Management of Inhalation Injury in an Adult Burn Patient

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Abstract

Inhalation injuries are the primary cause of fatalities from burn injuries, accounting for nearly 60-80% of all fire-related deaths. Inhalation injury is an independent injury on itself; however, combined with cutaneous burns, this injury is associated with increased fluid requirements, increased incidence of pulmonary complications, and an increase in burn mortality.

A 30 year old Hispanic female presented to a level one trauma, emergency department after sustaining a flash burns to the face and 3% total body surface area burn to left upper extremity and upper chest. The patient also presented with signs and symptoms of inhalation injury and acute hypoxia requiring mechanical ventilation. The patient developed acute respiratory distress syndrome on the second day stay in the intensive care unit. This article review illustrates the complex disease process of inhalation injury, the challenges and its impact to patient's recovery, and the collaborative role of an advanced practice nurse in the management of a burn patient with inhalation injury.

INTRODUCTION

The treatment and management of cutaneous burns has progress remarkably in recent decades; however, only slow improvements have occurred in the treatment and management of patients with inhalation injury.1,2 Inhalation injuries are the primary cause of fatalities from burn injuries, accounting for nearly 60-80% of all fire-related deaths. About 22% of all burn presentations have concurrent smoke inhalation injury.3 Inhalation injury is an independent injury on itself; however, combined with cutaneous burns, this injury is associated with increased fluid requirements, increased incidence of pulmonary complications, and an increase in burn mortality by 20%.4 The incidence of lung injury associated with smoke inhalation has been well documented in the literature and constitutes the major factor of increased morbidity and mortality for burn patients.4-6

The complex pathophysiology of inhalation injury is also well described in the literature.1,2,4,6,7 However, the standard diagnostic criteria for inhalation injury remains elusive, and treatment is largely supportive.2,8 Inhalation injury can be classified according to anatomic location: 1) thermal injury restricted to upper-airway structures (mouth, oropharynx, and larynx); 2) chemical injury to the lower airway and parenchyma; and 3) metabolic asphyxiation/systemic toxicity with inhalation of carbon monoxide or cyanide.2,9 All 3 forms of injury may occur in a given patient which may influence treatment management.9 The inhalation injury described in each phase has a different pathophysiology, treatment, and long-term implications.8 The purpose of the article review is to illustrate the complex disease process of inhalation injury and the collaborative role of an advanced practice nurse (APN) in the management of a burn patient with inhalation injury.

PATHOPHYSIOLOGY

Inhalation injury is caused by direct heat injury to airway mucosa and the toxins contained in smoke.4 The upper airway injuries are primarily caused by the inhalation of super-heated air (temperature in excess of 150° Celsius) resulting in direct thermal injury to the mouth, oropharynx, and larynx.8,9 The injured airway activates the inflammatory and complement cascade causing increased capillary permeability and increased pulmonary hydrostatic pressure leading to airway inflammation, and mucosal/airway edema.4,8 Unless the patient is exposed to steam, dry heat does not easily penetrate to the lower respiratory tract; thus, tissue damage in the lower airway is generally caused by chemical toxins and particulate matter.4

The lower airway and parenchymal injury is caused by exposure to the toxic constituents of smoke such as toxic gas and particulate matter produced by incomplete products of combustion. Chemical injury to lower airway caused by

inhaled particulate perpetuate local tissue injury by adhering to the mucosa and cause sloughing of the airway-lining epithelium, mucus secretion, inflammation, atelectasis, and airway obstruction.8 Carbon monoxide is an odorless, tasteless gas produced by partial oxidation of carbon-containing compounds such as wood, paper, coal, natural gases, and petroleum products.9 Inhaled carbon monoxide has a stronger affinity for hemoglobin (200 times greater than the affinity of oxygen) and competes with oxygen to form carboxyhemoglobin causing impaired oxygen delivery at the tissue level.2,7 Hydrogen cyanide is also a colorless gas, with an odor of a bitter almonds, produced during combustion of materials such as plastics, foam, paints, silk, carpets, paper products, and polyurethane. This gas binds to the terminal cytochrome oxidase and inhibits electron transport and cellular respiration. This process prevents cellular use of oxygen. Carbon monoxide and/or cyanide exposure cause impairment of oxygen delivery and utilization at the tissue level and the co-factor in inhalation injury process, accelerating the development of ventilation/perfusion mismatch resulting to hypoxia.9

