Hypoxic Respiratory Failure Further Complicated During Airway Exchange Catheter Placement

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Abstract

Introduction: An airway exchange catheter is a hollow-lumen tube able to deliver oxygen and maintain access to a difficult endotracheal airway. This case report demonstrates an undocumented complication associated with an airway exchange catheter and jet ventilation, particularly in a patient with reduced airway diameter due to thick endotracheal secretions. Due to the frequent use of airway exchange catheters in the intensive care unit, this report highlights an adverse event of bilateral pneumothoraces that can be encountered by clinicians. Case Presentation: This case report describes a 24-year-old female with severe adult respiratory distress syndrome and thick endotracheal secretions whose hospital course was complicated by bilateral pneumothoraces resulting from the use of an airway exchange catheter connected to jet ventilation. During the exchange, the catheter occluded the narrowed endotracheal tube to create a one-way valve that led to excessive lung inflation. Conclusion: Airway exchange catheters used with jet ventilation in a patient with a narrowed endotracheal tube and reduced lung compliance have the potential risk of causing a pneumothorax. Clinicians should avoid temporary concomitant oxygenation via jet ventilation in patients with these findings and reserve the use of airway exchange catheters for difficult airways.

Keywords: airway exchange catheter, pneumothorax, jet ventilation

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Introduction

The airway exchange catheter (AEC) is an essential component of the complicated airway algorithm. Due to its ability to deliver supplemental oxygen via an artificial lumen while maintaining access to the existing airway, the AEC is frequently used in patients who are potentially difficult to reintubate or have an existing endotracheal tube (ETT) requiring replacement [1]. Because reintubation rates often exceed 19%, an AEC is frequently used in the intensive care unit (ICU) [2–4]. Despite the frequent use of AECs and ETT exchangers, few data support their use or document their associated adverse events. In this case, we illustrate a never-before documented complication associated with AEC use.

Case Presentation

An otherwise healthy 24-year-old female marathon runner presented to University of Maryland Medical Center, a tertiary care teaching hospital in Maryland, USA, with acute hypoxic respiratory failure after being found unresponsive by her fiancé. In the week before admission, she experienced two episodes of non-bloody vomiting, fever, and myalgias. On arrival at the hospital, her trachea was intubated with a 7.5 mm ETT for hypoxemia and airway protection. A computed tomography angiography (CTA) scan of the chest was negative for suspected pulmonary embolism but showed left lower lobe dense consolidation, and bilateral patchy infiltrates throughout all other lobes. Laboratory studies, on admission, were significant for se-
rum sodium of 115 milliequivalents per litre (mEq/L) and a white blood cell count of 17,100 cells per microliter (μL). She was started on piperacillin-tazobactam (Pfizer, NY, USA) 3.375 g IV every six hours for suspected pneumonia. This was subsequently switched to vancomycin (Pfizer, NY, USA) 750 mg intravenously, every eight hours, and cefepime (Pfizer, NY, USA) 1 g intravenously twice daily the following day. Micafungin (Astellas Pharma US, Inc., IL, USA) 100 mg daily, intravenously, was added two days post-admission. Four days post-admission, the patient’s hypoxemia worsened with oxygen saturation (SpO\textsubscript{2}) of 60-70% despite high positive end-expiratory pressure (PEEP), a maximal fraction of inspired oxygen (FiO\textsubscript{2}), and deep sedation using propofol (Pfizer, NY, USA) and fentanyl drip (Akorn Inc., Lake Forest, IL). Neuromuscular blockade was initiated with vecuronium (Pfizer, NY, USA), and she was urgently transferred to the hospital’s medical ICU for further management of severe acute respiratory distress syndrome (ARDS).

On arrival at the ICU, the patient was ventilated on a pressure-regulated volume control mode with a set respiratory rate of 24 breaths per minute, tidal volume of 300 millilitres (mL), PEEP of 14 centimetres of water (cm H\textsubscript{2}O), and FiO\textsubscript{2} of 100%. Arterial blood gas revealed severe ARDS with a PaO\textsubscript{2}/FiO\textsubscript{2} ratio of 64. She was given a continuous infusion of fentanyl and propofol, which were titrated for adequate sedation and paralyzed with vecuronium. For empiric coverage of legionella and influenza, in addition to the already prescribed antimicrobial regimen, moxifloxacin ( Bayer AG, Leverkusen, Germany) 400 mg intravenously, every 24 hours, and oseltamivir (Hoffman-La Roche, Basel, Switzerland) 75 mg twice daily for five days, were added. A repeat chest CT completed seventy-four hours after her admission from the outside hospital revealed multifocal pneumonia with ground-glass opacities involving all five lobes (Figure 1a).

Because attempts to pass an endotracheal suction catheter were met with resistance and ventilator findings were also consistent with increased airway resistance, a flexible bronchoscopic examination was performed. The inspection identified a large, circumferential mucus concretion within the mid-ETT, but the procedure was quickly aborted due to a severe oxygen desaturation that rapidly resolved upon withdrawal of the bronchoscope.

The patient’s partially occluded ETT necessitated a tube change, but, due to her tenuous respiratory status, the decision was made to utilize an appropriately sized Cook® [Cook Medical; Bloomington, IN] AEC to oxygenate her during the procedure (Figure 1b).

After AEC placement in the ETT, supplemental oxygen was delivered at a rate of 15 litres per minute (L/min) and 50 pounds per square inch (PSI) of pressure. Despite encountering mild resistance at the area of the hardened secretions, the inserted AEC was placed beyond the partial obstruction to a depth of 26 centimetres (cm) measured from the patient’s lip. Within five seconds of its passage, the patient’s SpO\textsubscript{2} decreased, and subcutaneous emphysema was noted at the base of the neck. The AEC was immediately withdrawn, and mechanical ventilation was reinitiated through the existing ETT.

