Respiratory Failure Induced by Myxedema — A Case Report

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Respiratory failure in myxedema is a complex medical emergency and may require prolonged ventilatory assistance. Chronic hypothyroidism is easily neglected by clinician due to the lack of specific symptoms and signs. It is also easily misdiagnosed as heart failure. We report a 55-year-old woman with chronic hypothyroidism. She had been treated for congestive heart failure for years. Myxedematous coma was not diagnosed until respiratory failure occurred. After replacement with levothyroxine, she was successfully weaned from prolonged mechanical ventilation. ***(Thorac Med 2007; 22: 182-186)***

Key words: myxedema hypothyroidism respiratory failure

Introduction

Hypothyroidism is a common medical problem encountered in the primary care setting. The most common causes of hypothyroidism include autoimmune thyroiditis, previous thyroid ablation, lithium or amiodarone use, and rarely a pituitary tumor or hypothalamic dysfunction [1] Severe hypothyroidism may result in cardiovascular compromise, respiratory failure, psychosis and obtundation. Despite adequate hormonal replacement, patients who present with the severe manifestations of hypothyroidism often die. Profound hypothyroidism causes impaired ventilatory response to hypoxia and hypercapnia [2], neuromuscular dysfunction [3], hypoventilation [4], obstructive sleep apnea [5], and pleural effusions [6]. Respiratory failure as a component of myxedema coma has a high mortality.

Case Report

A 55-year-old woman treated as congestive heart failure was irregularly followed up at a local hospital for 3 years. She had been in relatively stable condition and was ambulatory. Her medical history was otherwise noncontributory.

She suffered from slowly progressive bilateral lower leg weakness and difficulty in breathing for about 2 months. She fell to the ground occurred on August 24, 2005 without major complications, and then visited our neurosurgery and orthopedic OPD for help. A lumbar computed tomography (CT) scan on September 2, 2005 showed herniation of the intervertebral disc over L5/S1. However, persistent progressive shortness

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of breath, dyspnea on exertion, and chest discomfort were noted. On the morning of September 3, 2005, she suffered from severe dyspnea, severe chest discomfort and consciousness disturbance. She was sent to the emergency department of our hospital where her blood pressure was 133/91 mmHg, heart rate was 74/minute, respiratory rate was 20 and body temperature was 35.3°C. There was periorbital edema. Heart sounds were distant with a regular rate and rhythm. Bilateral basilar rales were audible,and nonpitting edema of the extremities was present. Deep tendon reflexes had marked a relaxation delay. The Glasgow coma scale of the eye was 1, verbal was 1,and motor was 1. Endotracheal tube insertion was perfomed with mechanical ventilation .

Laboratory data revealed normocytic anemia, and no leukocytosis. Liver function test, renal function test, C-reactive protein and electrolyte revealed no abnormal findings. The arterial blood gas (ABG) showed a pH of 7.26, a PCO_2 of 89.4 mmHg, a PO_2 of 75.9 mmHg, a saturation of oxygen of 92%. Chest radiograph (Figure 1) revealed cardiomegaly and low lung volumes with intubation. Electrocardiogram showed low voltage and sinus bradycardia. Echocardiogram demonstrated severe hypokinesia in the inferior and posterior wall, a left ventricle ejection fraction (LVEF) around 32%, and a small to moderate amount of pericardial effusion without tamponade. The thyroid function study revealed thyroid stimulating hormone (TSH) of 68 uIU/ml (reference range, 0.4~4.0), and free thyroxine (free T4) of 0.12 ng/dL (reference range, 0.8~1.7). The clinical and laboratory pictures were compatible with myxedema coma and respiratory failure. Oral levothyroxine was given. The titer of antithyroid peroxidase (anti-TPO) was 228.40 U/ml (reference range <60) and the antithyroiglobulin (anti-TG) was 208.30 U/mL (hospital day 3). Hashimoto's disease was diagnosed. On hospital day 5, the patient's consciousness became clear. Levels of free thyroxine checked on hospital day 3 and day 12 were still low. The levothyroxine dose was adjusted twice. Free thyroxine checked on day 31 was in euthyroidism (Table 1). Tracheo-

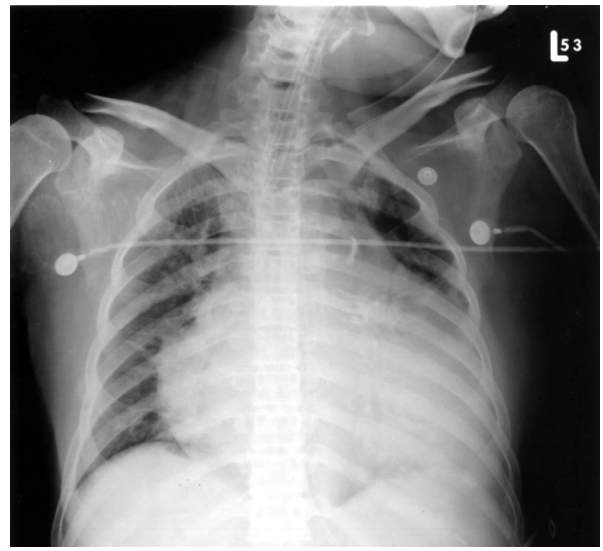

Fig. 1. Chest radiograph taken in emergency room revealing cardiomegaly, bilateral pleural effusion and low lung volumes with intubation.

