CORRESPONDENCE

Impending myxedema coma as the initial presentation of lung cancer

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Myxedema coma is an endocrine crisis,¹ and metastatic malignancy induced thyroid dysfunction is uncommon but receiving increased attention. Progressive destruction of the thyroid gland caused by metastatic malignancy can contribute to hypothyroidism and potentially eventual myxedema coma.² Here, we report a patient with impending myxedema coma diagnosed as lung cancer with thyroid-gland metastasis in conclusion.

A 72-year-old man presented to our Emergency Department with altered consciousness, progressive dyspnea, and general edema over 2 weeks. Chest radiograph revealed bilateral pleural effusion and pericardial effusion, which were lymphocyte-predominant exudate. Laboratory investigations showed that sodium levels had dropped to 110 mmol/L (normal range: 135–147 mmol/L). Serum free-T4 levels were also below the detectable level (<0.4 ng/dL) and thyroid-stimulating hormone levels had increased to 71.0 uIU/mL (normal range: 0.400–4.000 uIU/mL). Both anti-thyroglobulin antibody and anti-thyroid peroxidase antibody were negative. On thyroid sonography, 3.6- and 3.5-cm diameter heterogeneous masses with diffuse microcalcification were observed in the right and left lobes of the thyroid gland, respectively. Fine-needle aspiration revealed malignant cells. Following thyroxine and glucocorticoid replacement, the patient underwent total thyroidectomy. Pathology demonstrated that the normal thyroid gland had been almost completely replaced by metastatic carcinoma cells that were positive for TTF-1, CK7, and napsin A, but negative for thyroglobulin and PAX-8 (Figure 1). A metastatic tumor of pulmonary origin was favored. Chest computed tomography confirmed a 5.5-cm diameter mass in the right middle lobe of the lung, with multiple enlarged mediastinal lymph nodes. The final diagnosis was stage IV lung adenocarcinoma with thyroid-gland metastasis, causing secondary hypothyroidism and impending myxedema coma. Erlotinib was prescribed due to the presence of the EGFR L858R mutation in the biopsied tumor cells. The patient responded well, and the level of consciousness returned to normal status within 2 months. However, the disease progressed with pleural seeding, pericardial effusion, and massive pleural effusion after 10 months of treatment.

Myxedema coma constitutes an emergency situation, with high mortality rates ranging from ~25% to 60%.

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cases are related to thyroidectomy or with a previous history of radioiodine therapy. There are increasing case reports of hypothyroidism caused by treatment with a tyrosine kinase inhibitor, most notably sunitinib. The clinical presentation is systemic failure, including respiratory failure and altered mental status. The treatment consists of supportive care and emergent thyroid-hormone replacement.

Tumor metastasis as the cause of hypothyroidism is rare, with autopsy studies reporting the incidence of thyroid metastasis in patients with known malignancy ranging from 1.9% to 24%. The most common origins are kidney, breast, gastrointestinal tract, and lung. Pathology with an ancillary panel of immune-histochemical staining is needed to make a final diagnosis. In our case, the presence of a classical EGFR mutation supported the diagnosis. Surgical resection provides evidence for both diagnosis and treatment; however, the benefits and disadvantages remain controversial.

There is increased awareness of thyroid dysfunction caused by metastatic cancer-cell infiltration. In most cases, thyroid function remains normal; however, both thyrotoxicosis and hypothyroidism have been reported.

To clinical physicians, it is important to note that thyroid metastasis is among the differential diagnoses of hypothyroidism, especially in patients with thyroid nodules.

References