

A Rare Mechanical Cause of Extubation Failure After Short-Term Intubation and Outgoing with the Stridor Clinic: A Case Report

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Abstract

Stridor after extubation is generally a clinical symptom that may indicate laryngeal edema or vocal cord injury due to intubation, and hoarseness may accompany this. The presence of stridor may cause reintubation due to upper airway obstruction, a risk factor for patient mortality and morbidity. Laryngeal edema and vocal cord damage are mostly seen as the cause of stridor. In this case, OFTP (Obstructive fibrinous tracheal pseudomembrane) and stridor in the lower line of the vocal cords, which are rarely seen during short-term intubation, are presented.

Keywords: Obstructive fibrinous tracheal pseudomembrane; post extubation stridor; fiberoptic bronchoscopy; medical intensive care unit

Introduction

Stridor may cause reintubation due to upper airway obstruction, which is a risk factor for patient mortality and morbidity [1,2]. The incidence of stridor and hoarseness after extubation ranges from %1.5 to %26.3 [3]. Studies have shown that female gender, long-term intubation status, increased number of intubation attempts, younger age, trauma, and being hospitalized in the ICU (Intensive care unit) are risk factors for the development of laryngeal edema stridor after extubation [4-7]. OFTP (Obstructive fibrinous tracheal pseudomembrane) is a rare and little-known complication of endotracheal intubation that presents with stridor. In this case, OFTP in the lower line of the vocal cords and stridor, which are rarely seen during short-term intubation, are presented. Written informed consent was obtained from the patient for the case report.

Case report:

A 42-year-old female patient was admitted to the Anesthesia ICU in the postoperative period after McKeown esophagectomy operation by the general surgery clinic due to esophageal squamous cell carcinoma. After the routine examinations of the patient who was followed up with

right lung tube thoracostomy and arterial blood gas detection, sedative agents were discontinued and spontaneous breathing trials were performed. The patient was extubated on the 5th postoperative day without any problems. After extubation, the patient was followed up with an 8L/min oxygen mask with SaO₂ %95 and in the arterial blood gas, pH: 7.48 PaO₂: 60 mmHg PaCO₂: 36 mmHg HCO₃: 26 mmol/L SaO₂: %94. It was observed that approximately 48 hours after the patient's extubation, respiratory distress was observed, and accessory respiratory muscles were involved in respiration. The arterial blood gas of the patient whose respiratory rate was 20-22/min, showed pH: 7.52 PaCO₂: 41 mmHg PaO₂: 58 mmHg HCO₃: 33 mmol/L, SaO₂: %94. Intravenous 1mg/kg methylprednisolone, inhaler beta-2 agonist and inhaler steroid treatment was started to the patient. However, in the follow-up, the patient's oxygen demand and respiratory distress increased significantly despite medical treatment, and thorax computed tomography was performed. Computed tomography sections showed a lesion that could be a pseudomembrane extending towards the lumen, narrowing the tracheal lumen by more than 50%, just below the vocal cords (Figure 1).

FOB (Fiberoptic bronchoscopy procedure) was planned for the patient in the presence of current clinical and radiological findings. The respiratory rate of the patient was 28-

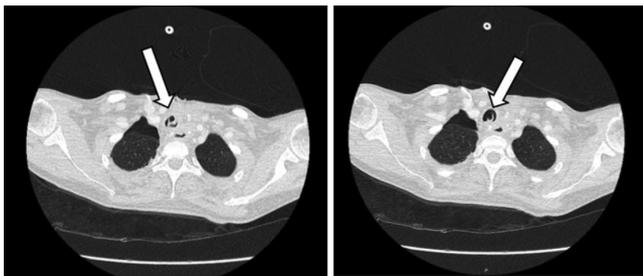


Figure 1: Pseudomembrane tissue in computed tomography

30/min, and the patient had significant dyspnea. The arterial blood gas of the patient showed that pH: 7.24 PaCO₂: 79 mmHg PaO₂: 72 mmHg HCO₃: 27mmol/L SaO₂: %88. There was a change in consciousness of the patient and elective intubation was decided. During the intubation of the patient with the intubation tube numbered 8.0 under the video laryngoscope, a yellow-grayish hard lesion was seen in the camera of the laryngoscope located just below the vocal cords, which prevented the advancement of the intubation tube and narrowed the trachea (Figure 2).

