

Ventilator Auto-Triggering in a Patient with Tuberculous Bronchopleural Fistula

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We report a case of ventilator auto-triggering resulting from tuberculous bronchopleural fistula being managed with chest tube suction. Early recognition of bronchopleural fistula-related auto-triggering is extremely important. Auto-triggering can lead to serious adverse effects, including severe hyperventilation and inappropriate escalation of sedatives and/or neuromuscular blockers (administered to reduce spontaneous breathing efforts). Auto-triggering was confirmed in our patient when tachypnea persisted despite pharmacologic neuromuscular paralysis. Auto-triggering can be reduced or eliminated by decreasing ventilator trigger sensitivity or by decreasing the air leak flow by reducing the degree of chest tube suction. [Respir Care 2003;48(5):519–521. © 2003 Daedalus Enterprises]

Introduction

Chest tube insertion is the cornerstone of medical management of bronchopleural fistula (BPF).^{1,2} Pleural drainage is essential when pulmonary tuberculosis is complicated by the presence of a BPF. This is best accomplished by using a large-diameter chest tube to relieve pneumothorax and promote drainage of the infected pleural space. Small-diameter chest tubes are an inferior alternative and may result in lung collapse and tension pneumothorax, especially if the patient has a mobile mediastinum. Some authors advocate using the least possible chest tube suction, to reduce tidal volume loss through fistula flow.^{3,4} Paradoxically, the chest tube can also have detrimental effects with a patient suffering a BPF. A large air leak through a BPF can result in failure of lung re-expansion, loss of applied positive end-expiratory pressure, and an inability to maintain alveolar ventilation.⁵

The volume of flow through a BPF is a function of the size of the air leak (resistance) and the transpulmonary

pressure gradient (airway pressure minus pleural pressure). Increasing chest tube suction proportionally increases transpulmonary pressure and thus increases flow through the BPF.^{5–7} This presents a difficult management and therapeutic challenge.

Case Summary

A 64-year-old woman presented to the hospital with dyspnea and a cough productive of yellow sputum. Her chest radiograph revealed right-sided apical consolidation with cavitation and left-sided alveolar infiltrates. The patient was isolated on the general medical floor with the presumptive diagnosis of tuberculosis. Sputum was positive for acid-fast bacilli, and the patient was started on isoniazid, rifampin, ethambutol, and pyrazinamide. Her history was notable for a left-sided malignant pleural effusion of unknown etiology, for which she had undergone pleurodesis approximately 6 months prior to admission. She also had a history of alcoholism, malnutrition, and hypertension. Her clinical condition deteriorated over the next few days as she developed worsening dyspnea and right-sided pneumothorax. A chest tube was placed, but the patient's respiratory status continued to worsen. Arterial blood gas analysis (while receiving 100% oxygen via nonrebreather mask) revealed a pH of 7.41, P_{aCO_2} of 29 mm Hg, P_{aO_2} of 67 mm Hg, and an oxygen saturation of 93%. The patient was transferred to the medical intensive care unit. She was endotracheally intubated and mechanical ventilation was instituted. Shortly after intubation, the

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VENTILATOR AUTO-TRIGGERING IN A PATIENT WITH TUBERCULOUS BRONCHOPLEURAL FISTULA

Table 1. Adjustments to Ventilator Sensitivity and Neuromuscular Blockade

Chest tube suction (cm H ₂ O)	Paralysis	V _T (mL)	Set RR (breaths/min)	Actual RR (breaths/min)	F _{IO₂}	Ventilator sensitivity (cm H ₂ O)
-20	No	450	15	35	0.6	-0.5
-20	Yes	450	15	35	0.6	-0.5
-20	Yes	450	15	15	0.6	-3

V_T = tidal volume
 RR = respiratory rate
 F_{IO₂} = fraction of inspired oxygen

patient developed hypotension, and a chest radiograph revealed left-sided mediastinal shift with right-sided apical and basilar pneumothoraces. Fluid resuscitation and vasopressor support helped stabilize her condition. Emergency chest decompression was conducted by placing additional chest tubes. There was a large and persistent air leak throughout the respiratory cycle, through all of the chest tubes. The initial ventilator settings in the assist control mode were tidal volume 450 mL, respiratory rate 15 breaths/min, and 100% oxygen. The patient was over-breathing the ventilator, with a respiratory rate around 35 breaths/min.

