CASE REPORT
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A Case of Myxedema Coma Presenting as a Brain Stem Infarct in a 74-Year-Old Korean Woman

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Myxedema coma is the extreme form of untreated hypothyroidism. In reality, few patients present comatose with severe myxedema. We describe a patient with myxedema coma which was initially misdiagnosed as a brain stem infarct. She presented to the hospital with alteration of the mental status, generalized edema, hypothermia, hypoventilation, and hypotension. Initially her brain stem reflexes were absent. After respiratory and circulatory support, her neurologic status was not improved soon. The diagnosis of myxedema coma was often missed or delayed due to various clinical findings and concomitant medical condition and precipitating factors. It is more difficult to diagnose when a patient has no medical history of hypothyroidism. A high index of clinical suspicion can make a timely diagnosis and initiate appropriate treatment. We report this case to alert clinicians considering diagnosis of myxedema coma in patients with severe decompensated metabolic state including mental change.

Key Words: Myxedema Coma; Hypothyroidism; Stroke

INTRODUCTION

Myxedema coma represents an extreme form of hypothyroidism and is associated with a high mortality rate (up to 60%) (1, 2). This rare clinical state presents as a life-threatening decompensated state that may include altered mental status, hypothermia, bradycardia, hypoventilation, and cardiovascular collapse (1-3). Coma results from either hypothyroidism or as a complication of hypothyroidism. In reality, few patients present as comatose with severe myxedema. Here we report a case of myxedema coma presenting with a severe decompensated metabolic state.

CASE REPORT

A 74-yr-old woman was transferred to our emergency department from the local general hospital with an initial diagnosis of brain stem infarct on January 17, 2009. The patient was found unconscious and in a bed-ridden state during the early morning on the presenting day. The patient's breathing was shallow and irregular. The patient was unable to walk well due to a coccyx fracture two years ago, but she was able to perform daily life activities with minor help. One week ago, her facial edema started and she complained dry cough and general weakness three days ago. No other medical, familial, or social history was reported, and the patient had not taken any medication. There was also no evidence of intoxication.

According to the records of the local general hospital, the patient had an impending respiratory arrest and comatose mental status. Her blood pressure was 71/48 mmHg, her body temperature was 36.0°C, and her pulse rate was 99 beats/min with a sinus rhythm by electrocardiography (ECG). Neurologic examination revealed pinpoint pupils and a lack of brain stem reflexes, including the light reflex, corneal reflex, gag reflex, and doll's-eye phenomenon. She also showed decerebrated rigidity in response to painful external stimuli. The patient was assigned a score of 5 on the Glasgow Coma Scale, and the Babinski sign was detected at both feet. Brain computed tomography (CT) produced unremarkable results. Endotracheal intubation was performed for protection of the patient's airway, assisted ventilation was initiated, and an inotropic agent was injected to support the patient's state of shock, which did not respond to fluid therapy (1 L of normal saline). Prior to transfer, the patient's blood pressure was increased, but her neurologic findings were not improved. She was transferred to our hospital for further evaluation and treatment of a brain stem infarct.

On arrival, the patient's blood pressure was 70/40 mmHg, her pulse rate was 59 beats/min, and an intravenous infusion of premixed dopamine was administered (500 mL/800 mg, 20 μg/kg/min). The patient's body temperature was 34.8°C according to a tympanic membrane thermometer, and her self-respiration was slow and shallow. The patient's lungs on auscultation were bilaterally clear, and cardiac examination showed a regular rate and rhythm without murmurs, gallops, or rubs. The patient's abdo-

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men was soft without signs of guarding, tenderness, or rebound tenderness. A rectal examination revealed no masses, and the patient's stool was guaiac negative. No soiling due to urine or feces was noted on her undergarments.

The patient's face and extremities were edematous, and she presented with non-pitting edema. Her skin was dry, cool, pale, and slightly mottled and desquamated; however, there was no evidence of petechiae or purpura (Fig. 1).

The patient's level of consciousness was stupor to semi-coma. A neurologic examination revealed intact cranial nerves and she didn't show lateralizing signs. The patient's pupils were 3 mm across and briskly reactive. It was impossible to check for visual abnormalities and focal cerebellar findings. The results of the Babinski reflex test were negative.

The patient's laboratory values were as follows: WBC count 6,000/μL (86% neutrophils), hemoglobin 11.8 g/dL, sodium 121 mEq/L, potassium 4.2 mEq/L, chloride 87 mEq/L, BUN 1.3 mg/dL, creatinine 0.6 mg/dL, glucose 104 mg/dL, ALT 113 U/L, AST 74 U/L, albumin 3.3 g/dL, CK 499 IU/L, CK-MB 21.4 ng/mL, troponin-I <0.02 ng/mL, lactate 1.2 mM/L, urine RBCs 1-4/HPF, and urine WBCs 0-1.5/HPF. Arterial blood gas analysis (FiO_2 1.0) revealed a pH of 7.486, a pCO_2 of 30.6 mmHg, a pO_2 of 36.4 mmHg, a $HCO3^-$ concentration of 22.9 mM/L, and an oxygen saturation of 77%.

