

# Comatose patient with hypothermia, dyspnea, and general edema in the emergency department: a case report

Journal of International Medical Research

2018, Vol. 46(10) 4338–4342

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DOI: 10.1177/0300060518791074

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## Abstract

Patients presenting to the emergency department with hypothermia are rare and often require prompt diagnosis and management. Myxedema coma, which may cause severe hypothermia, is a true endocrine emergency requiring early and appropriate treatment. We report on a 47-year-old woman with a history of hyperthyroidism who underwent thyroidectomy 5 years previously, with no regular medication or examinations. She presented to the emergency department with a 1-month history of progressive dyspnea associated with general weakness. She also showed hypothermia, decreased mental status, and general edema. Echocardiography revealed increased pericardial effusion without tamponade. Laboratory examination suggested myxedema coma and hypothyroidism. She received thyroxine, glucocorticoid supplement, and intensive supportive care, after which she gradually improved and was discharged. This case suggests that myxedema coma should be considered in patients with hypothyroidism or a history of thyroidectomy who present with change in consciousness, hypothermia, or other symptoms related to critical or slow presentation in multiple organs. Moreover, long-standing hypothyroidism or precipitating acute events such as sepsis, cerebrovascular accidents, gastrointestinal bleeding, cold exposure, trauma, and some medications may also cause myxedema coma. Myxedema coma is associated with a high mortality, and patients suspected to be suffering from this condition should be treated without delay.

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## Keywords

Hypothyroidism, myxedema coma, hypothermia, general edema, thyroxine, emergency department

Date received: 22 February 2018; accepted: 5 July 2018

## Introduction

This report describes a patient with myxedema coma who presented to the emergency department (ED) with hypothermia, dyspnea, and general edema. Their condition gradually improved after treatment with thyroxine, glucocorticoid supplement, and intensive supportive care, and they were finally discharged.

## Case report

A 47-year-old woman was brought to our ED with a 1-month history of progressive dyspnea associated with general weakness. The patient presented with chills, body weight gain, and hair loss, but had no signs of fever, cough, chest pain, neurological defects, bleeding focus, trauma history, or cold. She had a history of thalassemia but was not receiving any medication for this condition.

On arrival in the ED, the patient showed lethargy and poor response. Her vital signs were as follows: respiration rate 26 bpm, pulse rate 62 bpm, temperature 31.6°C, and blood pressure 111/66 mmHg. Her pulse rate subsequently decreased to 58 bpm and her blood pressure decreased to 82/44 mmHg. Physical examination showed pale conjunctiva and general edema (Figure 1) with no other findings such as bloody or tarry stools on digital examination. However, the patient had a surgical scar in the thyroid region of her neck. An initial finger-stick test indicated a blood glucose level of 158 mg/dL. Laboratory tests detected leukopenia (white

blood cell count, 3100/ $\mu$ L), severe anemia (hemoglobin level, 3.6 g/dL), mildly elevated C-reactive protein (6.59 mg/L) and aspartate transaminase levels (38 U/L), hyponatremia (sodium level, 129 mEq/L), and hypokalemia (potassium level, 3.3 mEq/L). Examination of renal and liver functions, as well as troponin-I, albumin/calcium, and cortisol levels showed no specific results. Electrocardiography indicated sinus bradycardia, and chest radiography indicated



**Figure 1.** The patient showed hair loss and brittle hair, facial and eyelid edema, a dull blank expression, extreme fatigue, apathy and lethargy, and dry skin



**Figure 2.** Chest radiography showed cardiomegaly with bilateral pleural effusion

cardiomegaly (Figure 2). Echocardiography showed increased pericardial effusion without tamponade.

Assessment of the history of the surgical scar indicated that the patient had developed hypothyroidism 5 years after undergoing thyroidectomy, but was receiving no regular medication for this condition. The results of the above analyses and the presence of hypothermia, decreased mental status, and general edema suggested that the patient had myxedema coma. She was administered oral thyroxine sodium immediately (two tablets, 0.1 mg thyroxine sodium/tablet) once daily for 10 days, followed by a maintenance dose of one tablet once daily; intravenous hydrocortisone (100 mg hydrocortisone/vial) once daily; and methylprednisolone (one vial, 40 mg methylprednisolone/vial) every 8 hours for 1 day. Hypothyroidism was confirmed by assessment of her thyroid hormone levels (thyroid-stimulating hormone, 90.540  $\mu$ IU/mL [reference range, 0.35–5.50  $\mu$ IU/mL] and free thyroxine, 0.22 ng/dL [reference range, 0.93–1.56 ng/dL]).