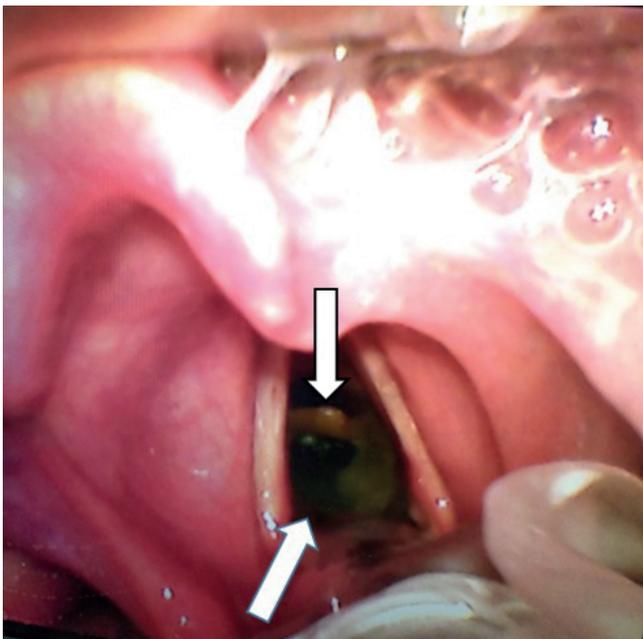


Figure 2: On the video laryngoscope camera, the appearance of a yellow-grayish hard pseudomembrane narrowing the trachea in the posterior location just below the vocal cords

The patient was intubated with the numbered 7.0 intubation tube. In order for the existing lesion not to progress to the lower part of the trachea, the balloon of the tube was inflated just below the vocal cords, and mechanical ventilation was supported. In the chest X-ray taken after intubation, it was observed that the left lung was total atelectatic (Figure 3).

In the FOB performed with the chest diseases clinic, intense purulent secretion and plug in the left lung bronchial structures were seen and aspirated. The lesion under the vocal cord was tried to be aspirated by FOB. Forceps were used



Figure 3: A/P (Antero-Posterior) chest X-ray on the left, atelectasis

to remove the lesion, which had a hardness and consistency that could not be aspirated. It was decided to terminate the FOB procedure and the patient was taken to the rigid bronchoscopy. With the rigid bronchoscopy, 5x1 cm lesion was excised from just below the vocal cord and sent to pathology (Figure 4,5).



Figure 4: Image of pseudomembrane adhered to the posterior wall of the trachea in rigid bronchoscopy

Pathological examination revealed inflamed fibrin tissue and squamous cell debris. After the procedures, in the chest X-ray of the patient whose hypoxia and hypercarbia had resolved, it was observed that the atelectatic area was totally opened (Figure 6). The patient who was extubated, was fol-

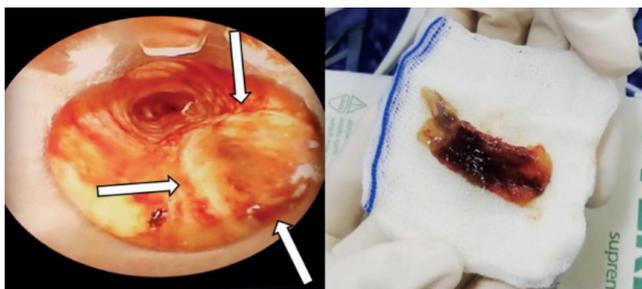


Figure 5: The appearance of the lesion after removal

lowed up under 4L/min simple oxygen mask treatment and transported to the service. After the follow-ups in the service, the patient was discharged without any problem.

