Household Products

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Objectives

• Know the various ingredient categories
• Identify the determinants of toxicity, symptoms, treatment, and monitoring considerations for each ingredient category

Examples-Household Products

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<th>Ammonia</th>
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<td>Hydrogen Peroxide</td>
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Household Product Categories

- Soaps
  - Detergents
    - nonionic
    - anionic
    - cationic
  - Builders
  - Acids
  - Alkalis
- Bleach
- Ammonia
- Hydrocarbons
- Brodifacoum
- Hydrofluoric Acid

Soap/Detergent Based Household Cleaning Products

- Soaps: Bar/Liquid soaps
- Detergents: Laundry Detergent, Fabric Softener, Dish Detergent, Hard Surface Cleaners, Scouring Cleansers
- 6% of exposures to poison centers

Soaps

- Na & K salts of Tallow/Fatty acids
- Other ingredients: biocides, perfumes, coloring agents, lotions
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<th>Detergents</th>
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<td><strong>Builders</strong></td>
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<td>• Bleaching Agents</td>
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<td>• Bactericidal Agents</td>
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<td>• Enzymes</td>
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<td>• Perfumes</td>
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<td>• Colorants</td>
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<tr>
<td>• Whitening Agents</td>
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<td>• Softeners</td>
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<tr>
<td>Surfactants are substances that lower the surface tension of water to enable it to “wet” surfaces more effectively, remove dirt, disperse soil, and emulsify oil or grease.</td>
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<tr>
<td>• Nonionic Surfactants</td>
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<tr>
<td>• Anionic Surfactants</td>
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<td>• Cationic Surfactants</td>
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<table>
<thead>
<tr>
<th>Nonionic Surfactant Examples</th>
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<tbody>
<tr>
<td>• alkyl ethoxylate</td>
</tr>
<tr>
<td>• nonyl phenol ethoxylate</td>
</tr>
<tr>
<td>• polyethylene glycol stearate</td>
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</tbody>
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<th>Anionic Surfactant Examples</th>
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<tr>
<td>• sodium lauryl sulfate</td>
</tr>
<tr>
<td>• linear alkyl benzene sulfonate</td>
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<tr>
<td>• dioctyl sodium sulfonate</td>
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<tr>
<td>• alcohol ether sulfate</td>
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<th>Cationic Surfactant Examples</th>
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<tr>
<td>• alkyl dimethyl ammonium chloride</td>
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<tr>
<td>• benzalkonium chloride</td>
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<tr>
<td>• cetylpyridinium chloride</td>
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<table>
<thead>
<tr>
<th>Cationic Surfactant Containing Products</th>
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<tr>
<td><strong>Product Types:</strong></td>
</tr>
<tr>
<td>• Disinfectants, mouthwash, fabric softeners, institutional cleaners</td>
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<tr>
<td><strong>Brands:</strong></td>
</tr>
<tr>
<td>• Bathroom Duck - 0.5%</td>
</tr>
<tr>
<td>• Dowbrands Bathroom Cleaner - 0.2%</td>
</tr>
<tr>
<td>• Lysol Basin, Tub and Tile Cleaner - 0.1%</td>
</tr>
<tr>
<td>• Lysol Brand Deodorizing Cleaner - 2.7%</td>
</tr>
<tr>
<td>• Amway Germicidal Concentrate - 15%</td>
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</tbody>
</table>
Toxicity of Soaps & Nonionic & Anionic Surfactants

- Mild mucous membrane and GI irritants
- Occasional nausea, vomiting and diarrhea
- Allergic/Contact Dermatitis
- Photosensitization

Toxicity of Cationic Surfactants

Determinants of Toxicity
- Concentration (<0.5% to 15-20%)
- Not pH dependent, but...alkalinity?
- Systemic concerns at >1 gram
- Single side chain substituted > double side chain substituted

Toxicity of Cationic Surfactants

- Ingestion
  - Burns to the mouth, esophagus & Stomach
  - Nausea, vomiting, & diarrhea
  - CNS effects (restlessness, confusion, seizures)
  - Respiratory paralysis (curare like effect)
  - Muscle weakness

- Dermal
  - Usually mild-severe irritation
  - >7.5% may cause severe burns
- Ocular
  - Mild conjunctival & corneal edema vs.
  - Corneal erosions, scarring, & blindness
  - Immediate pain/anesthetic effect
  - Watch for punctate burns from powders

Builders

Builders: inorganic salts that inactivate calcium and other minerals that interfere with detergency and maintain the proper pH of the washing solution

- Sodium carbonate
- Sodium metasilicate
- Sodium silicate
- Sodium tripolyphosphate

