Chapter 64 – Chemical Injuries

Episode Overview:

1. Describe the difference between Alkali and Acid injuries
2. Describe a HAZMAT response on scene
3. Describe the decontamination of an individual
4. List PPE for first responders or caregivers

Wisecracks:

1) Name two acids that cause damage through liquefactive necrosis
2) List Chemical Agents used in Warfare / Terrorism
3) What chemicals should you NOT irrigate with water?

Rosen’s in Perspective

- Chemicals are everywhere, >10,000 new ones are made every year → be afraid!
- Most susceptible organs: skin, eyes, lungs
- Most commonly release substances:
  - Volatile organic compounds (VOC’s)
  - Herbicides
  - Acids
  - Ammonia
  - Cement
  - Drain cleaner
  - Gasoline

Pathophysiology:

- Most cause damage through a chemical reaction with the skin

1) Describe the difference between Alkali and Acid injuries

- Acidic compounds:
  - Cause protein denaturation and coagulative necrosis with the skin
    - The necrosis forms eschar → which LIMITS the depth of acid penetration
    - Their free hydrogen ions are easily neutralized on the skin by copious water irrigation
- Alkali compounds:
  - Produce saponification and liquefactive necrosis of body fat - they produce soluble protein complexes which “permit the passage of hydroxyl ions deep into the tissue” and limiting the contact of the alkali complex on the surface of the skin.
  - Because there is no eschar formation they usually penetrate DEEPER into the tissue
2) Describe a HAZMAT response on scene

Because chemical disasters can occur at anytime and any place, the contingency plan for hazmat (hazardous materials) management has TWO parts:

1. Initiation of the site plan (using the incident command system)
   a. **Identify the offending substance(s)**
   b. **Assess and secure the surrounding environment**
      i. First responders should develop a perimeter
      ii. Use PPE before entering
      iii. Determine which victims need rescue and decontamination

2. Evacuation
   a. Secure the scene (extinguish fires, watch for other hazards)
   b. Decontamination

3) Describe the decontamination of an individual

**Should ideally occur BEFORE** the arrival to the ED:

1. First responder uses a scene survey approach
2. Attempt to determine which substance(s) may be involved
3. Don appropriate PPE (personal protective equipment)
4. Safely remove the victim from the hostile environment
5. ALL the victim’s clothing should be removed and placed in plastic bags
6. Dry agents should be brushed off; Wet agents should be sprayed off copiously
   a. Priority: eyes, mucous membranes, skin, hair
   b. Clean until a normal skin pH

**Hydrotherapy:**
- Application of large amounts of water to the skin - (dilution is the solution) under low pressure
  - Rosen’s: even after **1 hour of copious water irrigation** alkali exposures still often leave an abnormal pH for up to 12 hours!
  - Exception may be elemental metals (sodium) which are better removed with mineral oil

**Ocular therapy:**
- Alkali burns are more common - and severe causing coagulation necrosis deep into the corneal stroma
  - E.g. Anhydrous ammonia can penetrate into the anterior chamber in < 1min
- Classified into four categories:
  - I-II: hyperemia and conjunctival ecchymosis; some conjunctival haziness
  - III-IV: deeper penetration causing mydriasis, iris discoloration, and cataract formation
- Treatment:
  - Copious water irrigation!!
o Submerge eyes in running tap water - continuously open and close the eyes
  ■ Turn the head so that the other eye doesn’t get contaminated!!
o In the ER:
  ■ Continued tap water irrigation until morgan lens system setup
  ■ Repeated application of tetracaine / alcaine local anesthetic

3) List PPE for first responders or caregivers

- Chemical-resistant clothing with a hood
- Boots
- Eyewear
- Two layers of gloves
- Respiratory mask

Wisecracks:

1) Name two acids that cause damage through liquefactive necrosis

We’ll cover the underlined compounds on the podcast:

1. Specific chemical agents
   a. **Hydrofluoric acid**: - burns as small as 2.5% TBSA can be fatal
      i. Potential sources: glass etching, microelectronic production, rust remover, aluminum cleaner, cement and brick cleaner.
      ii. Can present with delayed onset pain.
      iii. Unique mechanism of action: although an acid, works by liquefactive necrosis. The free fluorine ion scavenges calcium and magnesium.
      1. Very dangerous because it can cause hypocalcemia, hypomagnesemia, inhibition of ATPase and Krebs cycle, hyperkalemia, QTC prolongation.
      iv. Most commonly *dermal exposure with sudden to delayed onset pain*. Coagulation and eschar can appear.
   v. Key management points:
      1. Irrigation for 15-30 mins with copious water
         a. If still painful, continue with:
      2. Blister removal
      3. Local 2.5% Calcium gluconate gel
      4. Intravenous and intra-ARTERIAL infusion vs. subcutaneous injection of calcium gluconate (both controversial)
      5. Require hospitalization and calcium gluconate infusions

