PULMONARY EDEMA FOLLOWING TRACHEAL STENT IMPLANTATION IN A PATIENT WITH AIRWAY OBSTRUCTION

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Abstract

We report the case of a 72-year-old man with a soft tissue mass that was located in the right mediastinum and compressed the tracheal lumen externally, causing airway obstruction. He presented with severe dyspnea and disturbed consciousness. Tracheal stent implantation was performed for relieving the obstructed airway. After the procedure, his breathing was assisted with a T-piece. A few minutes later, he developed oxygen desaturation, productive cough with pink foamy sputum, and bilateral basal crepitations. Therefore, acute pulmonary edema was suspected. Type II postobstructive pulmonary edema was diagnosed after the other types of pulmonary edema were ruled out. Awareness of the cause and mechanism of type II postobstructive pulmonary edema could facilitate timely and appropriate postoperative care such as progressively weaning the patient from mechanical ventilator by decreasing positive end-expiratory pressure gradually.

Key words: Airway obstruction, Tracheal stent implantation, Postobstructive pulmonary edema

Introduction

Airway obstruction is an emergent condition, which can result in hypoxia and even death. The treatment strategy of airway obstruction aims at removing the cause of obstruction and maintaining patency of the airway. However, postobstructive pulmonary edema (POPE) is known to occur after the treatment of upper airway obstruction.1 If pulmonary edema is left untreated, it can lead to fatal hypoxia. We observed this complication in a patient who underwent tracheal stent implantation for the relief of a critical endotracheal occlusion; no subsequent positive pressure ventilation was given to the patient after the stent implantation. Recognizing this potential complication will help identify and possibly avert its occurrence.

Case report

We report the case of a 72-year-old man from Taiwan who experienced the lump sensation and hoarseness in the throat, for several months.
before admission. He was previously healthy without any history of systemic illness or major surgery. Further, he developed blood-tinged sputum and dysphagia for 7 days. He was transferred to our emergency room for dyspnea and tightness in the chest that lasted for 3 days and exacerbated during that time. In the emergency room, arterial blood gas analysis showed the following results: $\text{pH} = 6.679$, $\text{PaCO}_2 = 82 \text{ mm Hg}$, $\text{HCO}_3^- = 13.4 \text{ mEq/L}$, $\text{PaO}_2 = 69.6 \text{ mm Hg}$, and $\text{SaO}_2 = 83.1\%$. The patient was not intubated because his family refused endotracheal intubation. Therefore, ventilation was assisted by biphasic positive airway pressure (BiPAP) ventilation for impending respiratory failure. Computed tomography of the chest showed a huge lobulated soft tissue mass (7 cm on an axial plane) with a large area of central necrosis and trapped air bubbles, located at the right mediastinum (the right aspect of esophagus), which markedly compressed the tracheal lumen and esophagus (with leftward displacement of the trachea and esophagus) and invaded the posterior wall of the trachea (Fig. 1).

About 48 h later, the patient was shifted to the operating room. He still showed disturbed consciousness, and pulse oximeter showed $\text{SaO}_2$ of 91% with BiPAP ventilation. An arterial line was inserted, and he was anesthetized with an incremented dose (50 $\mu$g) of fentanyl via awake endotracheal intubation performed using fiberoptic bronchoscopy.

After intubation, he was administered 30 mg rocuronium, and anesthetic state was maintained with intermittent 2% sevoflurane in 1 L/min oxygen and intravenous propofol bolus with a total dose of 60 mg. The fiberoptic bronchoscopy showed a 1.5 cm long protruding tumor located at the posterior membrane of the trachea, 2 cm above the carina. Tracheal stent implantation was performed to regain the patency of the trachea (Fig. 2). The procedure was carried out smoothly, and it lasted for 1 h. The total solution intake was 500 mL of normal saline, and urine output was 100 mL. Arterial blood gas analysis showed the following results: $\text{pH} = 7.334$, $\text{PaCO}_2 = 29.7$ mm Hg, $\text{HCO}_3^- = 13.4 \text{ mEq/L}$, $\text{PaO}_2 = 137.8 \text{ mm Hg}$, and $\text{SaO}_2 = 99.1\%$. Thereafter, the patient was transferred to a postanesthetic room.

In the postanesthetic room, the patient was given 6 L/min oxygen via a T-piece. Ten minutes later, he developed desaturation of oxygen ($\text{SaO}_2$, 70~89%), productive cough with pink foamy sputum, and bilateral basal crepitations. The patient was intubated and transferred to an intensive care unit (ICU) under the impression of pulmonary edema. In the ICU, the chest radiograph showed bilateral ground glass infiltrates (Fig. 3). Echocardiography showed normal cardiac function. The parameters of pulse contour cardiac output (PiCCO) monitoring were as follows: cardiac index (CI) = 2.36 (3.0-5.0) L·min$^{-1}·m^{-2}$, cardiac function index (CFI) = 3.3 (4.5~6.5) L·min$^{-1}$, global end-diastolic volume index (GEDVI) = 696 (680–800) mL·m$^{-2}$, intrathoracic blood volume index (ITBVI) = 870 (850–1000) mL·m$^{-2}$, stroke volume variation (SVV) = 27% (<10%), pulse
pressure variation (PVV) = 23% (<10%), systemic vascular resistance index (SVRI) = 1862 (1700-2400) dynes · s · cm⁻⁵ · m⁻², pulmonary vascular permeability index (PVPI) = 5.4 (1-3), and extra-vascular lung water index (EVLWI) = 19 (3-7) mL · kg⁻¹. In ICU, general supportive treatments were given.

