

Unexpected tracheal compression detected after immediate extubation failure

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Extubation failure is defined as the need for reinstitution of ventilatory support within 24–72 h of planned endotracheal tube removal. It occurs in 2–25% of extubated patients. Many causes, such as an imbalance between respiratory muscle capacity and work of breathing, upper airway obstruction, excess respiratory secretions, inadequate cough, encephalopathy and cardiac dysfunction, have been stated in the literature [1].

In patients on mechanical ventilation for exacerbations of chronic obstructive pulmonary disease (COPD), no exact extubation failure rates are known; however, occurrence rates will probably not be very low. Furthermore, the institution of non-invasive pressure ventilation for extubation failure has not been proven to be safe [2]. In COPD, the decision for reintubation is difficult and will be weighed against the chances of successful treatment of underlying causes. In case the respiratory reserve is marginal, reintubation sometimes will be foregone. We present a case of a 71-yr-old female with COPD and non-small cell lung cancer in which advanced COPD was suspected to be the major reason for extubation failure, but careful examination led to an unexpected and treatable underlying disorder.

A 71-yr-old female was encountered at home by paramedics in respiratory distress with reduced levels of consciousness. She was transported to the emergency room of our hospital. Her Glasgow Coma score was 4 and she was gasping. Her medical history revealed a T₂N₂ non-small cell bronchus carcinoma, treated with curative radiotherapy, and moderate COPD, dyspnoea with moderate exercise and FEV₁ <80% (COPD Gold class II). Physical findings were pulse 96 min⁻¹, blood pressure 180/100 mmHg and temperature 37.6°C. A large goitre was palpated in the neck with a deviation of the trachea to the right. She was tachypnoeic, and rhonchi were heard over all lung fields. Arterial blood gas analysis showed pH 7.04, PCO₂ 16.5 kPa, PO₂ 26.1 kPa, HCO₃⁻ 32.2 mmol L⁻¹,

base excess -2.7 mmol L⁻¹ and SaO₂ 100% (with oxygen mask). Leucocytes 12.2 × 10³ g L⁻¹ and C-reactive protein 156 mg L⁻¹ were indicative for infection.

The patient was immediately intubated and admitted to the intensive care unit (ICU). Insertion of the central venous line was very difficult because of the abnormal anatomy due to the large goitre. A chest X-ray showed infiltrations at the right middle lobe. Treatment was commenced with antibiotics for pneumonia and corticosteroids for exacerbation of COPD. *Haemophilus influenzae* was cultured from the sputum. Within 5 days infection parameters and chest X-ray were normalized. Extubation was immediately followed by progressive hypercapnia and severe respiratory failure. No inspiratory or expiratory stridor were noted. Immediate reintubation was performed. Because of the rapid extubation failure without clear cause, an upper airway obstruction was suspected. A chest computed tomography (CT) scan was ordered to find anatomic lesions responsible for airway obstruction. A large retrosternal mediastinal multinodular goitre was found. By endoscopy compression of the trachea was detected at the level of the mediastinal goitre. Thyroid hormones (T₃ and FT₄) and thyroid-stimulating hormone (TSH) were within normal ranges. A diagnosis was made of a non-toxic goitre causing upper airway obstruction and recurrent hypercapnic respiratory failure. A left-sided hemithyroidectomy was performed without complications. Pathological examination showed a multinodular goitre of 185 g. There were no signs of malignancy. On the first postoperative day, the patient was successfully extubated. The patient was discharged from the ICU after 2 days and from the hospital a week later.

After treatment for *H. influenzae* pneumonia and exacerbation of COPD, rapid extubation failure was encountered. Initially, progressive COPD and deteriorating pulmonary reserve was suspected. The discrepancy with the known pulmonary status forced us to consider other options. After diagnostic workup, an intrathoracic goitre was found that deviated and compressed the trachea causing upper airway obstruction. Hemithyroidectomy was performed and the patient could be extubated the next day.

Non-toxic goitre is a diffuse or nodular enlargement of the thyroid gland that does not result

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from an inflammatory or neoplastic process and is not associated with abnormal thyroid function. Endemic goitre is defined as thyroid enlargement that occurs in more than 10% of the population, and sporadic goitre results from environmental or genetic factors that do not affect the general population. Endemic goitres associated with iodine deficiency are associated with thyroid hypertrophy, hypothyroidism and cretinism. Sporadic goitres are generally asymptomatic and are found either by a clinician's physical examination or by the patient's observation of neck enlargement. Occasionally, the goitre may cause symptoms due to pressure on anterior neck structures, including the trachea (wheezing, cough), the oesophagus (dysphagia) and the recurrent laryngeal nerve causing vocal cord paralysis (hoarseness). The female-to-male incidence ratio is 4:1 [3,4].

The thyroid gland usually grows outward because of its location anterior to the trachea. Occasionally, the thyroid wraps around and compresses the trachea and/or the oesophagus, or extends inferiorly into the anterior mediastinum. In the diagnostic workup, all patients with goitre should be assessed for thyroid dysfunction with a serum thyrotropin (TSH) assay [5–8]. Assessment of size and extent of the goitre is necessary to determine if progressive growth of the thyroid is occurring. CT scan and magnetic resonance imaging are expensive but excellent for assessing tracheal compression and intrathoracic extension of the goitre [5,6,9]. Pulmonary function tests may be used as a functional assessment of tracheal compression [10]. Non-toxic goitres usually grow very slowly over decades without causing symptoms. Without evidence of rapid growth, obstructive symptoms (dysphagia, stridor, cough, shortness of breath) or thyrotoxicosis, no treatment is necessary. Therapy is considered if growth of the entire goitre or a specific nodule is present, especially if intrathoracic extension of the goitre, compressive symptoms or thyrotoxicosis exist [5,6,11]. The intrathoracic extension of the goitre cannot be assessed by palpation or biopsy. The goitre, if significant in size, should be removed surgically. Thyroidectomy or surgical decompression causes rapid relief of obstructive symptoms, as was demonstrated in our case. Most intrathoracic goitres may be removed from a cervical incision without sternotomy [11,12].

In conclusion, in ventilated patients with COPD and palpable goitre, extubation failure should not lead to the immediate assumption that only pulmonary factors might have led to the event, but compression of the trachea by intrathoracic extension of the goitre should be considered, as this may cause or add to the obstructive symptoms.

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