CLINICAL VIGNETTE

The Management of a Patient with Severe Inhalational Injury: Case Report and Literature Review

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Case Report

A 63-year-old man with no past medical history was found outside a burning building by firefighters. He had been trying to extricate a family member but was forced to flee when the building was overtaken with smoke. He was found outside the building covered in soot and disoriented. He was placed on a non-rebreather oxygen mask and transported to the Emergency Department of our burn center. In the ER, temperature was 36.8 degrees Celsius, blood pressure was 132/68, heart rate was 120 beats per minute, and pulse oximeter read 95% on a 15 liters per minute non-rebreather mask. He had deep second and third degree burns on his posterior torso, upper extremities, knees, feet, and face, which was estimated at 21% total body surface area. He had a singed forehead and facial and nasal hairs, as well as soot in his nasal passages and oropharynx. He was able to speak in complete sentences and was oriented to time, place, and person but was notably tachypneic. Arterial blood gas demonstrated a PH of 7.09, PCO2: 23, PaO2: 234, and base excess of -21. His carboxyhemoglobin was 14.5% and methemoglobin level was 0. He was emergently intubated in the emergency room for impending airway compromise due to laryngeal edema. On direct laryngoscopy, the vocal cords were covered with grey soot, and a size 7.5 endotracheal tube was passed through them without difficulty. Post-intubation chest X-ray showed clear lung fields.

ECG revealed sinus tachycardia with a rate of 120 beats per minute and no ischemic changes. Initial laboratory data revealed white blood cell count, 17,000 cells/mm^3; hemoglobin 14 g/dL; sodium 143 mEq/L; Potassium 4.1 mEq/L; chloride 106 mEq/L; bicarbonate 9 mEq/L; BUN 18 mg/dL; creatinine 1.6 mg/dL; glucose 142 mg/dL; and blood lactate 1.7 mEq/L. He was started on IV fluids and transferred to the intensive care unit for mechanical ventilator support and hemodynamic monitoring.

As per standard protocol for severe inhalational injury, early bronchoscopy was performed, which revealed extensive soot covering the trachea, main bronchi, and all proximal bronchopulmonary segments. During the first few days of admission, his oxygen requirements on conventional ventilator settings increased dramatically suggesting the onset of mucosal edema and ARDS. On hospital day 2, he was switched to a high frequency percussive ventilator (HFPV) initially set at: FiO2 60%, I:E ratio 1:1, respiratory rate 15 breaths per minute , pulse frequency of 450 oscillations per minute, mean airway pressure of 22, and peak inspiratory pressure of less than 35 cm of water. These settings were adjusted to maintain adequate O2 and CO2 levels. He was also started on vigorous chest physiotherapy and underwent bronchoscopy every other day with lavage and aspiration of the sooty debris sloughing from the airways. He was persistently febrile with negative bronchoalveolar lavage cultures and was nevertheless maintained on empiric broad spectrum antimicrobials. On ICU day 8, his gas exchange markedly improved, and the HFPV was transitioned back to conventional pressure control ventilation. On ICU day 16, he was extubated but had to be re-intubated several days later for severe neuromuscular weakness, ICU delirium, and pseudomonas pneumonia. He was managed with conventional ventilation and gradual weaning of sedative and analgesic medications and antibiotics. He also required multiple excision and auto/homografting procedures for his deep skin burns. He was extubated successfully on ICU day 28, transferred to a hospital floor and discharged home with no lingering respiratory impairment on hospital day 40.

Discussion

Smoke inhalation is the leading cause of death from fires and is often associated with high rates of acute respiratory failure, placing patients at-risk for prolonged need of mechanical ventilation. Smoke inhalation causes thermal injury to the upper airways, chemical injury to the tracheobronchial tree, and can cause systemic effects related to carbon monoxide and or cyanide poisoning.[1] When fire victims are brought to the Emergency Department, intubation should be performed immediately if the patients show signs of respiratory distress, any stridor, or if they have severe face or neck burns. If patients do not present with these signs but have erythema in the oropharynx, bronchoscopy, or laryngoscopy can be performed to assess for upper airway edema. If edema is visualized, patients should be prophylactically intubated as airway patency can quickly become compromised as edema worsens. If no edema is appreciated, patients can be observed closely and should not be discharged. All patients should have an ABG with co-oximetry to assess carboxyhemoglobin (CO) and methemoglobin concentrations and should be placed on 100% oxygen while waiting for results. This is done to displace possible CO from hemoglobin to which it has a high

affinity. Hyperbaric oxygen can be considered if CO level >25 or if patients have loss of consciousness, severe metabolic acidosis, or any evidence of end organ ischemia. Treating for CN poisoning should also be considered for patients who are exposed to smoke in a closed space fire with soot in nose/mouth, have altered mental status, or have hypotension. CXR should be performed; however, it may be negative even with severe inhalation injury.[2]