A portable chest radiography revealed infiltration of the right

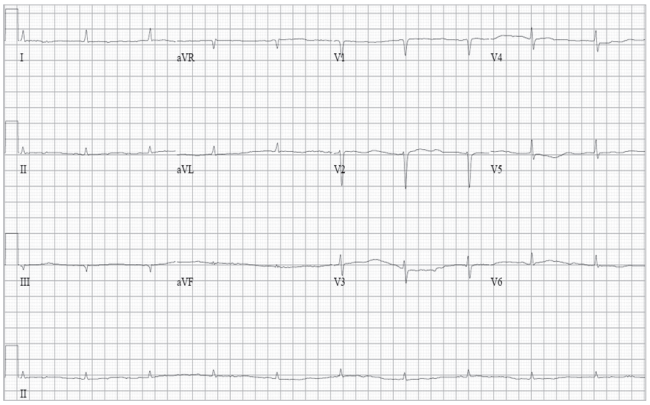

Fig. 2. Initial ECG showed sinus bradycardia (ventricular rate 59/min), low QRS voltage, and a prolonged QT interval (QTc >470 ms).

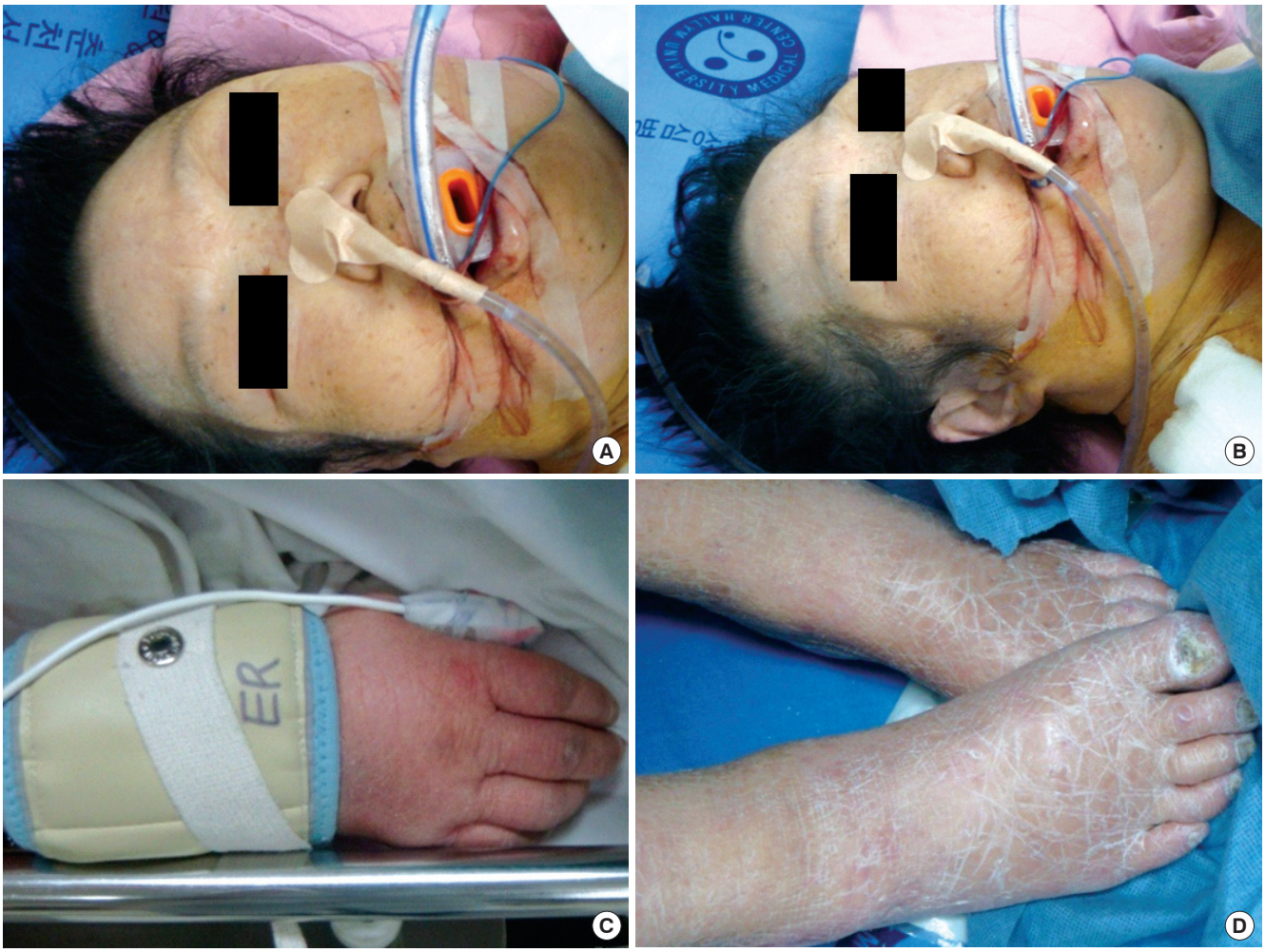

Fig. 1. The gross photos of patient's face and extremities. (**A**, **B**) Severe periorbital edema and thinned eyebrow. (**C**, **D**) Non-pitting edema and desquamation of the hands and feet.

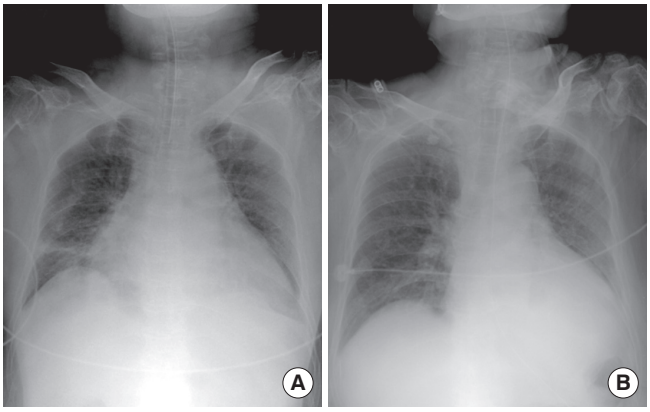

Fig. 3. Chest X-rays. (**A**) Initial chest X-ray revealed cardiomegaly and infiltration of the left lower lung zone. (**B**) Follow-up X-ray showed an improved state.

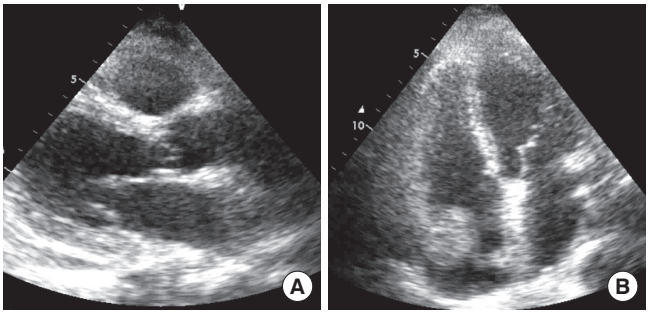

Fig. 4. Echocardiography. Parasternal long axis view (**A**) and apical four chamber view (**B**) revealed a moderate amount of pericardial effusion without hemodynamic significance; effusion was localized to the right atrium.

lower lung zone and cardiomegaly. ECG revealed sinus bradycardia with no evidence of ischemia; the intervals and axis were normal except for a prolonged QTc interval. Echocardiography revealed a small amount of pericardial effusion, but it did not affect the patient's hemodynamic status.

During our resuscitative efforts, the patient's blood pressure didn't respond and additional laboratory test results confirmed hypothyroidism. The patient's free thyroxine was 0.05 ng/dL (0.93-1.7) and her TSH was 30.12 μIU/mL (0.27-5.0). A diagnosis of myxedema coma exacerbated by community-acquired pneumonia was thus assigned.

