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# Myxedema Coma in the Emergency Department: A Diagnostic Challenge – A Case Report

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**Abstract**

Myxedema coma is an endocrine emergency and the most severe manifestation of decompensated hypothyroidism. We report the case of a 47-year-old woman admitted to the emergency department with acute neurological symptoms. Although she had no previous diagnosis of hypothyroidism, she exhibited multiple clinical features suggestive of myxedema coma. Delayed recognition of the condition resulted in postponed initiation of appropriate therapy, contributing to rapid clinical deterioration and ultimately death. This case highlights the diagnostic challenges of myxedema coma in the emergency setting and reviews its clinical presentation, diagnostic approach, and current management, emphasizing the importance of early recognition and prompt treatment to improve patient outcomes.

**Keywords:** myxedema coma, hypothyroidism, endocrine emergency, critical care

## Introduction

Myxedema coma is an endocrine emergency in which early recognition and prompt treatment are critical determinants of patient outcomes. It represents a rare but severe complication of decompensated hypothyroidism caused by profound and prolonged thyroid hormone deficiency, leading to multiorgan dysfunction. (1)

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The estimated incidence ranges from 0.22 to 1 case per million people annually, with a reported mortality rate of approximately 20–25%. The condition predominantly affects women and occurs more frequently during the winter months. Clinically, it is characterized by altered mental status, hypothermia, and bradycardia, accompanied by dysfunction in multiple organ systems. Biochemically, patients with primary hypothyroidism typically present with low serum T3 and T4 levels and elevated thyroid-stimulating hormone (TSH) levels, whereas patients with secondary hypothyroidism usually exhibit low or inappropriately normal TSH concentrations. (2) Diagnostic scoring systems have been developed to support clinical suspicion by assessing organ involvement, metabolic abnormalities, and precipitating factors. Management is based on thyroid hormone replacement therapy and glucocorticoid administration, both of which significantly improve outcomes when initiated promptly.

## Case report

A 47-year-old woman with a documented allergy to quinolones and no known chronic medical conditions presented to the emergency department on December 21, 2025. She presented with visual and auditory hallucinations, accompanied by paranoid ideation, and insomnia lasting approximately 48 hours. According to a family member, she had sustained minor head trauma two weeks earlier after falling from a height of approximately 50 cm, resulting in a right frontal scalp contusion.

On arrival, her oxygen saturation was 77% while breathing room air ($FiO_2$ 21%). Physical examination revealed a Glasgow Coma Scale (GCS) score of 9 (E1, V4, M4), generalized pallor, perioral cyanosis, dry oral mucosa, diminished breath sounds bilaterally, and 2+ generalized pitting edema.

Supplemental oxygen was administered via low-flow nasal cannula, resulting in an improvement in oxygen saturation to >90%. Given her recent history of head trauma, a non-contrast head computed tomography (CT) scan was performed, which showed no evidence of intracranial hemorrhage or other acute intracranial abnormalities (Figures 1 and 2).

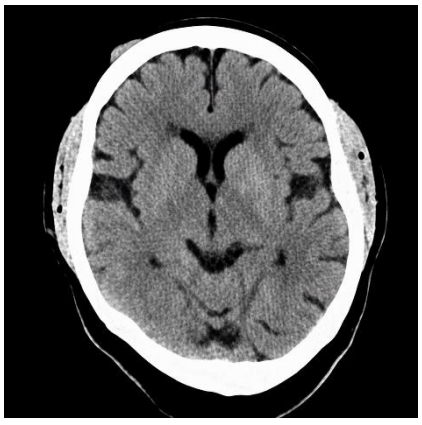

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Figure. 1 Non-contrast axial head CT at the level of the lateral ventricles, demonstrating preserved differentiation between gray and white matter. No focal hypodense or hyperdense lesions suggestive of ischemic or hemorrhagic injury are observed. More than 72 hours after symptom onset, the ventricular system preserves its usual size and morphology.

