

Unusual presentation of more common disease/injury

Bilateral pneumothoraces and pulmonary oedema following tracheostomy induced by acute tracheal obstruction

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Summary

We describe the presentation of bilateral pneumothoraces with pulmonary oedema following an elective tracheostomy. A 69-year-old man underwent panendoscopy following primary chemoradiotherapy for locally invasive vocal cord carcinoma. A tracheostomy was performed for upper airway oedema and necrosis. Postoperatively, acute airway obstruction with profound desaturation developed. Tracheostomy tube suctioning dislodged an airway clot with clinical improvement and restoration of bilateral breath sounds. A chest X-ray subsequently demonstrated bilateral pneumothoraces with marked pulmonary oedema. Management, including chest drain insertion, resulted in stabilisation and subsequent full recovery. This case highlights the potential for more than one cause of life-threatening complication following tracheostomy. The importance of considering multiple pathologies in the setting of severe hypoxia and to institute prompt management is emphasised.

BACKGROUND

Complications following tracheostomy are uncommon and generally controllable; however, some can be fatal.¹ Pneumothorax is a rare but expected complication of the procedure.² Hydrostatic pulmonary oedema can occur after upper airway obstruction, but has not been reported following tracheostomy. This case describes the simultaneous development of bilateral pneumothoraces and pulmonary oedema following tracheostomy.

CASE PRESENTATION

A 69-year-old man was scheduled for panendoscopy following chemoradiotherapy for a locally invasive (T3N0M0) vocal cord squamous cell carcinoma. History was notable for hypertension and chronic obstructive pulmonary disease. He was an ex-smoker with a 40-pack year's history. Examination revealed stridor but was otherwise normal.

A preoperative chest x-ray (CXR) demonstrated emphysematous lung but was otherwise normal. Significantly a CT of the thorax revealed no evidence of a pulmonary bleb. Cardiac echocardiogram was normal (left ventricle ejection fraction >56%). MRI of the larynx showed glottic carcinoma extending into the anterior commissure and lower supraglottic space. A fiberoptic endoscopy examination under topical anaesthesia showed glottic oedema.

In the operating room, routine monitors were applied and the airway was secured with a 6 mm cuffed microlaryngoscopy tube following an uneventful awake fiberoptic intubation. Anaesthesia was then induced with propofol, remifentanyl and mivacurium and maintained with oxygen, air, sevofurane and remifentanyl. Microlaryngoscopy revealed necrosis and oedema in the glottic and supraglottic regions. Multiple biopsies were taken. In view of these findings, an uneventful tracheostomy was performed with an 8 mm cuffed Shiley tracheostomy tube. At

the end of the procedure, the patient was awake, breathing comfortably and haemodynamically stable.

An hour later, respiratory difficulty progressing to acute obstructive apnoea with profound desaturation ($\text{SaO}_2 < 80\%$) developed. Tracheostomy tube suctioning dislodged an airway clot with prompt improvement in gas exchange and oxygen saturation (figure 1). Auscultation of the chest revealed diminished breath sounds bilaterally with bilateral basal crackles. Despite 100% oxygen, respiratory distress continued with increasing tachypnoea and SaO_2 ranging between 90 and 93%.

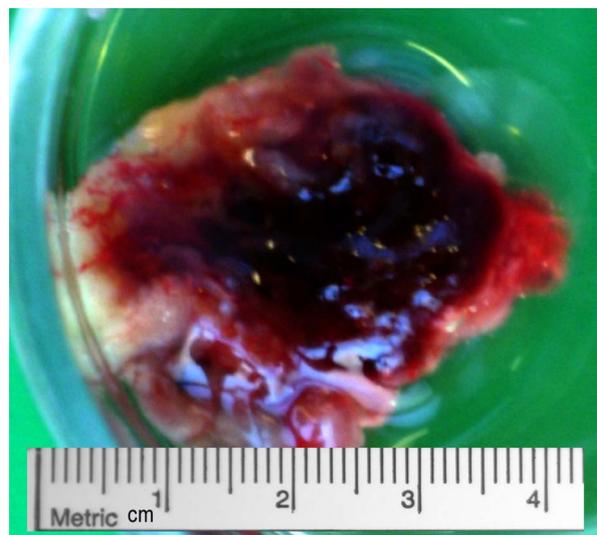


Figure 1 Haemorrhagic mucus plug, which was removed while changing tracheostomy tube. Histopathology was consistent with inflamed inspissated secretions.



