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Diagnosis and Management of Inhalation Injuries in U.S. Children

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Abstract
Inhalation injuries are the leading cause of mortality in fire related deaths in children, who because of their small and fragile airways have a greater risk of mortality than their adult counterparts. Direct injury to the lung parenchyma can be caused by a combination of heat and chemical damage, and their secondary effects. **Diagnosis.** Fiberoptic bronchoscopy is the current diagnostic standard when determining the presence and severity of an inhalation injury Chest CT (computed tomography) has been described as an adjunct to identifying parenchymal lung damage in inhalation injury patients. **Management.** The benefits of high tidal volume ventilation in this patient population include decreased ventilation days, decreased ARDS, and decreased atelectasis [The goals of medical therapy in patients with an inhalation injury are: 1) decrease bronchospasm, and 2) decrease airway edema. Bronchodilators are the mainstay of medical therapy for inhalation injuries. Chest physiotherapy including suctioning, coughing techniques, and early mobilization can be beneficial for patients with inhalation injuries. **Summary.** Most children who sustain a major burn injury survive. Although those who sustain an inhalation injury are at greater risk for morbidity and mortality, modern modes of airway management and ventilatory support generally result in good outcomes.

Keywords: *Inhalation injury, burn, children, pediatric, intubation, respiratory support*

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Introduction

Most pediatric burn injuries are minor and can be treated by any caregiver. Larger burns can cause significant morbidity and, if not treated appropriately, can lead to significant physiologic derangement, end organ damage, even death. The presence of an inhalation injury can markedly increase the risk of death, depending on the severity of the burns and the age of the patient.

Incidence

Inhalation injuries are present in 10 to 30% of all burn inpatients and are associated with a 16% greater risk of mortality for the same size burn [1, 2, 3, 4, 5]. They are the leading cause of mortality in fire related deaths in children [1, 4], who because of their small and fragile airways have a greater risk of mortality than their adult counterparts. In 2017, a total of 3,645 Americans died from fire/smoke inhalation injuries, of which 314 were children between the ages of 0–14 years [6, 7].

Mechanisms of Inhalation Injury

Inhalation injuries can damage the upper and lower airways, lung parenchyma and entire pulmonary system through a variety of mechanisms. Upper airway injuries (supraglottic) are usually caused by direct thermal injury [8]. Patients typically present with redness and swelling of the oropharynx, increased respiratory secretions, hoarseness and carbonaceous sputum,

which may herald symptoms of airway swelling and lead to airway obstruction [9, 8].

Lower airway injuries (infraglottic) are primarily caused by products of combustion, or chemical injury [9, 5]. The products of combustion that are most injurious include halogen acids, aldehydes, ammonia, hydrogen sulfide, sulfur dioxide, phosgene, nitrogen dioxide, and organic nitriles [10, 11]. Particulate matter can lead to increased inflammation in the lower airways, especially if the material is smaller than 5 mm in diameter [9, 10]. The specific mechanisms of injury in the lower airways involve: 1) increased bronchial blood flow causing increased pulmonary edema; 2) cast formation from solidified goblet cell secretions; 3) release of reactive oxygen species (ROS) causing broncho-constriction and irritation; 4) cilia loss causing increased airway plugging, cast build-up, increased bacterial load, and increased epithelial sloughing; and 5) increased inflammation via interleukin-1 (IL-1) release [12, 8, 13, 14, 15]. The classic presentation of a lower airway inhalation injury includes increased work of breathing, crackles/wheezes and decreased breath sounds on exam, together with low oxygen saturation [8].

Direct injury to the lung parenchyma can be caused by a combination of heat and chemical damage, and their secondary effects. These mechanisms include: 1) pulmonary edema secondary to increased vascular permeability; 2) alveolar collapse as a result of pulmonary edema; 3) cellular injury from ROS; 4) obstruction and occlusion by debris and edema; 5) decreased surfactant levels; 6) atelectasis; and 7) decreased phagocytosis by macrophages [9, 8, 13]. Together, these processes contribute to ventilation perfusion mismatch and diminished lung compliance [8, 5]. Parenchymal lung injury increases the risk of pneumonia in this patient population, especially in the setting of decreased phagocytosis by pulmonary macrophages [13]. Inhalation injury can also result in systemic toxicity via carbon monoxide and cyanide poisoning, which are discussed below [11].