Chemical injury to the tracheobronchial tree can cause severe lung injury and bronchospasm due to direct toxin damage to the airway. Often, several types of chemicals are inhaled concomitantly, which can have negative synergistic effects on the airways. For example, the inhalation of compounds such as acrolein, formaldehyde, chlorine, phosgene, nitric oxide, and others can cause an inflammatory response in the lower airways. Combustion of organic materials produces fine carbonaceous particulates (soot), which tends to adhere to mucosal surfaces. When soot and toxic particles are inhaled together, this often causes prolonged contact of chemicals with bronchial mucosa, producing a heightened inflammatory cascade.[3] Activation of inflammatory cytokines, capillary leak, and impaired mucocilliary clearance due to adherent soot on airways produce tenacious secretions, which cannot be effectively cleared causing VQ mismatch and gas exchange abnormalities. After injury to the tracheobronchial tree has occurred, necrotic epithelium will begin to slough, which also causes an increase in mucos plugging and atelectasis. This predisposes the inhalation injury patient to bronchial infections such as ventilator associated pneumonia, which is associated with increased mortality in burn patients.[4] The intense inflammatory response is also associated with a high rate of acute respiratory distress syndrome, which is also associated with significant morbidity, mortality, and prolonged need for mechanical ventilation.[4] Inflammation and pulmonary edema lead to decreased pulmonary compliance, and these patients often require high airway pressures from mechanical ventilators to provide adequate oxygenation. This can cause further complications related to barotrauma.

Inhalation injury patients should remain intubated until upper airway edema and lower airway damage has resolved. Deep suctioning is often done but may not be helpful to remove particles from the distal bronchial tree. Bronchoscopy, which is done frequently in these patients, can remove the foreign particles and secretions that worsen the inflammatory response to slow disease progression and improve ventilation.[3,5] It should be performed at least once in intubated patients, particularly if they develop pneumonia as this has been associated with shorter number of ventilator days as well as ICU and hospital length of stay.[6] This was done every other day in our patient to remove tenacious secretions and soot until his airway mucosa normalized.

High frequency percussive ventilation is a time cycled, pressure limited mode of mechanical ventilation with inspiratory and expiratory oscillation. With this mode, a piston mechanism is positioned at the end of the endotracheal tube, which delivers high frequency pulsations of gas (200-900 beats/min) that accumulates to form a low tidal volume breath (set at 10-15 breaths/min).[7] The operator sets a peak inspiratory pressure, positive end expiratory pressure, continuous positive airway pressure, inspiratory time, expiratory time, and percussive frequency and rate. In theory, these high frequency pulsations aid in the mobilization of pulmonary secretions and clear airway debris. Continuous stacking of tiny low tidal volume breaths may allow for improved alveolar recruitment, less atelectasis, and more efficient gas distribution in the lower airways at lower mean airway pressures.

In theory, it appears that HFPV should be beneficial in smoke inhalation patients; however, there are little data. Small studies have varying methodology and findings on its efficacy. There are very few randomized trials comparing HFPV to conventional ventilation. It appears to perhaps improve gas exchange, but there is little data to suggest a mortality or benefit in duration of mechanical ventilation or incidence of ventilator associated pneumonia.[8,9] A recent study showed that high tidal volume ventilation (15 ml/kg) compared to regular tidal volume (9 ml/kg) decreased ventilator days in pediatric burned patients with inhalation injury.[10] This needs more investigation in pediatric as well as adult patients.

Most patients with inhalation injury who survive to hospital discharge do not have long-term respiratory complications.[11] An exception is a subset of patients with chemical inhalations who develop bronchiolitis obliterans syndrome, a life threatening, non-reversible, obstructive, fibrotic lung disease.

Conclusion

Our patient presented with severe inhalational injury and was treated with early intubation, vigorous airway clearance methods, including frequent bronchoscopy and high frequency percussive ventilator. As expected due to the severity of his illness, he suffered from ventilator associated pneumonia, ICU delirium, and critical illness myopathy, despite aggressive measures to prevent these complications. He required aggressive physical therapy and left the hospital without respiratory compromise after 40 days.

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