Lung dysfunction after inhalation injury develops over the course of 24 to 72 hours.8 Pulmonary edema and airway obstruction causes ventilation perfusion mismatch as well as pulmonary shunting which is manifested by poor gas exchange and oxygenation. In addition, mucociliary action is impaired due to structural damage from heat and toxins in the smoke causing an impaired cough reflex with tenacious secretions. Inhalation injury is associated with a high risk of occlusion of smaller airways leading to atelectasis and infection.1

As a result, patients frequently develop acute lung injury, acute respiratory distress syndrome (ARDS), and pneumonia. The partially obstructed airways may result in air trapping and alveolar hyperinflation, leading to barotrauma.8 Airway and pulmonary edema combined with obstructive casts from cellular debris and fibrin clots causes airway obstruction and marked decrease in pulmonary compliance leading to respiratory failure. Smoke inhalation can cause a combination of direct pulmonary injury and systemic toxicity.

CASE PRESENTATION

A 30 year old Hispanic female is admitted to the intensive care unit (ICU) after sustaining flash burns to her face and 3% total burn surface area (TBSA) to her left upper extremity and upper chest from a house fire. The patient also presented with signs and symptoms of inhalation injury and acute hypoxia requiring mechanical ventilation.

History

Chief Complaint: Sustained facial flash burns and burns to upper chest and left upper extremity

History of Present Illness: A 30 year old Hispanic female presented to a level one trauma, emergency department (ED) after sustaining a flash burns to face and 3% TBSA burn to left upper extremity and upper chest. Per report, the patient was found down and extricated by friends from an enclosed space laundry room in a house fire. The extrication time is unknown. On initial assessment in the ED, the patient was noted with singed nasal hair, black soot in the nose and mouth and upper extremities, and the presence of carbonaceous debris in the sputum. The patient had a Glasgow Coma Scale (GCS) score of 15 on arrival, but with significant low oxygen saturation by pulse oximetry (SpO2) of 82% and hoarse voice during initial evaluation. She was emergently intubated for presumed smoke inhalation injury. In the ED, a carboxyhemoglobin level was also reported as 15% and an alcohol level of 37 milligrams/deciliter (mg/dl). The patient was transferred to the ICU for continuous high level of acute care and treatment of inhalation injury.

Past Medical/Surgical History: None reported by family; takes no medications

Social History: Patient lives with her 2 younger siblings. She completed high school and works full time at a grocery store. Patient is single and was spending the night at a friend/co-worker's house where the fire occurred. Per family, patient drinks alcohol occasionally, but denies any drug and tobacco use. She has good friends and never has been in trouble with the law.

Family History: Family reported positive for obesity and hypertension.

Allergies: No known drug allergies

Review of Systems: Unobtainable due to patient's current condition: intubated and sedated, but father reported that the patient is a healthy and active young adult with no health problem or issues reported.

Physical Examination

Vital signs: blood pressure (BP) = 112/58, heart rate (HR) = 90-134, respiratory rate (RR) = 14-36 breaths per minute

(bpm), temperature= 98.5o Fahrenheit, pulse oximeter oxygen saturation (SpO2) = 97% on mechanical ventilation with 70% fraction of inspired oxygen (FiO2) ; weight = 84.7 kilogram (kg)

General: intubated, sedated with occasional agitation on stimulation; Richmond Agitation Sedation Scale (RASS) score = -2

Neuro: GCS 8T with Eye opening (E) = 3, Verbal response (V) = Endotracheal tube (T), Motor response (M) = 5

