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A Rare Cause Of Respıratory Faılure In the Elderly: Myxedema Coma

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Cover Page Footnote
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A RareCause Of Respıratory Faılure In the Elderly: Myxedema Coma

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ABSTRACT

Some of the findings of hypothyroidism are very similar t othose of normal aging. Myxedema coma, the most severe presentation of hypothyroidism, has a mortality rate of 50-60% in the elderly. Although respirator yfailure is rare in myxedemacoma, it is an emergency due to the high mortality, especially in the elderly. This case report presents a geriatric patient who developed type 2 respiratory failure following myxedema coma associated with Hashimoto's thyroiditis and emphasizes the importance of early recognition and treatment of myxedema coma.

Keywords: hypothyroidism, elderly, respiratory failure

INTRODUCTION

Hypothyroidism is a systemic disease that affects the central and peripheral nervous systems and the musculoskeletalsystem. Neuromuscular effect sinclude findings of carpaltunnel syndrome, sensory and sensorimotorpolyneuropathyandmyopathy(1). Myopathic involvement usually presents as pronounced proxima lmuscle weakness, myalgia, cramps and elevated creatinine kinase (2, 3). Myxedemacoma is a life-threatening form of decompensated hypothyroidism; its mortality rate is between 25-60%, and it is considered an emergency (4-6). Respiratory failure is rare in myxedemacoma; however, due to it sespecially high mortality in the elderly, it is an emergency (7). In this case report, we present a geriatric patient who developed type 2 respiratory failure following myxedema coma associated with Hashimoto's thyroiditis; we aimed to emphasize the importance of early recognition and treatment in myxedema coma.

CASE REPORT

A 90-year-old male patient was admitted to our clinic with complaints of swelling in the legs, feet and around the eyes, dry skin, coarsening of the voice, cold intolerance, and constipation. The patient had been fully independent in his basic and instrumental daily activities until three months earlier when his complaints had first begun as weakness, fatigue, and widespread muscle pain. In the previous week, repeated falls had been added to the patients' complaints. He had a history of atrial fibrillation and hypertension and had been taking 400 mg dronedarone for the previous four years. His vital signs weres table on physical examination. His skin was dry and pale, his voice was hoarse, and his mentaland motor

responses were slow. He had nonpitting edema of the feet and periorbital area, rales in the bases of both lungs, and skin abrasions secondary to falls. On neurological examination, the proximal muscle strength in his lower extremities was 4/5. There were no known neuromuscular diseases in his or his family's history. His laboratory results were as follows (with normal reference ranges when applicable): creatinine kinase (CK) = 973 IU/L (41-171); myoglobin = 486.3 ng/mL (28-72); lactate dehydrogenase (LDH) = 508 U/L (135-225); aspartate transaminase (AST) = 39 U/L (<35); creatinine = 1.6 mg/dL (0.7-1.3); hemoglobin = 9.8 g/dL (12.6-17.4); leukocytes = 7,790/µL; glucose = 90 mg/dL (60-110); C-reactive protein (CRP) = 1.56 mg/dl (0-0.5); thyroid-stimulating hormone (TSH) = 122 µIU/mL (0.35-5.5); free T3 = 0.76 pg/mL (2.3-4.2); and free T4 = 0.2 ng/mL (0.74-1.52). Antithyroid peroxidase [(antiTPO) = 158 IU/mL (0-35)] and antithyroglobulin [(antiTG) = 1,845 IU/mL (0-40)] antibodies were positive. The patient's alanine transaminase, total bilirubin, direct bilirubin, albumin, sedimentation, alkaline phosphatase, and total protein values were normal. At 8:00 am his basal cortisol was 19.52 µg/dL (6.7-22.6) and adrenocorticotropic hormone (ACTH) was 21.8 pmol/L (10-46). The patient exhibited sinus rhythm on electrocardiogram, and minimal pericardial effusion was detected on echocardiogram. Treatment was started with 25 µg oral levothyroxine once daily and oral hydrocortisone twice daily at 10 a.m. and 5 p.m. However, the patient developed mental confusion while in our clinic; arterial blood gas analysis results were pH = 7.39 (7.35-7.45); pCO_2 = 67.5 mmHg (35-45); pO_2 = 97.8 mmHg (83-108); HCO_3 = 36.8 mmol/L (22-26); and oxygen saturation = 97.6% (95-98). The patient did not exhibit rhonchi on physical examination; type 2 respiratory failure was the initial diagnosis; the patient was transferred to the respiratory intensive care unit for non-invasive mechanical ventilator support. While in intensive care, 500 µg/day intramuscular levothyroxine was started and hydrocortisone was switched to 20 mg intravenous prednisolone. Respiratory myopathy secondary to hypothyroidism was believed to be the cause of the patient's respiratory failure. Monitoring of the patient's blood gas parameters is shown in Table 1.

