Joint Trauma System

Inhalation Injury and Toxic Industrial Chemical Exposure

Part of the Joint Trauma System (JTS) Clinical Practice Guideline (CPG) Training Series

Purpose

This CPG provides evidence–based guidelines for the management of the most common toxic industrial chemicals which lead to pulmonary injury.

Presentation is based on the JTS Inhalation Injury and Toxic Industrial Chemical Exposure CPG, 25 Jul 2016 (ID:25). It is a high-level review. Please refer to the complete CPG for detailed instructions. Information contained in this presentation is only a guideline and not a substitute for clinical judgment.

Agenda

1. Summary
2. Background
3. Evaluation and Treatment
4. Chlorine Overview
5. Phosgene Overview
6. Hydrogen Sulfide
7. Ammonia
8. Cyanide
9. Carbon Monoxide
10. Fire Suppressants
11. Performance Improvement (PI) Monitoring
12. References
13. Appendices
14. Contributors

Summary

- Patients with both burn and inhalation injury have significantly increased morbidity and mortality compared to those with burn injury alone.
- Care is often supportive.

Background

- Patients with both burn and inhalation injury have significantly increased morbidity and mortality compared to those with burn injury alone.
- During smoke inhalation, thermal and chemical injury are the primary initial toxicities.

Background

Chemicals come in a variety of irritants and asphyxiants

Mechanisms of Lung Injury of Gaseous Respiratory Irritants	
Irritant Gas	**Mechanism of Injury**
Ammonia (NH_2) Source: Agriculture, rain, plastic, explosive	Alkali burns
Hydrogen chloride (HCl) Source: Dyes, fertilizers, textiles, rubber, thermal degradation of polyvinyl chloride	Acid burns
Sulfur dioxide (SO_2) Source: Smelting, combustion of coal/oil, paper manufacturing, food preparation	Acid burns
Chlorine (Cl_2) Source: Paper textile manufacturing, sewage treatment	Acid burns, free radical
Oxides of nitrogen (NO, NO_2, N_2O_4) Source: Agriculture, mining, welding, manufacturing of dyes/lacquers	Acid burns, free radical
Phosgene ($COCl_2$) Source: Firefighters, welders, paint strippers, chemical intermediates (isocyanate, pesticides, dyes, pharmaceuticals)	Acid burns
Colors indicate water solubility – Red: High; Yellow: Intermediate; Green: Low	

Source: Medical Aspects of Chemical Warfare – Borden Institute

Evaluation & Treatment

- Toxic chemical inhalation injury treatment generally supportive, but some specific chemicals require **antidotes.**
  - Most critically ill patients require unique ventilation techniques used for Acute Respiratory Distress Syndrome (ARDS).
  - Patients are at a higher risk of developing ventilator-associated pneumonia.

Evaluation & Treatment

- ARDS management focuses on:
  - Airway management
  - Lung-protective ventilation strategies
  - Aggressive Pulmonary toilet
  - Avoidance of volume overload to prevent worsening pulmonary edema

Chlorine Overview

- Yellow-green gas with irritating smell commonly used in industry – found in industrial/chemical accidents and sometimes in IEDs
  - Dissolves in water to form hydrochloric and hypochlorous acids.
  - Clinical effect: tearing, skin burning, drooling, cough, shortness of breath, chest pain, hypoxia, respiratory distress.
  - If pulmonary toxicity, may worsen over days.

Photo by US Army – Dugway Proving Ground Public Affairs

Chlorine Overview

- Treatment: Skin decontamination, supplemental oxygen, beta agonists, and ARDS ventilatory techniques
  - Inhaled Corticosteroids (Fluticasone 200 mcg BID) may improve secondary outcomes and should be done if the patient requires intubation.
  - Consider IV steroids If unable to administer inhaled or has significant bronchoconstriction.

