



MYXEDEMA CRISIS IN AN ADOLESCENT FEMALE PRESENTING WITH SHOCK AND ERYTHEMA AB IGNE

Endocrinology

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ABSTRACT

Background: Myxedema crisis represents a profound, life-threatening manifestation of decompensated hypothyroidism. It is exceedingly rare in adolescents, typically occurring in elderly patients subjected to environmental or physiological stressors. Consequently, its clinical recognition in younger demographics is frequently delayed or missed entirely. **Case Presentation:** An 18-year-old female with a history of primary hypothyroidism presented with an acute three-day history of gastroenteritis complicated by severe hypovolemic/distributive shock and profound bradycardia. Physical examination revealed delayed relaxation of the ankle deep tendon reflexes and distinct, reticulated, hyperpigmented macular lesions spanning both calf muscles. This cutaneous manifestation was identified as erythema ab igne, arising from chronic, habitual close-proximity heat exposure to a traditional stove utilized by the patient to alleviate severe cold intolerance. Laboratory diagnostics confirmed profound primary hypothyroidism, demonstrating a markedly elevated thyroid-stimulating hormone (TSH) level (>150 mIU/L), critically depleted free thyroid hormones, and high titers of anti-thyroid peroxidase (anti-TPO) antibodies. **Conclusion:** A definitive diagnosis of myxedema crisis precipitated by acute infection and non-compliance with hormone therapy was established. Aggressive therapeutic intervention involving intravenous stress-dose corticosteroids, oral levothyroxine loading, fluid resuscitation, and inotropic support led to complete clinical recovery. This case highlights erythema ab igne as an invaluable, visible clinical indicator of underlying, poorly managed severe hypothyroidism.

KEYWORDS

INTRODUCTION

Myxedema crisis, historically referred to as myxedema coma, is an extreme, life-threatening metabolic emergency resulting from long-standing, uncorrected hypothyroidism. While the condition is characterized by systemic slowing of metabolic processes, patients infrequently present with actual coma; instead, they display altered mental status, hypothermia, bradycardia, and cardiovascular instability. The epidemiological profile of this crisis overwhelmingly skews toward elderly female patients, particularly during winter months when cold ambient temperatures tax a compromised thermoregulatory system.

Its occurrence in adolescents is extraordinarily uncommon, which poses a considerable diagnostic dilemma for clinicians due to a low index of clinical suspicion^[1].

The transition from severe hypothyroidism to an acute crisis is almost universally driven by an underlying precipitating event. Common triggers include systemic infections, cerebrovascular accidents, myocardial infarction, trauma, or the abrupt cessation of thyroid hormone replacement therapy^[2]. When a patient presents in decompensated shock, the subtle signs of underlying endocrine failure can easily be obscured by the prominent symptoms of the precipitating illness^[3].

Erythema ab igne is a localized, reticulated, erythematous-to-hyperpigmented dermatosis caused by repeated, prolonged exposure to infrared radiation or sub-maximal heat insufficient to produce a direct thermal burn^[4]. Historically noted in individuals sitting close to open fireplaces or wood-burning stoves, it is now occasionally seen with laptop computers, heating pads, or space heaters. In the context of endocrinology, severe cold intolerance is a hallmark feature of advanced hypothyroidism. Patients may seek external warmth through excessive, close-proximity heat exposure, unintentionally inducing erythema ab igne. Thus, this distinct dermatological finding can serve as an invaluable, visible diagnostic window suggesting severe, chronic thyroid hormone deficiency^[5].

Case Presentation

An 18-year-old female presented to our emergency department with a three-day history of persistent vomiting, profuse watery diarrhea, and high-grade fever. Her medical history revealed a diagnosis of primary hypothyroidism established three years prior. Although initially prescribed levothyroxine replacement therapy, the patient demonstrated non-compliance, discontinuing her conventional medication after a six-month period and transitioning entirely to indigenous, non-regulated alternative treatments. On detailed systemic inquiry, she had experienced profound, progressive lethargy, significant weight gain, and intense chronic cold intolerance. To mitigate her cold intolerance, she engaged in the daily habit of sitting in close proximity to a traditional open-flame cooking stove (*chulha*), exposing her bare lower extremities to direct radiant heat.