Six days later, the patient died because of complications of pneumonia with septic shock and multiple organ failure.

Discussion

Airway obstruction can be fatal if untreated, and therefore, the main strategy of the treatment is to relieve the obstruction. However, the patient may encounter another critical post-treatment complication, that is, pulmonary edema. In the present case, after tracheal stent implantation was performed to regain tracheal patency, the patient developed sudden oxygen desaturation, productive cough with foamy sputum, and bilateral basal

Fig. 3. Chest radiograph obtained before (A) and after (B) the tracheal stent implantation. (B) Bilateral ground glass infiltrate, which is consistent with pulmonary edema, can be seen in the aerated left lung.
crepitations, and bilateral ground glass infiltrate was seen in chest radiograph. The patient had no evidence of heart disease. In addition, the echocardiograph and PiCCO monitor showed no massive heart failure (CI = 2.36 L \cdot \text{min}^{-1} \cdot \text{m}^{-2}, CFI = 3.3 \text{ L} \cdot \text{min}^{-1}). Further, the PiCCO monitor also showed that GEDVI and ITBVI were close to the lower limit of the normal range, while SVV and PVV values were higher than the normal range. These data indicated that there was no fluid overload and even dehydration. Because the patient developed acute pulmonary edema after the tracheal stent was implanted for relieving the obstruction caused by the tumor, other causes can be ruled out; the pulmonary edema most likely arose after the relief of airway obstruction.

Airway obstruction-related pulmonary edema named POPE is divided into 2 types: type I POPE is associated with forceful inspiratory effort in acute airway obstruction, whereas type II occurs after the relief of a partial airway obstruction.\(^2\) In the present case, since the patient was very weak and had disturbed consciousness, there was no forceful inspiratory effort to cause type I POPE. Further, bronchoscopy examination, clinical presentation, and laboratory examination did not show evidence of pulmonary edema just before tracheal stent implantation. Therefore, in this case, the pulmonary edema developed after tracheal stent implantation, and therefore, the diagnosis was of type II POPE.

Type II POPE may develop after adenoidectomy, tonsillectomy, laryngeal mass resection, or reduction of a hypertrophic redundant uvula.\(^5\) The incidence of type II POPE in patients who underwent endotracheal intubation and tracheostomy to relieve airway obstruction is estimated to be approximately 11-12%\(^3\). Type II POPE has been postulated to occur via the following mechanism: airway obstruction reduces the venous return to the thorax during expiration; however, when the obstruction is relieved, the sudden influx of blood to the thorax leads to edema. After the removal of the obstruction, the left ventricular function may not be able to upturn output fittingly, increasing the hydrostatic pressure in the pulmonary circuit and resulting in the formation of edema.\(^4\) According to the aforementioned mechanism, positive pressure mechanical ventilation should have been given to the patient to avoid the sudden influx of blood to the thorax after the obstruction was removed. However, in the present case, the patient did not receive positive pressure mechanical ventilation support, and therefore, type II POPE may have developed. Therefore, this mechanism indicates that after the treatment procedure, the anesthetist should wean the patients with upper airway obstruction from the ventilation by gradually decreasing the positive end-expiratory pressure (PEEP).

Treatment for POPE consists of careful supportive treatments, including mechanical respiratory support and maintaining a negative balance for fluid intake and output. PEEP decreases venous return and improves oxygenation, and therefore, it may be advantageous in treating pulmonary edema. Excess intravenous fluid should be avoided; if necessary, diuretics and invasive hemodynamic monitors such as the PiCCO may be used. The usage of steroids is debatable.\(^5\)

Patients with external tracheal compression by a tumor who undergo tracheal stent implantation for the relief of airway obstruction are at a risk for type II POPE. Awareness of the cause and mechanism of type II POPE could facilitate early identification and appropriate prevention by weaning patients from mechanical ventilation. After upper airway obstruction is relieved, PEEP should be gradually decreased to prevent the development of type II POPE. If pulmonary edema has occurred, therapy with PEEP, oxygen supplementation, and diuretics should be given.

References


氣管支架緩解氣道阻塞後導致肺水腫 - 案例報告

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摘要

七十二歲男性，因右側縱膈腔腫瘤，壓迫氣管，造成呼吸困難，意識改變，因而接受氣管支架置放術。在手術後，病人留置氣管內管，使用T型接管(T-piece)且自發性呼吸，不久之後，病人血氧下降，抽痰發現有粉紅色泡沫痰，兩側聽診有嘔音，因此診斷為急性肺水腫。在排除其他造成肺水腫的原因之後，診斷為第二型氣道阻塞後引致之肺水腫 (postobstructive pulmonary edema type II)。了解第二型氣道阻塞後引致之肺水腫的成因及機轉，將有助於我們更能及時，適當的處理這類病人，例如慢慢調低吐氣末正壓 (positive end expiratory pressure)，讓病人慢慢脫離呼吸器。

關鍵詞：氣道阻塞，氣道阻塞後引致之肺水腫，氣管支架