Soaps & Detergents: Treatment Considerations

Ingestion:
- Liquid vs Powder
- Hand vs Automatic
- Use as emetics
- Standard irrigation protocols (less than a swallow, <1%, or >2 hours)
- Gastric Lavage? Emesis?
- Activated Charcoal/Cathartic?
- Burn Management
### Soaps & Detergents: Treatment Considerations

- **Dermal & Ocular**
  - Standard irrigation protocols
  - Beware of powders!
  - Antihistamines/steroids

### Acid-Based Cleaners

#### Product Types
- **Toilet Bowl Cleaners** (Sulfuric acid 8-10%, HCl 10-25%, oxalic acid 2-5%)
- **Drain Cleaners** (Sulfuric acid 95-99%, Sodium OH 50-99%)
- **Metal Cleaners/Antirust compounds** (numerous, watch HF)
- **Soldering Fluxes** (Zinc Cl 10-35%, HCl up to 40%)
- **Automobile Battery Fluid** (Sulfuric acid (25-30%)
- **Swimming Pool Sanitizers** (Ca or Na Hypochlorite 70%)

### Acids

#### Determinants of Toxicity
- Physical characteristics of the chemical
  - Liquids vs crystals/powders, viscosity
- **pH**
  - <2
- **Titratable Acid/Alkaline Reserve/PKa**
- **Concentration**
- **Contact Time**
- **Exposure Volume**
- **Pre-existing barriers to contact (food/liquids)**
- **Pre-existing condition of the GI tract**

#### Mechanism of Action
- Coagulation Necrosis
  - Precipitates proteins
  - Limits penetrability
- **Hydrofluoric acid is the exception**
- **Upper GI more resistant to damage**

#### Titratable Acid Reserve (TAR):
- The amount of standard alkali that must be added to an acid to restore pH to neutral
- Higher the TAR, the greater potential for causticity
- **Ex:** 1% ammonia soln., pH 9.6, TAR of 10, produces burns
  - 1% bleach soln., pH 9.5, TAR of 1, no burns
**Acids-Toxicity**

**Initial effects**
- Pain on contact vs delayed
- Drooling
- Dysphagia
- Vomiting
- Hematemesis
- Substernal and abdominal pain
- Melena

**Gastrointestinal Complications**
- Oropharyngeal burns vs Esophageal-gastric involvement
- Acids vs. alkalis
- Esophageal-gastric burns without Oropharyngeal burns or Sxs.
- Acids vs. alkalis

**Respiratory Complications**
- Upper Airway obstruction from laryngeal edema
- Tachypnea, Stridor, & Dyspnea
- Aspiration pneumonitis

**Other Complications**
- Systemic acidemia
  - HCl non-anion gap metabolic acidosis
  - Sulfuric acid anion gap

**Determination of Toxicity**
- Liquid vs. crystals
- pH >12
- Titratable Alkaline Reserve
- Liquifaction Necrosis
  - continuing damage on penetration
  - 3.8% soln of NaOH causes necrosis of submucosa of esophagus and the muscular layer in 10 sec.
  - 22% soln causes complete necrosis of esophagus in 10 sec
- Same variables as Acids

**Acids**

**Brands**
- Lewis Red Devil Lye Drano (100%)
- Liquid Plumer (5% NaOH)
- Easy Off Oven Cleaner
- Crystalline Drano (57% NaOH)
- Household Ammonia (3-5% NH₃OH)
- Automatic Dish Detergents
- Bleaches (3-6% Na Hypochlorite)
- Disc Batteries

**Alkalis-Based Cleaners**

- Sodium hydroxide
- Potassium hydroxide
- Sodium metasilicate
- Sodium Carbonate

**Alkalis**

**Brands**
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- Disc Batteries
Alkalis

- Solids
  - oral and esophageal burns
- Liquids
  - esophageal without oral burns
  - circumferential burns; stricture formation
  - 30% with oral burns have esophageal burns
  - 2-15% with esophageal burns have no oral burns

Acids-Alkalis: Treatment Considerations

Ingestion
- Evaluate and Stabilize the Airway!
  - Evaluate for swelling of oropharynx, stridor, difficulty handling secretions, hoarseness
  - Intubate if symptoms suggest threatened upper airway patency BEFORE obstruction
- Triage?
- Dilution? pH change? Neutralization?
- Nasogastric suction
- Endoscopy
  - within 24-48 hours
  - to first area of ulceration
  - All with symptoms or oral burns-scope!

