Acute respiratory failure in the ward: mind the oesophagus!

Katie Wren[1], Ashraf Roshdy[2,3]

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Correspondence to:
Ashraf Roshdy
Critical Care Unit
Queen Elizabeth Hospital
Stadium Road
Woolwich
London SE18 4QH
UK

Email:
ashraf.roshdy@nhs.net

A 77-year-old male presented with dyspnoea, cough and left-sided chest pain. He had past medical history of achalasia (oesophageal myomectomy in 1992), Parkinson's disease (PD) and asthma. Hours after admission to the ward he collapsed suddenly (Glasgow Coma Scale: E1M3V1) and appeared clammy and pale. His blood pressure was 200/120 mmHg. Arterial blood gas showed an unrecordable high partial pressure of carbon dioxide (normal range: 5.1–5.6 kPa), a partial pressure of oxygen of 54 kPa (normal range: 10.5–13.5 kPa) on fraction of inspired oxygen of 0.85 (room air equals 0.21) and pH of 7.0 (normal value: 7.35–7.45). He was intubated and admitted to the intensive care unit (ICU). Chest X-ray (Figure 1) showed enlargement of the oesophagus obscuring the right hemithorax. CT pulmonary angiogram (Figure 2) showed massive achalasia impacted with debris filling the right hemithorax, a small pulmonary embolism (PE) in the right distal branch and patchy changes in left lower lung zone.

The patient had possibly poorly controlled PD due to impaired medication transit. Oesophageal endoscopy and fluoroscopy were performed to aspirate the debris and to insert a nasogastric tube. The patient did not develop pneumonia and was extubated 48 hours later. A radiologically inserted percutaneous gastrostomy was then inserted before discharge home with dietician follow up. Three months after discharge, the patient described a significant improvement in his respiratory symptoms.

This case highlights the complexity of managing acute respiratory failure (ARF) with multiple pathologies. Achalasia and PD had probably contributed to the sudden deterioration of the patient. PE was considered not to be responsible for the ARF owing to its small size and the fact that the patient presented with type 2 (hypercapnia) rather than type 1 ARF (hypoxia). It is less clear if the dilated oesophagus contributed to the PE/thrombus in situ by compressing the pulmonary arteries.

Achalasia is a disease that causes impaired peristalsis of the oesophageal wall and relaxation of the lower oesophageal sphincter.[1] Despite oesophageal pathologies being known to be associated with many respiratory pathologies (e.g. gastro-oesophageal reflux and hiatus hernia), tracheal compression by a hugely dilated oesophagus has been rarely reported (approximately 50 cases since the 1950s).[2] Achalasia can cause both chronic respiratory failure and ARF by different mechanisms: aspiration pneumonia/pneumonitis, airway obstruction secondary to aspiration or external compression, interstitial lung fibrosis, lung abscess and isolated bronchospasm.[3] In this context, the condition can be misdiagnosed as asthma. It remains resistant to treatment until achalasia is diagnosed and properly managed.[1]

In our case the diameter of the oesophagus reached 151 mm and was filled with long-standing solid food debris. The rapid improvement after intubation and positive pressure ventilation combined with the absence of signs of pneumonitis or pneumonia indicates that the ARF was most probably secondary to tracheal compression by the massively dilated oesophagus.

The term mega-oesophagus had been recommended when the oesophageal diameter exceeds 70 mm.[4] In the long term, continuous pressure can lead to tracheomalacia rendering the trachea even more prone to collapse.[1] The trachea is protected by C-shaped cartilaginous rings where the posterior

[1]Foundation Year 1, Critical Care Unit, Queen Elizabeth Hospital, Lewisham and Greenwich NHS Trust, London, UK; [2]Consultant of Intensive Care Medicine, Critical Care Unit, Queen Elizabeth Hospital, Lewisham and Greenwich NHS Trust, London, UK; [3]University Lecturer and Consultant of Critical Care Medicine, Critical Care Department, Alexandria University, Alexandria, Egypt

Figure 1 Anteroposterior semi-erect chest X-ray of the patient on admission to the ward

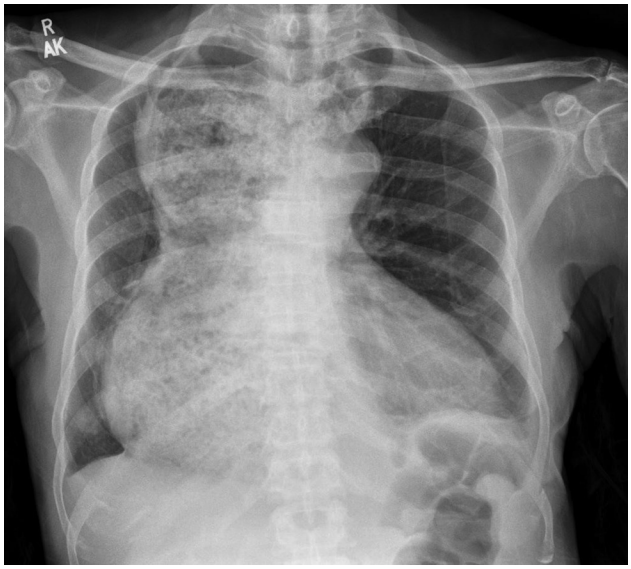
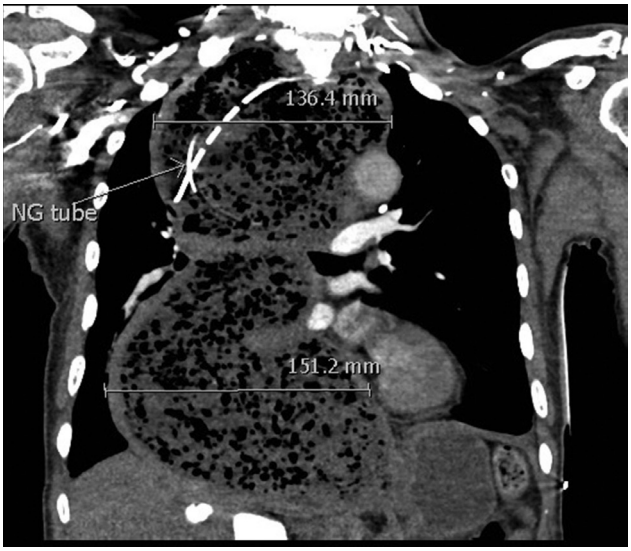


Figure 2 Coronal CT pulmonary angiogram reconstruction showing grossly enlarged oesophagus secondary to acahlasia and filled of food, note the coiled nasogastric tube (NG) in the upper oesophagus (arrow)



wall is membranous. As such, the mega-oesophagus, especially when filled with food, can easily compress the posterior soft wall of the trachea. This may be the cause of why ARF has been mostly reported shortly after oral feeding.[5] In our case, the trachea was narrowed to 5 mm below the lower level of the endotracheal tube and just above the carina (normal diameter 13–25 mm).[4]

PD had probably played a significant role in the progression of the patient's condition. While clinicians are generally aware of the impact PD has on the oral and pharyngeal stages of deglutition, the oesophageal affection is less known.[6] This occurs in different ways: impaired peristalsis, oesophageal spasms, slower oesophageal transit, and deficit in sphincter relaxation and pressure.[6]

Medication management merits special interest in our case. The patient may well have entered a worsening cycle wherein he was poorly absorbing his Parkinson's medications due to unreliable transit. Poorly controlled PD may have worsened the dilatation of his already very enlarged oesophagus. This is of usual concern when PD patients are admitted to the ICU and have impaired absorption.

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