Head, Eyes, Ears, Nose, Throat (HEENT): superficial flash burns to face and ears, Pupils equal, round, and reactive to light and accommodation (PERRLA), no icteric sclera, neck supple, no lymphadenopathy, no mass/carotid bruit, unable to assess oral cavity due to presence of endotracheal tube, dry and mild swollen lips

Cardiovascular: S1/S2 with regular rate and rhythm, no murmurs/rubs/gallops, sinus tachycardia (116 bpm per monitor)

Respiratory: course bilateral breath sounds on airway pressure release ventilation (APRV) ventilator mode with 70% FiO2, peak inspiratory pressure (PIP) = 19 centimeter of water pressure (cmH20)

Gastrointestinal: abdomen soft, non-tender, non-distended, normal active bowel sounds in 4 quadrants

Extremities: Palpable 2+ bilateral radial pulses, 2+ pulses bilateral lower extremities

Skin: 2% TBSA superficial partial-thickness burn to left upper extremity, superficial burn to upper chest, superficial facial burns anteriorly with sloughing of skin as well as ears

Diagnosis

A bronchoscopy was done on arrival to the ICU and revealed mild diffuse erythema to distal segments with carbonaceous material in proximal bronchi. This finding confirmed the diagnosis of smoke inhalation injury in combination with the patient's history and clinical presentation.

The APN practiced collaboratively with the interprofessional team in collecting pertinent patient history, physical examination, laboratory, and diagnostic evaluation in developing a comprehensive treatment management for a patient with inhalation injury. Employing good communications was critical to inform clinicians involved in the direct patient care regarding patient health status, any changes in the patient's condition, implementation of a treatment plan, and patient response to treatment provided. Family participation was also necessary to provide a patient centered plan of care that supports short-term and long-term goal achievement.

DIAGNOSIS OF INHALATION INJURY

The clinical diagnosis of inhalation injury can be imprecise and challenging due to the lack of standard criteria for diagnosis and grading of severity.1,8 However, inhalation injury should be considered based on the constellation of a patient's history of an enclosed space fire, physical findings including facial burns, singed nasal hair, cough, carbonaceous sputum, and/or evidence of upper airway edema, hoarseness, stridor, or wheezing. Diagnosis of inhalation injury is best confirmed by fiberoptic bronchoscopy (FOB) in conjunction with a patient's history and physical examination.2,4

Fiberoptic bronchoscopy

FOB allows for direct visualization and evaluation of the airway from the oropharynx to the lower bronchi. This procedure is considered the "current standard" for establishing the diagnosis of inhalation injury.9 FOB is also used to determine and evaluate the severity of inhalation injury with development of pulmonary complication, detect late pulmonary sequelae, as well as assess airway status/improvement through direct visualization. Diagnosis of inhalation injury requires a patient's history of exposure to smoke and bronchoscopy revealing below the glottis evidence of carbonaceous material, and signs of edema or ulceration.10

An initial finding of injury such as erythema/edema and carbonaceous deposits in the airway mucosa are indicative of the presence of inhalation injury; however, the degree of severity of injury is diagnosed based on the course of the disease process rather than on initial findings from a fiberoptic bronchoscopy.6 FOB is also done as needed to directly visualize pulmonary status and any changes or improvement. If the patient is suspected with pulmonary infection, a bronchoalveolar lavage (BAL) is necessary to identify and determine the presence of invading microorganisms.

Chest radiograph

Admission/initial chest x-ray (CXR) is not used for

diagnostic purpose, but is important for baseline evaluations. Chest radiograph can provide useful clinical information such as new or worsening opacities, consolidation, development of pneumothorax, and atelectasis suggestive of worsening status secondary to lung injury, aspiration, ARDS, volume overload, or infection. CXR can provide useful clinical information suggestive of progressive changes or improvement and can guide treatment as well as detect complications from various therapeutic interventions.11

Arterial blood gas (ABG) and co-oximeter.