Acids-Alkalis: Treatment Considerations

Cont.,
- Contraindications to esophagoscopy:
  - Upper Airway obstruction
  - Signs and symptoms of perforation (risk highest from day 3-14)
  - >48 hours since exposure
- Strictures may not be evident for 3-6 wks
- Strictures may require lifetime dilation or esophageal transplant

Acids-Alkalis: Treatment Considerations

Cont.,
- Guaiac stools
- Surgical intervention?
- Corticosteroids?
- Antibiotics?

Acids-Alkalis: Treatment Considerations

Eye Irrigation
- Use anesthetics and retraction of lids as necessary
- Routine Irrigation guidelines
- Irrigate for minimum of 20-30 minutes
- Check for particulates
- Check pH of sac
- Slit lamp with fluorescein stain

Bleach
- Most bleaches are the sodium hypochlorite type.
- Symptoms:
  - mild to moderate irritant
  - nausea, vomiting, abdominal pain
  - mild burns (rare)
- New bleaches may have 5-7% NaOH!
Bleach: Chemical Reactivity

Bleach + Strong acid = ?

Bleach + Ammonia = ?

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Bleach: Chemical Reactivity

- Symptoms:
  - upper respiratory irritation
  - coughing, choking, SOB, wheezing, ocular stinging, pain on deep inspiration, nausea and pulmonary edema
- Groups at Risk:
  - smokers
  - chronic lung disease

Bleach: Chemical Reactivity

- Treatment:
  - fresh air
  - fluids
  - humidified air
  - occasionally ED treatment (oxygen, respiratory support, nebulizer treatments)

Ammonia

- Household ammonia (3-10%) = irritant
- Industrial/farm concentrations (10-80%) = corrosive
- Chloramine gas is less soluble than chlorine gas, so it may persist, cause more damage, and a more prolonged course of healing.

Hydrocarbons

- Aliphatic hydrocarbons: propane, butane, isobutane
- Aromatic hydrocarbons: benzene, toluene, xylene
- Petroleum distillates: kerosene, gasoline, mineral spirits, mineral seal oil
- Pine oils: pine oil, turpentine
- 5% of all exposures to poison centers
Hydrocarbons

• 18% of all pediatric poisoning admissions
• Leading cause of death in children resulting from household products (29%)
• Ingestion vs. Aspiration
• Used as vehicles for heavy metals and pesticides

Risk of Aspiration

• High volatility and low surface tension
• Low viscosity
  – Measured in SUS (Saybolt's Universal Seconds)
  – Substances with SUS <60 are easily aspirated, those > 100 are not

Substances <60 SUS
- mineral seal oil (furn. Polish)
- gasoline
- turpentine & kerosene
- petroleum naptha (lighter fl.)
- aromatic hydrocarbons
- mineral spirits

Substances >100 SUS
- lubrication oil (engine)
- petroleum jelly
- grease
- rubber cement
- paraffin wax
- mineral oil
- Paints

Non-Respiratory Effects

• Bloating
• Burning in mouth and throat
• Diarrhea
• Spontaneous vomiting: risk factor for aspiration

Aspiration leads to Chemical Pneumonitis

• Signs and Symptoms
  – coughing/choking
  – gasping/SOB
  – tachypnea
  – cyanosis
  – respiratory distress/arrest
• Of 115 patients, all that showed no respiratory sx.
  by 2 hours postexposure, remained asx.

Monitoring and Treatment

– Obtain: baseline CXR, ABG’s, CBC.
– CXR’s may develop 12 hours after exposure
– Often appear worse on day 3; clear days later
– Correlate poorly with clinical sx.
– Asx. patients may have abnormal CXR (35% in one series)
– Sx. patients may initially have nl CXR
– Monitor: breath sounds, LOC, respiratory rate
– Ventilator support if necessary, antibiotics and nebulizer treatments
# Hydrocarbons

## Triage and Hospitalization
- Parent transport alone vs parent with assistance vs ACLS Rig
- Admit:
  - Sx. children with abnormal CXR
  - Suicidal patients
  - Hypoxic or obtunded patients
  - Patients with a substantially abnormal CXR

## Discharge after 6 hours of observation
- Asx children with a nl CXR
- Asx children with mildly abnormal CXR who do not develop sx during observation

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# Hydrocarbons

## Admit after 6 hour observation period
- Asx children with abnormal CXR & develop sx during observation
- Asx children who develop sx from other chemicals
- Sx children with a nl CXR whose sx do not improve
- All patients whom you cannot f/u closely

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# Rodenticides/Brodifacoum

## Talon G, Weather Block
- All contain 0.005%
- Half-life ~ 25 days

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# Brodifacoum

## Mechanism of Action:
Inhibits Vitamin K1 epoxide reductase enzyme & production of Vit K1 dependant clotting factors, thus prolonging PT, PTT, INR