b. Formic acid
   i. Causes damage leading to acidosis, hemolysis, hemoglobinuria.
   ii. Treatment: copious irrigation, NaHCO3 for acidosis. Severe cases may need hemodialysis or exchange transfusion.
c. **Anhydrous ammonia:**
   i. Colourless, pungent gas used in fertilizer settings (also in petroleum, plastics, explosives, synthetic fibers).
      1. Some forms of methamphetamine production use anhydrous ammonia as a precursor - so watch for this!
   ii. Ammonia liquid causes toxicity in two ways:
      1. Freeze any tissue it touches (-33°C)
      2. Vapor dissolves easily in skin, eyes, oropharynx, lungs
         a. The ammonia vapours **form hydroxyl ions can cause burns through liquefactive necrosis**
         b. Concentration and duration of exposure determines toxicity
            ➔ Huge morbidity in proximal airway exposures
   iii. Treatment:
      1. Irrigation of eyes, skin with water
      2. Secure airway if necessary with large bore tube

d. **Cement**
   i. When in contact with water → forms a basic solution
   ii. Can cause heat-related or blast induced burns
   iii. Needs copious irrigation, skin often needs grafting

![Hand with cement](https://c1.staticflickr.com/2/1245/1027393112_7cd83fb81c.jpg)

e. **Phenol and derivatives**
   i. “Starting materials for organic polymers and plastics”
   ii. Highly reactive, corrosive proteins causing cell wall destruction and coagulative necrosis.
   iii. **Dilute** phenol solutions are more readily absorbed (and more toxic) because it doesn’t cause the same necrosis of the papillary dermis**
   iv. Cause direct CNS and CV toxicity
   v. Some are used by plastic surgeons for “chemical face peels” - so watch out for dysrhythmias
vi. Treatment:
   1. Polyethylene glycol therapy
      a. Wiping down skin and tissue exposed to phenol with PEG sponges decreases toxicity
   2. Use LARGE amounts of water
   3. Use bicarb for acidosis

f. Phosphorous
   i. Can cause chemical and thermal burns
   ii. Absorption also leads to electrolyte (calcium, phosphate) and cardiac toxicity
   iii. Causes toxicity in three different stages
   iv. Treat with copious irrigation, decontamination, supportive care.

g. Nitrates and nitrites
   i. Used in many settings
      1. Medical, fertilization, food industry
   ii. Known to cause methemoglobinemia
      1. results when the ferrous ion in hemoglobin becomes oxidized to the ferric (3+) state, and can no longer carry oxygen.
      2. The toxicity exists on a spectrum, but when nonanemic patients get a level >20% → anxiety, headache, dyspnea, tachycardia occur
         a. Severe coma and death occur at levels approaching 70%
   iii. ****any cyanotic patient with a SPO2 of 85-88% should be investigated for methemoglobinemia in the setting of exposure or no response to oxygen or has a chocolate brown ABG.
   iv. Treatment:
      1. For symptomatic patients (who don’t have G6PD) 2mL of 1% methylene blue per Kg is given over 5 mins.
      2. People with G6PD need an exchange transfusion.

h. Hydrocarbons
   i. Found in fuels, solvents, paints, spot removers, dry cleaning solutions, lubricants
   ii. Toxicity usually relates to highly volatile inhalational/aspirational injury to the lungs
   iii. Treatment: dizziness, nausea, wheezing usually resolves through removal from substance
      1. Observe for at least 6 hrs for signs of aspiration: coughing, gagging, vomiting, wheezing, tachypnea, hypoxia
      2. Supportive care

i. Tar
   i. Roofing asphalt causes serious thermal and chemical burns, and the tar hardens with the hair
   ii. Treatment: direct cooling and then removal of tar (it doesn’t form a bond with the epidermis; instead it sticks on by enmeshing with the hair)
1. Sunflower oil can help remove tar from the skin
2. Antibiotic ointment may also help remove tar and asphalt

j. Elemental metals
   i. Produce volatile exothermic reactions when in contact with water

k. Gases
   i. Watch out for:
      1. Chlorine, chloramine
      2. Phosgene, nitrogen oxides / nitrogen dioxide gases, phosphene, and zinc bombs can cause delayed pulmonary edema.