Table 1. Progression of the patient's blood gas values during levothyroxine treatment

		pH	**CO_2 (mmHg)**	**PO_2 (mmHg)**	**HCO_3 (mmol/L)**	**O_2 saturation (%)**
Oral treatment	**Day 1**	**7.39**	**67.5**	**97.8**	**36.8**	**97.6**
	Day 4	**7.34**	**65.5**	**87.7**	**35.5**	**97.5**
Intramuscular treatment	**Day 5**	**7.38**	**65.3**	**53**	**37.8**	**89**
	Day 7	**7.44**	**46.4**	**65.9**	**31.3**	**92**
	Day 10	**7.49**	**39.1**	**103**	**29.3**	**93**

While in the intensive care unit, the patient developed Acinetobacter pneumonia; he was unresponsive to treatment and died.

DISCUSSION

The most common causes of hypothyroidism in the elderly are Hashimoto's disease or autoimmune thyroiditis due to atrophic thyroiditis. Other causes are Graves' disease treated with I^{131} or iatrogenic causes such as thyroidectomy (8). Myxedema coma, the most severe

presentation of hypothyroidism, has a 50-60% mortality rate in the elderly (4, 9).Triggering factors include infection, trauma, surgical procedures and medication (10). The main findings of myxedema coma are deterioration of mental status, hypothermia and cardiopulmonary decompensation (11). Some of the findings of hypothyroidism are very similar to those of normal aging, such as cold intolerance, dry skin, a tendency toward constipation and muscle weakness, which can make the diagnostic approach complex (12). Type 2 respiratory failure should be kept in mind when evaluating the thyroid function tests of a patient who appears hypothermic and myxedematous. Hypothyroidism causes respiratory failure through slowing of the central respiratory center, weakening of the respiratory muscles, alveolar hypoventilation and low diffusion capacity, narrowing of the upper respiratory tracts, sleep apnea and pericardial/pleural effusion; hypoxia and hypercapnia occur as a result. With thyroid hormone replacement therapy, respiratory failure gradually resolves (5, 13, 14). The early recognition of hypothyroidism and myxedema coma in elderly patients is critical for its prognosis (15). Respiratory failure generally occurs in the elderly due to congestive heart failure or respiratory diseases (16). In an elderly patient, acute respiratory failure resulting from severe hypothyroidism may be evaluated as a symptom of heart or lung disease and therefore misdiagnosed. Delayed or wrong diagnosis of profound hypothyroidism Findings of hypercapnia, hypoxia, changes in mental status, obstructive sleep apnea syndrome (OSAS) and pericardial/pleural effusion due to are similar to findings in chronic obstructive pulmonary disease (COPD) exacerbation, cardiogenic pulmonary edema, pneumonia, and obesity. In hypothyroidism, respiratory failure is caused by insufficient central ventilation center response to hypercapnia and hypoxia, hypoventilation associated with neuromuscular dysfunction, and OSAS (7, 17). Furthermore, hypothyroidism can cause symptoms ranging from exercise intolerance to diaphragm dysfunction severe enough to mimic diaphragm paralysis, and this dysfunction returns to normal following thyroid hormone replacement therapy (14). Treatment of myxedema coma usually begins with four doses of 100-500 µg intravenous levothyroxine, followed by 50-100 µg/day given intravenously or orally. Respiratory support with non-invasive ventilation may be required in the treatment of elderly patients with profound hypothyroidism(9, 18). Thyroid hormone replacement therapy may increase cortisol clearance, thus aggravating cortisol deficiency; therefore, intravenous administration of 50 mg hydrocortisone at 6-hour intervals is recommended (19). In elderly patients who develop hypoventilatory respiratory failure that cannot be explained by a cardiac, pulmonary or systemic pathology, as in our case, hypothyroidism should be considered in the differential diagnosis, even if the patient has no previous history of thyroid dysfunction (16).

CONCLUSION

Our case demonstrates that hypothyroidism should be considered in the differential diagnosis of respiratory failure in elderly patients when it cannot be explained by pulmonary, cardiac and central nervous system pathologies.

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