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Obstructive Fibrinous Tracheal Pseudomembrane:

A Rare condition in post-extubation stridor

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Conflict of interest

None

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Abstract

Obstructive fibrinous tracheal pseudomembrane (OFTP) is an airway complication which occurs after endotracheal intubation. It originates from superficial mucosal abrasion and desquamation of necrotic tracheal epithelium at the site of cuff pressure. This condition is a rare cause of post-extubation stridor. We present a case of post-extubation stridor secondary to OFTP, resulting in clinical features of upper airway obstruction over 8 hours after extubation. The case features complete obstruction at tip of endotracheal tube from OFTP.

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Introduction

Post-extubation stridor is a life-threatening condition. Causes include obstructed secretion, vocal cord edema, vocal cord dysfunction, and tracheomalacia.[1] This report describes a rare case of post-extubation stridor due to obstructive fibrinous tracheal pseudomembrane (OFTP) that blocked the tip of an endotracheal tube (ETT) completely requiring reintubation.

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Case Report

A 29-year-old man was admitted with respiratory failure and diabetic ketoacidosis. 5 days before admission, he presented with fever, productive cough and worsening shortness of breathing. He had a 15 pack-year smoking history and continued to smoke eight to ten cigarettes per day. On the first day of admission, he developed respiratory failure requiring endotracheal intubation with low-pressure/high-volume ETT, and was transferred to intensive care unit (ICU) for ventilatory support and hemodynamic monitoring. Physical examination revealed a temperature of 39 degree Celsius, cyanosis and bilateral inspiratory fine crackles over lower lung zones. Chest radiograph revealed opacification predominately at the right upper lobe and right lower lobe areas, consistent with pneumonia. He was treated with broad spectrum antibiotics and intravenous insulin infusion. In the ICU, the endotracheal cuff pressure was checked routinely and adjusted to be less than 25 cmH2O. He was liberated from mechanical ventilation 10 days after admission, however eight hours after extubation he complained of dyspnea and examination revealed inspiratory stridor. Direct laryngoscopy was performed and revealed no evidence of angioedema or vocal cord dysfunction. His clinical picture worsened, and the patient was re-intubated. However, ventilation was not possible due to significantly elevated airway pressure. Suction through ETT was not attempted due to severe restlessness and oxygen desaturation. The endotracheal tube was immediately removed. At the tip of endotracheal tube, there was a tubular membrane of necrotic debris (approximately 3 cm long and 1.5 cm thick) (Fig 1 and 2). This membrane completely obstructed the tip of the ETT. His stridor resolved immediately after ETT removal within few minutes. Repeat bronchoscopy was considered in order to demonstrate residual tissue and tracheal mucosa. Unfortunately, the patient absolutely

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refused any additional procedures. The patient was closely observed for any respiratory compromise over a period of 24 hours without any further need for reintubation. Histopathology of the necrotic debris revealed “membranous fragments of fibrino-inflammatory debris with focal areas of reactive squamous epithelium”.

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Discussion

Several etiologies of post-extubation stridor have been described. OFTP is a rare cause of this condition. We present a case of a worsening dyspnea and inspiratory stridor in the post-extubation period, with OFTP diagnosed by observation of a thick red-whitish tubular pseudomembrane molding within the tip of the endotracheal tube leading to complete luminal obstruction.

Post-intubation tracheal complication is a result of airway damage, with the most common entities being tracheal stenosis and tracheomalacia[2], while OFTP is rare and little known. Etiologically, high endotracheal cuff pressure leads to local ischemia, subsequent infarction and ultimately necrosis of the tracheal mucosa.[2] It is postulated that OFTP may be the initial process that can lead to tracheal stenosis afterwards.[3] This process results in superficial tracheal damage leading to macroscopic visible tissue plug at the site of the endotracheal cuff. Pathological findings include superficial abrasions of the mucosa and desquamated necrotic tracheal epithelium.[2] Several factors have been proposed which predispose patients to tracheal injury after intubation[4], however the risk factors for OFTP remains difficult to ascertain due to rarity of the condition. We postulate that diabetes may be a contributing factor to development of OFTP in this patient, as microcirculatory changes in diabetes which are well described may directly affect tissue healing.