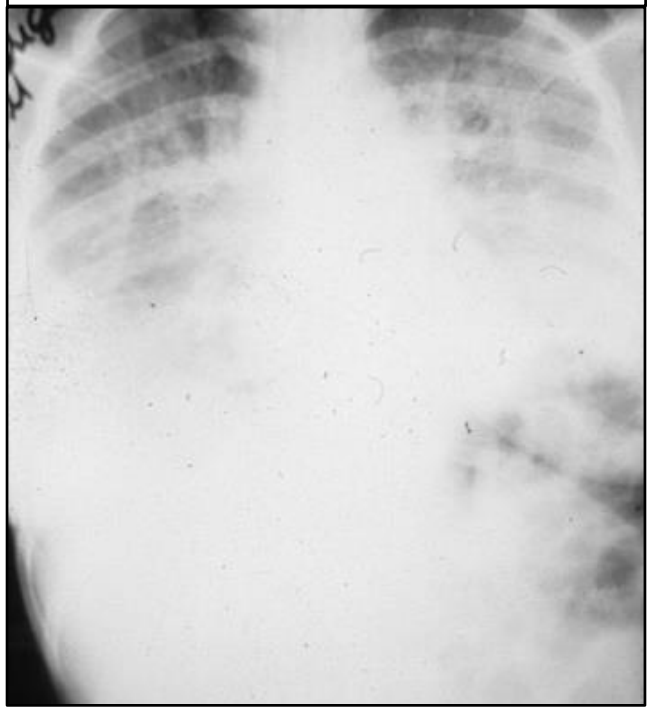
Phosgene Overview

- Sweet, pleasant, smell of mown hay – does not prompt escape
- Combustion of chlorinated hydrocarbons (welding, fires) and synthesis of solvents (degreasers, cleaners)
- Clinical effect: Delayed ARDS (up to a day)

Phosgene Treatment

- Treatment: Observation, supplemental oxygen, and ARDS ventilation techniques
- Decontamination typically not needed

Chest radiograph 2 hours post phosgene exposure. Patient died 6 hours post exposure.



Source: Medical Aspects of Chemical Warfare – Borden Institute

Hydrogen Sulfide

- Smells like rotten eggs
  - Exposure occurs in waste management, petroleum, natural gas industries, and asphalt/rubber factories
- Clinical effects
  - Low Concentrations: Skin and mucous membrane irritation
  - High Concentrations: Sudden loss of consciousness, seizure, myocardial ischemia, keratoconjunctivitis, and upper airway and pulmonary injury
- Treatment: Skin irrigation, supplemental oxygen, removal from exposure, **intravenous sodium nitrite (300 mg)**, and supportive care
  - Sodium nitrite associated with methemoglobinemia, and hypotension – infuse over 5-7 minutes

Ammonia

- Pungent odor
  - Common industrial and household cleaner – fertilizer, refrigerant, cleaning agent, plastic and explosive synthesis.
  - Often transported under pressure at sub-zero, liquid form
- Clinical effect: Tearing, skin irritation, eye pain/burning, severe upper airway irritation, and alkali skin burn
  - High concentrations or prolonged Exposure: Tracheobronchial and pulmonary inflammation, respiratory failure at 2-5 minutes of exposure
- Treatment: Skin and eye irrigation, alkali burn skin care, supplemental oxygen, ARDS ventilatory techniques, supportive care

Cyanide

- Colorless, often odorless or bitter almond smell
  - Manufacturing of pesticides and synthetic materials, metal extraction, and in chemical laboratories
- Clinical effects:
  - Early or mild effects: Dizziness, headache, nausea, and anxiety
  - Late or severe effects: Coma, seizure, respiratory depression, hypotension, tachycardia, ARDS, pulmonary edema
- Treatment: Oxygen, mechanical ventilation, rapid administration of **hydroxocobalamin** (5g over 15 minutes)
  - Second dose of hydroxocobalamin can be administered in patients with severe toxicity or poor clinical response

Carbon Monoxide

- Colorless and odorless
  - Combustion of carbon containing compounds – combustion engines and cooking stoves in enclosed spaces
- Clinical effects: confusion, stupor, coma, seizure, and myocardial infarction - may have normal PaO2 and SpO2 readings
  - CO levels traditionally measured using CO-oximeter in a blood gas sample
  - Newer non-invasive CO-oximetry may allow for early diagnosis and better monitoring
- Treatment: 100% oxygen (If available - hyperbaric oxygen therapy)

Fire Suppressants

- Generally a simple asphyxiant (displaces oxygen) often used in military vehicles during fires
  - Most common is HFC227 – Heptafluoropropane – colorless/odorless
  - Small amount can convert to hydrogen fluoride during a fire – which can result in rapidly progressive or fatal respiratory failure
- Clinical effect: shortness of breath, cough, or hypoxia
- Treatment: Supportive
  - If hypocalcemia present: nebulized calcium gluconate (1.5 ml of 10% Ca Gluconate in 4.5 ml water) every 4 hours until normalization of serum calcium
  - If no significant burns, can consider steroids

PI Monitoring

- **Intent (Expected Outcomes)**

  All patients who suffer severe toxic or chemical inhalation injuries will receive appropriate supportive care including intubation and mechanical ventilation when indicated.

- **Performance/Adherence Measures**
  - ❑ All patients with severe toxic or chemical inhalation injuries received appropriate supportive care, including intubation and mechanical ventilation.
  - ❑ Appropriate evaluation of the posterior pharynx and mucosal inflammation of the airway using standard bronchoscopy assessment.

