Tracheal Rupture Resulting in Life-Threatening Subcutaneous Emphysema

Cynthia J Gries MD and David J Pierson MD FAARC

We present a case of a patient with severe chronic obstructive pulmonary disease who developed dramatic mediastinal and subcutaneous emphysema, without pneumothorax, following a difficult intubation. Misdiagnosis of tracheal rupture as barotrauma from alveolar overdistention initially delayed intervention and caused persistence of subcutaneous emphysema. Despite efforts to minimize tidal volume and airway pressure, the large airway disruption and positive-pressure ventilation resulted in tension subcutaneous emphysema with near-fatal hemodynamic compromise, oliguria, and respiratory acidosis. Decompression with subcutaneous vents immediately reversed the life-threatening circulatory and respiratory compromise and stabilized the patient until surgical correction of the tracheal tear could be accomplished. *Key words: tracheal laceration, difficult intubation, mechanical ventilation, pneumomediastinum, subcutaneous emphysema, barotrauma, chronic obstructive pulmonary disease, COPD, intrinsic positive end-expiratory pressure.* [Respir Care 2007; 52(2):191–195. © 2007 Daedalus Enterprises]

Introduction

Subcutaneous emphysema in patients receiving positive-pressure mechanical ventilation is generally considered a benign condition that does not require specific measures to vent the subcutaneous gas.[1,2] Rarely, however, this form of extra-alveolar air can be a threat to life requiring immediate intervention.[3,4] We present a patient who developed hypotension, oliguria, and increased airway pressure that prevented effective ventilation, due to massive subcutaneous emphysema. With incision of the skin and placement of bilateral thoracostomy tubes, a dramatic amount of subcutaneous air was released, with decreased airway pressure, increased urine output, and improved hemodynamics. This patient proved to have a tracheal laceration secondary to repeated unsuccessful in-

tubation attempts in the field. Tracheal rupture was initially misdiagnosed as barotrauma in a mechanically ventilated patient with severe chronic obstructive pulmonary disease (COPD).

Case Summary

A 64-year-old woman presented with a 2-week history of increased dyspnea, cough, and yellow sputum production. The patient was initially treated with antibiotics for a COPD exacerbation, but developed progressive respiratory distress. Paramedics found her tachypneic, with a pulse-oximetry-measured oxyhemoglobin saturation of 80%. After several unsuccessful intubation attempts in the field, a 7.0-mm endotracheal tube was placed without difficulty in the emergency room at an outside hospital. Immediately after intubation the patient was noted to develop neck and facial swelling, and she was transferred to our institution for further care.

The patient had known severe COPD, with a history of multiple previous intubations for respiratory distress. Her pulmonary function tests from one year prior to this admission revealed a forced expiratory volume in the first second (FEV_1) of 0.46 L (20% of predicted), forced vital capacity (FVC) of 1.08 L, and FEV_1/FVC of 0.43. At home she was on oxygen (2 L/min) and nocturnal contin-

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Fig. 1. Supine anteroposterior chest radiograph taken in the emergency department, showing extensive mediastinal and subcutaneous emphysema. An endotracheal tube is present, with its distal tip in the mid-trachea.

uous positive airway pressure for obstructive sleep apnea. She also had a history of type 2 diabetes.

On admission the patient was intubated, ventilated, and sedated. She was obese, with a body mass index of 34 kg/m^2, height 160 cm, and weight 86.7 kg. She was afebrile, with a heart rate of 104 beats/min, blood pressure of 118/86 mm Hg, and respiratory rate of 12 breaths/min. She had marked subcutaneous emphysema of the chest, breasts, neck, and face. Her breath sounds were distant, without crackles or wheezes. Her heart sounds were barely audible. The remainder of the examination was unremarkable.

On admission, her arterial blood gas values while receiving a 0.50 fraction of inspired oxygen (F_{IO_2}) were pH 7.32, P_{aCO_2} 64 mm Hg, and P_{aO_2} 159 mm Hg. The hematocrit was 40% and the white-blood-cell count was 13.7×10^3 cells/μL. Other blood chemistries, including blood urea nitrogen and creatinine, were normal. A chest radiograph (Fig. 1) and chest computed tomogram (Fig. 2) obtained in the emergency department showed pneumomediastinum and massive subcutaneous emphysema in the anterior chest wall and breasts, but no pneumothorax.

Her respiratory distress was thought to be secondary to a COPD exacerbation, and the patient was treated with intravenous corticosteroids, antibiotics, albuterol, and ipratropium bromide via aerosol. Pneumomediastinum and subcutaneous emphysema were attributed to barotrauma from alveolar rupture and overinflation during mechanical ventilation before, during, and after intubation.

