Pulmonary Hyperinflation and Respiratory Distress Following Solvent Aspiration in a Patient With Asthma: Expectoration of Bronchial Casts and Clinical Improvement With High-Frequency Chest Wall Oscillation

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An 18-year-old student with a history of asthma accidentally inhaled organic solvent during a class, with immediate cough and dyspnea that worsened over several hours. He presented in severe respiratory distress, with hypoxemia and marked pulmonary hyperinflation. Administration of inhaled bronchodilator was ineffective because of agitation, and the patient could not be positioned for chest physiotherapy to treat presumed widespread mucus plugging. High-frequency chest wall oscillation (HFCWO) in the sitting position initially caused increased distress but was subsequently tolerated when noninvasive positive-pressure ventilation (NPPV) via nasal mask was initiated. Almost immediately, the patient began expectorating bronchial mucus casts, with concomitant clinical improvement. Endotracheal intubation was avoided, and with aggressive pharmacologic treatment for acute severe asthma and continuation of intermittent HFCWO-NPPV, the patient made a full recovery over the next several days. This case suggests that the combination of HFCWO and NPPV may be helpful in the presence of mucus plugging as a complication of acute inhalation injury or acute severe asthma. *Key words: pulmonary, respiratory distress syndrome, high-frequency ventilation, asthma, mucus, toluene, solvent, inhalation injury.* [Respir Care 2004;49(11):1335–1338.

Introduction

Acute severe asthma is characterized by bronchospasm, inflammatory edema, and excessive mucus production, which can lead to widespread plugging of the airways.[1,2] In this setting, a cascade of events, including hypoxia, hypercapnia, acidosis, lung hyperinflation, increased work of breathing, and ventilatory muscle fatigue may lead to life-threatening circulatory depression and respiratory arrest.[3] We report the case of a young asthmatic who experienced widespread airway mucus plugging with acute pulmonary hyperinflation and severe respiratory distress after accidental organic solvent inhalation, in whom expectoration of bronchial casts and dramatic clinical improvement were associated with the application of high-frequency chest wall oscillation (HFCWO) and noninvasive positive-pressure ventilation (NPPV).

Case Presentation

An 18-year-old student presented to the Kawazu Respiratory Clinic with cough and increasing dyspnea after accidentally inhaling an unknown quantity of a toluene-containing solvent. Asthma had been diagnosed 2 years previously, at which time the patient was hospitalized and treated with inhaled β agonist, oral prednisolone, oral theophylline, and inhaled fluticasone. Subsequently, his symptoms were seasonal and intermittent, and he stopped all medications except for occasional β agonist administered by metered-dose inhaler. An exacerbation 2 weeks prior to admission had been treated with the same regimen as before, and with improvement over the next several days the

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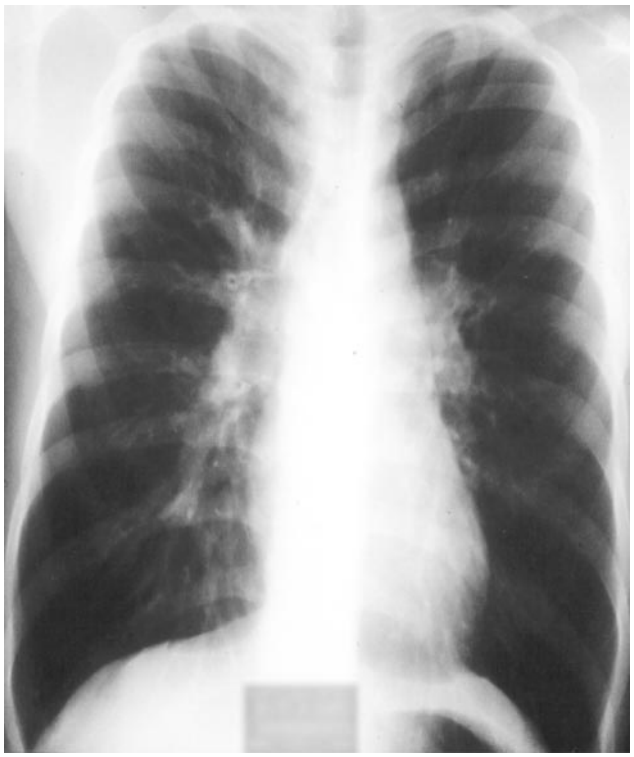