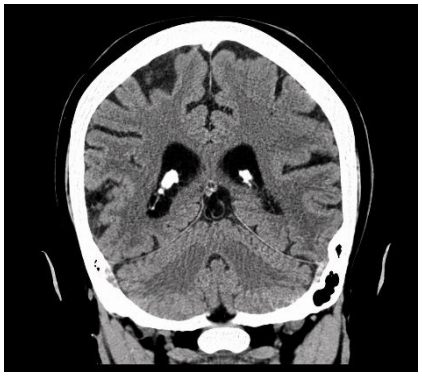

Figure. 2 Non-contrast coronal head CT demonstrating preserved gray-white matter differentiation. Bilateral, symmetric hyperattenuating foci are identified in the basal ganglia region, consistent with physiologic calcifications. Ventricular system with preserved morphology and size. Basal cisterns are visible and patent. No focal hypodense lesions suggestive of acute ischemic stroke are identified. No hyperdense extra-axial collections suggestive of hemorrhage are observed.

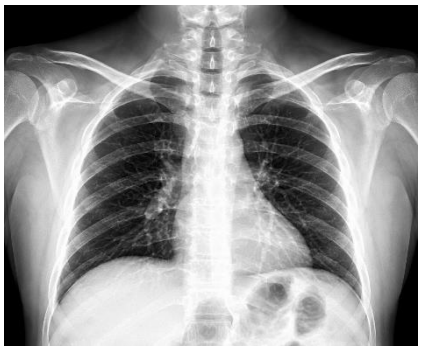

Figure 3. Anteroposterior chest radiograph obtained on admission, demonstrating clear lung fields without focal air space consolidation or interstitial infiltrates. Mild global cardiomegaly is present.

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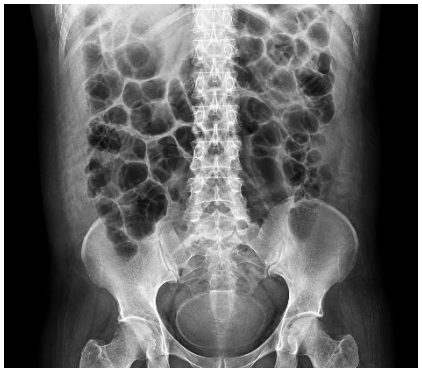

Figure. 4. Anteroposterior abdominal radiograph demonstrating dilation of the small bowel and colonic loops, with abundant fecal material and scant distal gas. These findings are consistent with non-obstructive ileus in the setting of an underlying metabolic disorder.

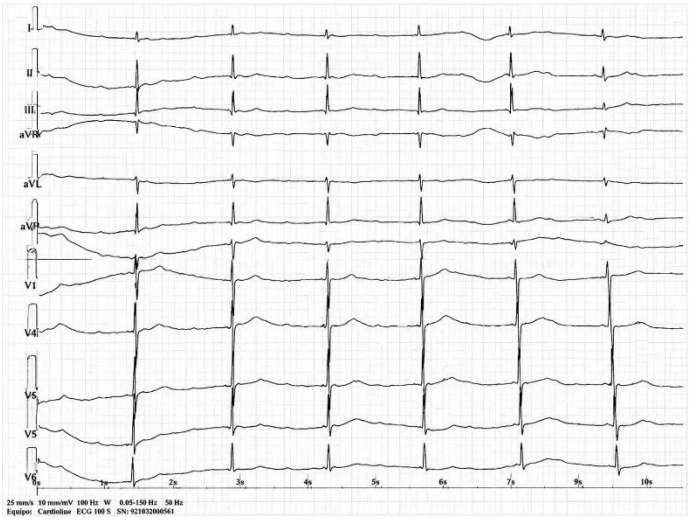


Figure 5. Standard 12-lead electrocardiogram demonstrating a heart rate of 42 bpm and electrocardiographic findings consistent with complete (third-degree) atrioventricular block.

Laboratory evaluation demonstrated markedly elevated TSH levels with severely decreased free T4 and T3 concentrations (Table 1), establishing the diagnosis of primary hypothyroidism. Oral levothyroxine was initiated at dose of 50 µg once daily.

During her prolonged stay in the emergency department, her neurological status fluctuated between agitation and somnolence. Mixed delirium related to prolonged hospitalization and limited family support was initially suspected. Despite adequate crystalloid therapy, she developed oliguria, persistent generalized edema, increasing oxygen requirements, progressive facial swelling, and worsening pallor. In response to these findings, the oral levothyroxine dose was increased to 100 mcg once daily.

