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Title: Obstructive fibrinous pseudomembrane tracheitis after double lumen tube intubation: a case report

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Running title: Obstructive pseudomembrane tracheitis

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Obstructive fibrinous pseudomembrane tracheitis after double lumen tube intubation: a case report

Running title: Obstructive pseudomembrane tracheitis
Abstract

Background: Obstructive fibrinous pseudomembrane tracheitis [OFPT] is a rare complication of endotracheal intubation.

Case: We describe a 73-year-old woman who had undergone a short duration of intubation for video-assisted thoracotomy and developed acute life-threatening stridor 2 days after extubation from OFPT. She required emergency tracheostomy to maintain airway patency and bronchoscopy for removal of the obstructing pseudomembrane. She also suffered a non-ST elevation myocardial infarction as a result of this episode. She recovered and the tracheostomy was subsequently decannulated after a few weeks. Histology returned to reveal mucosal ulceration and inflammatory changes.

Conclusions: OFPT is an uncommon cause of life-threatening airway obstruction after extubation that is often not immediately recognized, but is usually readily treatable with early bronchoscopic intervention.

Keywords: Airway management; Airway obstruction; Respiratory insufficiency; Respiratory tract diseases; Tracheal stenosis; Tracheitis.
Endotracheal intubation is widely performed for airway control during anesthesia. Obstructive fibrinous pseudomembrane tracheitis [OFPT] is a rare but life-threatening complication of endotracheal intubation, and can mimic other pathology, such as vocal cord palsy, laryngeal edema, and tracheomalacia. Patients usually present with stridor and voice hoarseness hours to days after intubation. Here, we report a patient who had respiratory distress 2 days after thoracic surgery. Written informed consent for publication was obtained from the patient.
Case report

A 73 year-old woman [height: 154cm, weight: 55kg] was diagnosed with left lower lobe malignant lung nodule incidentally discovered on computed-tomography scan. She was a nonsmoker, with a history of hypertension, non-insulin-dependent diabetes mellitus on oral-hypoglycaemic agents, epilepsy on sodium valproate, and a previous laparoscopic cholecystectomy performed 11 years ago, which was uncomplicated according to the patient, but there were no old charts that could be retrieved. She also had a history of hyperthyroidism, and had radioactive iodine treatment more than 10 years ago, and was on oral levo-thyroxine replacement. The preoperative thyroid function tests were normal and she was clinically euthyroid. There were no preexisting voice hoarseness or dyspnea. Functional capacity was 4-10 METS as she was able to walk 200m at normal speed.

Preoperative assessment showed a normal airway [Mallampati 2, interincisor distance was at least 5cm and full neck range of movement, no neck masses] and the patient was edentulous. Routine tests for thoracic surgery showed that her spirometry was normal [FEV1 1.44L, 85% of predicted. FVC1 1.72L, 92% of predicted. DLCO 94% of predicted]. Transthoracic echocardiography revealed an ejection fraction of 66%, and absence of regional wall motion abnormalities. There was aortic valve sclerosis, but no stenosis and all other valves were normal.

She underwent video-assisted thoracoscopic surgery for left lower lobectomy. Induction of anesthesia was uneventful, direct laryngoscopy was performed which showed a grade 1 Cormack-Lehane view with no abnormality of the vocal cords. A size 37 left double-lumen endotracheal tube [DLT] was inserted easily on first attempt, no excessive pressure or force was used. A DLT of this size was chosen, instead of one of smaller bore, in order to facilitate fiberoptic bronchoscopy and tracheal suctioning. The bronchial tip was directed into the left mainstem bronchus on first attempt atraumatically with fiberoptic bronchoscope guidance. The tracheal cuff was maintained at 20cmH2O. The DLT was in place for 2h 10mins and surgery took place uneventfully. Anesthesia
was reversed fully and she was then extubated to face mask. She had some sore throat but no hoarse voice was noted, and she was subsequently discharged from the post anesthesia care unit.

However, after 48hrs, she was noted to have a hoarse voice with soft stridor in the ward. There were no swallowing problems. A bedside nasoendoscopy was performed which showed anterior and posterior webbing was seen in the subglottic area, just inferior to the level of the true cords, corresponding to Cotton-Myer grade II stenosis [Figure 1]. The membrane was not removed as the nasoendoscope was narrow-bored which did not have features to allow therapeutic excision to be performed, and monitoring was also limited at the bedside for such procedures to be performed safely. At this time, the patient only had acute hoarseness of voice, she did not have any dyspnea or respiratory distress, and was able to maintain adequate oxygenation on room air. For fear of aggravating laryngeal edema, bronchoscopy was avoided. She was subsequently started on inhaled budesonide puffs.