Diagnosis

On clinical exam, there are specific stigmata suggestive of an inhalation injury. These include burns on the face, singed nasal hairs, bronchorrhea, sooty sputum, and wheezing or rales [5]. Fiberoptic bronchoscopy is the current diagnostic standard when determining the presence and severity of an inhalation injury [5]. The severity of inhalation injury is determined based on the "Abbreviated Injury Score" developed by Endorf and Gemeilli, which ranges from 0 to 4 (0, no injury; 4, massive injury) [16, 17]. Patients with higher abbreviated injury scores have increased mortality rates, ventilation days, and ICU lengths of stay [16, 18].

Chest CT (computed tomography) has been described as an adjunct to identifying parenchymal lung damage in inhalation injury patients. A grading severity system called the RADS score (Radiologist's Score) was developed in an ovine model, to assess the extent of inhalation injury [19, 20]. The RADS score provides 0 points for normal appearing parenchyma, 1 point for increased interstitial lung markings, 2 points for ground glass opacification, and 3 points for consolidation [19]. This grading system is applied to each quadrant of the left and right lung fields. In a study by Oh et al, an admission CT RADS score >8 and abnormal findings on bronchoscopy were associated with inhalation injury [19]. The advantage of using chest CT in the evaluation of inhalation injury is its more thorough assessment of the lung parenchyma and distal airways. CT can also detect lung injury as early as six hours after insult [20]. Other modalities to evaluate for inhalation injury include Xenon 133 lung scans and pulmonary function tests; however, these diagnostic methods are seldom used in the acute setting and are only included here for their historical significance.

Management

Airway Management

Bronchoscopy has diagnostic and therapeutic purposes in the management of patients with inhalation injuries. The severity of mucosal injury can be gleaned from diagnostic bronchoscopy, and therapeutic bronchoscopy can be used to clear secretions and remove particulate matter from the upper and lower airways [21]. In fact, multiple bronchoscopies may be necessary for airway clearance and symptomatic relief.

Initial management of inhalation injuries includes airway assessment and 100% high flow oxygen via mask. Early intubation may be necessary, especially in patients with large and deep burns, burns to the face, and clinically significant smoke inhalation injury. These types of patients should be intubated early, because the airway can swell to the point of total obstruction within 12 to 24 hours following injury. In 2011, the American Burn Association (ABA) outlined the following criteria for intubation: presence of "full

facial burns, stridor, respiratory distress, swelling on laryngoscopy, upper airway trauma, altered mentation, hypoxia/hypercarbia, and hemodynamic instability" [22]. The Denver Criteria were recently introduced by Badulak et. al. [22], adding singed facial hair and suspected smoke inhalation to the ABA criteria. In a study in adult burn patients at the University of Colorado, the Denver criteria were found to have increased sensitivity in predicting the need for intubation [3]. In other studies, however, the presence of singed nasal hair on its own has not been found to be a predictor of early intubation [23]. Some studies cite a high clinical index of suspicion for inhalation injury or a TBSA (total body surface area) burn injury of >40% as indications for intubation [24]. There is no consensus on intubation criteria in pediatric inhalation injury patients.

Ventilation Strategies

There is no consensus on the preferred ventilation strategy in pediatric patients with an inhalation injury [21]. Current strategies include SIMV (Synchronized Intermittent Mandatory Ventilation), HFV (high frequency ventilation), HFPV (high frequency percussive ventilation), APRV (Airway pressure release ventilation), low or high tidal volume ventilation, and HFOV (high frequency oscillatory ventilation). In a survey of burn providers, SIMV was found to be more commonly utilized as a ventilation strategy at high patient volume burn centers, as compared to low volume centers [25]. In HFPV, breaths are delivered at a rate of up to 500–600 breaths per minute [25]. The advantages to using HFPV include lower airway peak pressures, increased functional reserve capacity (FRC), increased secretion clearance, and increased plug clearance [27, 28, 11]. Cioffi et al [29] compared HFV and HFPV in patients with an inhalation injury and found lower rates of pneumonia in the HFPV group [29]. APRV has been used in the adult population, however, there is limited information on its use in pediatric patients. One of the drawbacks to the use of APRV is that it can increase the mean airway pressure.