Table 1. Thyroid Function Laboratory Data

Hospital day	Day 1	Day 3	Day 12	Day 31
T3 ng/dL (reference 60~181)		40		
T4 ug/dL (reference 4.5~10.9)		1		
Free T4 ng/dL (reference 0.8~1.7)	0.12	0.16	1.07	2.17
TSH uIU/ml (reference 0.4~4.0)	68	30.6	13.2	0.77

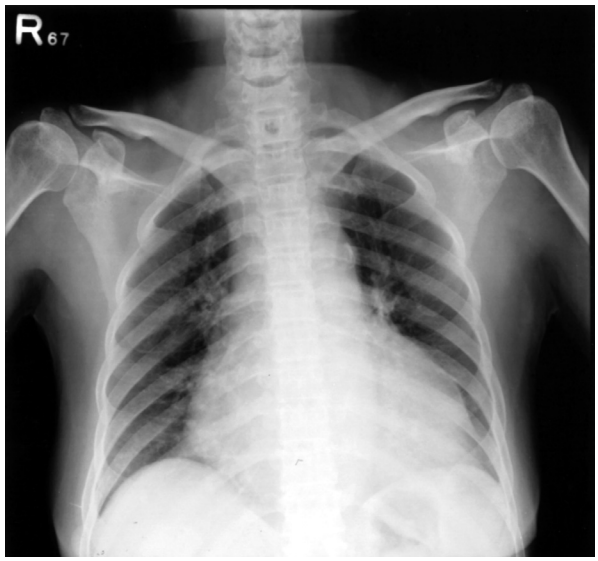

Fig. 2. Chest X-ray on day 44 showing mild cardiomegaly, but no pleural effusion.

stomy was suggested to the patient due to poor expectoration and excess sputum. Prolong intubation and ventilator dependence were also found after the weaning profile was not passed. A tracheostomy was done on day 27. Repeated echocardiogram demonstrated moderate hypokinesia in the inferior and posterior wall, LVEF around 57%, and no evidence of pericardial effusion. A T-piece was tried on hospital day 32, and a tracheal button was inserted on day 34. She was then transferred to the general ward. Chest X-ray (Figure 2) on day 44 showed mild cardiomegaly, but no pleural effusion. She was discharged on hospital day 47.

Discussion

This case clearly demonstrated that severe chronic hypothyroidism was easily neglected and treated as congestive heart failure for years and highlights the need for physicians to understand the respiratory consequences of hypothyroidism: impaired ventilatory response to hypercapnia and hypoxemia; subsequent respiratory alkalosis despite low mechanical minute ventilation and a prolonged ventilatory course from severe neuromuscular compromise.

Hypothyroidism alters ventilation in 2 ways. One feature is diminished central response to hypoxia and hypercapnia, resulting in respiratory acidosis. Nordqvist *et al.* first reported CO_2 narcosis in myxedema in 1960 [2]. The second feature of hypothyroidism is a propensity for respiratory alkalosis. Iatrogenic alkalosis is common after the initiation of overaggressive mechanical ventilation. Immediately after induction and intubation, the end-tidal CO_2 was between 18 and 20 mmHg. These investigators attributed these findings to a reduced basal metabolic rate and diminished CO_2 production brought about by depressed levels of thyroid hormone [7]. Decreased levels of PCO_2 may trigger an adjustment in the central respiratory drive [3]. No investigations on humans have determined the duration of hormonal replacement needed to normalize metabolic rates and consequently, CO_2 production and minute ventilation. Our patient had a similar clinical presentation. At presentation, her acid-base disorder was consistent with chronic respiratory acidosis. After mechanical ventilation use, she developed respiratory alkalosis. This was caused by iatrogenic hyperventilation and by a very low metabolic rate with diminished CO_2 production. We were unable to correct respiratory alkalosis even by using low minute ventilation in this patient. Added anatomic dead space was used with some improvement.

In addition to the altering ventilation, hypothyroidism also affects the neuromuscular system by causing weakness of the diaphragm and other respiratory muscles. Diaphragmatic dysfunction causes a restrictive respiratory pattern that may contribute to hypoxia and hypercapnia [8]. Defici-

ency of thyroid hormone causes demyelination and fibrosis of the phrenic nerve. Hormone replacement improves phrenic nerve conduction and normalizes transdiaphragmatic pressure within 3 months [9].

Several other factors may contribute to difficult weaning and should be investgated in severely hypothyroid patients. Patients may have a combination of hypothyroid-induced diaphragmatic and peripheral neuropathy, myopathy, or concomitant critical illness polyneuropathy [10]. Other complicating factors include congestive heart failure [11], anemia [12], and electrolyte imbalance. Hypothyroidism may exacerbate angina or congestive heart failure. It may also induce a normochronic nomocytic anemia that reduces oxygen-caring capacity [13]. As with all patients requiring prolonged mechanical ventilation, electrolyte should be monitored and corrected if abnormality is present. Balanced nutrition is also necessary. Physical therapy is necessary to prevent further muscular atrophy.

In conclusion, this case suggests that in a patient with symptoms and signs of heart failure, thyroid function needs to be checked. Myxedematous patients with respiratory failure need hormone replacement as soon as possible. Tracheostomy should be considered due to prolonged mechanical ventilator assistance. Full recovery may take several months.

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黏液水腫導致呼吸衰竭—病例報告

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呼吸衰竭發生在黏液水腫的病人身上是極具複雜性的急症而且治療需要長期的呼吸器協助。慢性甲狀腺功能低下由於沒有明顯的症狀，常常被臨床醫師忽略，並以心臟衰竭的藥物來治療。我們報告一個 55 歲女性病人因為長期慢性甲狀腺功能低下，卻以心臟衰竭藥物來治療，直到呼吸衰竭，意識不清，插管治療並使用呼吸器後才診斷出來。經過荷爾蒙的補充及長期時間呼吸器輔助,我們成功的將她脫離呼吸器。*(胸腔醫學* ***2007; 22: 182-186)***

關鍵詞：黏液水腫，甲狀腺功能低下，呼吸衰竭

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