Inhalation Injuries

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Des Moines
4/21/16
Objetives

• Discuss the types of inhalation injury
• Describe the pathophysiology of inhalation injury
• Outline the principles of the management of inhalation injury
Disclosure

I have nothing to disclose....

Except...

A strong bias towards ensuring quality and safe care to our patients
• Pathophysiology
• Clinical features
• Diagnosis
• Initial management
• Subsequent management
The Problem

- Treatment of inhalation injury has not kept pace with improvements in the care of cutaneous burns
  - Other mechanism of injury to the lung
  - Lack of uniform diagnosis criteria
- Improved mortality over the years
Inhalation Injury

• Damage to the respiratory tract or lung tissue
  – Heat
  – Smoke
  – Chemicals

• Fires – Leading cause of death
Epidemiology

- 372000 fires in residential buildings
  - 2530 deaths
  - 13125 injuries
  - 7 Billion in property loss
- Inhalation injury responsible for 77% of all deaths – CO poisoning
- Independent mortality predictor in burn patients
Notable Events Leading to Smoke Inhalation Knowledge

World War I
Cleveland Clinic Fire, 1929
Use of poisonous gases and the effect on the lungs
Effect of the inhalation of volatile products from burning X-ray film

Coconut Grove Fire, 1942
Effect of volatile products in smoke causing early and late respiratory distress, initially from upper airway compromise and then airway plugging

Mid-1940s World War II
Pathophysiologic; time, course, and treatment using respiratory assistance

1950s–1960s
Vietnam War, 1960s
Development of blood–gas monitoring and intensive care
Identification of adult respiratory distress syndrome caused by alveolar capillary membrane damage

1970s
Better understanding of smoke inhalation causing post-traumatic pulmonary insufficiency (PTPI, ARDS), improved ventilator management, and the toxicology of smoke

1980s–1990s casualties from hotel fire in Las Vegas and Kings Cross Underground Station Fire
Role of the effect of airway inflammation in smoke inhalation injury

2000s mass casualties, World Trade Center Disaster 2001
Long-term effects of smoke exposure changes in airways epithelium

Rhode Island Nightclub Fire, 2003
Biochemical and cell biologic changes; improved ventilatory strategies
Pathophysiology

- Complicates burns in 10-20% of patients
- Thermal injury is usually limited to supraglottic structures
- Injury to lower airway is chemical in nature
- Degree of injury
  - Gas components inhaled
  - Presence of particulate matter
  - Magnitude of exposure
  - Individual host factors
Smoke Toxicity

• Asphyxiation
• Systemic toxicity
• Direct effect on respiratory tissue
Combustion Gases

- CO
- Hydrogen Cyanide
- Halogen acids
- Formaldehyde
- Unsaturated aldehydes
Particles containing:

- HCl
- Aldehydes
- Acrolein
- O₂ radicals
- NO₂
- NH₃OH

Water-soluble gases:

- CO₂
- SO₂
- Cl₂
- NH₃

Mucosal edema
Ulceration
Ciliary damage
Cell-membrane damage

Surfactant inactivation damage

Absorbed toxins
Carbon Monoxide
Cyanide
Pulmonary edema
Diagnosis

• Subjective and clinical findings
• Exposure to flame, smoke, or chemicals, duration, enclosed space, and loss of consciousness or disability
• Facial burns, singed facial or nasal hair, carbonaceous material on the face or in the sputum, and signs of airway obstruction including stridor, edema, or mucosal damage
**Table 1 Bronchoscopic criteria used to grade inhalation injury**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0 (No Injury):</td>
<td>Absence of carbonaceous deposits, erythema, edema, bronchorrhea, or obstruction.</td>
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<tr>
<td>Grade 1 (Mild Injury):</td>
<td>Minor or patchy areas of erythema, carbonaceous deposits in proximal or distal bronchi. [any or combination]</td>
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<tr>
<td>Grade 2 (Moderate Injury):</td>
<td>Moderate degree of erythema, carbonaceous deposits, bronchorrhea, with or without compromise of the bronchi. [any or combination]</td>
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<tr>
<td>Grade 3 (Severe Injury):</td>
<td>Severe inflammation with friability, copious carbonaceous deposits, bronchorrhea, bronchial obstruction. [any or combination]</td>
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<tr>
<td>Grade 4 (Massive Injury):</td>
<td>Evidence of mucosal sloughing, necrosis, endoluminal obliteration. [any or combination]</td>
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Endorf and Gamelli [25].
*Reproduced with permission from J Burn Care Res and Endorf, et al.*
Upper Airway Injury