In the ARDS (acute respiratory distress) literature, there has been a rise in the use of low tidal volume ventilation, defined as 6–8 cc/kg. In the landmark ARDSnet study, however, patients younger than 16 years old and patients with TBSA burns >30% were excluded. When low tidal volume ventilation has been used in children with ALI (acute lung injury) or ARDS, there has not been a decrease in mortality when compared to high volume ventilation [28]. On the contrary, in one study in the pediatric burn population, high tidal volume ventilation was associated with fewer ventilation days but an increased mortality and increased peak inspiratory and plateau pressures, when compared to low tidal volume ventilation [15]. The benefits of high tidal volume ventilation in this patient population include decreased ventilation days, decreased ARDS, and decreased atelectasis [15]. The drawbacks to the use of high tidal volume ventilation include potentially higher mortality, increased incidence of pneumothorax, and increased likelihood of barotrauma [15]. Additional research is necessary to determine the optimal mode of ventilation for pediatric burn patients with inhalation injuries.

There is substantial literature recommending against the use of HFOV (high frequency oscillatory ventilation) [30, 27]. The disadvantages to HFOV include difficulty in clearing secretions, wide variations in lung recruitment, gas trapping, bronchospasm, and increased development of exudates and casts [30, 31]. Other drawbacks to the use of HFOV in children with inhalation injuries include difficulty administering nebulized therapy and a higher incidence of barotrauma [31].

Medical Management

The goals of medical therapy in patients with an inhalation injury are: 1) decrease bronchospasm, and 2) decrease airway edema [12]. Bronchodilators are the mainstay of medical therapy for inhalation injuries. They are grouped into several classes of medication, including nebulized beta 2 agonists (albuterol, salmeterol, and racemic epinephrine) and muscarinic receptor antagonists (tiotropium) [5]. Beta 2 agonists cause smooth muscle relaxation and can decrease peak and plateau airway pressures, decrease V/Q mismatch, and improve lung compliance [11]. Presently, the use of beta blockers is considered the standard of care. Muscarinic receptor antagonists such as tiotropium can also decrease airway pressures and decrease mucus production by inhibiting smooth muscle constriction and decreasing cytokine release [21,27, 15].

Mucolytic agents, such as N-acetylcysteine, form another class of medications that can be used to treat inhalation injuries. They have the potential to break down mucus and decrease free radical levels, however, they are uncommonly used in the management of pediatric inhalation injuries [27].

Inhaled heparin is another medication that has been described in animal and adult models to treat inhalation injury. Inhaled heparin is used to decrease fibrin and cast formation. In the adult literature, the use of inhaled heparin and N-acetylcysteine is associated with improved survival and improved lung injury severity scores [32]. Unfortunately, there are no large studies evaluating the use of inhaled heparin in pediatric burn patients.

Prophylactic steroid use is not recommended for patients with inhalation injuries. In fact, patients who have received steroids for an inhalation injury have been found to have higher mortality rates [2]. There are other rare therapies that have been described in animal models and small studies to treat inhalation injuries including tissue plasminogen activator (tPA) to breakdown casts, inhaled nitric oxide, and surfactant [38]. There are, however, few studies describing their use children and none are considered standard of care.

Respiratory Therapy

Chest physiotherapy including suctioning, coughing techniques, and early mobilization can be beneficial for patients with inhalation injuries [12]. Head of bed elevation to prevent aspiration pneumonia is also recommended [21]. Antibiotics for pneumonia prophylaxis are not indicated in this patient population. In severe cases with ARDS, prone positioning should be considered [21].

Labs

Initial blood tests include a complete metabolic panel, lactate, CO-oximetry and arterial blood gases. In severe burns, metabolic acidosis may be caused by hypoxia, inadequate resuscitation, or methemoglobinemia. High lactate levels, above 10 mmol/L, have been shown to be associated with cyanide poisoning [33].

CO toxicity

Carbon monoxide (CO) is an odorless, tasteless, and colorless gas that is a leading cause of death in house fires. CO binds to hemoglobin with a much higher affinity than oxygen, forming carboxyhemoglobin. CO binding to hemoglobin causes a leftward shift of the oxygen-hemoglobin dissociation curve, thus preventing unloading of oxygen in the tissues, resulting in hypoxia and ischemia. Physicians should have a low threshold to test for CO toxicity, such as a history of combustion exposure (house fire, charcoal or gas grill, improper gas or oil heating). Symptoms of CO toxicity often start as nonspecific neurological symptoms and may include headache, confusion, fatigue, dizziness, nausea, and vomiting. They can quickly progress to hallucinations and a comatose state [34]. Standard pulse oximetry (SpO_2) is not a reliable screen for CO exposure, because it does not differentiate carboxyhemoglobin from oxyhemoglobin [29].