The patient was referred to the Department of Endocrinology and admitted to the intensive care unit for treatment and ventilatory support. The patient received 1,000 μg of levothyroxine orally for two days, followed by 300 μg/day for an additional three days and finally maintained on levothyroxine 0.1 mg orally each day. Following the administration of synthetic thyroid hormones for hypothyroidism and antibiotics for pneumonia, the patient regained consciousness and respiration. She was transferred to a general ward after two weeks in the intensive care unit and discharged from the hospital one week later.

DISCUSSION

The patient's initial presenting conditions, which were misdiagnosed as a brain stem infarct at the local general hospital, may have been due to low blood perfusion to her brain. Comas are caused by a wide variety of disorders, ranging from structural central nervous system problems to diffuse systemic disease. Approximately 15% of coma cases are caused by structural lesions (1, 2). According to the records of the local general hospital, the patient's major brain stem reflexes were absent both initially and after their attempts at resuscitation; however, her neurologic status improved following our resuscitative efforts, so shock was our main concern. Septic shock due to pneumonia was the initial diagnosis; however, the patient was relatively unresponsive to our treatment efforts and her heart rate was slow despite the fact that her blood pressure was low. The patient's compensatory mechanisms were therefore not working and her clinical presentation was one of a severe decompensated metabolic state. She showed typical non-pitting edema, especially on the face and the result of routine laboratory test like albumin didn't explain the cause of her edema; therefore, we considered the diagnosis of myxedema and ordered thyroid and adrenal function tests.