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Table 1. Thyroid hormone and serum cortisol levels obtained at admission and 2 days after the initiation of levothyroxine therapy, demonstrating persistently elevated TSH levels with profoundly reduced free T4 and free T3 concentrations.

| Laboratory studies | 22/12/2025 | 24/12/2025 |
|---|---|---|
| **FT4** | 0.07 ng/ dL | 0.00 ng/ dL |
| **Thyroid- stimulating hormone** | 124.216 UI/ml | 99.775 UI/ml |
| **Total T3** | 1.07 µg/ml | 0.63 µg/ml |
| **Total T4** | 0.55 µg/dL | 0.38 µg/dL |
| **FT3** | 1.50 pg/ml | 1.35 pg/ml |
| **Serum cortisol** | 23.57 µg/dL | |

Table 2. Blood gas parameters measured at admission to the Emergency Department and upon admission to the Internal Medicine ward, showing hypercapnia and hyperlactatemia in the latter measurement

| Venous Blood Gas Analysis | 22/12/2025 | 01/01/2026 |
|---|---|---|
| **pH** | 7.39 | 7.24 |
| **pCO2** | 53 | 57 |
| **pO2** | 40 | 26 |
| **HCO3** | 28.5 | 24.5 |
| **Lactate** | 1.6 | 10.6 |
| **BE** | 6.8 | -4.6 |

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Table 3. Laboratory studies at admission to the Emergency Department and upon admission to the Internal Medicine service, with the latter demonstrating renal, hepatic, and hematologic dysfunction.

| Laboratory studies | 21/12/2025 | 30/12/2025 | 31/12/2025 |
|---|---|---|---|
| **WBC** | 2.16 x 10^3 $mm^3$ | 1.96 x 10^3 $mm^3$ | 5.93 x 10^3 $mm^3$ |
| **Neutrophils** | 75 % | 75.76 % | 88.45% |
| **Lymphocytes** | 25 % | 14.32 % | 5.01% |
| **Hemoglobin** | 11.23 g/dL | 11.65 g/dL | 9.52 g/dL |
| **Hematocrit** | 33.05 % | 34.27 % | 28.20% |
| **Platelet count** | 209 x 10^3 $mm^3$ | 138.8 x10^3 $mm^3$ | 122.2x10^3 $mm^3$ |
| **D- dimer** | | | 114 ng/mL |
| **PT** | | 10.8 s | 10.8 s |
| **aPTT** | | 35.1 s | 35.5 s |
| **INR** | | 1.02 s | 1.02 s |
| **Procalcitonin** | | | 0.21 ng/Ml |
| **Glucose** | | 63.1 mg/dL | 134.8 mg/dL |
| **BUN** | 26.3 mg/dL | 17.71 mg/dL | 40.56 mg/dL |
| **Urea** | 56.8 mg/dL | 37.9 mg/dL | 86.8 mg/dL |
| **Creatinine** | 0.7 mg/dL | 0.6 mg/dL | 1.8 mg/dL |
| **Triglycerides** | 210.1 mg/dL | | 62.88 mg/dL |
| **Total Bilirrubin** | 0.3 mg/dL | | 0.3 mg/dL |
| **Sodium** | 134.6 mEq/L | 138.8 mEq/L | 134.8 mEq/L |
| **Potassium** | 4.27 mEq/L | 3.72 mEq/L | 3.95 mEq/L |
| **Cl** | 96.2 mEq/L | 96.5 mEq/L | 97.3 mEq/L |
| **ALT** | 39.3 mg/dL | | 85.3 UI/L |
| **AST** | 36.8 UI/L | | 50.3 UI/L |

On the ninth hospital day, the patient was transferred to the internal medicine ward and evaluated by psychiatry because of persistent visual and auditory hallucinations associated with mixed delirium. She was diagnosed with depressive disorder likely secondary to longstanding hypothyroidism with psychotic features, and treatment with selective serotonin reuptake inhibitors and a second-generation antipsychotic was initiated.

On the second day after transfer to the internal medicine ward, the patient experienced sudden neurological deterioration. She presented with a GCS score of 3, miosis, bradycardia of up to 44 beats/minute, atrioventricular block on electrocardiography (Figure 5), hypotension, cold and dry skin, and severe generalized edema (+++/+++). Advanced airway management and central venous catheter placement were performed. Venous blood gas analysis demonstrated hypercapnia and hyperlactatemia (Table 2).

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The Popoveniuc score was calculated at 95 points, strongly supporting the diagnosis of myxedema coma. At that time, the patient had already developed multiorgan failure involving the renal, hematologic, neurologic, respiratory, and hemodynamic systems (Table 3), with a Sequential Organ Failure Assessment (SOFA) score of 14 and an Acute Physiology and Chronic Health Evaluation II (APACHE II) score of 30, corresponding to an estimated mortality risk greater than 80%.