Altered gas exchange is reflected in blood-gas analysis.6 ABG combined with other diagnostic studies can provide useful clinical information about the patient's oxygenation status and response to treatment. Serial measurements can help identify and evaluate pulmonary status and oxygenation. The partial pressure of oxygen in the arterial blood (PaO2)/FiO2 ratio information from the ABG analysis can gauge the degree of pulmonary shunting and predict impending respiratory failure. In addition, a carboxyhemoglobin blood level is readily available with arterial blood samples using a co-oximeter to assess systemic/asphyxiation injury.11

TREATMENT

Airway Management

Early airway management is crucial in patients with inhalation injury. Endotracheal intubation is indicated in patients who have evidence of upper airway edema, decreased mental status, and signs of deteriorating respiratory status. Airway edema increases over 12-18 hours after injury and early recognition of airway deterioration from mild pharyngeal edema to complete airway obstruction is important.9 However, prophylactic intubation without good indications should be avoided because intubation may otherwise increase pulmonary complications in burn patients.7

Pharmacologic Intervention

Carbon monoxide management. An elevated carboxyhemoglobin level is an indication of carbon monoxide toxicity/exposure. Carbon monoxide poisoning bind competitively to hemoglobin and reducing oxygen carrying capacity resulting to hypoxia. High flow 100% oxygen via facemask or 100% oxygen by means of an artificial airway should be administered.2 Patient with evidence of carbon monoxide exposure maybe predispose to cardiac injury; thus, may require electrocardiogram and cardiac enzymes measurement.2

Cyanide management. The management of cyanide exposure remains controversial. However, an antidote such as hydroxycobalamine is used to manage cyanide exposure.7 Hydroxycobalamine binds cyanide by forming cyanocobalamin and is directly excreted by the kidney. The administration of this drug may be associated with side effects such as headache, allergic reactions, skin and urine discoloration, hypertension, or reflex bradycardia.

Beta-agonists. Another cause of impaired oxygenation in patients with inhalation injury is due to severe bronchoconstriction from increase airflow resistance and peak airway pressure. Beta-agonists are bronchodilators, and have the potential to improve patient outcomes by multiple mechanisms. Beta- agonists such as ipratropium and albuterol possess several properties that can improve dynamic compliance, promote bronchial smooth muscle relaxation, provide anti-inflammatory properties, and improve airspace fluid clearance thereby improving gas exchange.12

Aerosolized heparin. Airway obstruction is one of the major causative factors profoundly contributing to pulmonary dysfunction secondary to burn and smoke inhalation. The obstructive cast material is composed of cellular debris, neutrophils, mucus, and fibrin. The fibrin content solidifies and formed a firm cast that is hard to remove even by aggressive airway toilet resulting to airway obstruction.13 The benefit of using aerosolized heparin is to inhibit airway fibrin clot formation without systemic anticoagulation effect, thereby preventing obstructive cast formation.9

Sedation. For patients requiring mechanical ventilation, the use of a sedative is needed to reduce anxiety, facilitate mechanical ventilation, and enhance patient safety from self extubation. Accidental removal of an endotracheal tube can be fatal because facial swelling occurs within 24-48 hours.7 Different sedation medications are available such as propofol, dexmedetomidine (Precedex), and midazolam. Sedation is commonly used to achieve patient comfort during mechanical ventilation. The use of sedation also carries risks attributed to prolonged use, patient tolerance, production of delirium, and other alterations in central nervous system function.

Opioid analgesic. Opioid analgesic is needed to control pain

from burn injury. Pain from burn injury generates unusually high levels of pain and opioid agonists are the most commonly and effective analgesic to treat burn pain.14 Pain may aggravate pulmonary dysfunction. In addition, if the patient is intubated, the presence of an endotracheal tube, invasive monitor devices, suctioning, and mechanical ventilation further increases a patient's discomfort and increases agitation. Administering opiates such as fentanyl as a continuous infusion are commonly used rather than on an as needed basis.