The patient also received supportive care, including intubation for progressive respiratory failure and hypercapnia, inotropic agents including intravenous dopamine followed by norepinephrine for hypotension, blood transfusion for severe anemia,

rewarming with a heat lamp, and general intensive care unit arrangements. Her hypothermia resolved within 1 day and her condition then improved gradually after extubation for 11 days, and she was subsequently discharged. The patient provided written informed consent for publication of this report.

## Discussion

Hypothermic patients are rarely encountered in the ED but may require prompt diagnosis and management. Mechanisms underlying the development of hypothermia include increased heat loss, decreased heat production, and impaired thermoregulation. Hypoglycemia always develops in hypothermic patients in the ED who show a change in consciousness. Cold exposure, medication-associated vasodilation, and skin disorders such as burn injury may increase heat loss and lead to hypothermia. Moreover, endocrine diseases such as hypothyroidism, hypoadrenalism or malnutrition, hypoglycemia, and neuromuscular inefficiency, which decrease heat production, may also cause hypothermia. Other possible causes of hypothermia include impaired peripheral regulation induced by diabetes mellitus and neuropathy, impaired central regulation induced by cerebrovascular accidents, intracranial hemorrhages, and drugs, and sepsis, uremia, and trauma.<sup>1–4</sup> Patients admitted to the ED should be given a finger-stick test, and any history of drug use, alcohol consumption, and any relevant medical history should be determined promptly to facilitate the diagnosis of hypothermia.

Myxedema coma is characterized by decreased mental status, hypothermia, and other symptoms associated with the poor functioning of multiple organs due to severe hypothyroidism. This condition should be considered in patients with hypothyroidism or a history of thyroidectomy,

especially elderly female patients. Patients with myxedema coma may develop hypotension, hyponatremia, hypoglycemia, bradycardia, and hypoventilation/hypercapnia. Myxedema coma may be precipitated by acute events such as sepsis, cerebrovascular accidents, myocardial infarction, gastrointestinal bleeding, cold exposure, trauma, or medication use, especially involving lithium, amiodarone, and opioids.<sup>5,6</sup> Furthermore, patients with myxedema coma may also develop congestive heart failure and pericardial effusion in the absence of any preexisting cardiac disease.<sup>7</sup> Myxedema coma should be diagnosed based on the patient's clinical presentation and medical history and should be treated without delay, given that the results of endocrine and thyroid function tests are usually not immediately available.

Treatment for myxedema coma includes thyroid hormone therapy and intensive supportive care. However, although thyroid hormone therapy is the main treatment option, there is currently no consensus on the standard therapeutic regimen. Patients with myxedema coma are generally administered thyroxine and triiodothyronine orally and intravenously.<sup>5,8</sup> Guidelines suggest that patients with myxedema coma should receive 200 to 400  $\mu\text{g}$  (0.2–0.4 mg) thyroxine intravenously followed by a daily dose of 50 to 100  $\mu\text{g}$ , and 5 to 20  $\mu\text{g}$  triiodothyronine intravenously followed by 2.5 to 10  $\mu\text{g}$  every 8 hours, which can be changed to an equivalent oral dose of thyroxine (oral dose = intravenous dose  $\div$  0.75) once the patient can tolerate oral medication. Although some current studies recommend intravenous rather than oral hormone replacement therapy, others have shown no difference in outcome between these administration routes, though recovery may be delayed following oral administration because of the effect of intestinal absorption.<sup>9</sup> Thyroid hormone therapy has also been associated with an increased

risk of myocardial infarction and atrial arrhythmias.<sup>9</sup>

Stress doses of glucocorticoids (e.g., intravenous hydrocortisone 100 mg every 8 hours) are recommended in patients with coexisting adrenal insufficiency. Other supportive care, including intensive care unit arrangements, intubation, electrolyte correction, inotropic agents, judicious fluid supplementation, rewarming, and empirical antibiotic use, is also recommended as required.

Myxedema coma is associated with a high mortality rate of approximately 20% to 60%. Older age, cardiac complications, poor consciousness, persistent hypothermia, sepsis, and delayed treatment have been identified as possible risk factors for a poor prognosis in patients with myxedema coma.<sup>10,11</sup> These findings confirm that myxedema coma represents a true endocrine emergency requiring early and appropriate treatment.

## Conclusions

In this study, we report on a female patient with myxedema coma who presented to the ED with hypothermia, dyspnea, and general edema. Although myxedema coma is associated with high mortality, this patient's condition improved gradually after receiving thyroxine, glucocorticoid supplement, and intensive supportive care, and she was subsequently discharged. Doctors should be aware of the possibility of myxedema coma, especially in elderly females or patients with previous thyroidectomy who present at the ED with 'cold', 'slow', and 'weak' symptom presentations. Treatment should be administered without delay in patients with suspected myxedema coma.

## Declaration of conflicting interest

The authors declare that there is no conflict of interest.

## Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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