Fig. 1. Frontal chest radiograph taken on initial presentation. The lungs are markedly hyperinflated, with downward displacement of the hemidiaphragms. Increased bronchial and vascular markings are present in the right lower lung field.

patient once again discontinued all medications except inhaled β agonist.

The aspiration incident occurred during a woodworking class and was followed by increasing dyspnea and cough. Having obtained no relief from inhaled β agonist, the patient presented to our clinic later that afternoon. On physical examination he was in obvious respiratory distress, sitting upright with rapid shallow breathing, nasal flaring, and the use of accessory respiratory muscles. Breath sounds were diminished on auscultation, and rhonchi were present. Oxygen saturation measured by pulse oximetry with the patient breathing room air was 82%. A chest radiograph (Fig. 1) revealed marked pulmonary hyperinflation.

Treatment for acute severe asthma with presumed widespread mucus plugging was initiated with supplemental oxygen, intramuscular epinephrine, intravenous theophylline, and intravenous hydrocortisone. Attempts to administer β-agonist aerosol by metered-dose inhaler and nebulizer were unsuccessful because of the patient's agitation and distress. Because he was unable to tolerate positioning for chest physiotherapy, we decided to apply HFCWO to facilitate removal of mucus plugs from the airways.

Treatment was initiated using an inflatable vest connected by tubes to an air-pulse generator (HM-Airway Clearance System, technologically identical to *The Vest*, model 104, both manufactured by Advanced Respiratory Inc, St Paul, Minnesota). The apparatus' pressure control was set at 1.5, with which peak pressure inside the vest was 0.28–0.30 psi, with a waveform amplitude (maximum minus minimum pressure) of approximately 0.20 psi, according to the manufacturer's specifications. Oscillation frequency was set at 10 Hz. Arterial blood gas values at the time HFCWO was begun, with the patient breathing oxygen at 5 L/min, were pH 7.40, P_{CO_2} 42 mm Hg, P_{O_2} 71 mm Hg, and arterial oxygen saturation 94%.

Because the patient developed increased agitation and signs of severe fatigue after only a few seconds of HFCWO, we decided to add NPPV in an attempt to reduce work of breathing and to avoid endotracheal intubation. Using a bi-level pressure-pre-set ventilator (Eclipse BP, Taema, Antony, France), we initiated NPPV with peak inspiratory pressure 10 cm H_2O and end-expiratory pressure 4 cm H_2O. With the addition of NPPV, the patient was able to tolerate HFCWO for increasing periods, from 1 minute initially to a target treatment period of 10 min.

Within a few minutes of starting combined HFCWO-NPPV, the patient began to expectorate bronchial casts (Fig. 2). This was accompanied by subjective improvement and improved oxygenation, such that his oxygen saturation (measured by pulse oximetry) was 93% during a brief period breathing room air after the first hour of treatment. Three 10-min HFCWO treatments were administered on the day of admission, and the patient was maintained on NPPV with oxygen bled into the circuit at 2 L/min until the next morning. Pharmacologic therapy for acute severe asthma was continued.

With improvement in the patient's condition, NPPV was discontinued, but twice-daily 10-min HFCWO treatments were administered for the next 7 days. Supplemental oxygen was no longer required after day 5. At the time of discharge from the clinic 8 days after initial presentation, the patient's symptoms had resolved and the marked hyperinflation demonstrated on the initial chest radiograph had improved substantially (Fig. 3).