2) List chemical agents used in warfare/terrorism

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<td>Distilled mustard, sulfur mustard (HD)</td>
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<td>Nitrogen mustard (HN1, HN2, HN3)</td>
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<td>Organic arsenical agents (e.g., Lewisite; L)</td>
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<td>Halogenated oxide agents (e.g., phosgene oxide; CX)</td>
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<td>Choking agents</td>
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<td>Sodium thiosulfate</td>
<td>Hydrosolubulin</td>
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*Chemical or common name (military chemical symbol).

Chemical Agents

Most choking agents (CG, CL, HC, PS) stimulate excessive lacrimation and only require supportive care (copious eye irrigation, bronchodilators, nebulized 4% sodium bicarbonate); rarely intubation for bronchospasms and pulmonary edema.

Other classes are discussed below.

1) Nerve agents
   a) Naturally occur as liquids at room temperature - so they need to be aerosolized into a gas
   b) Work by preventing acetylcholinesterase from hydrolyzing ACh.
      i) Lead to DUMBELS or SLUDGE and the deadly B’s
   c) Treatment: decontaminate, atropine (tapered to secretions - start at 2 mg), consider pralidoxime, use benzo’s for seizures.

2) Vesicants: “blistering agents” that form skin reactions at the site of contact.
   Aka “mustard gas”
i. Above 14 deg C solid mustard becomes gaseous. The gas form is heavier than air
ii. Up to 30 minutes of exposure can cause respiratory toxicity and death
iii. Exposure doesn’t cause immediate pain, and may present delayed up to 24 hrs.
iv. Treatment: remove from environment, decontaminate with hypochlorite solution (or household bleach diluted to 1:9)

3) Cyanide

- Notes taken from Ch. 64
  - Cyanide gas form is liberated from burning plastic-containing compounds
    - It is a cellular toxin - and inactivates cytochrome oxidase
      - The electron transport chain and inhibits oxidative phosphorylation
    - Leading to cellular hypoxia and death
  - If the salts/acid are ingested (metal cleaners, photographic production) usually results in sudden CV collapse, coma and acidosis.
    - “May smell a note of bitter almonds on their breath” -- not often clinically noted though
  - Most telling laboratory findings is:
    - ***Profound lactic acidosis***
    - Elevated mixed venous O2 saturation (cellular utilization of O2 is blocked)
    - Shortened QT interval
    - Normal pulse oximeter - despite cellular hypoxia
  - Treatment:
    - PPE and decontamination
    - ABC’s
    - Antiarrhythmics
    - Vasopressors
    - **Antidotes:**
      - 1) Trifecta of pimping:
        - Amyl nitrite, sodium nitrite, sodium thiosulfate
          - Amyl nitrite: pearls are broken open and breathed for 30 seconds on 30 sec. Off
          - Sodium nitrite: 300 mg IV dose over 5 minutes
          - Sodium thiosulfate: 12.5 g IV dose
        - The Nitrites induce methemoglobinemia
        - The thiosulfate causes transsulfuration of cyanide - so it can be renally excreted as thiocyanate
        - ***if coexisting carbon monoxide inhalation toxicity is suspected (smoke inhalation) the nitrites should be avoided***
- 2) Hydroxocobalamin (Cyanokit) - IM / IV
  - Binds cyanide to form cyanocobalamin
    - This is then renally excreted
    - administer 5g hydroxocobalamin diluted in 200 mL of 5% dextrose IV over 30 minutes (binds 100mg cyanide — use a larger initial dose if necessary) (from life in the fast lane)
  - Can cause reddening of the skin
  - Causes serum laboratory measurements to be unreadable
  - May safely be used with the "trifecta kit", but usually only thiosulfate is given due to the risks associated with the nitrites.

3) What chemicals should you NOT irrigate in water?

A) **Dry lime**: brush off the skin prior to irrigation. Lime contains calcium oxide, which when combined with H2O forms calcium hydroxide, a strong alkali. This turns bad into really BAD

![image from: http://images.slideplayer.com/14/4331842/slides/slide_73.jpg](http://images.slideplayer.com/14/4331842/slides/slide_73.jpg)

If you have already started to irrigate and recognise its dry lime… STOP. Brush off any obvious concretions/particulate matter off before restarting irrigation with water.

B) **Elemental metals/reactive metal compounds** combust or release hazardous byproducts when exposed to water.
Examples include: sodium, potassium, magnesium, phosphorous, lithium, cesium, and titanium tetrachloride. Remove obvious metals with forceps, and apply mineral oil. Wipe oil, then re-apply until all the metal is removed. You may need to ask your surgical colleagues to help out if metal has penetrated tissues.

C) **Phenol** is not water soluble. Take a sponge, and if you can soak it with 50 percent polyethylene glycol (PEG). THIS IS NOT THE PHARMACY OR GI PREP DOSE. If not, mega large doses of water, as phenol becomes more dilute, it penetrates skin better.