Despite supportive management, the patient experienced progressive hemodynamic deterioration and experienced cardiorespiratory arrest on January 2, 2026. Because she had previously signed advance directives declining resuscitative measures, advanced life support was withheld, and the patient died.

**Pathophysiology**

Patients with hypothyroidism generally develop adaptive neurovascular mechanisms that maintain metabolic homeostasis. However, in the presence of a precipitating factor, such as those listed in Table 4, these compensatory mechanisms fail, resulting in profound reductions in T4 and consequently T3 levels. This leads to elevated TSH levels in primary hypothyroidism, whereas TSH concentrations may remain low or normal in secondary hypothyroidism.

The resulting hormonal imbalance produces dysfunction across multiple organ systems, particularly the central nervous, respiratory, cardiovascular, and renal systems. Importantly, thyroid hormone levels are not necessarily proportional to the severity of clinical manifestations. (3,4)

According to a 2025 systematic review by Zhang, Chu, and Han, infectious and inflammatory conditions are among the most common precipitating factors. Cytokines such as interleukin-1 and tumor necrosis factor-alpha (TNF-α) alter thyroid hormone metabolism by regulating proteins involved in hormone transport, receptor activity, and metabolism through inflammatory pathways such as nuclear factor kappa-B (NF-κB) and activator protein-1. (2)

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Table 4. Precipitating factors for myxedema coma.

| Precipitating Factors | |
|---|---|
| Infections | Surgical procedures |
| Sepsis | Hypercalcemia |
| Pulmonary thromboembolism | Hyponatremia |
| Stroke | Hypoxemia |
| Acute Myocardial Infarction | Hypercapnia |
| Winter season (cold exposure) | Anesthetic procedures |
| Heart Failure | Trauma |
| Discontinuation of hypothyroidism treatment | Medications: amiodarone, lithium, tyrosine kinase inhibitors |
| Gastrointestinal bleeding | Consumption of Bok Choy |

## Clinical Manifestations

The manifestations of myxedema coma generally represent severe exacerbations of hypothyroid symptoms. In the previously mentioned systematic review, 88.9% of patients presented with neurological abnormalities, 71.9% with hypothermia, and 66.2% with bradycardia, making these the most frequently reported findings. (2)

## Central Nervous System

Historically, the diagnosis was limited to patients presenting in coma; however, current understanding recognizes a broad spectrum of neurological manifestations, including seizures, status epilepticus, depression, lethargy, confusion, drowsiness, and coma. These abnormalities are primarily related to decreased cerebral blood flow, impaired glucose metabolism, and electrolyte disturbances. (4,5)

Cases of “myxedematous psychosis,” characterized by behavioral disturbances and persecutory delusions, have also been reported. (5)

## Myxedema

Myxedema is considered a pathognomonic feature of severe hypothyroidism. It results from the abnormal accumulation of hyaluronic acid and other glycosaminoglycans in tissues throughout the body, particularly in the skin, intestines, lungs, and muscles. Reduced thyroid hormone activity promotes fibroblast-mediated synthesis of hyaluronic acid, fibronectin, and collagen, leading to interstitial edema and organ dysfunction. (1)

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### Hypothermia

Impaired thermogenesis secondary to hypothyroid-induced hypometabolism may reduce body temperature to as low as 24°C. Temperatures ≤32°C are associated with increased mortality and are considered a poor prognostic indicator. (5)

### Cardiovascular System

Reduced inotropic and chronotropic cardiac activity predisposes patients to shock and fatal arrhythmias. Bradycardia, hypotension, hypertension, and QT interval abnormalities are commonly observed. (6)

The term "myxedematous heart" has been used to describe the combination of cardiomegaly, electrocardiographic abnormalities, and hemodynamic compromise associated with severe hypothyroidism. (5)

### Respiratory System

Depression of the respiratory center results in a reduced ventilatory response to hypoxia and hypercapnia. Mechanical airway obstruction may also occur because of laryngeal edema, macroglossia, and goiter, potentially necessitating advanced airway management. (6)

### Gastrointestinal System

Patients commonly present with abdominal pain, nausea, and reduced intestinal motility, which may progress to paralytic ileus or toxic megacolon. (5,6) This is particularly relevant in countries where intravenous thyroid hormone formulations are unavailable, as impaired gastrointestinal absorption may reduce the efficacy of oral therapy.