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# Brodifacoum

Any amount greater than the equivalent warfarin therapeutic dose is considered potentially toxic
### Brodifacoum

#### Warfarin Dose - Adults
- 70kg adult = 20mg
- Brodifacoum equivalent
  - 0.25mg
- 0.25mg = 25 large pellets or 150 mini pellets

#### Warfarin Dose - Children
- 10kg child = 2.85mg
- Brodifacoum equivalent
  - 0.036mg
- 0.036mg = 4 large pellets or 22 mini pellets

#### Adverse Effects
- BLEEDING!
  - Easy bruising
  - Gum bleeding
  - GI bleeding
  - Nose bleeds

#### Decontamination:
- Unknown ingestion
  - induce vomiting
- Late presenters/intentional ingestion
  - lavage or activated charcoal
- Avoid prophylactic Vitamin K use

#### When to get coagulation studies:
- Asx, accidental pediatric ingestion: probably only needed at 48 hours
- Symptomatic or intentional ingestions: obtain stat, then at 24 and 48 hours
- Serious cases should be typed and cross matched should transfusion become necessary

#### Treatment:
- Vitamin K1 is antidotal - administer to any pt. with significant prolonged PT or INR
  - Dose: start with 10mg IV and can increase for serious cases (100-250mg/day)
  - Can repeat in 6-8 hours
  - PT may not normalize for 3-4 days and therapy may take weeks to months

#### Treatment (cont.):
- Vitamin K1
  - For less serious cases, can use PO 15-25mg per day for adults and 5-10mg/day for children
- Cholistyramine
  - 12-16g qd in divided doses has been shown to decrease half life & increase clearance of warfarin ingestions
### Brodifacoum

**Treatment (cont.):**
- Transfusions
  - in pts. with serious hemorrhage, blood product will be needed.
  - use fresh frozen plasma

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### Carbon Monoxide

**Sources:**
- Endogenous Production
- Fires
- Tobacco Smokers
- Automobile Exhaust
- Furnaces
- Natural Sources
- Methylene Chloride (Paint strippers)

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### Carbon Monoxide

**Characteristics:**
- Colorless, odorless, tasteless, non-irritating
- Slightly less dense than air (0.97)
- Toxic effects are increased by:
  - decreased barometric pressure
  - increased alveolar ventilation
  - high metabolic rate
  - reduced cardiac output

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### Carbon Monoxide

**Characteristics (cont.):**
- Toxic effects are increased by:
  - preexisting cardiovascular or cerebral vascular disease
  - anemia
  - hypovolemia
  - impaired diffusion capacity

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### Carbon Monoxide

**General Pathophysiology:**
- CO is rapidly absorbed from the lung into the blood
- Displacement of oxygen from Hb results in hypoxia eventually leading to tissue hypoxia
- CO affinity for Hb 250 times that of oxygen
- The COHb concentration reflects the red cell load and not the tissue concentration

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### Carbon Monoxide

- Leading cause of death from poisoning
- ~3800 die annually
- Difficult diagnosis (missed in 30% of cases)
Carbon Monoxide

General Pathophysiology:
• Myoglobin is an O2 reservoir and provides O2 when the PAO2<60mmHg
• CO saturates cardiac myoglobin 3x greater than skeletal muscle
• This leads to myocardial depression, hypotension, hypoxia, and ischemia

Clinical Presentation:
• Misdiagnosed 30% of the time as the “flu” or gastro-enteritis
• Misdiagnoses:
  – food poisoning, hysteria, delirium, intoxication, and migraines

Clinical Presentation:
• Individuals with cardiovascular disease have a lower tolerance for CO.
  – Sudden death occurs when COHb level exceeds 20%.
  – Conduction defects, PVC’s, A-fib, MI and accelerated atherosclerosis

Clinical Presentation:
• Pulmonary complications
  – aspiration pneumonia, ARDS, pulmonary edema
• Bullae can occur over pressure areas and their appearance may be related to the severity of poisoning
• Cherry red skin or lips?

Delayed & Chronic Neuropsychiatric Effects:
• 3-40% incidence
• Complications post-recovery:
  – visual loss, dementia, retardation, disorientation, memory loss, personality changes, and frank psychosis
  – rarely: Parkinson’s disease
• 2-4 weeks post-exposure:
  – apathy, mutism, amnesia, HA, irritability, memory loss, fecal and urinary incontinence, personality changes and visual changes
Carbon Monoxide

Delayed & Chronic Neuropsychiatric Effects:
- Most frequently, mental deterioration, mutism, incontinence, and gait disturbances are found in middle age or older pts.
- Level of consciousness loosely correlates with subsequent development of gross neuropsychiatric sequela.