• Above vocal cords
• Thermal Injury
• Erythema, ulcerations, and edema
• Ciliary dysfunction
• Increased production of thick secretions
• Complicated airway management
Tracheobronchial Injury

- Cause by chemicals in smoke
- Persistent coughing and wheezing, soot-containing airway secretions, increased work of breathing, hypoventilation
- Increased production of neuropeptides
  - Bronchoconstriction, increased vascular permeability, and vasodilation
- Loss of bronchial epithelium
- Ventilation-perfusion mismatch as a primary mechanism of hypoxemia
Parenchymal Injury

- Delayed
- Time difference / Severity of Injury
- Atelectasis, increased transvascular fluid flux, decrease in surfactant, impaired oxygenation
- Decreased antifibrinolytic activity
- Pneumonia
Systemic Toxicity

- Headaches
- Nausea
- Dizziness
- Breathlessness
- Collapse
- Loss of consciousness
Systemic Toxicity

- CO poisoning
  - 15,000 emergency department (ED) visits and 500 unintentional deaths each year
CARBON MONOXIDE (CO) POISONING

CAN’T BE SEEN
CAN’T BE SMELLED
CAN’T BE HEARD
CAN BE STOPPED
• Interferes with oxygen transport
  • 200 x greater affinity to Hb
• Concentration, duration and underlying health status
  • >15 – Admit to hospital

• Carboxyhemoglobin
  • 250 min RA
  • 40 - 60 min 100% oxygen
  • Lower with hyperbaric oxygen
Increasing % of Carbon Monoxide in the Blood

Death (especially if not removed from exposure)

Seizure, Unconsciousness, Heart Attack

Confusion, Blurred Vision, Shortness of Breath, Pounding Headache, Uncontrolled “Sleep”, Vertigo, Loss of Coordination, Chest Pain, Memory Loss

Impaired Judgment, Difficulty Breathing, Blurring of Vision, Bad Headache, Increasing Drowsiness, Stomach Pain

Drowsiness, Headache, slight increase in Respiratory Rate, Dizziness

Slight Headache, Nausea

None

Typical Symptoms of Carbon Monoxide Poisoning.

Note: Headache is the most common symptom.

Caution: People experience different symptoms of different intensities and in different orders. This is only a guide.
Systemic Toxicity

• Hydrogen Cyanide
  – Nonspecific symptoms
  – Cyanide level
  – Unexplained lactic acidosis, low or declining \( \text{PaCO}_2 \), depressed level of consciousness, cardiac arrest, or cardiac decompensation
Other Irritants

Smoke
Chlorine
Phosgene
Ammonia
Sulfur
Chloramine
Methyl isocyanate
Chlorine

• Sanitation
• Bleaching

• Strong oxidizing element
  • Oxygen
  • Hydrogen chloride
Chlorine gas leak at Pimpri sewage plant affects 82

Siddharth Gaikwad, TNN | Jan 19, 2011, 06.11AM IST

PUNE: Panic gripped the residents of the Ramabainagar slum area near Bhatnagar in Pimpri on Tuesday morning after chlorine gas leaked from the municipal corporation-run sewage treatment plant (STP) located along the Pimpri-Chinchwad link road. At least 82 persons were affected due to the leak, which began at around 3 am. The residents complained of eye and throat irritation, breathing problems and vomiting. While 56 people were given medical treatment on the spot, 26 others had to be hospitalized but were discharged later in the evening.

The gas, which leaked from a one-tonne cylinder in the chlorinator room, spread into the slum located behind the sewage plant. Surprisingly, the workers at the plant realized that the gas was leaking only at 4.30 am. The workers threw the cylinder into water in an attempt to reduce the effects of the leak. However, this later caused problems in controlling the leak, fire-brigade personnel said.