### Renal System

Reduced renal blood flow and glomerular filtration rate are common findings. In addition, impaired free water excretion caused by excess antidiuretic hormone activity contributes to hyponatremia. (3,6)

### Diagnosis

Diagnosing myxedema coma in the emergency department is particularly challenging because patients often present with altered mental status and incomplete or unavailable medical histories. Nevertheless, a high index of clinical suspicion, together with biochemical abnormalities can support prompt diagnosis.

In patients with suspected myxedema coma, treatment should not be delayed while awaiting laboratory confirmation. Currently, two diagnostic scoring systems have been proposed.

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Popoveniuc et al. proposed a scoring system for myxedema coma (Table 5) in which scores <25 make the diagnosis unlikely, scores between 25 and 59 indicate risk, and scores ≥60 strongly support the diagnosis. The reported sensitivity and specificity of 100% and 85%, respectively. (7)

Another scoring system proposed by Chiong et al. in 2015 incorporates six variables, including TSH and free T4 levels (Table 6), although it demonstrates lower sensitivity and specificity (<80%). (8)

Biochemically, primary hypothyroidism is typically characterized by elevated TSH levels and reduced free T3 and T4 concentrations. However, in secondary hypothyroidism, TSH levels may remain normal or decreased.

Table 5. Scoring System for the Diagnosis of Myxedema Coma Proposed by Popoveniuc.

| **Thermoregulatory System Dysfunction** | |
|---|---|
| Body temperature >35°C | 0 |
| Body temperature 32–35°C | 10 |
| Body temperature <32°C | 20 |
| Neurological Status | |
| Alert | 0 |
| Somnolence/lethargy | 10 |
| Obtunded | 15 |
| Stupor | 20 |
| Coma/ seizures | 30 |
| **Gastrointestinal Findings** | |
| Anorexia/ abdominal pain/constipation | 5 |
| Reduced intestinal motility | 15 |
| Paralytic ileus | 20 |
| **Precipitating Event** | |
| Absent | 0 |
| Present | 10 |
| **Cardiovascular Dysfunction** | |
| No bradycardia | 0 |
| HR: 50-59 bpm | 10 |
| HR: 40-49 bpm | 20 |
| HR: <40 bpm | 30 |
| Electrocardiographic changes (QT interval prolongation, conduction blocks, nonspecific ST-segment changes) | 10 |

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| | |
|---|---|
| Pericardial effusion / Pleural effusion | 10 |
| Pulmonary edema | 15 |
| Cardiomegaly | 15 |
| Hypotension | 20 |
| **Metabolic Disturbances** | |
| Hyponatremia | 10 |
| Hypoglucemia | 10 |
| Hypoxemia | 10 |
| Hypercapnia | 10 |
| Decreased glomerular filtration rate (GFR) | 10 |
| Source: Adapted by the authors from Reference 5. | |

Table 6. Scoring System for the Diagnosis of Myxedema Coma Proposed by Chiong

| **Glasgow Coma Scale** | |
|---|---|
| 0-10 points | 4 |
| 11-13 points | 3 |
| 14 points | 2 |
| 15 points | 0 |
| **TSH levels** | |
| >30 mIU/L | 2 |
| 15-30 mIU/L | 1 |
| **Reduced FT4 levels (<0.6 ng/dL)** | 1 |
| **Clinical Findings** | |
| Hypothermia | 1 |
| Bradycardia | 1 |
| Precipitating event | 1 |
| Adapted by the authors from Reference. | |

**Treatment**

Management of myxedema coma requires a multidisciplinary approach in the intensive care unit because of the high risk of multiorgan failure. Treatment should be initiated immediately, while simultaneously identifying and correcting the underlying precipitating factors.

The cornerstone of management includes continuous hemodynamic monitoring, supportive care, glucocorticoid administration, and thyroid hormone replacement therapy. (3)

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**First-line management: Glucocorticoids**

Empiric glucocorticoid therapy should be initiated before thyroid hormone replacement. Patients with severe hypothyroidism may have impaired adrenal reserve, and initiation of thyroid hormone therapy increases cortisol metabolism, potentially precipitating an adrenal crisis in those with unrecognized adrenal insufficiency. (3,5,9)

Serum cortisol levels should ideally be measured before treatment initiation. The recommended regimen consists of hydrocortisone 100 mg administered intravenously as a loading dose, followed by 50–100 mg intravenously every 6–8 hours. The dose should be gradually tapered after adrenal insufficiency has been excluded and the patient has achieved hemodynamic stability. (3,5,9)

Thyroid Hormone Replacement: Levothyroxine

Most clinical guidelines recommend high-dose levothyroxine (LT4) as first-line therapy to rapidly replenish thyroid hormone stores and promote peripheral conversion to triiodothyronine (T3).