However, she deteriorated quickly the next day and required emergency surgical intervention for critical upper airway obstruction [Figure 2]. The oral pharynx was topicalised with lidocaine and an awake videolaryngoscopy was performed but visualization of the glottic opening was poor, and an awake tracheostomy was performed under local anaesthesia with oxygenation via a Hudson mask. On securing the airway with tracheostomy, a direct laryngoscopy was performed which showed white adherent fibrinous material at the glottis and subglottic area with residual 2mm pinpoint glottic gap posteriorly, corresponding to a Cotton-Myer grade III subglottic stenosis. The adherent fibrinous material appeared fixed at first but on palpation, it was removable. After peeling off the material with a bronchoscope, subglottic area appears less than 50% stenotic, improving to a Cotton-Myer grade I stenosis. The underlying mucosa appears healthy anteriorly but the posterior commissure mucosa was edematous. Nasoendoscopic evaluation during the subsequent days showed that the pseudomembrane had cleared.
Subsequently, she suffered a non-ST elevation myocardial infarction the next day, when she presented with T-wave inversion without any angina or evidence of hypoxaemia. She underwent percutaneous coronary intervention, and had stenting of the culprit left anterior descending artery occlusion, which was an underlying structural lesion that was undetected preoperatively. She was subsequently discharged 2 weeks after hospital admission. Serial outpatient nasoendoscopy done showed no reaccumulation of the subglottic pseudomembrane and the tracheostomy was successfully decannulated two months later.

Histological analysis of the subglottic material returned to show mainly inflammatory changes. There was squamous-lined mucosa with extensive ulceration and formation of organizing fibrinopurulent acute inflammation in the lamina propria. No bacteria or fungi demonstrated on stains.
Discussion

OFPT is a known but rare and potentially fatal complication of endotracheal intubation. The exact pathophysiology of pseudomembrane formation remains unclear. One hypothesis states that the subglottic mucosa is prone to injury during intubation as it is the narrowest part of the adult larynx and it is completely surrounded by the complete ring of the cricoid cartilage. After the injury, accumulation of the desquamated epithelial cells may result in a membranous-like material [1]. In support of this, the most common location of the tracheal pseudomembrane is in the subglottic area, but can also be found distally, such as in the mid-trachea or carina [2-5].

Risk factors may include female gender, tracheal tube cuff pressure >25cmH₂O, traumatic intubation, use of double-lumen endotracheal tube, prolonged endotracheal intubation, airway stenting, immunocompromised host, bacterial or fungal infection [1,2,6-16]. Additionally, aspiration of gastric contents leading to caustic injury has also been proposed as a contributory factor to the formation of OFPT after the initial disruption to the tracheal mucosa, followed by abnormal healing process [1,14]. A high clinical vigilance should be present as OFPT can occur in the absence of prolonged intubation, and there can be a large variation to the time to onset of symptoms after extubation [3,5]. Before confirmation of the diagnosis, patients usually received empirical treatments consisting of salbutamol bronchodilator, epinephrine, and corticosteroids.

Bronchoscopy has to be performed in order to confirm diagnosis [1,5]. Histology typically shows the presence of inflammatory infiltrates, fibrin and ulceration. Bacteria, virus or fungi may also be seen as they may also cause OFPT. Treatment typically involves removal of the pseudomembrane via rigid bronchoscopy or flexible bronchoscopy to relief the obstruction, and rigid bronchoscopy is the safer option as it can be used to maintain ventilation during the intervention. Although recurrence of the tracheal membrane is possible but uncommon, it can result in repeated obstruction [1].
In conclusion, OFPT is an uncommon cause of airway obstruction after extubation that is often not immediately recognized, but usually readily treatable with bronchoscopic intervention.
References


Figure 1: Thick rubbery whitish pseudomembrane at the anterior and posterior region, in the subglottic area, just inferior to the level of the true vocal cords.
Figure 2: Near-complete upper airway obstruction requiring emergency tracheostomy.

Credit to co-author Brenda L H Sim for providing the figures above.