Tracheal Stenosis and Failure to Wean from Mechanical Ventilation due to Herpetic Tracheitis*

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A 64-year-old man with chronic obstructive pulmonary disease presented with pneumococcal pneumonia that progressed to respiratory failure within one week, requiring mechanical ventilation. Despite a low minute ventilation and clear chest roentgenogram, multiple weaning attempts failed. Bronchoscopy revealed significant narrowing of the distal trachea with erythema, edema, and ulceration of the mucosa. Cytology of tracheal washings was consistent with herpes simplex virus, and the patient was successfully extubated following treatment with intravenous acyclovir. Bronchoscopy following acyclovir therapy demonstrated resolution of the inflammation and narrowing. Herpetic tracheitis is a rarely recognized reversible cause of tracheal stenosis, especially in a nonimmunocompromised patient. It should be suspected in patients without an obvious cause of failure to wean from mechanical ventilation, and can be successfully treated with acyclovir.

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\[ \text{HSV} = \text{herpes simplex virus} \]

The majority of patients who require mechanical ventilation are successfully removed from the ventilator in less than seven days. However, a small group of ventilated patients (approximately 9 percent), require mechanical support for longer than one week, and as a consequence, are at increased risk for developing numerous complications such as infection, barotrauma, and GI bleeding. In addition, prolonged mechanical ventilation results in significant financial and emotional burdens, and is associated with both a limited survival (less than 30 percent at one year), as well as a poor functional status in those that do survive. We describe a patient who required prolonged mechanical ventilation, which was subsequently proven to be secondary to herpetic tracheitis and resultant tracheal stenosis. Treatment with intravenous acyclovir resulted in extubation, resolution of the tracheal inflammation and narrowing, and ultimately, discharge from the hospital.

CASE REPORT

This 64-year-old white man had a long history of chronic bronchitis due to a 100 pack-year smoking history. He was receiving home oxygen, and had a prior history of multiple hospital admissions for "exacerbations" of his obstructive lung disease, although he had never required mechanical ventilation. He presented to his local hospital with a one-week history of increasing productive cough and dyspnea. Chest roentgenogram revealed a right lower lobe infiltrate, and a diagnosis of pneumococcal pneumonia was made based on a compatible Gram stain and positive sputum culture. Despite treatment with intravenous antibiotics, corticosteroids, aminophylline, and an aerosolized beta-agonist, the patient was transferred to The Ohio State University Hospitals due to worsening respiratory status.

Upon presentation to our institution, the patient was noted to be lethargic, and arterial blood gas values on room air revealed a pH of 7.30, a PaO\(_2\) of 60 mm Hg, and a PaCO\(_2\) of 93 mm Hg. He was intubated, placed on mechanical ventilation, and admitted to the medical intensive care unit. Examination revealed a rectal temperature of 37.7°C, a pulse rate of 140 beats per minute, and a blood pressure of 140/94. There was no evidence of cutaneous or oral mucosal herpetic lesions. The patient had an increased anterior-posterior chest diameter, and auscultation of the lungs revealed diffuse wheezing. The cardiac examination was unremarkable, with no murmurs or gallops. A chest roentgenogram revealed changes of obstructive lung disease, but the lungfields were clear, with no evidence of the previous right lower lobe infiltrate. An ECG demonstrated sinus tachycardia, left anterior hemiblock, and occasional PACs. The WBC was 13,900/cu mm, with 78 percent neutrophils, 18 percent lymphocytes, and 4 percent monocytes. Results of routine chemistries and liver function tests were normal, except for a carbon dioxide content of 38 mmol/L. The theophylline level was 11.7 mg/dl. An arterial blood gas on a fraction of inspired oxygen of 0.4 revealed a pH of 7.36, a PaO\(_2\) of 72 mm Hg, and a PaCO\(_2\) of 73 mm Hg.

The patient was initially treated with the following intravenous medications; hydrocortisone (100 mg every six hours), cefizoxime (2 g every eight hours), and a continuous infusion of aminophylline to maintain a therapeutic level. He also received aerosolized albuterol every four hours. The chest roentgenogram continued to reveal clear lungfields, his arterial blood gas values remained stable, and his minute ventilation was only six to seven liters. However, multiple attempts at weaning from the ventilator were unsuccessful due to the development of tachypnea and hypopnea. Due to copious secretions and difficulty passing the suction catheter, bronchoscopy was performed on the tenth hospital day, and demonstrated significant narrowing of the distal trachea, with erythema, edema, and mucosal ulceration (Fig 1). The remainder of the tracheobronchial tree was unremarkable. Cytologic findings of tracheal washings were consistent with herpes simplex virus and did not demonstrate any evidence of malignancy (Fig 2). Viral culture of tracheal washings grew herpes simplex virus. Intravenous acyclovir was immediately started (8 mg/kg, every eight hours), and a 14-day course of therapy was completed. Eight days after the institution of acyclovir, the patient was successfully extubated, and subsequently was transferred out of the intensive care unit.

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FIGURE 1. Bronchoscopic view of the distal tracheal stenosis. Note significant erythema, exudate and mucosal ulceration producing narrowing of trachea.
care unit. Arterial blood gas values at that time revealed a PaO2 of 63 mm Hg and a PaCO2 of 45 mm Hg on 3 L of oxygen. Five days after completion of acyclovir, bronchoscopy demonstrated improvement of the inflammation and tracheal narrowing. One month after discharge, bronchoscopy was essentially normal, with resolution of the above findings (Fig 3).

