Acute upper airway obstruction by a goitre due to Hashimoto’s thyroiditis

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DESCRIPTION
An 85-year-old woman who lived in Kanuma, Tochigi prefecture, Japan, presented to the emergency department of a hospital with dyspnoea, which developed 3 hours before the visit. At the time of presentation, she was in respiratory distress, with a blood pressure of 171/88 mm Hg, heart rate of 105 beats per minute, respiratory rate of 40 breaths per minute, body temperature was 35.6°C and oxygen saturation was 82% on ambient air. Her oxygen saturation improved to 100% on oxygen at 10 L/min; however, the patient remained tachypnoic and became cyanotic. Therefore, the patient was transferred to the intensive care unit of our hospital for intensive care. The patient had hypertension but did not have any history of autoimmune or thyroid diseases. The patient did not take any medication and had no allergy. There was no family history of thyroid disease. The patient never smoked and did not take alcohol and there was no history of excessive iodine intake or radiation exposure during the Fukushima Daiichi Nuclear Power Plant accident.

At the time of the transfer, the patient was alert and oriented to time, place and person. Blood pressure was 130/82 mm Hg, heart rate was 81 beats per minute with an irregular rhythm, respiratory rate was 32 breaths per minute, body temperature was 35.3°C and oxygen saturation was 94% on oxygen at 10 L/min. There was no jugular venous distention. A diffuse goitre was noted. Marked wheezes were heard over the entire chest. There was no sign of deterioration of hypothyroidism such as coma, bradycardia, hypotension or hypothermia after the CT scan. A diffusely enlarged thyroid extended to the retrosternal, encircling most of the trachea and possibly compressing the trachea on the CT scan (figure 1A). There was no other abnormal finding. Therefore, upper airway obstruction by the diffusely enlarged thyroid was suspected as a cause of her respiratory failure. Additional laboratory tests revealed highly elevated antithyroglobulin antibody (2040.0 IU/mL) and antithyroperoxidase antibody (322.0 IU/mL) levels. The ultrasound of the thyroid showed no findings suggesting malignancy. The arterial blood gas 30 min after intubation revealed respiratory acidosis (pH 7.287, PaO2 155 mm Hg, PaCO2 65.7 mm Hg and HCO3 28.4 mmol/L). Chest X-ray revealed no abnormalities. An ECG showed atrial fibrillation without specific ST-T segment changes. An echocardiogram showed no abnormal finding in cardiac function.

At the time, severe asthma was suspected. The patient was treated with supplementary oxygen, bronchodilator nebulizer and systemic steroid infusion. However, respiratory condition of the patient progressively deteriorated within the next hours and finally, the patient was intubated. Although the patient had a goitre and hypothyroidism, a contrast-enhanced chest and abdominal CT scan was performed since massive pulmonary thromboembolism was considered. There were no signs of deterioration of hypothyroidism such as coma, bradycardia, hypotension or hypothermia after the CT scan. A diffusely enlarged thyroid extended to the retrosternal, encircling most of the trachea and possibly compressing the trachea on the CT scan (figure 1A). There was no other abnormal finding. Therefore, upper airway obstruction by the diffusely enlarged thyroid was suspected as a cause of her respiratory failure. Additional laboratory tests revealed highly elevated antithyroglobulin antibody (2040.0 IU/mL) and antithyroperoxidase antibody (322.0 IU/mL) levels. The ultrasound of the thyroid showed no findings suggesting malignancy. The arterial blood gas 30 min after intubation revealed respiratory acidosis (pH 7.287, PaO2 155 mm Hg, PaCO2 65.7 mm Hg and HCO3 28.4 mmol/L). Chest X-ray revealed no abnormalities. An ECG showed atrial fibrillation without specific ST-T segment changes. An echocardiogram showed no abnormal finding in cardiac function.

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Goitre is recognised as a possible cause of acute airway obstruction through severe airway compression from masses regardless of malignant or benign as seen in our case.1–3 Patients with goitre are usually asymptomatic even when goitres compress their airway tract1 and acute respiratory failure secondary to tracheal compression by goitre is infrequent.2 Therefore, this condition has a risk that physicians cannot consider as a cause of acute respiratory failure even when recognising a goitre. However, physicians should consider goitre as a cause of acute respiratory failure in patients with goitres which have the characteristics such as diffuse, extending to substernal and encircling most of the trachea.1–3 Physicians should also recognise that not the size of goitres but the anatomy and shape of goitres may play a role developing in critical airway obstruction.1 Indeed, our patient had a diffuse goitre that extended substernal and encircled most of the trachea. For the treatment, endotracheal intubation and emergent thyroidectomy have been recommended as the best treatment for patients with life-threatening airway compromise secondary to benign goitres as conducted in our case.2,3

Learning points

► Acute airway obstruction should be considered in patients with severe progressive dyspnoea or respiratory failure.
► Goitre can be a cause of critical airway obstruction.
► Goitres with characteristics such as diffuse, extending to substernal and surrounding almost the entire trachea carry the risks of airway obstruction by goitres.

REFERENCES


Figure 1  (A) A contrast-enhanced CT scan at the time immediately after intubation. An enlarged thyroidal mass encircling most of the trachea is seen. (B) Macroscopic findings of a resected thyroid. The thyroid is diffusely enlarged and no apparent nodule or cyst is seen.

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