

Obstructive Fibrinous Tracheal Pseudomembrane After Tracheal Intubation: A Case Report

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Obstructive fibrinous tracheal pseudomembrane is a rare, but potentially fatal complication associated with endotracheal intubation. It has been known that the formation of tracheal pseudomembrane is related with intracuff pressure during endotracheal intubation or infectious cause. But in the patient described in this case, pseudomembrane formation in the trachea was associated with subglottic epithelial trauma or caustic injuries to the trachea caused by aspirated gastric contents during intubation rather than tracheal ischemia due to high cuff pressure. We report a patient with obstructive fibrinous tracheal pseudomembrane after endotracheal intubation who presented with dyspnea and stridor and was treated successfully with mechanical removal using rigid bronchoscopy.

Key Words: Airway Obstruction; Intubation; Bronchoscopy

INTRODUCTION

Obstructive fibrinous tracheal pseudomembrane (OFTP) is a rare condition associated with endotracheal intubation in which a pseudomembrane that encircles the tracheal wall is formed. Little is known about the mechanisms involved in the development of this fibrinous pseudomembrane, but it requires early detection and urgent management, as it causes a life-threatening tracheal obstruction. Here, we present a case of OFTP that developed 3 days after extubation in a patient who was intubated for 3 days.

CASE REPORT

A previously healthy, nonsmoking 66-yr-old woman (height 160 cm, weight 58 kg) was admitted to the hospital because of altered mental status on November 8, 2007. According to her family, she was vomiting and becoming increasingly lethargic when found, and empty sleeping pill bottles were discovered beside her. On arrival to the hospital, the patient was somnolent and agitated, and unable to follow commands. Her blood pressure was 110/70 mmHg, pulse rate 80/min, body temperature 36°C, and respiration rate 24/min. Physical examination revealed no evidence of trauma. The lungs were clear on auscultation. The results of urinalysis and routine hematologic and blood

chemistry tests were normal. Brain computed tomography (CT) was normal. The arterial pH was 7.23, PaO₂ 35.3 mmHg, PaCO₂ 62.7 mmHg, and SaO₂ 55.8% while the patient was breathing room air.

After several unsuccessful and traumatic attempts, her trachea was intubated with a 7.5-mm cuffed tracheal tube (Hi-Lo™, Mallinckrodt Medical, Athlone, Ireland) to treat acute respiratory distress. After mechanical ventilation, the patient's condition improved rapidly. Suctioning via the endotracheal tube revealed scanty amount of whitish sputum that showed no evidence of microorganisms. Seventy-two hours after intubation, the trachea was extubated. While intubated, the intra-cuff pressure was monitored and maintained below 15 cmH₂O. Three days after extubation, she complained of dyspnea and inspiratory wheezing. On examination, she was afebrile and tachypneic. Despite treatment with a short-acting bronchodilator and steroids, the symptoms became progressively worse. There were no signs suggestive of infection on a chest radiography, blood cultures, urinalysis and sputum cultures. But chest CT revealed an irregular luminal narrowing in the proximal trachea just below the vocal cords (Fig. 1).

On flexible bronchoscopy under conscious sedation, a white, rubbery membrane encircling the trachea was seen just below the vocal cords, which resulted in narrowing of the tracheal lumen by approximately 80% (Fig. 2). This pseudomembrane was

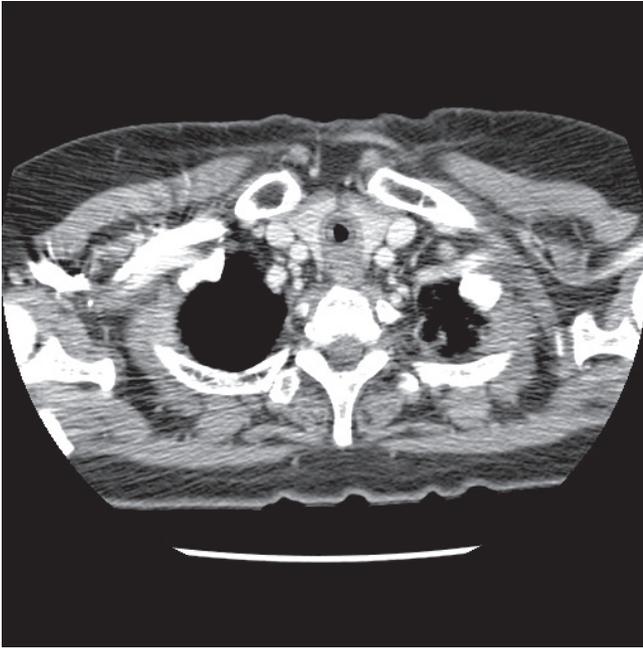


Fig. 1. Chest computed tomography image at the level of the trachea just below the vocal cords showing luminal narrowing.