Non-Pharmacological Intervention

Mechanical ventilation. The management of inhalation injury has been largely supportive and providing mechanical ventilation support with pulmonary toilet has been the mainstay of intensive care.7 No standard mode of mechanical ventilation is used for the treatment of inhalation injury.9 However, the ventilator strategy selected must support oxygenation and ventilation and reduce ventilator-induced lung injury. The clinician must be familiar with the type of ventilator mode used designed to achieve respiratory support goal.2,4

The use of a Volumetric Diffusive Respirator (VDR) has been reported to facilitate a lung protective strategy by providing ventilation at lower mean airway pressures.4 After inhalation injury, lungs are less compliant and have increased airway resistance. Therefore, the primary goal of ventilation support must focus on recruiting and stabilized collapsed alveoli while preventing further lung injury.9

Compared to other conventional ventilator modes, the benefit of using High Frequency Percussive Ventilation (HFPV) such as VDR is that this mode improves oxygenation and provides adequate ventilation at lower peak pressures as well as promotes clearance of airway debris.9 The use of HFPV in inhalation injury was associated with a significant decrease in both overall morbidity and mortality in patients with less than 40% TBSA.5 The practice of using VDR in the management of inhalation injury has been limited by its complexity, lack of availability, labor intensive monitoring, and maintenance requirements.8

Therapeutic bronchoscopy. Bronchoscopy can also be used therapeutically to clear obstruction and remove mucus plugs that may impede ventilation as well as obtain bronchial fluid for culture. In addition, bronchoscopy can be done as clinically indicated based on the patient's pulmonary/oxygenation status and to assess progression or improvement of lung injury.11

Monitoring for complications. Inhalation injury increases the risk of developing respiratory failure from acute lung injury/ARDS and pneumonia. The cardiopulmonary hemodynamics, pulmonary status, and gas exchange of the patient should be frequently re-evaluated to trend any progressive changes and provide timely intervention. The clinician needs to monitor for complications such as ICU delirium, infection (wound, respiratory, device acquired infection), and multi-organ dysfunction. Evaluation of the patient's response to treatment should be an ongoing assessment.

BURN CENTER ROLE

Burn center referral criteria have been established by the American Burn Association (ABA) to guide healthcare providers and improve patient outcomes. The presence of inhalation injury is one of the criteria identified for burn center referral. Inhalation injury is highly fatal if not treated early and appropriately. Burn centers have the dedicated resources, rehabilitation, and support services that may not routinely available at non-burn centers as well as being staffed by a highly trained healthcare team that specializes in this type of injury. Burn center care has been associated with improved survival, and shorter lengths of stay.15

EDUCATION AND COUNSELING

The patient and family must be educated about the recovery process, long term outcomes, and impact on physical and neurocognitive function as well as potential pulmonary complications. The patient's family is the primary support and should be included in all aspect of care and recovery for the patient. Patients receiving prolonged ventilator support, may need psychological counseling and emotional support to assist the patient in developing effective coping strategies. Family conferences with the multidisciplinary team should be held to discuss the patient's status/progress, discharge planning, and expected needs after hospitalization as well as long term goals and outcomes. In a patient with prolonged ventilator use and immobility, an in-patient rehabilitation or long term acute care program should be considered to provide continued pulmonary and physical rehabilitation after hospitalization.

LONG TERM IMPLICATIONS

The impact of inhalation injury is influenced by the patient's premorbid state, degree of injury and its sequelae. These factors are independent of age and total burn area involved.8

Depending on the extent of the inhalation injury, a patient may require longer ventilator support resulting in a tracheostomy. Longer use of sedation, opioids, and additional adjunct therapy may result in longer immobility and muscle weakness. Inhalation injury and its sequelae also have physical/physiological and psychosocial effect on patient's recovery.

