Myxedema coma with cardiac tamponade and severe cardiomyopathy

Abdulla Majid-Moosa, DO, Jeffrey M. Schussler, MD, and Adan Mora, MD

Myxedema coma is an infrequent but potentially fatal complication of hypothyroidism. We present a rare case of previously undiagnosed hypothyroidism presenting in cardiogenic shock from pericardial tamponade and depressed myocardial contractility in myxedema coma. Here, we focus on cardiovascular complications associated with the condition.

CASE PRESENTATION

A 57-year-old white woman without chronic medical problems presented with complaints of midepigastria pain for a few days. She denied any nausea, vomiting, or other gastrointestinal symptoms. She was not on any medications and had no allergies. In the emergency department, her body temperature was 94°F. Her earliest recorded blood pressure was 90/70 mm Hg on 5 mcg/kg/min of dobutamine and 0.04 mcg/kg/h of norepinephrine. She required 80% oxygen on biphasic noninvasive positive pressure ventilation of 16/8 cm H₂O to maintain an oxygen saturation of 95%. She was pale and somnolent but able to be aroused despite slow mentation. Her mucous membranes were dry, her heart sounds were distant, her lungs were clear, her abdomen was obese, and her lower extremity pulses were depressed. An electrocardiogram revealed attenuated QRS complexes (*Figure 1a*).

Her serum creatinine was 2.1 mg/dL; bicarbonate, 20 mEq/L; aspartate aminotransferase, 1087 U/L; alanine aminotransferase, 799 U/L; alkaline phosphatase, 287 U/L; white blood cell count, 9000 10³/L; hemoglobin, 10.9 g/dL; and hematocrit, 32%. Her initial cardiac troponin I level was 24.1 ng/mL; thyroid-stimulating hormone, 68.2 uIU/mL; and free thyroxine, 0.3 ng/dL. The lactic acid level was 6.2 mmol/L. An arterial blood gas had a pH of 7.19, partial pressure of carbon dioxide was 42.9 mm Hg, and partial pressure of oxygen was 65 mm Hg on the noninvasive positive pressure ventilation.

Imaging revealed cardiomegaly (*Figure 2a*), a large pericardial effusion and pneumatosis intestinalis of the ascending colon (*Figure 2b*), and global cardiomyopathy with an ejection fraction of 25% to 30% (*Figures 2c, 2d*). Compression of the right atrium and ventricle by a large circumferential pericardial effusion was noted, with collapse in <50% of the cardiac cycle. The patient was taken to the operating room for drainage of the pericardial effusion.

A workup of the fluid, including infectious studies, was negative. In addition, in a panel of autoimmune antibodies—

including antinuclear antibodies, antineutrophil cytoplasm antibodies, rheumatoid factors, hepatitis antibodies, and extractable nuclear antigen antibodies—all tests were negative with the exception of an isolated positive U1 ribonucleoprotein antibody.

Our patient's hospital stay spanned almost 3 months, most of which was in the intensive care unit, and she suffered several complications of myxedema coma. After draining the pericardial fluid, a drain remained in place for almost 5 days with minimal additional fluid formation once thyroid replacement was initiated. Her cardiac function improved and she remained hemodynamically independent after replacement of thyroid hormone. She suffered from respiratory failure that required a tracheostomy with a slow prolonged weaning course off the ventilator. She also developed embolic ischemic strokes, required a percutaneous endoscopic gastrostomy (PEG) tube, and was treated with antibiotics and antifungals through her recovery. She was eventually decannulated, her PEG tube was removed, and plans were made for her to be transferred to a long-term care facility.

DISCUSSION

Thyroid hormone plays a ubiquitous role in the body's metabolic processes, and hypothyroidism is the clinical manifestation of a deficiency in thyroid hormone. This process affects all organ systems. The extent of clinical impact depends on the degree of hormone deficiency. Myxedema coma is a rare life-threatening complication of hypothyroidism. It either occurs as a natural progression of the disease or is precipitated by an infection or other events such as drug overdose (in particular, sedative drugs like opioids), myocardial infarction, or cold exposure in the setting of previously existing chronic hypothyroidism (1). It most commonly affects elderly women. Hypothyroidism is present in about 6% of the population to varying degrees, but myxedema coma is seen in only 0.1% of this subgroup of patients (1). Early and widespread use of thyroid-stimulating hormone

From the Department of Internal Medicine (Majid-Moosa), the Division of Cardiology (Schussler), and the Division of Pulmonary and Critical Care Medicine (Mora), Baylor University Medical Center at Dallas; the Jack and Jane Hamilton Heart and Vascular Hospital (Schussler); and the Texas A&M College of Medicine (Schussler).

Corresponding author: Adan Mora, MD, Division of Pulmonary and Critical Care Medicine, Baylor University Medical Center at Dallas, 3500 Gaston Avenue, Dallas, TX 75246 (e-mail: Adan.Mora@BaylorHealth.edu).