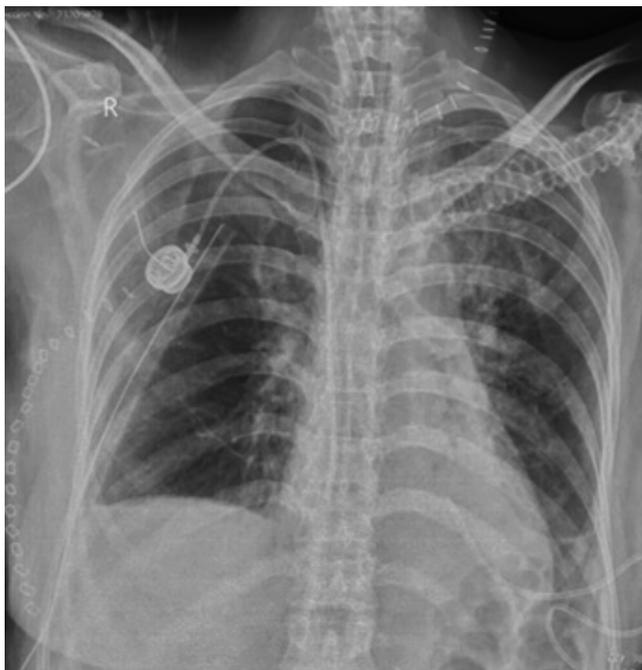


Figure 6: A/P chest X-ray taken after removal of the lesion

Discussion

Stridor after extubation are generally clinical signs that may indicate laryngeal edema or vocal cord injury due to intubation. Stridor may cause reintubation due to upper airway obstruction, with stridor after extubation ranges from %35 to %69 [8,9]. In our case, after esophageal surgery, the patient was extubated in the intensive care unit as a result of suitable extubation conditions and stridor developed after extubation.

The stridor clinic of OFTP is a rare and little-known complication of endotracheal intubation. It was first reported in 1981 [10]. Although chest radiography and tomography are used in the diagnosis and treatment of OFTP, bronchoscopy is often preferred. In one of systematic review; rigid bronchoscopy was used in %46.3 of cases and FOB was used in %31.5 of cases in the treatment of 53 cases with OFTP

development. While rigid bronchoscopy was used more frequently in adults (%56.4) to relieve airway obstruction, FOB was used more frequently in pediatric cases (%60) [11]. Lins et al. reported the use of bronchoscopy in 22 of 24 cases diagnosed with OFTP (rigid bronchoscopy in 18 cases, FOB in 4 cases) [12]. In one of the review where 58 cases with signs of obstruction due to tracheal pseudomembrane were reported after extubation between 1981 and 2015, it was found that rigid bronchoscopy was used in 31 cases and FOB was used in 18 cases [13]. Kang et al. primarily used FOB for the removal of the lesion in the case of OFTP they reported, and after their failure, they removed the lesion with rigid bronchoscopy [14]. In our case, we primarily used FOB, but we failed because the pseudomembrane was highly adherent to the tracheal wall.

Then, we ensured the complete removal of the pseudomembrane with rigid bronchoscopy. OFTP is thought to represent the first step in a process that can lead to tracheal stenosis and is caused by ischemic injury from cuff pressure. Despite the use of a low-pressure high-volume cuff with intensive monitoring of cuff pressure in our case, we have no explanation for why OFTP occurs. We have routinely used high-volume, low-pressure endotracheal tubes for many years. After the endotracheal tube is in place, the cuff is inflated with enough air to reach a leak-free spot. Following placement of the endotracheal tube, the cuff pressure is adjusted to less than 25 cm H₂O by controlling the amount of cuff leakage, as excessive cuff pressure can cause ischemia of the tracheal mucosa. The concern with increasing cuff pressure above this threshold is that mucosal blood flow is compromised, eventually resulting in subglottic stenosis. Cuff pressure is checked daily. In our case, OFTP developed at a level that endangered airway safety even during a short intubation period. It was determined that the pathology causing stridor was a pseudomembrane consisting of inflamed fibrin tissue and squamous cell fragments at the subglottic level. The lesion, which caused more than %50 intraluminal stenosis in the trachea, resulted in reintubation by causing significant dyspnea in the patient. Due to the current secretions, left lung atelectasis was added to the clinical situation in the patient. In conclusion, OFTP, a rare but potentially life-threatening complication of endotracheal intubation, requires rapid diagnosis (FOB) and treatment (usually rigid bronchoscopy). It is thought that avoiding long-term intubation of patients, avoiding high cuff pressure levels during intubation, spontaneous breathing trials and effective secretion excretion will reduce the mechanical obstruction caused by post-extubation secretions.

Ethics approval and consent to participate: The approval has been received.

Consent for publication: The approval has been received

Availability of data and materials: *The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.*

Competing interests: *The authors declare that they have no competing interests”*

Funding: *None*

Authors’ contributions: *Damla Ernur has significant contributions to the design of the work and the acquisition, analysis or interpretation of data for the study, final approval of the version to be published. She is responsible for all aspects of the work to ensure that questions regarding the accuracy or completeness of any part of the work are properly investigated and resolved.*

Ali Necati Gökmen has significant contributions to the design of the work and the acquisition, analysis or interpretation of data for the study, final approval of the version to be published. He is responsible for all aspects of the work to ensure that questions regarding the accuracy or completeness of any part of the work are properly investigated and resolved. All authors read and approved the final manuscript.

Acknowledgements: *We thank the anesthesia intensive care team for their support in treatment of the case.*

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