Figure 2 Bilateral pneumothoraces with lung collapse and pulmonary oedema.

CXR revealed bilateral pneumothoraces, subcutaneous emphysema and bilateral pulmonary infiltrates consistent with pulmonary oedema (figure 2). A left chest drain was inserted with immediate improvement in respiratory effort and oxygen saturation (>90%). Treatment with oxygen, diuretics, bronchodilators and chest physiotherapy resulted in gradual resolution of both pneumothoraces. This was confirmed by CXR (figure 3). The chest drain was removed on day 2 and the patient was discharged on day 10.

DISCUSSION

Tracheostomy is one of the oldest surgical procedures dating back to 3000 years. A Greek physician, Asklepiades is credited with undertaking a tracheostomy in 100 BC.³ Today, the complication rate ranges between 4% and 40% depending on the study design,^{1 2 4} and may relate to the procedure itself or to the equipment used. The commonest early complication is haemorrhage (3–6% of cases); others include tube displacement or obstruction, infection, pneumomediastinum and pneumothorax.^{1 2 4 5}



Figure 3 Chest x-ray following left chest drain insertion showing partial re-expansion of the lung on that side. The right-sided pneumothorax was resolved. Bilateral pulmonary oedema is evident.

The incidence of subcutaneous emphysema and pneumothorax is less than 1% following tracheostomy, but it carries significant morbidity and mortality.^{2 4} The mechanism usually involves damage to the tissues around the stoma with consequent entrainment of air.⁶

In the setting of upper airway obstruction, powerful inspiratory effort generates a marked negative intrathoracic pressure. This pressure ‘sucks’ air from the atmosphere into the mediastinum through the edges of the tracheostomy wound, or (in this case) possibly through the laryngeal biopsy sites. Air accumulates in the mediastinum, when this air reaches a sufficient pressure it ruptures through the pleura.⁷ The resulting pneumothorax leads to hypoxaemia by reducing alveolar ventilation following collapse of the lung. Prevention of the problem requires avoidance of airway obstruction and meticulous surgical technique, including limiting the time interval between dividing the cervical fascia and inserting the tracheostomy tube.⁷

Other less common causes of a pneumothorax in this setting include rupture of a pulmonary bleb, barotrauma or an iatrogenic problem (lung biopsy, central vein cannulation, etc).⁸ The possibility of pulmonary bleb rupture is remote in this case, given the absence of a pre-existing bleb on the preoperative CXR or CT of the thorax.

The pathogenesis of negative pressure pulmonary oedema is multifactorial and well described.^{9 10} It occurs with forced inspiration against a closed or obstructed airway. The mechanism appears to be transmission of negative intrapleural pressure to the pulmonary capillary bed leading to transudation of fluid from the pulmonary capillaries into the interstitial space. This results in the clinical picture of interstitial and alveolar oedema with impairment of gas exchange.¹¹

Our patient developed airway obstruction due to mucus plug formation in the tracheostomy tube. The consequent marked negative intrathoracic pressure resulted in interstitial oedema. This negative pressure also entrained air around the stoma and/or laryngeal biopsy sites, causing air to enter the mediastinum and subsequently rupture into the intrapleural space resulting in bilateral pneumothoraces. These dual pathologies led to severe hypoxaemia by different mechanisms—pulmonary oedema impairing gas exchange and bilateral pneumothoraces reducing alveolar ventilation. Both the pneumothorax and the interstitial oedema required prompt therapy in the post-anaesthesia care unit (PACU) to stabilise the patient.

In summary, better understanding of the potential causes of hypoxia following tracheostomy along with optimal surgical practice and heightened vigilance may help prevent such occurrences with their potentially fatal consequences.

Learning points

- ▶ Better understanding of the potential causes of hypoxia following tracheostomy along with optimal surgical practice and heightened vigilance may help prevent such occurrences with their potentially fatal consequences.

Competing interests None.

Patient consent Obtained.

Correction notice This article has been corrected since it was published online on the 20 August 2012. The author list has been amended.

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