Although levothyroxine has a relatively gradual onset of action and is generally well tolerated, gastrointestinal dysfunction frequently observed in myxedema coma may impair oral absorption. This limitation is particularly relevant in Mexico, where intravenous levothyroxine is currently unavailable.

The recommended initial dose is 200–500 mcg administered intravenously as a loading dose, followed by 50–100 mcg intravenously once daily or approximately 1.6 mcg/kg/day. When intravenous administration is unavailable, oral administration through a nasogastric or orogastric tube is recommended, recognizing the potential for impaired absorption. (2,3)

Under these circumstances, the highest feasible oral dose should be administered. A practical conversion from intravenous to oral therapy may be estimated using the following formula:
Oral levothyroxine dose = Intravenous dose ÷ 0.75

Treatment should be initiated under continuous cardiac monitoring, particularly in elderly patients or those with cardiovascular disease. (2,3)

Thyroid Hormone Replacement: Liothyronine

Liothyronine (LT3) has a faster onset of action because it does not require peripheral conversion and is up to four times more potent than levothyroxine. However, its short half-life and fluctuating serum levels limit its use in patients with cardiovascular disease. (3,10)

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Several sources recommend combined therapy with LT4 and LT3. Suggested regimens include:
• Initial dose: LT4 200–300 mcg plus LT3 25 mcg administered intravenously.
• After 24 hours: LT4 100 mcg plus LT3 25 mcg administered intravenously.
• Maintenance therapy: LT4 50 mcg intravenously once daily until oral administration is feasible. (2, 3, 10)

According to the 2025 British Thyroid Association guidelines, liothyronine monotherapy is not recommended unless levothyroxine intolerance exists, and its use should be avoided during pregnancy. (10)

An observational study conducted by Dutta et al. comparing intravenous and oral thyroid hormone replacement found no significant differences in survival or functional outcomes between both treatment approaches. (11) These findings suggest that the lack of intravenous formulations in certain countries may not necessarily worsen prognosis.

In our patient, intravenous thyroid hormone replacement was unavailable because no intravenous formulation is currently marketed in Mexico. Consequently, oral levothyroxine was administered despite concerns regarding impaired gastrointestinal absorption associated with myxedema coma.

**Discussion**

This case illustrates how delayed recognition of myxedema coma, prolonged emergency department stay, and limited availability of intravenous thyroid hormone replacement in resource-constrained settings may collectively contribute to poor clinical outcomes.

Myxedema coma represents the most severe manifestation of decompensated hypothyroidism and remains a true endocrine emergency. Although rare, its clinical significance lies in its nonspecific presentation and the difficulty of distinguishing it from conditions such as sepsis, cerebrovascular disease, metabolic encephalopathy, shock, or psychiatric illness.

Importantly, despite the term “coma,” many patients do not initially present with deep unconsciousness. Instead, they often demonstrate progressive deterioration in mental status, ranging from confusion and lethargy to frank coma.

Diagnosis remains primarily clinical, and treatment should not be delayed while awaiting biochemical confirmation when clinical suspicion is high. Levothyroxine administration, empiric glucocorticoid therapy, and comprehensive intensive supportive care remain the cornerstones of management.

The prognosis of myxedema coma depends largely on early recognition and timely initiation of appropriate therapy rather than on the severity of biochemical abnormalities alone. Indeed, the degree of thyroid hormone deficiency does not necessarily correlate with the extent of clinical

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deterioration, emphasizing the importance of recognizing the syndrome based on its overall clinical presentation rather than laboratory findings alone.

## Conclusion

Myxedema coma remains a rare but highly lethal endocrine emergency whose diagnosis is particularly challenging in emergency medicine because its clinical presentation often mimics a wide range of critical conditions.

Maintaining a high index of clinical suspicion is essential for early recognition. Prompt initiation of thyroid hormone replacement, empiric glucocorticoid therapy, and aggressive multiorgan supportive care remain the cornerstone of treatment and offer the greatest opportunity to improve patient outcomes.

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