Discussion

Recent evidence indicates that the hypersecretion and retention of airway mucus contribute to the development of acute severe asthma[4,5] and that mucociliary clearance is decreased both because of the adherent nature of the mucus and as a consequence of the reduced airflow.[6,7] Diffuse airway obstruction by mucus plugging may play a critical role in some cases of fatal asthma.[8–10]

In acute respiratory distress caused by diffuse airway obstruction and excessive airway mucus, it seems rational to include measures directed at facilitating secretion clearance in addition to aggressive pharmacologic therapy. Studies by Bateman et al[11,12] and by Sutton et al[13] showed that

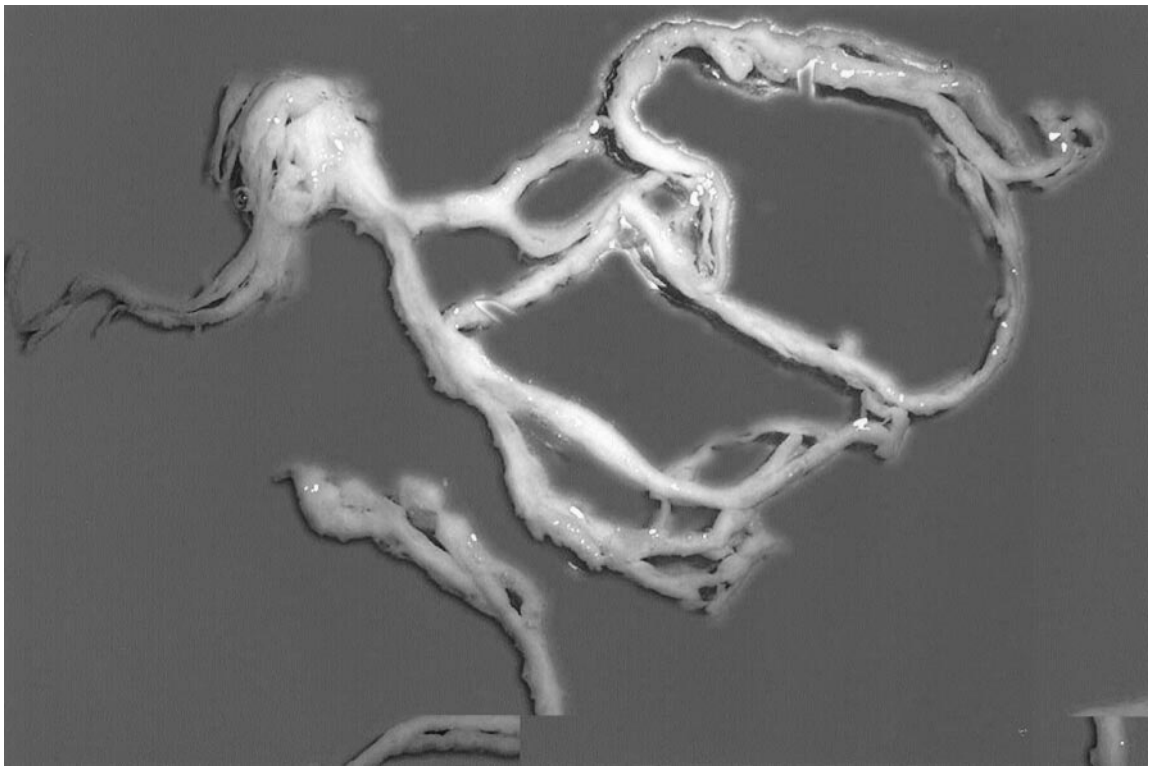

Fig. 2. Bronchial casts expectorated by the patient shortly after initiation of combined HFCWO-NPPV therapy.

