LIFE-THREATENING ACUTE RESPIRATORY DISTRESS IN LATE PREGNANCY

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SUMMARY
Acute severe ventilatory failure from any cause is always a critical emergency. In advanced pregnancy, such an episode is particularly hazardous. The management of a pregnant patient who presented with an acute episode of respiratory distress is described. Although this was thought initially to be an asthmatic attack, there was evidence of upper airway obstruction caused by an enlarged thyroid. The major complicating factors and aetiology are discussed.

KEY WORDS

CASE REPORT
The duty anaesthetists were forewarned of a patient en route to the emergency department. She was a 28-yr-old at term in her third pregnancy with a history of asthma, and presenting in severe respiratory distress.

On arrival, she was severely dyspnoeic with marked cyanosis and tachycardia, but with a good pulse pressure and peripheral perfusion. She appeared awake, but was unresponsive. Although little air entry could be heard, there was a wheeze, which was more marked on expiration than inspiration. She was given oxygen and salbutamol by nebulizer and i.v. infusion. A left lateral tilt was maintained to avoid aorto-caval compression. The fetal heart rate was normal. Arterial blood-gas analysis (which was not received until some time later) showed pH 6.82, Pao₂ 6.0 kPa, Paco₂ 14.6 kPa, base excess —22 mmol litre⁻¹ and SaO₂ 42%.

A brief history was obtained from the husband, who confirmed a past history of asthma, with recent problems strongly suggestive of an acute asthmatic attack. There was also a 2- or 3-yr history of mild nodular thyroid enlargement.

As the patient's condition did not improve immediately with bronchodilator therapy, because of the severity of her respiratory distress we proceeded to tracheal intubation and assisted ventilation without further delay. It was now noted that there was a moderately enlarged thyroid which interfered with the application of cricoid pressure. Anaesthesia was induced with etomidate 6 mg and tracheal intubation was facilitated by suxamethonium 100 mg. The mouth, pharynx and glottis were normal, in particular there was no laryngeal oedema, and the tracheal tube passed easily and without obstruction.

The lungs were easy to ventilate and the cyanosis resolved rapidly, excluding an acute asthmatic attack. The thyroid seemed to be larger. There was now fetal bradycardia and, as the mother's condition had been stabilized, arrangements were made for delivery by Caesarean section and an ENT surgeon was called. A live male infant was delivered; tracheal intubation and artificial ventilation were required.

During the Caesarean section the thyroid continued to expand in size and an ultrasound scan was performed in the theatre; this demonstrated an enlarged thyroid containing a cystic mass. Surgical exploration confirmed an enlarged thyroid with massive expansion of the isthmus, some retrosternal extension and relative sparing of the upper poles. After subtotal thyroidectomy, it was found that the isthmus contained a large haemorrhage. Tracheotomy was performed as there was a degree of tracheomalacia. Direct laryngoscopy confirmed normal mouth and pharynx, and glottis.

After a short period of ventilation in the Intensive Care Unit, spontaneous ventilation was re-established and the patient regained full consciousness with no neurological deficit. The tracheostomy was closed on the 5th day and the patient was discharged home later. On review in outpatients clinics, she was continuing to make a good recovery. The tracheostomy had healed and there has been no further dyspnoea.

Unfortunately, the baby showed evidence of hypoxic brain damage. A scan showed several intracranial bleeds and he died on the 3rd day after operation.

DISCUSSION
This patient presented in extremis, so only an extremely limited history was immediately available and this strongly indicated a severe asthmatic attack;
clinical examination was compatible with this diagnosis. Our initial treatment was directed, therefore, at treating an asthmatic attack and the correct treatment to relieve her airways obstruction was delayed. She failed to respond rapidly to pharmacological treatment and so the trachea was intubated in the emergency department within a few minutes of her arrival.

Had it been possible to question the patient, we would probably still have been misled. After operation, she recalled that she had felt a tightness in her chest, similar to an early asthmatic attack (similar indeed to one she had had the previous week). She denied any abnormal symptoms such as dysphagia. The dyspnoea increased rapidly and she was taken via the general practitioner’s surgery to hospital with minimal delay, during which time she lost consciousness. Two years previously, she had developed a small benign multicystic goitre, but no intervention was required. There had been no further thyroid enlargement noted at the antenatal booking clinic or when she was visited at home the day before admission, by a midwife who knew her well. Interestingly, her mother had required a thyroid operation about a year previously and had undergone tracheotomy. There had been no symptoms or signs of toxaemia of pregnancy, or of any other cause of laryngeal oedema.

The aetiology of this event remains rather obscure. Although the history indicated asthma, the operative finding was of an acute expansion of an already enlarged thyroid as a result of haemorrhage. There are several possible explanations.

First, the acute expansion, particularly of the thyroid isthmus, may have compressed the trachea, causing obstruction. The trachea was rather soft and the bulk of the haematoma was in the isthmus. This sequence is supported by the surgical evidence, but not by the history. A previous report of tracheal obstruction by an enlarged thyroid had an associated clear history of goitre, dysphagia and dyspnoea [1]. It is interesting that the tracheal tube passed the obstruction so easily, and that the thyroid was only moderately enlarged when the patient was first seen in hospital.

Second, the acute swelling of the isthmus might have increased the pressure caused by the retrosternal extension of the gland, resulting in obstruction at this point. This would explain why the symptoms and signs suggested an intrathoracic problem. However, at operation the retrosternal component did not appear significant.

Third, it may be that the initial event was an acute asthmatic attack which was compounded by tracheal obstruction caused by tracheomalacia, and that this episode triggered bleeding into the nodular goitre. This more complex sequence provides a fuller explanation of the clinical picture.

*There was no hypertension and no oedema obvious on tracheal intubation, so it seems unlikely that toxaemia of pregnancy was a significant contributory factor.*

Tracheomalacia is a well recognized cause of acute airway obstruction. There is a previous report in which this appears to have resulted in a clinical picture resembling asthma in a pregnant patient [2]. In that case, a massive goitre had been excised some years before.

Both asthma and thyroid disease may exist during pregnancy, and both may be affected by pregnancy. Asthma tends to exhibit a small overall improvement, but there is considerable individual variability [3]. It has been estimated that a life-threatening attack of asthma may be expected in 0.05–0.2% of all pregnancies [4].

Pregnancy has a goitrogenic effect. Part of this increase in thyroid size is caused by the increased body mass, but hormone concentrations are increased also [5]. In a review of a large series of patients presenting for thyroid surgery, 2% had significant respiratory compromise before operation, and 0.76% had severe dyspnoea resulting in episodes of suffocation, or of permanent dyspnoea with cyanosis [6]. Another group reported 24 patients requiring emergency admission caused by thyroid disease over a 4-yr period; nine of the patients needed immediate tracheal intubation and two were in the last trimester of pregnancy [7]. In most cases of respiratory obstruction caused by an enlarged thyroid, the symptoms are long standing and include feelings of pressure and dysphagia, in addition to dyspnoea. However, haemorrhage into a nodule may occur at any time, causing acute symptoms [8].

This case serves as a reminder that most medical emergencies may also occur during pregnancy, which further complicates the management. The true cause of an emergency may not be immediately obvious.

REFERENCES