Diagnosis of OFTP can be challenging because clinicians are often misled to diagnose patients with other causes of post-extubation stridor. We believe that positional and intermittent stridor due to ball-valve obstruction from the pseudomembrane may be an important clinical clue. Onset of respiratory distress in OFTP varies, ranging from within the first hour to 14 days after extubation.[3, 5-9] High pressure cuff and ETT type may directly affect development of OFTP, however we

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have no explanation of why OFTP still occurred in our patient despite the fact that low-pressure/high-volume cuff was used in conjunction with intensive monitoring of cuff pressure.

Early detection of OFTP is important in order to avoid further deterioration of the patient. However, diagnosis of this condition remains difficult. In recent reports, flexible or rigid bronchoscopy appears to be of value diagnostically and may also allow removal of the lesion.[5] Macroscopically, bronchoscopy usually reveals a thick, circular, rubber-like membrane adhering to the tracheal wall at the site of the cuff. However, our patient was surprisingly diagnosed with visible pseudomembrane blocking the ETT lumen. We suppose that reintubation may have detached the tissue from the tracheal wall. Unfortunately, repeat bronchoscopy was not performed to evaluate the residual tissue and tracheal mucosa due to patient refusal. However, in most cases OFTP are easily removed by bronchoscopy and patients almost always recover without residual lesions.[7-9] Besides bronchoscopy, the chest imaging, particularly computed tomography of chest, is also a helpful in diagnosing OFTP.[8, 9] Occasionally, this condition may be diagnosed by spontaneous expectoration of the OFTP tissue. However, prompt recognition of this condition is vital as the management of the patient would be completely altered.

Conclusion

In conclusion, this report highlights OFTP as a potentially fatal condition in patients presenting with dyspnea and post-extubation stridor. Clinicians should be aware of this condition, and bronchoscopy should be performed to allow early detection of this condition.

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Figure legends

Figure 1. Necrotic debris completely obstructing the tip of the endotracheal tube (arrow).

Figure 2. A thick, circular, rubber-like pseudomembrane (arrow) was revealed.

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Necrotic debris completely obstructing the tip of the endotracheal tube (arrow).
914x682mm (72 x 72 DPI)

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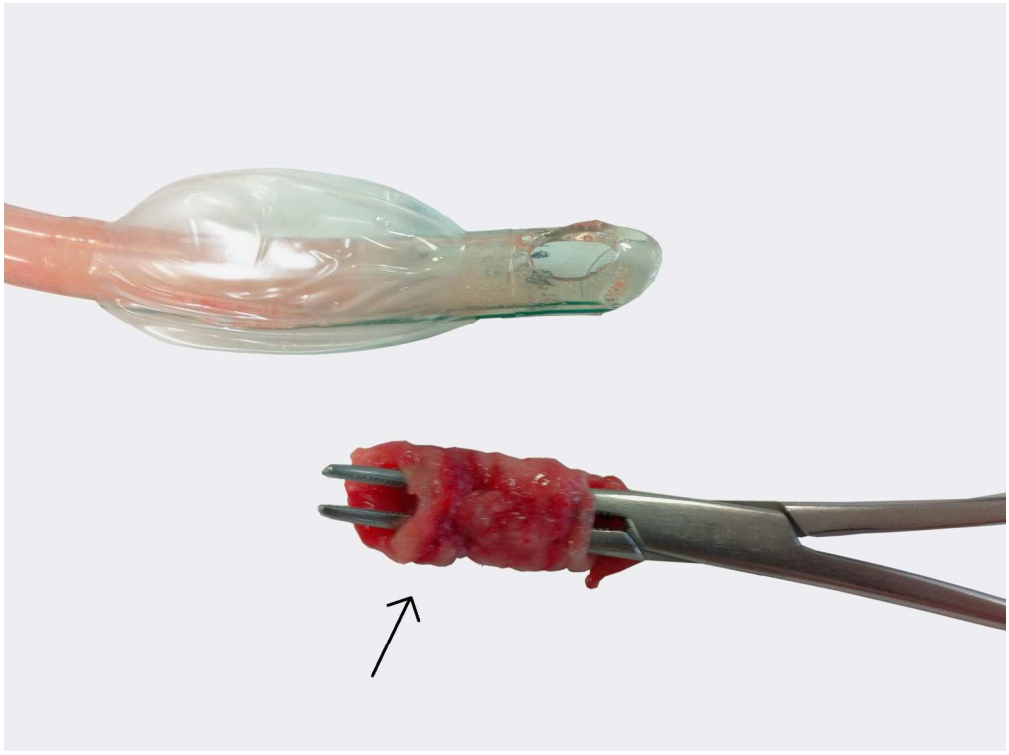

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