- **Data Source**
  - ❑ Patient Record
  - ❑ Department of Defense Trauma Registry (DoDTR)

References (1 0f 2)

1. Albright JM, Davis CS, Bird MD, Ramirez L, Kim H, Burnham EL et al. The acute pulmonary inflammatory response to the graded severity of smoke inhalation injury. Critical care medicine. 2012;40 (4):1113-21.
2. Holstege CP, Kirk MA. Smoke inhalation. In: Goldfrank LR, ed. Goldfrank's toxicologic emergencies. 7th ed New York: McGraw-Hill, Medical Pub. Division; 2002:1469-1477.
3. Nelson LS. Simple asphyxiants and pulmonary irritants. In: Goldfrank LR, ed. Goldfrank's toxicologic emergencies. 7th ed ed. New York: McGraw-Hill, Medical Pub. Division; 2002:1453-1468.
4. Wang J, Zhang L, Walther SM. Administration of aerosolized terbutaline and budesonide reduces chlorine gas-induced acute lung injury. J Trauma. Apr 2004;56(4):850-862.
5. DeLange DW, Meulenbelt J. Do Corticosteroids have a role in preventingor reducing acute toxic lung injury caused by inhalation of chemical agents? Clinical Toxicology. Vol 49, no 2. February 2011. pp 61-71
6. Wang J, Abu-Zidan FM, Walther SM. Effects of prone and supine posture on cardiopulmonary function after experimental chlorine gas lung injury. Acta Anaesthesiol Scand. Oct 2002;46 (9):1094-1102.
7. Vinsel PJ. Treatment of acute chlorine gas inhalation with nebulized sodium bicarbonate. J Emerg Med. 1990 May-Jun 1990;8(3):327-329.
8. Pascuzzi TA, Storrow AB. Mass casualties from acute inhalation of chloramine gas. Mil Med. 1998 Feb 1998;163(2):102-104.
9. Baud FJ, Borron SW, Megarbane B, et al. Value of lactic acidosis in the assessment of the severity of acute cyanide poisoning. Crit Care Med. 2002;30 (9):2044-2050.
10. Bebarta VS, Pitotti RL, Dixon P, Lairet JR, Bush A, Tanen DA. Hydroxocobalamin versus sodium thiosulfate for the treatment of acute cyanide toxicity in a swine (Sus scrofa) model. Annals of emergency medicine. 2012;59 (6):532-9.

References (2 0f 2)

11. Kealey GP. Carbon monoxide toxicity. Journal of burn care & research: official publication of the American Burn Association. 2009;30(1):146-7.
12. Henry CR, Satran D, Lindgren B, Adkinson C, Nicholson CI, Henry TD. Myocardial injury and long-term mortality following moderate to severe carbon monoxide poisoning. JAMA. 2006;295 (4):398-402.
13. Hampson NB. Noninvasive pulse CO-oximetry expedites evaluation and management of patients with carbon monoxide poisoning. The American journal of emergency medicine. 2012;30 (9):2021-4.
14. Weaver LK. Hyperbaric oxygen therapy for carbon monoxide poisoning. Undersea & hyperbaric medicine : journal of the Undersea and Hyperbaric Medical Society, Inc. 2014;41(4):339-54.
15. Bebarta VS, Tanen DA, Lairet J, Dixon PS, Valtier S, Bush A. Hydroxocobalamin and sodium thiosulfate versus sodium nitrite and sodium thiosulfate in the treatment of acute cyanide toxicity in a swine (Sus scrofa) model. Ann Emerg Med. Apr;55 (4):345-351.
16. Mcdougal JN, Dodd DE. Air Force approach to risk assessment for Halon replacements. Toxicology Letter. 1993;68 (1-2):31-35.
17. Emmen HH, Hoogendijk EM, Klopping-Ketelaars WA, et al. Human saftey and pharmacokinetics of the CFC alternative propellants HFC 134a (1,1,1,2- tetrafluoroethane) and HFC227 (1,1,1,2,3,3,3heptafluoropropane) following whole-body exposure. Regul Toxicol Pharmacol. 2000;31 (1):22-35.
18. Robin ML. Review of thermal decomposition product formation from halocarbon fire suppression agents: suppression of class A fires. West Lafayette, IN1999.
19. Chung KK, Wolf SE, Renz EM, Allan PF, Aden JK, Merrill GA et al. High-frequency percussive ventilation and low tidal volume ventilation in burns: a randomized controlled trial. Critical care medicine. 2010;38 (10):1970-7

Appendices

- **Appendix A**: Chlorine Inhalation
- **Appendix B**: Additional Information Regarding Off-Label Uses in CPGs

Contributors

- LCDR Omar Saeed, MC, USN
- CPT Nathan Boyer, MC, USA
- LTC Jeremy Pamplin, MC, USA
- MAJ Ian Driscoll, MC, USA
- MAJ Jeff DellaVolpe, USAF, MC
- LtCol Jeremy Cannon, USAFR, MC
- COL (ret) Leopoldo Cancio, MC, USA

Slides: Maj Andrew Hall, MC, USAF
Photos are part of the JTS image library unless otherwise noted.