The patient was transferred from the outside hospital on volume-assist/control ventilation at 12 breaths/min, tidal volume (V_T) 12 mL/kg of predicted body weight, peak inspiratory flow 56 L/min, F_{IO_2} 0.50, and positive end-expiratory pressure (PEEP) 5 cm H$_2$O. Measured total

PEEP was initially 20 cm H$_2$O. To reduce air-trapping and intrinsic PEEP, the V_T was reduced to 6 mL/kg of predicted body weight (312 mL), with a minute ventilation of 3.7 L/min. Arterial blood gas values while on F_{IO_2} of 0.40 and PEEP 5 cm H$_2$O were pH 7.18, P_{aCO_2} 105 mm Hg, and P_{aO_2} 83 mm Hg.

Total PEEP initially fell to 14 cm H$_2$O on the reduced V_T, but subsequently increased to 28 cm H$_2$O. As her respiratory status declined, the patient was sedated and paralyzed. An infusion of sodium bicarbonate was started to compensate for her respiratory acidosis. Even after decreasing the peak inspiratory flow from 72 L/min to 50 L/min, peak inspiratory pressure was measured as high as 118 cm H$_2$O. Aggressive fluid resuscitation was performed, but despite a normal central venous pressure, the patient's systolic blood pressure decreased to 80–90 mm Hg and urine output fell from 50–100 mL/h to < 10 mL/h. Physical examination revealed progression of subcutaneous emphysema that was tense to palpation and extended from her face to thighs. Arterial blood gas analysis while on F_{IO_2} of 1.0 showed pH 7.08, P_{aCO_2} 115 mm Hg, and P_{aO_2} 265 mm Hg. A repeat portable chest radiograph showed further extension of the subcutaneous emphysema, but no pneumothorax (Fig. 3).

Bronchoscopy to evaluate the possibility of tracheobronchial injury was considered, but was deferred because of the patient's severe hemodynamic and respiratory instability. At this point, thoracostomy tubes were inserted bilaterally. Initial incision of the skin prior to entering the pleural space resulted in a dramatic release of subcutaneous air. Intermittent air leaks from the chest tubes were subsequently noted. Without any ventilator adjustments, peak airway pressure fell to 50 cm H$_2$O, with end-inspiratory static pressure of 27 cm H$_2$O and total PEEP of 14 cm H$_2$O. The systemic blood pressure immediately increased to 116/58 mm Hg and the patient's urine output subsequently increased to 45 mL/h. A repeat arterial blood gas analysis showed pH 7.22, P_{aCO_2} 86 mm Hg, and P_{aO_2} 181 mm Hg, while on F_{IO_2} of 0.50.

Despite this initial improvement, the patient continued to have respiratory instability, with a total PEEP of 25 cm H$_2$O and severe respiratory acidosis. The patient was sedated and intermittently paralyzed as she was aggressively managed for a COPD exacerbation. Although the signs of hemodynamic compromise did not recur, the patient continued to have massive subcutaneous emphysema (Fig. 4) and air leaks through the chest tubes. Pneumothoraces were now noted on chest radiograph. In the diagnostic evaluation to explain her lack of improvement, a further review of the initial chest computed tomogram revealed a discontinuity in the lower posterior trachea (Fig. 5). Bronchoscopy confirmed the presence of a 2–3-cm posterior tracheal tear (Fig. 6). The patient was taken to the operating room, where surgical repair was accomplished
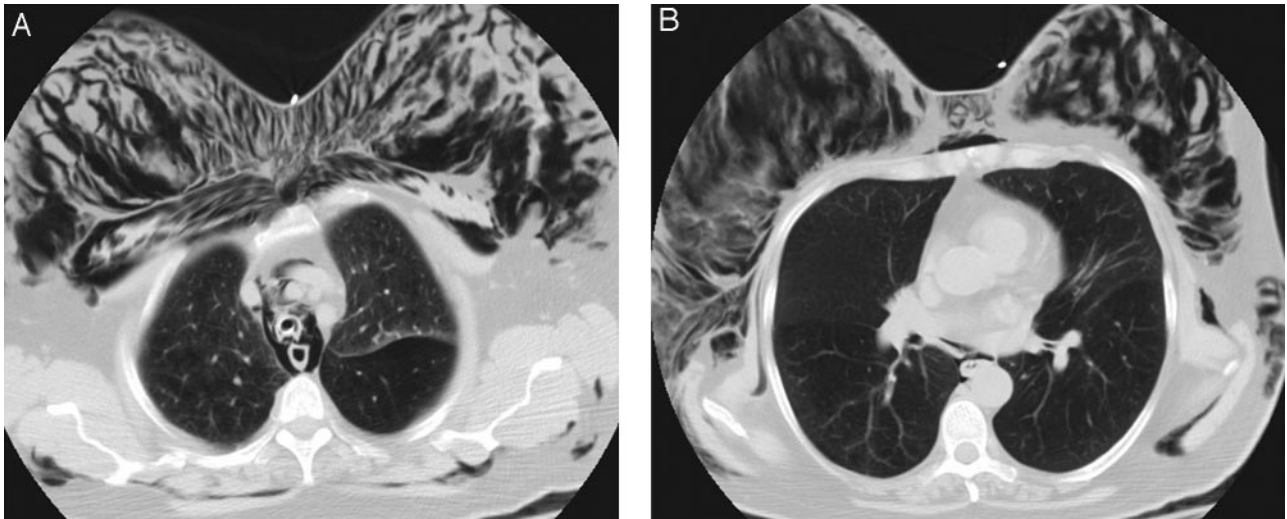
Fig. 2. Chest computed tomogram obtained in the emergency department. Left: Representative slice through the upper lobes, showing extensive mediastinal and subcutaneous emphysema. The trachea (with the distal end of the endotracheal tube) and esophagus are outlined by gas. Right: Representative slice through the lower lobes, showing extensive, mainly anterior, subcutaneous emphysema. There is no pneumothorax.