The diagnosis of carbon monoxide toxicity should be confirmed by an elevated carboxyhemoglobin as measured by CO oximetry on a blood gas, but it is important to note that carboxyhemoglobin levels do not correlate precisely with the degree of CO poisoning. The role of imaging studies is unclear in diagnosing CO toxicity, but computed tomography (CT) of the head can aid in ruling out other causes of neurological decompensation. There are rare reports of finding hemorrhagic infarction of the globus pallidus and the deep white matter on head CT or MRI (magnetic resonance imaging) in the setting of delayed neuropsychiatric syndrome (DNS). DNS has been reported in up to 40 percent of patients with significant CO exposure and can arise 3 to 240 days after initial recovery [35]. DNS is characterized by variable degrees of cognitive deficits, personality changes, movement disorders, and focal neurologic deficits.

The initial management of CO toxicity should focus on rapid administration of 100% fractional inspiration of oxygen ($Fi0_2$). The efficacy of hyperbaric oxygen in the management of CO toxicity remains unclear, but it should be considered in children with severe metabolic acidosis or evidence of end-organ ischemia.

Cyanide toxicity

Cyanide exposure is caused by the combustion of synthetics including plastics, foam, varnish, paints, wool, and silk. Cyanide toxicity should be suspected if a person involved in a closed-space fire presents with a decreased level of consciousness, low blood pressure, and/or high blood lactate level. The incidence of cyanide toxicity is underestimated, and the symptoms associated with cyanide toxicity are similar to inhalation injuries [1]. Patient may present with dyspnea, tachypnea, vomiting, bradycardia, hypotension, giddiness, coma, and/or seizures. One of the pathognomonic characteristics of cyanide poisoning is the smell of bitter almonds on the patient's breath, but this symptom is not always present. Cyanide toxicity is dose dependent and the mechanism of action involves cyanide binding to

cytochrome c oxidase, resulting in electron uncoupling in the mitochondria. It can be difficult to diagnose, because there is no rapid assay for its detection. Clinical features associated with cyanide toxicity include elevated lactate levels (>10 mmol/L), metabolic acidosis, and an elevated mixed venous saturation on blood gas. Patients who are worked up for cyanide toxicity should also be evaluated for carbon monoxide poisoning.

The treatment for cyanide toxicity is supportive care with high flow oxygen, correction of the metabolic acidosis, and administration of a cyanide antidote kit or hydroxocobalamin. The cyanide antidote kit is available at most institutions and comprises amyl nitrate pearls, sodium thiosulfate, and amyl nitrite [21]. The mechanism of action of amyl nitrate pearls and sodium nitrate is induction of methemoglobin, while sodium thiosulfate induces the conversion of cyanide into thiocyanate and facilitates renal excretion. Adverse effects of the components of the cyanide antidote kit include hypotension, gastrointestinal irritation, and injection site reactions. Hydroxocobalamin is another treatment for cyanide toxicity and functions by binding with cyanide to form cyanocobalamin. It has been widely used for the treatment of cyanide toxicity in adults and children [1].

Outcomes

Advances in care burn care are allowing greater numbers of children with severe burns and inhalation injuries to survive. The mortality rate for pediatric burns is low, approximately two percent [36]. Pediatric patients cared for at high patient volume burn centers have been shown to have improved outcomes, including lower mortality rates [36]. The most common complication that arises is respiratory tract infection, specifically pneumonia, which has been found to increase mortality following an inhalation injury [37, 11].

Most pediatric patients do not suffer long-term functional disability following inhalation injury; however, rare long-term sequelae include subglottic stenosis (secondary to thermal injury or pressure injury from prolonged intubation and/or an over-inflated endotracheal balloon), tracheal stenosis, and bronchiectasis [11]. Over the past few decades, pediatric patients with severe burn injuries have seen a significant improvement in overall survival. The prompt recognition and treatment of inhalational injuries remains paramount to the successful management of these children.

Summary

Most children who sustain a major burn injury survive. Although those who sustain an inhalation injury are at greater risk for morbidity and mortality, modern modes of airway management and ventilatory support generally result in good outcomes. Thus, the current focus of pediatric burn care is to optimize the child's functional, cosmetic, and psychological outcomes. To that end, there may be severe psychological and emotional hurdles that must be overcome. Fortunately, there are support organizations and burn camp programs to aid in recovery. Burn injured children who participate in these programs learn they are more alike than different, and their burn injury does not define them. They are defined by their story: their approach to life, how they overcome obstacles, and their confidence in the face of hardship.

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