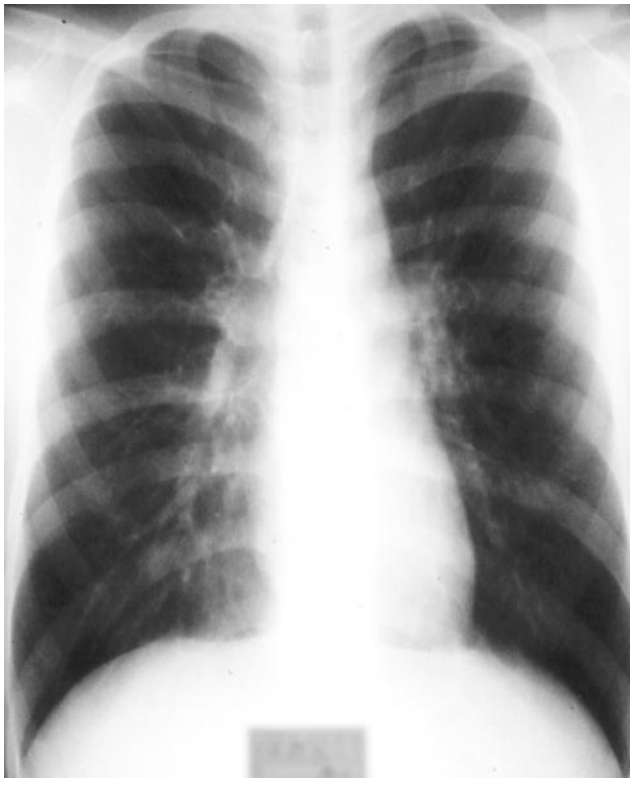

Fig. 3. Frontal chest radiograph taken on discharge, showing marked improvement in the previous signs of hyperinflation.

chest physiotherapy is effective in promoting the clearance of both inhaled radioactive aerosol and airway secretions. In both human and animal studies, HFCWO has been demonstrated to enhance tracheal mucus clearance.[14,15] Radioaerosol studies show that HFCWO can be effective in clearing secretions from the peripheral as well as from the central airways[14,16] That HFCWO reduces the viscoelastic and cohesive properties of mucus is supported by experimental evidence.[17] In patients hospitalized because of exacerbations of cystic fibrosis, Varekojis et al showed HFCWO to be at least as effective as vigorous, professionally administered postural drainage and chest percussion in clearing secretions.[18] It might be rational to select HFCWO instead of chest physiotherapy for hospitalized patients when trained personnel are unavailable or if the patient cannot tolerate the latter. Treatment times with HFCWO may be shorter than with thorough, rigorous chest physiotherapy, and treatments can be administered in the sitting position.

Although its benefits in acute severe asthma are not so firmly established as in exacerbations of chronic obstructive pulmonary disease, NPPV may improve gas exchange, unload the respiratory muscles, and augment circulatory function, reducing the need for intubation and decreasing ventilator-associated complications, duration of stay in the intensive care unit, and overall mortality.[19] Some investi-

gators maintain that NPPV is effective in correcting gas-exchange abnormalities and in preventing intubation in acute severe asthma.[20] On the other hand, the inability of a patient to adequately clear airway secretions is considered a contraindication to NPPV.[21]

In the context of acute inhalation injury and underlying asthma, our patient developed widespread mucus plugging, and specific measures to augment airway clearance appeared crucial to his management. Because he was unable to tolerate positioning for chest physiotherapy, we tried HFCWO. This too caused intolerable respiratory distress, until NPPV was added. However, with combined HFCWO-NPPV the patient was able to tolerate repeated therapy sessions and to expectorate a large number of bronchial casts, with dramatic improvement in his condition.

Although anecdotal, our experience with this patient suggests that the combination of HFCWO and NPPV may be a helpful or even life-saving intervention in the presence of severe mucus plugging complicating acute inhalation injury or acute asthma. Whether this adjunctive treatment should be added to standard therapies for these conditions should be established by appropriately designed clinical trials.

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