Fig. 3. Supine anteroposterior portable chest radiograph taken in the intensive care unit several hours after admission. Gas has spread more extensively throughout the soft tissues of the chest and neck, but no pneumothorax is evident.

via a right posterolateral thoracotomy and direct closure of the tracheal defect.

Postoperatively there were no further air leaks through the chest tubes, and the subcutaneous emphysema gradually resolved. Despite clinical improvement, however, persistent dynamic hyperinflation and intrinsic PEEP prevented discontinuation of ventilatory support. The patient was transferred on hospital day 40 to a long-term acute care facility where she was eventually liberated from the ventilator and discharged home.

Discussion

We present a case of a patient with severe COPD who developed massive subcutaneous emphysema that resulted in life-threatening hemodynamic instability and respiratory compromise that resolved after decompression with skin incision. These complications resulted from a tracheal tear. The symptoms were initially misdiagnosed as barotrauma secondary to alveolar rupture. Although most instances of pneumomediastinum and subcutaneous emphysema are physiologically unimportant, these forms of extra-alveolar air can occasionally pose a threat to life.

Massive subcutaneous emphysema can be life-threatening if it involves the deeper tissues of the thoracic outlet, chest, and abdominal wall. Compression of the thoracic outlet can result in airflow obstruction, decreased venous return to the heart, and poor perfusion to the head and neck.[5] Rigidity of the chest wall secondary to subcutaneous air restricts full expansion of the lungs and can lead to high airway pressure, severe respiratory acidosis, and ventilatory failure.

When these potentially fatal complications result from subcutaneous emphysema, surgical intervention seems indicated. Previous case reports have described successful treatment with tracheostomy, infraclavicular "blow holes," skin incisions, and subcutaneous drains.[6-8] In our case, a dramatic resolution in circulatory collapse and ventilatory failure occurred after bilateral skin incisions were made.