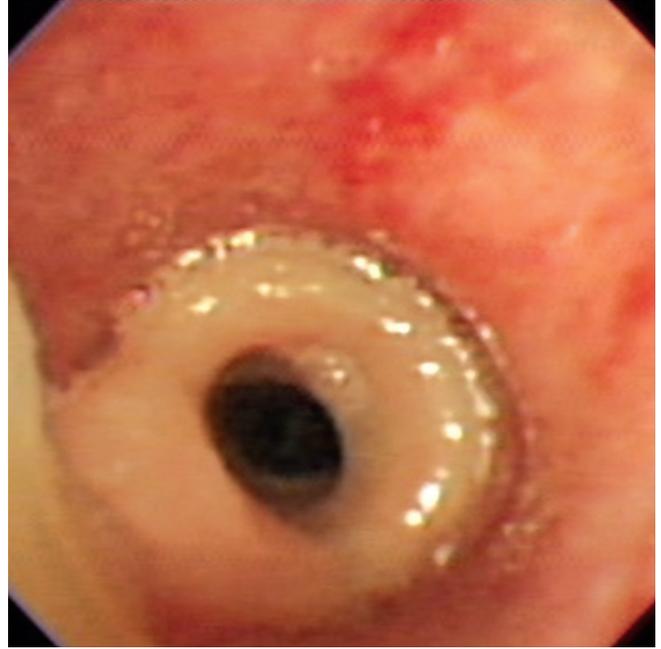


Fig. 2. Bronchoscopic image showing a thick, annular, rubberlike pseudomembrane encircling the tracheal wall.

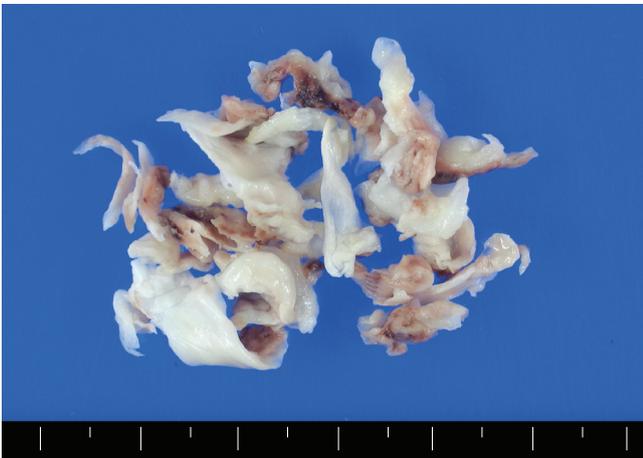


Fig. 3. Gross view of the pseudomembrane removed at rigid bronchoscopy.

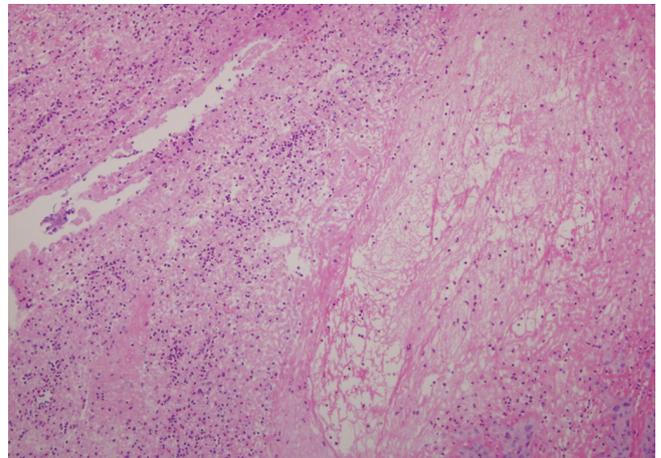


Fig. 4. Microscopic examination showing that the pseudomembrane consists of fibrinous material with polymorphonuclear cell infiltration (H&E stain $\times 100$).

tubular in shape, 4 cm long, and adherent to the tracheal wall. We tried to remove the pseudomembrane using bronchoscopic forceps, but the lesion was cut to pieces. Subsequently, rigid bronchoscopy was performed under general anesthesia and the pseudomembrane was removed entirely with mechanical forceps (Fig. 3). After the procedure, no findings suggestive of tracheomalacia or granulation tissue formation in the tracheal wall were detected. The patient's symptoms improved immediately after removing the pseudomembrane. The biopsy specimen showed fibrinoid material with eosinophils and acute inflammatory cells (Fig. 4). Tissue cultures grew no bacteria or fungus.