Limited data exist regarding the long term impact of severe inhalation injury on pulmonary function or quality of life in burn survivors from various treatment regimens.8 However, patients with inhalation injury are at risk for long term glottic, subglottic, laryngeal, and trachea-bronchial complications and chronic/restrictive airway disease from the combination of lung injury, intubation, infection, and chronic inflammation.4,9 The risk increases with extended days of intubation.9

Long term follow-up should include pulmonary function tests and laryngeal examination to detect early complications and treatment.9 Inhalation lung injury decreases exercise endurance.8 The patient who has limited physical activities from prolonged hospitalization, and the presence of tracheostomy has significant risk factors for depression. Patients should be monitored closely for signs and symptoms of depression, and pharmacologic and behavior therapy should be initiated when appropriate. Referral to counseling for psychological support may also be considered.

Limited data have been reported regarding the long term effects from the various therapies provided with inhalation injury such as effects and treatment of toxic gases, inhalation therapies, ventilator support, systemic therapies, airway maintenance, and anti-inflammatory therapies. Several studies have examined the efficacy of the treatment regimen during the hospitalization period, but not the long-term outcome.8 Long-term goals should include return of optimal pulmonary function, prevention of pulmonary complication, and physical function and performance improvement.

SUMMARY OF HOSPITAL STAY

Patients with inhalation injury greatly increase the incidence of pulmonary complications. On ICU day 2, this patient developed moderate ARDS based on the onset, severity of hypoxemia, and radiographic result (Berlin definition for ARDS). The patient demonstrated evidence of respiratory deterioration and hemodynamic instability and required escalating ventilator and vasopressor support on ICU day 2 (P/F ratio of 178, bilateral pulmonary infiltrates on chest x-ray). To improve oxygenation and enhance mobilization of secretion, the ventilator mode was changed to the volumetric diffusive respirator mode. The BAL culture grew Staphylococcus aureus, and the patient was treated with vancomycin and piperacillin/tazobactam for 10 days.

The patient responded well to the combination of different treatment modalities including therapeutic bronchoscopy, use of HFPV by means of VDR, pharmacological adjuncts with inhalation therapy, and aggressive pulmonary hygiene therapy. On ICU day 5, she was weaned from vasopressor support, her chest x-ray showed decreasing bilateral pulmonary infiltrates, and she had a decreased FiO2 requirement with increasing PaO2/FiO2 ratio of 210. The patient demonstrated improvement of pulmonary status and lung injury per bronchoscopy after 10 days of aggressive pulmonary treatment. She slowly recovered from ARDS, and the burn wound area to the left upper extremities and upper chest wall healed well. She tolerated slow weaning of FiO2 requirement to 40%. The patient's ventilator status has improved, but she had difficulty weaning from the mechanical ventilator support.

On ICU day 13, she underwent a tracheostomy. With continued pulmonary hygiene and physical/occupational therapy, her respiratory status continued to improve and she was weaned from the ventilator to tracheostomy collar on ICU day 17. The variable effects of hypoxia and carbon monoxide toxicity contributes to longer hospitalization and recovery. She was discharged to an in-patient rehabilitation facility on ICU day 18 for continued pulmonary and rehabilitation care. The patient did not have any further complications after discharge to in-patient rehabilitation. Her respiratory function gradually returned to normal, and the tracheostomy was decannulated on hospital day 28. With continued physical rehabilitation, she demonstrated increase physical function and exercise tolerance due to prolonged hospitalization. The patient was discharged to her home 1 month later. She was to be followed by her primary care physician 3 days after discharge from the hospital. The patient will have a pulmonary function test at a 3-month and 6-month follow up examination.

The care and management of patient with inhalation injury can be highly complex necessitating the participation of an interprofessional team of clinicians including nurses, advanced practice nurses, physicians, respiratory therapists, physical/occupational therapists, nutritionists, case manager/social workers, pharmacists, and other ancillary

staff. Early engagement of family should also be included in the short and long-term goals to improve a patient's compliance and support. Respiratory and physical/occupational therapy, nutrition, and aftercare are the key components of the long-term recovery of this patient. Collaboration is an essential function of an APN to deliver the highest quality of care. The APN can facilitate interprofessional collaboration to provide a comprehensive plan of care as well as supporting the transition of care to ensure optimal patient outcome.

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