Recovery is variable - 75% resolve in one year (memory and gait disturbances may remain)
- No clinical indicators to predict incidence or occurrence of this syndrome
- Psychometric testing can be used for assessment and treatment.

Assessment
- The clinical features approx. correspond to COHb.
- Death frequently at >50% COHb

COHb may be low because…
- rapid onset of poisoning
- pts. inability to tolerate hypoxia
- other drugs present
- resuscitation attempts, ie, O2 given
- sample accuracy

Treatment
- Remove from exposure and stabilize
- ABCs
- 100% O2 by non-rebreathing mask not nasal cannula; IV’s; cardiac monitor
- minimize activity for 2-4 weeks after episode
- Consider Admission for
  - pregnant females >10% COHb

Consider Admission if:
- individuals with cardiac compromise, >15% COHb
- Pt. with sxs >25% COHb
- Hx of decreased LOC, abnormal neuroexam, ischemic chest pain
- hypothermia, metabolic acidosis, CXR abnormal, abnormal ECG, myoglobinuria, or abnormal neuropsychological testing
Carbon Monoxide

**Treatment**
- Consider admission if:
  - pt. with sxs following 100% O2 >4hrs
  - symptomatic pt. w/clinically suspected CO poisoning
- Hyperbaric Chamber
  - enhances elimination of COHb & increases amount of O2 dissolved in plasma; increases tissue clearance of residual CO; reduces cerebral edema

**Hyperbaric Chamber**
- attempt to start HBO within 6 hours
- appears to reduce delayed neurological effects
- recommended HBO treatment if patient is/has
  - comatose, neurological impairment, cardiovascular involvement, >40% COHb, pregnant & >15% COHb, fetal distress, hx of ischemic heart disease, recurrent sxs up to 3 weeks after initial tx, sxs do not resolve 6 hrs. after 100% O2, unconscious > short period

**Monitoring**
- Monitor cardiac & respiratory status
- Consider other drugs, trauma, hypoxic encephalopathy
- Beware of cerebral edema
- Treat acidosis conservatively
- Hypothermia is not recommended

**Prevention**
- Implement programs to target new drivers, males, and the elderly.
- Do not run car motor in closed garage or park for long periods of time.
- Recognize excessive sleepiness in a car as a possible sign of CO poisoning.
- CO detectors with audible alarms in home and garage.
- Homeowners should check gas appliances, furnace, and chimney for proper function & vent.
- Think about CO when someone appears to have flu sxs - especially in the winter months.

**Hydrofluoric Acid**
- Household products usually 6-12% (Wink)
  - Not usually life-threatening
  - Pain at the site may be delayed up to 24 hours
- Industrial greater than 20%
- Biammonium Fluoride
- Treat all exposures seriously and aggressively
- Early symptoms are a poor sign
Hydrofluoric Acid

Mechanism of Toxicity

• Causes liquifaction necrosis
• Unique ability to penetrate deep tissue
• Fluoride binds to Ca and Mg
• Can see systemic fluoride poisoning secondary to dermal exposure

Hydrofluoric Acid

Presentation

• Usually the hand/fingers exposed
• Pain at the site may be delayed up to 24 hours
• Early symptoms are a poor sign
• Severity of symptoms may help assess prognosis
• Tissue becomes hyperemic, then blanched discoloration
• Necrosis ensues with ulceration

Hydrofluoric Acid

Treatment Considerations

• Irrigation/Prevent systemic absorption
• Aggressively correct electrolyte imbalances
• Rapid airway assessment and protection
• EKG
• IV access
• Electrolytes with Ca & Mg
• Calcium gluconate gel 2.5%/Magnesium solutions
  (consider use of sterile gloves-Monitor relief of pain)
• Do Not use Anesthesia
• Intradermal Ca gluconate injections (5%), 0.5ml/cm²

Hydrofluoric Acid

Treatment Considerations, Cont.

• Assess the patient in 4-6 hours post pain relief
• Debride necrotic areas
• Remove nails if subungal involvement
• If area of exposure is large or confined to an area which cannot be injected-use intra-arterial infusions
• Use radial or brachial artery
• Use 10ml of 10% Ca Gluconate in 50ml D5W and run over 4 hours

Hydrofluoric Acid

Treatment Considerations, Cont.,

• Ocular
  – standard eye wash
  – 1% calcium gluconate drops q 2-3 hours for 2-3 days (some authors suggest)
  – MgSO4 oph. ointment, irrigation with 0.2% benzalkonium chloride or calcium chloride, and calcium injections increased damage