Bronchoscopy performed 3 months later showed no residual

lesion. The patient has remained asymptomatic for 17 months.

DISCUSSION

Tracheal complications associated with endotracheal intubation include tracheal stenosis, ulcers, granuloma, and tracheomalacia (1). Of these, tracheal stenosis is one of the most dire complications and requires appropriate evaluation and management, such as endotracheal stenting or tracheal surgery.

Tracheal pseudomembranes are rare, but can occur following endotracheal intubation. Sigrist et al. (2) reported a rare, fatal case involving the development of a tubelike formation in the upper trachea after intubation. Harbison et al. (3) also described

a patient who developed a tracheal fibrinoid membrane 3 days after extubation. Deslée et al. (4) reported a series of ten patients with tubular, rubberlike pseudomembranes molding the trachea after a short duration of intubation. In these patients, the mean duration of endotracheal intubation was 6.2 ± 1.8 days, but in four cases it was less than 24 hr. Seven patients developed symptoms of acute airway obstruction and these symptoms occurred shortly after extubation (59 ± 27 hr). As in the reported cases, our patient was intubated for 72 hr and developed dyspnea and stridor 3 days after extubation.

Most reported cases of pseudomembrane formation in the trachea are associated with an infectious cause. *Diphtheria* is a well-known cause of pseudomembrane formation in the respiratory tract and other infectious etiologies have also been implicated, including fungi, bacteria, and viruses (5). Membranous laryngotracheobronchitis (membranous croup) may be considered in the differential diagnosis of OFTP, but it is predominantly associated with signs of infection including high fever, a greater degree of toxicity and presence of pathogenic bacteria in tracheal secretions or membrane (6).

The mechanism involved in the development of a pseudomembrane following endotracheal intubation is not clear, although tracheal ischemia due to cuff pressure injury of the endotracheal tube has been suggested as the etiology (4). When the cuff pressure exceeds 30 mmHg, it can result in mucosal ischemia, which causes ulceration and shedding of the tracheal epithelium. The pathological findings of tracheal pseudomembranes, such as superficial abrasions of the mucosa and desquamated necrotic tracheal epithelium, support this hypothesis (4).

However, we suggest that a pseudomembrane can be formed independently of cuff pressure. Indeed, the cuff pressure in our case was maintained below 15 cmH₂O while she was intubated. Considering the history of vomiting and several traumatic attempts to intubate the trachea, acid injury or traumatic injury to the tracheal mucosa, rather than ischemic injury, may have triggered the development of a pseudomembrane in our patient. When we tried to intubate the trachea, the endotracheal tube might have been smeared with gastric acid in her oral cavity, which may eventually have damaged the tracheal mucosa in contact with the cuff, and trauma might have increased the damage to the tracheal mucosa. Several papers support our hypothesis that the formation of a pseudomembrane is associated with subglottic epithelial trauma or caustic injuries to the trachea

caused by aspirated gastric contents during intubation (7, 8).

If left untreated, OFTP may cause acute tracheal obstruction, which can be life-threatening. Once a diagnosis of OFTP is established, the lesion should be removed immediately. In many cases, the only successful treatment is excision of the lesion via rigid bronchoscopy (4). In our case, we tried to remove the pseudomembrane with flexible forceps, but they were cumbersome and the procedure took too long. Subsequently, we performed rigid bronchoscopy and removed the pseudomembrane easily.

In summary, we described a case of OFTP that was removed via rigid bronchoscopy. OFTP is a very rare condition, but should be considered in every patient who develops symptoms of acute airway obstruction shortly after extubation. Tracheal ischemia has been considered as the main etiology in the development of a tracheal pseudomembrane, but we postulate that acidic or traumatic injury to the tracheal mucosa was a more likely cause in our case. Physicians should be alert to this fatal complication and bronchoscopy should be performed for the precise diagnosis to allow early detection. Mechanical ablation using a rigid bronchoscope can cure this rare entity.

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