Figure 1. (a) An admission electrocardiogram revealed attenuated QRS complexes, which (b) improved by the time of discharge.

assays has led to a progressive decline in the prevalence of this condition (2). It still retains a high mortality rate of almost 40% despite aggressive treatment (1).

Key to diagnosis is understanding that myxedema coma is diagnosed clinically based on presentation and is supported by other laboratory values. As expected, the clinical manifestations are not restricted to one organ system. The cardiovascular complications of this condition are varied and well known. Sinus bradycardia, low voltage complexes, bundle branch blocks, complete heart blocks, and nonspecific ST-T changes in the electrocardiogram have been recorded in myxedema crisis (1). Hypothyroid patients have dia-

stolic hypertension even in the setting of decreased cardiac output. This is thought to be due to the development of an imbalance in the alpha and beta adrenergic receptors with long-term hypothyroidism (3). The number of beta-adrenergic receptors is reduced, usually with preservation of alpha-adrenergic receptors and circulating catecholamines, causing beta/alpha-adrenergic imbalance, diastolic hypertension, reduced total blood volume, and other manifestations (3). In severe hypothyroidism, bradycardia and decreased myocardial contractility can alone result in hypotension (1).

A pericardial effusion is a notable finding that is seen uncommonly in hypothyroidism but more frequently in patients with

Figure 2. (a) Presenting posteroanterior portable chest radiograph demonstrating an enlarged cardiac silhouette. (b) Anteroposterior view of computed tomography scan revealing a large circumferential pericardial effusion. (c) Subcostal view of bedside transthoracic echocardiogram revealing compression of the right atrium and right ventricle by a large pericardial effusion consistent with tamponade physiology. (d) Four-chamber view of bedside transthoracic echocardiogram demonstrating complete collapse of the right atrium and partial collapse of the right ventricle.

more severe thyroid hormone deficiency, such as in myxedema (4). Rarely, pericardial effusion progresses to cardiac tamponade and impairs ventricular function. Case reports over the years have described myxedema coma—associated pericardial effusions causing cardiac tamponade (5, 6).

The backbone of treatment for myxedema coma is thyroid replacement. Aggressive supportive therapy and management of comorbidities is also critical for survival. Thyroid hormone is usually replaced intravenously as it is hypothesized that gut edema from a myxedematous state may not allow adequate and prompt absorption (7). Reports have suggested that replacement of thyroid hormone by nasogastric tube may also be sufficient (1). Parenteral administration of thyroid hormones presents the risk of arrhythmia from rapidly increasing serum concentrations and is an accepted risk of necessary therapy. Debate still exists about the type of thyroid replacement and dosage. A combination of T3 and T4 thyroxine is usually preferred, as deiodinase conversion of T4 to T3 is thought to be compromised in these settings (2). Daily doses of T4 in the range of 1.6 mcg/kg are recommended after a loading dose of 200 mcg. T3 is usually administered in doses of 5 to 10 mg loading, followed by doses of 2.5 to 10 mcg every 8 hours given the short half-life of T3. Administering T3 does not remain without risk, as its more

immediate action is even more likely to cause arrhythmia, particularly in patients with previous heart disease (8). As seen with this patient, the complications of myxedema are typically reversible with hormone replacement. Supplemental steroids are administered unless proven to not be adrenal insufficient. Supportive therapy for comorbid infections and adequate fluid resuscitation are critical.

1. Mathew V, Misgar RA, Ghosh S, Mukhopadhyay P, Roychowdhury P, Pandit K, Mukhopadhyay S, Chowdhury S. Myxedema coma: a new look into an old crisis. *J Thyroid Res* 2011;2011:493462.
2. Kwaku MP, Burman KD. Myxedema coma. *J Intensive Care Med* 2007;22(4):224–231.
3. Nicoloff JT, LoPresti JS. Myxedema coma. A form of decompensated hypothyroidism. *Endocrinol Metab Clin North Am* 1993;22(2):279–290.
4. Kabadi UM, Kumar SP. Pericardial effusion in primary hypothyroidism. *Am Heart J* 1990;120(6 Pt 1):1393–1395.
5. Patil VC, Patil HV, Agrawal V, Patil S. Cardiac tamponade in a patient with primary hypothyroidism. *Indian J Endocrinol Metab* 2011;15(Suppl 2):S144–S146.
6. Martin L, Spathis GS. Case of myxoedema with a huge pericardial effusion and cardiac tamponade. *Br Med J* 1965;2(5453):83–85.
7. Rehman SU, Cope DW, Senseney AD, Brzezinski W. Thyroid disorders in elderly patients. *South Med J* 2005;98(5):543–549.
8. Hylander B, Rosenqvist U. Treatment of myxoedema coma—factors associated with fatal outcome. *Acta Endocrinol (Copenh)* 1985;108(1):65–71.