Some workers at the plant suspected that someone could have attempted to steal the brass cork on the cylinder, which caused the leak. They said similar attempts to steal brass corks were reported in the past. However, municipal commissioner Ashish Sharma said negligence by the workers could be one of the likely reasons for the leak. The workers need to follow proper drills for handling the cylinders, he said, adding that the municipal corporation was probing the incident.
Phosgene

- Valued industrial gas
  - Pharmaceuticals
  - Organic compounds

- Increase in capillary permeability
- Hydrochloric acid
  - Alveoli cell death
  - Pulmonary edema
Ammonia

- Household cleaning
- Farming
- Industrial

- Ammonium Hydroxide
  - Tissue necrosis
  - Alveolar edema
Respiratory Support

• 20 to 33% experience some degree of upper airway obstruction due to pharyngeal edema
• Early ambulation, chest physiotherapy, airway suctioning, and therapeutic bronchoscopy
• Humidified 100 percent oxygen by face mask

The American Burn Association's Advanced Life Support protocol suggests that if there is any question about the security of the patient’s airway, the patient should be intubated prior to transfer to a trauma or burn center
Pulmonary Care

• Supportive
• Clean the airway !!
  – Bronchodilators
  – Mucolytics, aerosolized heparin
  – CPT, postural drainage
  – Bronchoscopy
Does a Nebulized Heparin/N-acetylcysteine Protocol Improve Outcomes in Adult Smoke Inhalation?

• Reduced morbidity, or were associated with increased survival
• Significantly reduced reintubation rates, incidence of atelectasis, and improved mortality
• Better lung compliance, less pulmonary edema, and less airway obstruction
• A placebo controlled clinical trial (NCT01773083) is pending
Does a Nebulized Heparin/N-acetylcysteine Protocol Improve Outcomes in Adult Smoke Inhalation?

Natalie S. Kashefi, MS
Jonathan I. Nathan, MD, MBA
Sharmila Dissanayke, MD, FACS

**Background:** Smoke inhalation is a major source of morbidity and mortality. Heparin and N-acetylcysteine treatment has potential efficacy in inhalation injury. We investigated the impact of a heparin/N-acetylcysteine/albuterol nebulization protocol in adult patients with inhalation injury.

**Methods:** A retrospective review was performed of adult inhalation injury patients, admitted to a regional burn center between January 2011 and July 2012, who underwent a protocol of alternating treatments of heparin and N-acetylcysteine/albuterol nebulization every 4 hours. The study cohort was matched 1:1 by age, sex, and burn size to a control cohort admitted within 5 years before protocol implementation.

**Results:** The study ($n = 20$) and control cohorts ($n = 20$) were well matched, with nearly identical age (50 vs 49 years), sex distribution (70% male), burn size (total body surface area, 22% vs 21%), and inhalation injury, except grade I injuries (79% vs 47%, $P = 0.01$). The protocol did not change mortality (30% vs 25%, $P = 0.72$) or duration of mechanical ventilation (8.5 vs 8.8 days, $P = 0.9$). There was no difference in development of sepsis (40% vs 23%, $P = 0.7$) or acute respiratory distress syndrome (15% vs 10%, $P = 1$); however, those who received the protocol were more likely to develop pneumonia (45% vs 11%, $P = 0.03$).

**Conclusions:** The implementation of a heparin/N-acetylcysteine/albuterol protocol did not reduce mortality or duration of mechanical ventilation in this cohort of adults with inhalation injury and resulted in a significant increase in pneumonia rates. Larger prospective studies are necessary, with close attention paid to minimizing the infection risk incurred from frequent administration of nebulized medications. (Plast Reconstr Surg Glob Open 2014;2:e165; doi: 10.1097/GOX.0000000000000121; Published online 10 June 2014.)