Approximately ten seconds after the initial desaturation, the patient’s hypoxia worsened, and she became bradycardic and pulseless. Bag-valve mask ventilation was initiated via her ETT, and she required fifteen seconds of chest compressions before obtaining the return of spontaneous circulation (ROSC). Contemporaneous to the timing of ROSC, bilateral needle decompressions resulted in instant rushes of air exiting the pleural spaces. Bilateral tube thoracotomies were performed within one minute of ROSC, which resolved the hemodynamic instability (Figure 2a). A prompt bronchoscopic inspection did not reveal any substantial airway damage during AEC insertion. The patient remained persistently hypoxic despite bag-valve-mask ventilation and attempts at various ventilator settings. Venovenous extracorporeal membrane oxygenation (V-V ECMO) was successfully initiated at the bedside via her right internal jugular and right femoral veins. She was subsequently transferred to the cardiac surgery ICU for continued ECMO management.

**Figure 1.** a. Admission chest CT (coronal view) showing multilobar consolidation and areas of ground-glass opacification in all five lobes. b. Cook® Airway Exchange Catheter & Rapi-Fit® adapter with 15-mm connector for oxygenation during exchange procedures.
The patient was decannulated on day three of her hospitalization, and the bilateral chest tubes were removed on day nine post-admission. (Figure 2b).

Her final diagnosis was severe ARDS due to culture-negative multilobar pneumonia, and she was subsequently discharged home eleven days post-admission to the hospital. Approximately one month following her discharge, she was seen at a local beer festival in good health.

**DISCUSSION**

Trauma to the tracheobronchial tree may occur when the AEC passes through the side port (Murphy’s eye) of the ETT [5]. The resulting damage can cause tracheal laceration, perforation, and pneumothorax, and the risk of this type of trauma is inversely proportional to the diameter of the AEC [5,6]. Reduced ondiameter of the ETT, high-pressure O₂ delivery (e.g., >25 psi), and excess inspiratory time (>1.25 sec) can all lead to air trapping, lung overinflation, and pneumothorax [5–9]. Additionally, jet ventilation via an AEC, particularly when positioned beyond the carina, can overinflate a single lung to cause a unilateral barotrauma- and volutrauma-induced pneumothorax [8]. Given that the patient had bilateral pneumothoraces and no evidence of tracheobronchial trauma on bronchoscopy, the AEC was unlikely to have been placed at an improper depth or location. Most likely, the AEC became lodged in the middle of the large, circumferential, flow-limiting concretion in the ETT tube, impeding the route of oxygen backflow. This obstruction effectively created a one-way valve that trapped 15 L/min of O₂ flowing through the AEC at 50 PSI in the patient’s lungs. With only three to five seconds at this flow rate, the patient received 750-1250 mL of oxygen, which, coupled with her ARDS-induced poor lung compliance, resulted in bilateral pneumothoraces.

The AEC maintains an important role in the complicated airway algorithm; however, one must be aware of potentially fatal iatrogenic events such as a tension pneumothorax [10]. In one case series of forty-five patients who underwent extubation by jet ventilation (15-50 psi) through an AEC, 11% of patients developed barotrauma [7,10]. Awareness of this complication and prompt recognition of the clinical signs and symptoms of a pneumothorax are essential when utilizing the AEC. Attention to the insertion depth is critical, and the presence of any airway resistance warrants prompt retraction of the exchanger by a few centimetres [11]. In one case series of 40 adult ICU patients with potentially difficult airways, O₂ insufflation of 2-8 L/min through an AEC during extubation did not lead to any pneumothoraces or hypoxemia (SpO₂ <90%).

![Fig. 2. a. Chest x-ray AP view following bilateral chest tube placement that shows bilateral patchy opacification with moderate right pneumothorax and diffuse subcutaneous emphysema along the thorax and neck. b. Chest x-ray AP view, day 9 of hospitalization after removal of bilateral chest tubes.](image-url)
In summary, the use of an airway exchange catheter was thought to be optimal due to the patient’s severe hypoxic respiratory failure from ARDS. It was believed that the patient would benefit from an airway exchange catheter with positive pressure ventilation to prevent worsening hypoxia and atelectasis. However, the AEC very likely completely occluded the narrowed mid-ETT to create a one-way valve through which high-flow $O_2$ excessively inflated the patient’s poorly compliant lungs, causing bilateral pneumothoraces.

**Conclusion**

This case report is considered to be the first report to describe the development of bilateral pneumothoraces from delivering jet ventilation through an AEC. The case particularly highlights the risk of placing an AEC through a narrowed ETT, which can cause a one-way valve when providing supplemental air. Given the frequent use of AECs, clinicians should avoid concomitant $O_2$ administration in patients with significant ETT narrowing, understanding that excessively inflating poorly compliant lungs can cause a pneumothorax. Moreover, an AEC should be reserved for those patients with a high-risk airway, requiring ETT exchange or extubation, and not as a means to improve oxygenation in those with transient isolated pulmonary pathology.

**Abbreviation List**

AEC- airway exchange catheter  
CT- computed tomography  
CTA- computed tomography angiography  
ETT- endotracheal tube  
FiO2- fraction of inspired oxygen  
ICU- intensive care unit  
PSI- pounds per square inch  
ROSC- return of spontaneous circulation  
VV ECMO- venovenous extracorporeal membrane oxygenation

**CONFLICT OF INTEREST**

None to declare.

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