**DISCUSSION**

Tracheal stenosis is infrequently encountered in the medical intensive care unit and is most commonly caused by tumors, extrinsic compression, or trauma (tracheostomy, intubation). The case presented demonstrates an unusual and uncommonly recognized cause of tracheal obstruction due to herpetic tracheitis, with subsequent failure to wean from mechanical ventilation.

Manifestations of herpes virus infection of the lower respiratory tract includes tracheobronchitis and pneumonia. Herpetic tracheobronchitis and pneumonia occur most commonly in patients with impaired immunity. However, a recent study of intubated patients demonstrated that although tracheal trauma is associated with intubation, it was unlikely to be the primary cause of infection. Underlying acute or chronic lung disease has also been thought to predispose to herpes virus airways infection.

The usual routes of herpes simplex virus infection of the respiratory tract are thought to be either aspiration or contiguous spread, in association with oral or pharyngeal herpetic lesions. The HSV is a part of the normal oral flora and is often cultured from oral secretions in the absence of oral lesions, but is more frequently present in the sputum or throat cultures of adults with any type of symptomatic respiratory illness. Tracheobronchial infection with herpes virus causes inflammatory, obstructing airway lesions, with erythema, edema, exudate, and mucosal ulceration. Direct visualization by bronchoscopy, in conjunction with cytology and culture, are diagnostic. Tissue histology may also be useful, although not necessary, and serologies are less helpful.

In a recent study, Sherry et al. described nine elderly (ages 61 to 88), nonimmunocompromised patients, with no evidence of chronic lung disease, who had herpetic tracheobronchitis. No patient had evidence of oral herpetic lesions. Eight patients presented with acute, refractory bronchospasm and no prior history of asthma or chronic bronchitis. Chest roentgenograms were normal in most of the patients, and six patients required mechanical ventilation. All patients had bronchoscopic findings characteristic of herpetic tracheobronchitis. Other authors have also reported herpetic tracheobronchitis as a cause of acute respiratory failure requiring mechanical ventilation in patients with malignancy or underlying chronic obstructive lung disease. In addition, HSV has been associated with the adult respiratory distress syndrome, with these patients requiring prolonged ventilatory support and demonstrating an increased late mortality.

The patient presented had several risk factors for the development of herpetic tracheitis, including underlying chronic lung disease due to cigarette smoking, recent pneumonia, treatment with corticosteroids, and tracheal intubation, although it is not clear whether the infection was present prior to intubation. It seems apparent, however, in view of a clear chest roentgenogram and a stable minute...
ventilation, that this patient's failure to wean from prolonged mechanical ventilation was a direct result of herpetic tracheitis and tracheal stenosis. Resolution following treatment with acyclovir allowed discontinuation of ventilatory support, and ultimately, discharge from the hospital.

Herpetic tracheitis is not a commonly recognized cause of large airway narrowing or obstruction, respiratory failure, or of failure to wean from mechanical ventilation. It should be suspected in patients with clear chest roentgenograms who are unable to be weaned from mechanical ventilation. Bronchoscopy should be performed in consideration of this diagnosis, which can be successfully treated with acyclovir.1,2

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Osler-Weber-Rendu Disease and Pulmonary Arteriovenous Fistulas*

Deterioration and Embolotherapy during Pregnancy

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Several reports have implicated pregnancy as a cause of deterioration in patients with pulmonary arteriovenous fistulas. We report a 27-year-old woman with multiple pulmonary arteriovenous fistulas who required coil spring embolotherapy in her 24th week of pregnancy due to a spontaneous hemothorax and hypoxemia.

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AVF = arteriovenous fistula

Pulmonary AVFs occur in approximately 15 percent of patients with Osler-Weber-Rendu disease1 and bleeding complications, such as hemoptysis and rarely hemothorax, are recognized.1-3 Several case reports also have associated complications of pulmonary AVFs with pregnancy.5-11 We report the case of a young woman with previously undiagnosed Osler-Weber-Rendu disease who presented in the 24th week of pregnancy with hypoxemia and a spontaneous hemothorax due to intrapleural rupture of a pulmonary AVF. She underwent successful coil spring occlusion of her AVFs, the first reported use of embolotherapy in a pregnant patient.

CASE REPORT

A 27-year-old woman was admitted to a hospital in her 24th week of pregnancy with left-sided chest pain, dyspnea and scant hemoptysis. A chest roentgenogram revealed bilateral pleural effusions and mild interstitial edema, and arterial blood gas levels were as follows: pH 7.50; Pco2, 23 mm Hg, and Paco2, 54 mm Hg while the patient was breathing room air. She was treated for presumed congestive heart failure with diuretics and digoxin. The workup included an echocardiogram which showed severe mitral regurgitation and mild mitral stenosis. A VQ scan was interpreted as low probability for pulmonary embolism; however, tracer activity was noted in the kidneys, suggesting a right-to-left shunt. On the fourth hospital day she developed a large left pleural effusion and was transferred to our institution.

Past medical history was remarkable for rheumatic heart disease as a child. Family history was remarkable for recurrent nosebleeds in her father and two children.

Physical examination was notable for diminished breath sounds...