As illustrated by this case, tracheal rupture can lead to severe morbidity and possible mortality. Tracheal rupture classically presents with massive subcutaneous air, pneumomediastinum, pneumopericardium, and/or pneumotho-
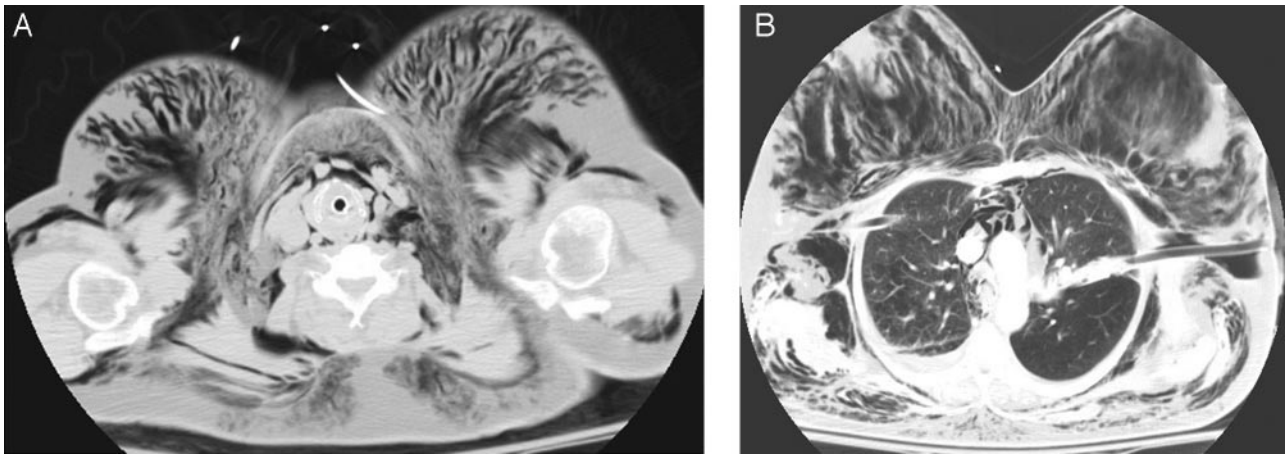
Fig. 4. Chest computed tomogram obtained on the 9th hospital day. Left: Representative slice from the level of the larynx, showing continued pneumomediastinum plus extensive dissection by gas, throughout the deep and superficial soft tissues, both anteriorly and posteriorly. Right: Representative slice at the level of the aortic arch, showing pneumomediastinum, extensive soft-tissue gas in all areas, and bilateral thoracostomy tubes.

Fig. 5. Chest computed tomogram obtained in the emergency department on the night of admission. This slice at the level of the aortic arch shows a discontinuity in the posterior tracheal wall (arrow), with partial extension of the esophagus into the opening.
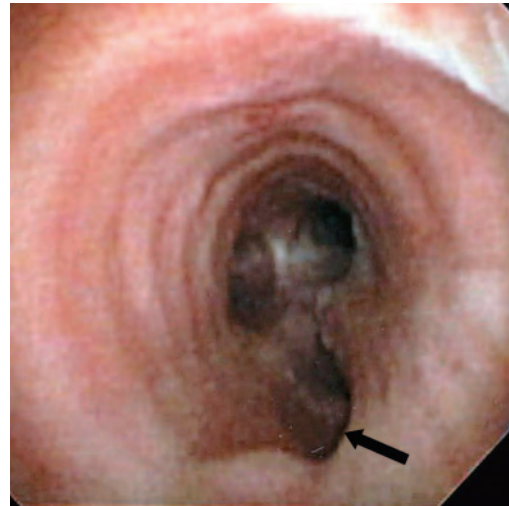

Fig. 6. Photograph of the mid-trachea, obtained during bronchoscopy, showing a 2–3-cm rent in the posterior tracheal wall (arrow), splayed widely open during inspiration.

rax. Symptoms from the tear itself can include cough, dyspnea, hemoptysis, and vocal change.[9] If diagnosis is delayed, the patient can also develop mediastinitis and sepsis from the tracheal injury.[10]

Patients who are female, older, have short stature, or have a history of corticosteroid use, COPD, or tracheal anatomic abnormalities are predisposed to tracheal injury during endotracheal intubation.[11] Although tracheal rupture can occur in uneventful intubations, technical problems such as multiple intubation attempts, overinflation of the cuff, malposition of the tube, use of a stylet, use of a double-lumen tube, or improper tube size can also increase the risk of this complication.[11]

Because tracheobronchial rupture after intubation is a rare occurrence, it is frequently misdiagnosed as barotrauma. In our case, we believed that the initial management with high V_T and high total PEEP led to alveolar rupture and subcutaneous emphysema. Although our patient was predisposed to barotrauma, she also had risk factors for tracheobronchial rupture, including short stature, obesity, steroid use, and multiple intubation attempts in the field. In addition, the worsening of subcutaneous emphysema and the presence of air leaks without evidence of pneumothorax suggested that tracheobronchial rupture was a possibility. Although bronchoscopy was delayed because of respiratory and hemodynamic instability, we were able to diagnose tracheobronchial rupture after a care-

ful secondary review of her initial computed tomogram, specifically looking for this injury.

Because the risk factors for developing tracheal rupture may be similar to those for barotrauma secondary to alveolar rupture, diagnosis may be delayed, as in this case. We recommend consideration of tracheal rupture when a patient presents with profound subcutaneous emphysema shortly after intubation, particularly if intubation was difficult and there is evidence of continued air leak.

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