Upper Airway Obstruction due to Inhalation of a Tracheal T-Tube Resulting in Pulmonary Edema*

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Acute upper airway obstruction may present with pulmonary edema. Following is a report of pulmonary edema secondary to acute upper airway obstruction due to inhalation of a Montgomery tracheal T-tube. The principal factor causing pulmonary edema is the generation of large negative transpulmonary pressures. This may be enhanced by changes in the cardiovascular function due to the Müller maneuver.

(Chest 1992; 102:644-45)

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P_{aCO_2} = \text{arterial carbon dioxide pressure; } P_{aO_2} = \text{arterial oxygen pressure; } pH = \text{negative logarithm of hydrogen ion activity}
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Pulmonary edema may be the presenting symptom of acute upper airway obstruction due to inhalation of foreign bodies. We present a case of pulmonary edema caused by the inhalation of a Montgomery tracheal T-tube.

CASE REPORT

A 65-year-old morbidly obese woman presented to the emergency department complaining of suffocation after allegedly inhaling a Montgomery tracheal T-tube while manipulating it. She has a history of subglottic stenosis secondary to prolonged intubation, diabetes mellitus and chronic obstructive pulmonary disease.

The patient was in acute respiratory distress and cyanotic. Respiratory rate was 10 breaths per minute; pulse, 133 beats per minute; and blood pressure was not obtained due to her obesity. Auscultation of the lungs disclosed bilateral rales, inspiratory stridor and expiratory wheezes. There was jugular venous distension and moderate pitting edema of the lower extremities. Arterial blood gas analysis with an undocumented concentration of oxygen showed a pH value of 7.08; \( P_{aCO_2} \) of 82 mm Hg; and \( P_{aO_2} \) of 86 mm Hg. A 2.5 mm pediatric endotracheal tube was inserted through the tracheostomy and the patient was mechanically ventilated.

A chest x-ray film (Fig 1) showed a correctly placed endotracheal tube and bilateral pulmonary shadows compatible with pulmonary edema but no evidence of a foreign body in the upper airways. Blood gas analysis on mechanical ventilation with a concentration of oxygen of 100 percent showed a pH value of 7.28; \( P_{aCO_2} \), 49 mm Hg; and \( P_{aO_2} \), 140 mm Hg. The respiratory rate was 30 breaths per minute; pulse, 125 beats per minute; and blood pressure, 149/117 mm Hg. She continued to complain of suffocation and insisted the T-tube was in her airways.

A bronchoscopy was performed with a flexible laryngoscope through the tracheostomy and the T-tube was found lodged at the level of the carina. After removing the T-tube (Fig 2) the patient improved significantly and mechanical ventilation was discontinued in a few hours. She was treated judiciously with diuretics and the lung infiltrates progressively cleared. She was discharged from the hospital three days later with the following room air blood gas values: pH, 7.49; \( P_{aCO_2} \), 33 mm Hg; and \( P_{aO_2} \), 71 mm Hg.

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Figure 1. Anteroposterior chest radiograph at presentation demonstrating pulmonary edema.

DISCUSSION

Recently Willms and Shure reviewed 25 cases reported in the English literature up to 1987. The most common cause of upper airway obstruction leading to pulmonary edema is postobstructive laryngospasm. The inhalation of tracheostomy tubes has been reported, although not as a cause of pulmonary edema. We believe ours is the first report of such a case.

Pulmonary edema due to upper airway obstruction appears to be caused by a combination of mechanisms. The principal factor is the generation of large negative transpulmonary pressures due to forceful inspiration against an obstructed airway. The drop in pleural pressure to oppose the increased airway resistance is transmitted to interstitial pressures which are thus lowered, while intravascular pressures are maintained. This favors the transudation of fluid from the capillaries to the interstitium and alveoli. Furthermore, this effect may be enhanced by the changes in cardiovascular function due to the Müller maneuver; left ventricular afterload increases, left ventricular ejection fraction decreases and right ventricular preload increases, all of which favor an increase in left atrial pressure and, therefore, the development of pulmonary edema.

*The authors would like to thank Dr. Shure, Department of Medicine, Boston University Medical Center, for his review of the manuscript.

Figure 2. Montgomery tracheal T-tube recovered from the patient's upper airways (original size, 14 mm).
accumulation of fluid in the interstitium leads to narrowing of small airways causing an increase in airway resistance, which will require greater negative pleural pressures, resulting in a vicious cycle.1,2

The extraluminal limb of the Montgomery tracheal T-tube is designed with ridges and grooves that allow the attachment of a ring washer to prevent posterior displacement of the tube.3,4 Patients should be instructed not to remove this ring washer to avoid the risk of inhalation. On the other hand, the T-tube can be modified for each patient simply by cutting with scissors,3,5 and this may make an arm too short and promote dislodgement.

In summary, the inhalation of foreign bodies may present with pulmonary edema, probably more likely in patients with underlying heart disease and may occur in those with tracheostomy tubes. Management of these patients is best accomplished by removal of the obstruction, adequate oxygenation and the judicious use of diuretics. Montgomery T-tubes are used frequently and physicians should be aware of this potential complication.

ACKNOWLEDGEMENTS: We want to thank Mark Roth, M.D., for referring this patient, and Toribio Flores, M.D., for assisting with the fiberoptic bronchoscopy.

REFERENCES

Pulmonary Vein Obstruction Following Single Lung Transplantation*
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Angiographic documentation after single lung transplant showed pulmonary venous obstruction due to compression by the omental pedicle. Retransplantation is described, and this complication is examined.

(Chest 1992; 102:645-47)

Bronchial anastomotic complications occur with moderate frequency following both single and bilateral lung transplantation and historically were responsible for most of the deaths in the early transplant experience.1 Using a pedicle of gastrocolic omentum to wrap the bronchial anastomosis promotes early revascularization, improves bronchial healing, and protects against dehiscence.2 The improved bronchial healing and decreased incidence of dehiscence that resulted from utilizing the omental wrap ushered in the current era in lung transplantation. However, complications from the omental pedicle do occur, and we present a case where the imaging studies demonstrate pulmonary venous obstruction secondary to omental displacement.

CASE REPORT
A 54-year-old man first noted the onset of significant dyspnea with exertion in 1986, at which time a diagnosis of emphysema was made. His alpha-1 antitrypsin level was normal and he had a 30 pack-year smoking history. Treatment included oral and inhaled bronchodilators, periodic antibiotics, several courses of steroids, and smoking cessation. He noted progressive worsening of his symptoms which severely restricted his performance of daily activities, and eventually he required continuous oxygen therapy. He was unable to work. In May 1989, he was referred to the Washington University Lung Transplant Program. On evaluation, he had an FEV1 of .57 L; FEV1/FVC, 32 percent predicted; and Dco, 5.4 (26 percent predicted). Arterial blood gas values on 3 L O2 showed a pH of 7.41; Pco2, 73.4; and Paco2, 45.5. Coronary angiography showed mild atherosclerotic coronary vessel disease. He underwent left single lung transplantation on July 26, 1989, utilizing a technique which has been previously described.3 Following completion of the bronchial anastomosis, a pedicle of omentum, having been mobilized off the transverse colon down to the level of the hepatic flexure, was passed substernally into the pleural space and wrapped circumferentially around the anastomosis. Several sutures secured the pedicle in place. The arterial and atrial anastomosis were then completed. Cardiopulmonary bypass was not required as oxygenation could be maintained by the native right lung while the left was implanted.

Postoperative Course
The early postoperative course was complicated by hypotension which responded poorly to maximal inotropic support. An intraaortic balloon pump (IABP) was inserted approximately 20 h following

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