponatremia, normocytic normochromic anemia, hypoglycemia, and high cholesterol. On the electrocardiogram, look for low-voltage QRS complexes, electrical alternans, nonspecific ST/T segment abnormalities, second-degree AV block, QT prolongation, and torsades de pointes. On chest X-rays, look for cardiomegaly, the "water jug sign", and bilateral pleural effusion. Transthoracic echocardiography is the method of choice for evaluating pericardial effusion, left ventricular function, right heart chamber collapse, signs of cardiac tamponade, diastolic dysfunction (increased isovolumic relaxation time), and reversible dilated cardiomyopathy.

Stages in the approach:

1) Initial resuscitation and hemodynamic stabilization:

- Admit all patients with suspected Myxedema State to intensive care, especially if there are signs of low cardiac output.
- Oxygen therapy, temperature control and fluid replacement as needed.
- Hydrocortisone 50 - 100 mg/IV every 8 hours until adrenal involvement is ruled out [8].

2) Thyroid hormone replacement:

- Levothyroxine IV: loading dose of 200 - 400 mcg/IV, followed by calculated doses of 1.6 mcg/kg/day until T4L levels equal to or greater than 1 are reached (some authors propose 0.85 ng/dL).
- If an intravenous formulation is not available, use the oral route or via a feeding tube (orogastric or nasogastric). In patients with bariatric surgery or gastric malabsorption, use the liquid or gel formulation [13]. In exceptional cases, use the intrarectal route, although it may cause diarrhea [14].
- Liothyronine (T3) in refractory cases or with severe neurological compromise (hemodynamic instability) at a dose of 2.5 - 10 mcg IV/8h within the first 48 - 72 hours [1].

Monitoring and tracking:

During the acute phase of Myxedema State with cardiac involvement, daily clinical and hemodynamic evaluation is recommended, supplemented by free T4 measurement every 72 hours, until a sustained increase in thyroid hormone levels is observed. Transthoracic echocardiography should be performed upon admission for all patients, and the frequency of subsequent follow-up should be adjusted according to the severity of the pericardial effusion.

All three patients showed clinical and paraclinical improvement after initiating hormone replacement therapy. In the first case, free T4 normalized after two weeks, with an improvement in left ventricular ejection fraction from 12% to 35% and resolution of symptoms. In the second case, free T4 levels increased to values greater than 1 ng/dL after 10 days of treatment, followed by stabilization and a decrease in pericardial effusion. In the third case, symptom improvement was achieved after 8 days of treatment, but normalization of TSH levels was reached at 6 weeks. Follow-up echocardiography in all patients demonstrated a significant

reduction after initiating treatment.

4. Conclusion

Pericardial effusion associated with myxedema remains an uncommon but clinically relevant complication of severe hypothyroidism, given its high risk of hemodynamic instability (due to cardiac tamponade) and a significant increase in mortality. Currently, guidelines exist for the management of severe hypothyroidism and myxedema crisis, but specific recommendations for managing pericardial effusion in this clinical setting remain limited. These cases highlight the importance of timely diagnosis, multimodal cardiovascular evaluation, and individualized management, taking into account the severity and hemodynamic compromise of the patient, rather than relying solely on echocardiographic findings.

Author Contributions

All authors contributed equally to the description of the cases, the creation of the manuscript, and the final review.

Conflicts of Interest

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

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