Myxedema coma is a life-threatening form of hypothyroidism with physiological decompensation, including mental changes (1-4). Myxedema coma is 4-8 times more common in women, and such patients show an altered mental status, including lethargy, stupor, delirium, or coma. Secondary insults such as climate-induced hypothermia, infection, additional systemic conditions, or drug therapy can worsen the patient's condition. The patient presented at our hospital with diffuse edema especially face and extremities, alterations in mental status, hypothermia, hypoventilation, hypotension, and bradycardia. The clinical presentation was one of a severe decompensated metabolic state (1). The habitus of a myxedema comatose patient is characteristic and usually recognizable; however, hypothermia and a depressed mental state are often seen in other conditions, which may be mistaken for hypothyroidism. Sepsis and accidental hypothermia, for example, may mimic hypothyroidism (2). In addition to physiologic decompensation, the patient showed generalized nonpitting edema. Facial edema started one week from the presenting date and progressed to all extremities. Her edema was most prominent at the periorbital area and her conjunctivae were also swollen. The patient's skin was smooth, dry, desquamated, and cool. Congestive heart failure or nephrotic syndrome with renal failure may initially be confused with myxedema coma due to generalized edema. However, generalized nonpitting edema, particularly with a periorbital distribution, is typical. Edema is secondary to hyaluronic acid deposition and is not characteristically seen initially in dependent areas (2). Similar to the patient described, laboratory evaluations may reveal anemia, hyponatremia, hypoglycemia, arterial blood gas abnor-

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malities, and elevated levels of transaminases, creatine phosphokinase, and lactate dehydrogenase. ECG may demonstrate sinus bradycardia, prolongation of the QT interval, and low voltage. Chest radiography may demonstrate an increased cardiac contour due to pericardial effusion (1, 5).

Poor prognostic factors include persistent hypothermia that is unresponsive to 72 hr of therapy, advanced age, bradycardia (<44 beats per min), sepsis, myocardial infarction, and hypotension. Predictive values of survival are the patient's level of consciousness at admission and his/her score on the Glasgow Coma Scale and on the Acute Physiology and Chronic Health Evaluation (APACHE) II (6). The three principles of management are rapid institution of thyroid hormone replacement, treatment of the precipitating cause, and the provision of ventilatory support (1, 5).

Missing a diagnosis of myxedema coma is a major cause of increased mortality. Despite readily available, sensitive thyrotropin assays, however, the recognition and treatment of myxedema coma remains a challenge.

Hypothyroidism in adults most frequently causes dementia and depression. Other less common clinical symptoms include myxedema coma and dysfunction of the cerebellum and cranial nerves. Hypothyroidism also increases one's risk of stroke. Peripheral diseases frequently include polyneuropathy, carpal tunnel syndrome, a myalgic state, and, rarely, myokymia. In particular, little is known regarding the underlying pathology of cognitive impairment and depression in hypothyroidism, which may differ from that seen in euthyroid patients. About 40% of hypothyroid patients have signs of sensorimotor axonal neuropathy early in the course of the disease. Little is known about the reversibility of changes in cerebral blood flow following thyroid replacement therapy (7, 8).

As considered patient's age and cardiovascular risks, the patient was dosed 1,000 μg of levothyroxine orally for two days, followed by 300 μg/day for an additional three days and finally maintained on levothyroxine 0.1 mg orally each day. Like this patient, prompt thyroid hormone replacement is critical for patient survival from myxedema coma, although the most effective regimen is unclear. The most widely published approach to myxedema coma involves the intravenous administration of T_4 or T_3 in a dose of 300 to 500 μg that depends on patient weight and cardiac risks. It is suggested that this dose replaces total body stores of T_4, and the pool can be maintained by 50 to 100 μg/day (2). There were no available IV drugs in our hospital, so we administered higher dose (double dose) of oral regimen. For elderly and patients with cardiac comorbidity, the use of T_4 alone should be considered. For critically ill younger patients without cardiac disease, where a more rapid correction of hormone levels is desired, the use of T_3 alone should be considered (2). A recent case showed that a young patient who was treated successfully with a large oral dose (triple dose) of levothyroxine (9).

When assessing a patient, even if there are physical findings of stroke or other neurologic diseases, endocrinal problems must be considered. These include not only diabetes, adrenal disorders, and pituitary disorders, but also thyroid disorders. Thyroid disorders must be ruled out, even though they are very rare. If such conditions are not considered, the survival rate will be low.

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