Upon initial physical examination, the patient was in a state of severe circulatory shock. Her blood pressure was profoundly hypotensive at 82/50 mmHg, and she exhibited a regular but marked sinus bradycardia at 44 beats per minute. Her respiratory rate was 14 breaths per minute, her oxygen saturation was 94% on room air, and her core body temperature was notably hypothermic at 35.2°C (95.4°F). She appeared lethargic, though she remained oriented to person and place.

Cutaneous evaluation revealed a striking, fixed, reticulated, serpiginous brownish hyperpigmented macular network distributed symmetrically across the anterior and lateral aspects of both calves.

The skin with these lesions was non tender, non blanching and lacked signs of acute ulceration or blistering, characteristic of advanced erythema ab igne secondary to chronic thermal radiation exposure. Neurological assessment was notable for a significantly delayed relaxation phase of her deep tendon reflexes, particularly the calcaneal (ankle) reflex.

An immediate electrocardiogram (ECG) confirmed severe sinus bradycardia with low-voltage QRS complexes but without acute ST-T

segment changes or prolonged QT interval prolongation. Emergency laboratory evaluations demonstrated a regular leukocytosis with a left shift. Her metabolic profile showed mild hyponatremia (serum sodium: 131 mEq/L) and a normal blood urea nitrogen and serum creatinine. Given her clinical presentation of refractory shock, hypothermia, bradycardia, and a known history of discontinued levothyroxine, a comprehensive serum thyroid profile was urgently requested.

The endocrinological assays confirmed a profound state of primary hypothyroidism:

- **Thyroid-Stimulating Hormone (TSH):** >150 mIU/L (Normal range: 0.4–4.5 mIU/L)
- **Free Triiodothyronine (fT3):** <0.07 pg/mL (Normal range: 2.0–4.4 pg/mL)
- **Free Thyroxine (fT4):** 0.17 ng/dL (Normal range: 0.8–1.8 ng/dL)
- **Anti-Thyroid Peroxidase (Anti-TPO) Antibodies:** Markedly elevated at 482 IU/mL (Normal range: <34 IU/mL), confirming underlying Hashimoto's thyroiditis.

Based on the combination of severe clinical hypometabolism, hypothermia, bradycardia, cardiovascular collapse out of proportion to the acute gastroenteritis, and extreme laboratory alterations, a definitive diagnosis of myxedema crisis precipitated by acute infectious gastroenteritis and profound medication non-compliance was formulated.



Figure : Erythema Ab Igne Over Calf Muscles

Clinical photographs demonstrating fixed, reticulated, and serpiginous brownish hyperpigmented macular networks distributed symmetrically over the calf muscles, secondary to chronic thermal radiation exposure from a traditional open-flame cooking stove.

Management

The patient was immediately admitted to the Medical Intensive Care Unit (MICU). Initial therapeutic priority focused on aggressive fluid resuscitation with warmed intravenous normal saline to address her hypovolemic and distributive shock components. Due to persistent hypotension despite adequate fluid challenges, continuous inotropic support via a central venous catheter was initiated using an infusion of norepinephrine, which successfully titrated her mean arterial pressure to >65 mmHg.

Simultaneously, management for suspected concomitant secondary adrenal insufficiency or blunted adrenal response—a critical safety step before thyroid hormone administration to prevent acute adrenal crisis—was implemented. She was administered an initial intravenous bolus of hydrocortisone 100 mg, followed by a maintenance regimen of 50 mg intravenously every 6 hours.

Because intravenous levothyroxine formulations were unavailable at our center, thyroid hormone replacement was initiated via an oral loading strategy. A loading dose of 300 micrograms (μg) of crushed levothyroxine tablet was administered via a nasogastric tube. This was followed by a daily maintenance dose of 100 μg administered orally. Passive rewarming using thermal blankets was carefully applied to correct her hypothermia without inducing rapid peripheral vasodilation, which could worsen her shock state. Empiric broad-spectrum intravenous antibiotic therapy (ceftriaxone) was instituted to target the precipitating gastrointestinal infection.

The patient exhibited a steady, highly favorable clinical response. Within 48 hours, her hemodynamics stabilized, allowing for the gradual weaning and eventual cessation of norepinephrine support. Her core body temperature normalized to 36.8°C, and her heart rate improved to a stable sinus rhythm fluctuating between 68 and 76 beats per minute. Her gastrointestinal symptoms completely resolved. On day five of admission, the intravenous hydrocortisone was

systematically tapered down and discontinued as her clinical status was robustly stable. Follow-up laboratory evaluations prior to discharge demonstrated an improving free T4 trend (0.68 ng/dL) and a downward trajectory of serum TSH. She was discharged in excellent clinical condition on day nine, with explicit counseling regarding medication adherence, the hazards of unverified alternative therapies, and a scheduled follow-up in the endocrine outpatient clinic.

