Inhalation Injury
Pathophysiology, Diagnosis, and Treatment

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KEYWORDS

• Inhalation injury • Pneumonia • Respiratory failure • Bronchodilators • Heparin

KEY POINTS

- Determinants of mortality in burns are size of burn, age, and the presence of inhalation injury.
- Inhalation injury with or without cutaneous burn increases morbidity and mortality for burn survivors.
- Resuscitation efforts are significantly altered by the presence of inhalation injury.
- There is no consensus among leading burn centers on the optimal mechanical ventilation modes for these patients. Supportive care remains the mainstay of treatment.
- Despite research gains in nutrition, and the hypermetabolic response to burn injury, there remains a lack of understanding of the pathophysiology of inhalation injury and the long-term physiologic consequences.

INTRODUCTION

There is no greater trauma than a large burn. No single injury affects more organ systems than a severe burn injury. The subsequent supraphysiologic responses to that injury lead to full-body catabolism and increased morbidity and mortality. Advances in critical care management, nutrition, wound coverage, and antimicrobial therapies have substantially improved outcomes for burn survivors regardless of burn size. However, when burns are accompanied by inhalation injury, health care providers and clinical scientists have yet to make major impacts on survival.

Inhalation injury is present in up to one-third of all burn injuries; however, it accounts for up to 90% of all burn-related mortality.[1–3] Inhalation injury causes localized damage via direct cellular damage, changes in regional blood flow and perfusion, airway obstruction, as well as toxin and proinflammatory cytokine release.[2,4] Inhalation injuries significantly incapacitate mucociliary clearance and impair alveolar macrophages.[5] They predispose patients to bacterial infection, specifically and primarily pneumonia, a leading cause of death for patients with burns.[6,7] Burn critical care units have the highest rates of ventilator-associated pneumonia in the country, and those patients with concomitant cutaneous injuries with inhalation injury have double the rates of ventilator-associated pneumonia.[5] Moreover, the probability of death increases from 40% to 60% when a burned patient with inhalation injury has pneumonia compared with patients with just cutaneous injuries.[8]

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PATHOPHYSIOLOGY

The mechanism of destruction can be classified in 4 ways: (1) upper airway injury, (2) lower airway injury, (3) pulmonary parenchymal injury, and (4) systemic toxicity. The extent of damage from an inhalation injury depends on the environment and the host: the source of injury, temperature, concentration, and solubility of the toxic gases generated, and the response to that injury by the individual.[9] Inhalation injuries cause formation of casts, reduction of available surfactant, increased airway resistance, and decreased pulmonary compliance,[10] leading to acute lung injury and acute respiratory distress syndrome (ARDS).[11]

The major pathophysiology seen in upper airway inhalation injuries is induced by microvascular changes from direct thermal injury and chemical irritation.[6] The heat denatures protein, which subsequently activates the complement cascade causing the release of histamine.[9,12] Subsequently, there is the formation of xanthine oxidase and release of reactive oxygen species (ROS), which combine with nitric oxide in the endothelium to induce upper airway edema by increasing the microvascular pressure and local permeability.[9,13,14] Proinflammatory cytokines, ROS, and eicosanoids attract polymorphonuclear cells to the area, further amplifying ROS and signaling proteases.[15–17] There is a substantial increase in microvascular hydrostatic pressure, a decrease in interstitial hydrostatic pressure, and an increase in interstitial oncotic pressure.[9] The hallmark of burn resuscitation is the administration of large amounts of crystalloid, which reduces plasma oncotic pressure affecting the oncotic pressure gradient in the microcirculation causing significantly more airway edema.[9] Barring steam inhalation injuries and blast injuries, the upper airway efficiently protects the lower airway via heat exchange to limit distal damage to the lower airway.

Injury to the lower airway is caused by the chemicals in smoke. The heat capacity of air is low and the bronchial circulation very efficient in warming or cooling the airway gases, so that most gases are at body temperature as they pass the glottis.[18] In order to induce thermal injury to the airway, flames must be in direct contact.[19] Accelerants, or burned biological materials, are caustic to the airways and induce an initial response to trigger a proinflammatory response. There is a 10-fold increase in bronchial blood flow within minutes of an inhalation injury,[20] which is sustained and causes increased permeability and destruction of the bronchial epithelium.[9] There is a subsequent increase in pulmonary transvascular fluid and a decrease in Pa_{O_2}/fraction of inspired oxygen (Fi_{O_2}) ratio less than or equal to 200 nearly 24 hours after injury.[21] There is a subsequent hyperemia of the tracheobronchial tree and lower airways, and that very prevalent clinical finding is used to diagnose the injury.[22–25] Early in the injury, the secretions from goblet cells are copious and foamy. In hours to days these secretions solidify, forming casts and airway obstruction.[9]

Changes to lung parenchyma are delayed, and depend on the severity of injury and the patient's response to the injury. Parenchymal injuries are associated with an increase in pulmonary transvascular fluid, which is directly proportional to the duration of exposure of smoke and toxins. As stated previously, injury to the lower airways and lung parenchyma is rarely caused by direct thermal contact. Only steam can overcome the very efficient upper airway heat dissipating capabilities.[6] There is a reduction to the permeability of protein, an increase in the permeability to small particles, an increase in pressure in the pulmonary microvasculature pressure, and a loss of hypoxic pulmonary vasoconstriction.[9] The key pathologic derangements in inhalation injury are edema, decreased pulmonary compliance from extravascular lung water and pulmonary lymph, and immediate inactivation of surfactant. There is a subsequent ventilation-perfusion mismatch that can lead to profound hypoxemia and ARDS.[6]

Systemic toxic changes are caused by the inhalation of chemicals and cytotoxic liquids, mists, fumes, and gases. Smoke combines with these toxins and increases mortality by increasing tissue hypoxia, metabolic acidosis, and decreasing cerebral oxygen consumption and metabolism.[26,27]

DIAGNOSIS

In the past, the diagnosis of inhalation injury has rested on both subjective and objective measures. History and physical are important factors because they may help prognosticate the host response and comorbidities. For example, the elderly population that is unable to escape from danger may have prolonged exposure to smoke and toxins. Key factors in diagnosis from history are the mechanism (flame and smoke or steam), exposure (duration), location (enclosed space), and disability. For the physical examination, facial burns, singed nasal or facial hair, carbonaceous sputum, soot, stridor, or edema.[6,23] There are no changes in chest radiograph on admission. Oxygen saturation by pulse oximetry (Sp_{O_2}) is usually not initially affected and may be misleading, even in the presence of carbon monoxide poisoning, in which the Sp_{O_2} is typically normal. Similarly, arterial blood gases are nondiagnostic. Even in