Smoke inhalation continues to be a major source of morbidity and mortality in burn patients, increasing the likelihood of mortality by as much as 25% despite the many advances in burn care over the past decades. Although inhalation injury is not uncommon among patients with burns, there remains no standard for its diagnosis, scoring, and subsequent treatment, which makes it difficult to evaluate the results of studies and treatments. For these reasons, inhalation injury was designated a top 10 research priority at the 2007 American Burn Association (ABA) Consensus Conference.

Smoke inhalation occurs through a variety of mechanisms, including thermal injury to the respiratory tract and the inhalation of toxic products of combustion.
Conclusions: The implementation of a heparin/N-acetylcysteine/albuterol protocol did not reduce mortality or duration of mechanical ventilation in this cohort of adults with inhalation injury and resulted in a significant increase in pneumonia rates. Larger prospective studies are necessary, with close attention paid to minimizing the infection risk incurred from frequent administration of nebulized medications. (Plast Reconstr Surg Glob Open 2014;2:e165; doi: 10.1097/GOX.0000000000000121; Published online 10 June 2014.)
Treatment

• Bronchoscopy
• Carbon Monoxide
  • Normobaric oxygen
  • Intubation
• Hyperbaric oxygen
  • Carboxyhemoglobin > 25%
  • Unconscious
• pH < 7.1
• Cyanide antidote kit
Noninvasive Ventilation

- Awake, cooperative, spontaneous breathing and able to protect airway

- More contraindication in burn patient

- Most serious complication - failure to recognize when therapy is inadequate
Lung-Protective Ventilation

• Vol - < 7ml/kg
• High PEEP
• Plateau pressure < 30 cm water

• Difficult
  – Fibrin casts
  – Extensive chest wall thermal injuries
  – High volumes of resuscitative fluid
High-frequency percussive ventilation

- Standard in many burn centers
- Improve secretion management
- Improve morbidity and mortality when burns <40% TBSA
Controversy
HTV ventilation may be better than traditional LTV ventilation

• Inhalation injury
• Fewer vent days
• Lower atelectasis and ARDS
• Higher pneumothorax

• Pediatric patients!!
APRV

• $P_aO_2/F_iO_2$ ratios were initially lower on APRV compared with conventional mechanical ventilation
• This equilibrated at 48 h
• Higher mean airway pressures needed to maintain oxygenation
• In the end, no survival difference was seen between APRV and conventional mechanical ventilation
• Tendency towards survival
  – < 200 hrs on ECMO
Medical Adjuncts

- Beta-agonist
- Pulmonary blood flow ligation
- Anticoagulants
- Anti-inflammatory agents
- Steroids
<table>
<thead>
<tr>
<th>TABLE 1 Advanced Burn Life Support (ABLS) burn center referral criteria</th>
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<tbody>
<tr>
<td>• Partial-thickness burns &gt;10% of total body surface area</td>
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<tr>
<td>• Burns on face, hands, feet, genitalia, perineum, or major joints</td>
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<tr>
<td>• Third-degree burns in any age group</td>
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<tr>
<td>• Electrical burns, including lightning burns</td>
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<tr>
<td>• Chemical burns</td>
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<tr>
<td>• Inhalation burns</td>
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<tr>
<td>• Burn injury in patients with preexisting medical conditions that could complicate management, prolong recovery, or affect mortality</td>
</tr>
<tr>
<td>• Any patient with burns and concomitant trauma (eg, fractures) in which the burn injury poses the greater risk of morbidity or mortality. If trauma poses the greater immediate risk, the patient may be stabilized in a trauma center before transfer to a burn unit. Physician judgment in these cases should reflect the regional medical control plan and triage protocols.</td>
</tr>
<tr>
<td>• Children with burns in hospitals without qualified personnel or equipment for pediatric care</td>
</tr>
<tr>
<td>• Burn injury in patients who will require social, emotional, or rehabilitative interventions</td>
</tr>
</tbody>
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Adapted from American Burn Association.³
Conclusion

• Inhalation injury remains a significant cause of morbidity and mortality
• Treatment of inhalation injury remains largely supportive
• Recent research has led to substantial gains in the understanding of the molecular pathophysiology of inhalation injury
• These advances as well as preclinical studies on targeted therapies provide hope for reversal of specific mechanisms of morbidity and improvement in outcomes