Physical examination on admission to the intensive care unit revealed a thin, wasted female who was intubated and sedated. Her blood pressure while on vasopressors ranged from 80/60 to 100/70 mm Hg, with a heart rate of 110 beats/min. She had extensive subcutaneous air in the chest wall and neck. There were diminished breath sounds on the right side, compared to the left. Her cardiac and abdominal examinations were normal. The air leak persisted, and returned tidal volume to the ventilator ranged from 200 to 250 mL. The drain tubes (Pleur-Evac, Genzyme Biosurgery, Cambridge, Massachusetts) were set on -20 cm H₂O suction. A few hours into her intensive care course the patient again developed worsening hypotension and extreme agitation, with low exhaled tidal volumes (< 250 mL). A repeat radiograph showed a recurrent pneumothorax on the right. The chest tubes were repositioned and her hemodynamics improved. Arterial blood gas analysis (while on 100% oxygen) revealed a pH of 7.45, P_{aCO₂} of 25 mm Hg, P_{aO₂} of 100 mm Hg, and an oxygen saturation of 99%. Despite heavy sedation and effective neuromuscular blockade, as judged by train-of-4 nerve stimulation, the total respiratory rate continued to far exceed the set ventilator rate (Table 1). This confirmed the suspicion that the persistent tachypnea was due to ventilator auto-triggering.

Discussion

This case presents an example of ventilator auto-triggering related to chest tube suction in a case of tuberculous

BPF. To our knowledge there are only 4 other reported cases of ventilator auto-triggering secondary to chest tube suction in patients suffering BPF.⁵⁻⁸

Ventilator auto-triggering caused by endotracheal tube cuff leak and ventilator gas leak have also been reported.⁹⁻¹¹ The mechanism of ventilator auto-triggering is similar in each of these circumstances: air leak from the proximal or distal airway lowers proximal airway pressure below the set trigger sensitivity and thus triggers inspiration. The increase in minute ventilation may cause severe respiratory alkalosis and/or intrinsic positive end-expiratory pressure.

In this case respiratory alkalosis and a normal P_{aO₂} ruled out the possibility that the tachypnea was caused by gas exchange abnormalities. The suspicion of ventilator auto-triggering was confirmed when the tachypnea did not abate with deep sedation and neuromuscular paralysis, since effective neuromuscular blockade eliminated the possibility of spontaneous respiratory muscle activity.

Early recognition of ventilator auto-triggering is imperative in order to avoid adverse outcomes that can result directly from excessive hyperventilation or indirectly from clinician attempts to pharmacologically suppress respiratory drive. This point is extremely important, because failure to recognize auto-triggering caused by a BPF could lead to inappropriate escalation of sedatives and/or neuromuscular blockers, because the patient might be assumed to be breathing spontaneously. Higher doses of sedatives and/or neuromuscular blockers are clearly associated with adverse outcomes.^{12,13}

Auto-triggering can be reduced or eliminated by decreasing the ventilator trigger sensitivity and/or by reducing the flow of the air leak, until the primary cause of the leak can be eliminated. In this case auto-triggering was eliminated by decreasing ventilator sensitivity (see Table 1). Of note, for this method to be effective, trigger sensitivity must be reduced to a value more negative than the reduction in airway pressure induced by chest tube suction. An alternative strategy is to reduce the degree of chest tube suction to decrease the transpulmonary pressure gradient for flow through the BPF, but this may only be possible if the lung has fully expanded.

In conclusion, management of BPF in mechanically ventilated patients poses a clinical challenge and requires a thorough understanding of all of the potential problems that can arise in the management of this type of patient. Clinicians should know that chest tube suction can cause ventilator auto-triggering in a BPF patient, so they can recognize and treat the problem early and avoid the associated adverse outcomes.

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