DISCUSSION

Myxedema crisis represents the absolute zenith of uncontrolled hypothyroidism, carrying a historical mortality rate approaching 30% to 50% even with aggressive modern intensive care [2]. Structurally, it is characterized by a global down-regulation of all cellular metabolic pathways, leading to multisystem degradation.

Thermoregulation fails due to decreased basal metabolic rate, cardiac output plummets owing to negative inotropy and chronotropy, and central nervous system drive diminishes [1,5]. While classically presenting in patients over the age of 60, this case serves as a crucial clinical reminder that the condition can occur across any age group, including adolescents, when chronic treatment default matches with severe physiological stressors.

In this specific case, the etiology of the primary hypothyroidism was autoimmune Hashimoto's thyroiditis, evidenced by the markedly high anti-TPO antibody titers. The progression to an acute crisis was driven by a dual-hit mechanism: the absolute cessation of prescribed levothyroxine for indigenous options, followed by the acute physiological stress of infectious gastroenteritis. Severe dehydration from vomiting and diarrhea, acting upon an already compromised cardiovascular reserve lacking adequate thyroid hormones, precipitated severe circulatory shock.

A remarkable diagnostic feature of this case was the presentation of erythema ab igne across the patient's calf muscles. Erythema ab igne is a classic, though now less frequent, cutaneous manifestation of chronic, localized heat exposure [4]. Because thyroid hormones are responsible for maintaining core temperature, severe hypothyroidism induces an intense, constant perception of cold. This patient's continuous exposure to a traditional open-flame cooking stove to seek comfort created the characteristic hyperpigmented, reticulated vascular-pattern lesions. Finding these fixed, serpiginous markings on an acutely ill adolescent presenting in shock should immediately orient the clinician toward a chronic illness characterized by intense cold intolerance, specifically severe hypothyroidism.

The therapeutic protocol utilized here highlights successful management under resource-limited circumstances where intravenous thyroid preparations are unavailable. While guideline recommendations frequently favor intravenous levothyroxine (T^4) or liothyronine (T^3) to ensure rapid, predictable systemic availability, oral administration via loading doses remains an effective alternative. A high initial loading dose (300 to 500mcg) overcomes the potential limitations of altered gastrointestinal absorption due to myxedematous bowel wall edema [3]. Furthermore, the empirical inclusion of stress-dose hydrocortisone before or concurrent with thyroid hormone initiation is mandatory. Accelerated metabolic clearance of cortisol induced by thyroid hormone replacement in a patient with limited adrenal reserve can trigger a fatal adrenal crisis [1].

Learning Points

- **Adolescent Vulnerability:** Although classically considered a disease of the elderly, myxedema crisis can develop in adolescents and young adults who demonstrate long-term non-compliance with thyroid hormone replacement therapy.
- **Dermatological Diagnostic Clues:** Erythema ab igne can serve as an invaluable, visible clinical indicator of profound, long-standing cold intolerance secondary to underlying decompensated hypothyroidism.
- **Sequential Endocrine Management:** In patients presenting with suspected myxedema crisis and shock, empirical stress-dose corticosteroids must always be administered prior to or concurrently with the first dose of thyroid hormone to avoid precipitating an acute adrenal crisis.
- **Efficacy of Oral Loading:** When intravenous thyroid hormone preparations are inaccessible, an aggressive oral levothyroxine loading strategy is a viable, lifesaving treatment methodology.

REFERENCES

1. Wartofsky L, Burman KD. Altered thyroid function in critically ill patients. *Endocrine Reviews*. 1982;3(2): 164–217.
2. Ono Y, Ono S, Yasunaga H, et al. Clinical characteristics and outcomes of myxedema

- coma: An analysis of a national inpatient database in Japan. *Journal of Epidemiology*. 2017;27(3):117–122.
3. Wall CR. Myxedema coma: Diagnosis and treatment. *American Family Physician*. 2000;62(11):2485–2490.
 4. Gibbs S. Erythema ab igne. *British Medical Journal*. 1999;318(7193):1244.
 5. Nicoloff JT, LoPresti JS. Myxedema coma: A form of severe decompensated hypothyroidism.
 6. *Endocrinology and Metabolism Clinics of North America*. 1993;22(2):279–290.