TOXINS WITH DIRECT INJURIOUS EFFECT

BUTTON BATTERIES:

ALMOST EXCLUSIVELY A PAEDIATRIC PROBLEM

THE MAJORITY PASS WITHOUT COMPLICATION, BUT LARGER BATTERIES MAY LODGE IN THE OESOPHAGUS, CAUSING MULTIPLE SERIOUS PROBLEMS

RISK ASSESSMENT:

- If diameter < 15mm, almost never lodge
- If >15mm (usually >20mm) → get stuck and cause severe local injury → mucosal injury, burns/strictures, perforation, haemorrhage and tracheo-oesoephageal fistulae
- Insufficient quantity of heavy metal to cause systemic toxicity through absorption

MECHANISM OF INJURY:

- **LOCAL INJURY OCCURS FROM DIRECT PRESSURE NECROSIS AND LEAKAGE OF ALKALI** → leads to injury pattern and complication mentioned above

CLINICAL FEATURES:

- Majority are asymptomatic initially, but present as ingestion was witnessed
- Most common problems are dysphagia and pain, but this may be delayed for several days

INVESTIGATIONS AND MANAGEMENT:

- AP/lateral of chest and abdomen to localize object (specifically above/below diaphragm) and thus to guide further management

MANAGEMENT:

- Resuscitation is rarely required unless presentation is delayed and the patient presents in haemorrhagic shock (following oesophageal haemorrhage) or from septic shock (following perforation)
- Patient may also be in respiratory distress due to tracheo-oesophageal fistula formation
- A BUTTON BATTERY LODGED IN THE OESOPHAGUS REQUIRES ENDOSCOPIC REMOVAL, IDEALLY WITHIN 6 HOURS OF INGESTION
    - Mucosal burns have been documented as early as 4 hours
    - Endoscopy allows both removal of battery as well as examination for local complications that will guide further management
    - A button battery located beyond the oesophagus in an asymptomatic child can be allowed to pass naturally
- If lodged in the nose or ears → REMOVE URGENTLY

- Some authors suggest serial radiograph if battery is in the stomach but not past the pylorus → controversial, but some suggest endoscopic removal if it has not past the pylorus by 48 hours

CORROSIVE AGENTS:

I.E. ACIDS/ALKALIS

CAUSE INJURY TO UPPER AIRWAY AND GIT → AIRWAY INJURY IS LIFE-THREATENING

ENDOSCOPY STRATIFIES RISK FOR DELAYED SEQUEALE IN SYMPTOMATIC PATIENTS

RISK ASSESSMENT:

- Ingestion of concentrated sulfuric acid and sodium hydroxide cause SEVERE CORROSIVE INJURY without systemic toxicity
- Ingestion of >60mL of concentrated hydrochloric acid leads to severy injury to the stomach and duodenum with necrosis and perforation with rapid onset of severe multi-organ failure and is usually fatal
- Ingestion of <150mL of household bleach (dilute sodium hypochlorite) does not cause significant corrosive injury
- SEVERE SYSTEMIC TOXICITY ASSOCIATED WITH THE FOLLOWING CORROSIVE AGENTS → glyphosate, mercuric chloride, paraquat, potassium permangante zince
- Stridor, dyspnoea, dyspnoia or throat pain indicate airway injury
- Significant gastro-oesophageal injury is indicated by stridor, drooling, vomiting

TOXIC MECHANISM:

- Cause direct chemical injury to tissues
- Extent of injury depends on pH, concentration and volume ingested
- Alkaline agents cause LIQUEFACTIVE NECROSIS, resulting in deep and progressive mucosal damage
- Acids cause protein denaturation and COAGULATIVE NECROSIS, which does not extend as deep

CLINICAL FEATURES:

- Patients may experience immediate mouth/throat pain, drooling, odynophagia, vomiting and abdominal pain
- Laryngeal oedema may cause rapidly progressive symptoms suggestive of airway obstruction
- Oesophageal perforation and mediastinits are associated with chest pain, SOB, fever, subcutaneous emphysema and pleural rub
- Grading of oesophageal injury at endoscopy predictive of future carcinoma, strictures
- GIT perforation complicated by septic shock/peritonitis and MOF

INVESTIGATIONS:

- ENDOSCOPY:

- Performed in all patients with persistent vomiting, oral burns, drooling or abdominal pain
- Defines extent of injury and risk for immediate (perforation) and delayed (strictures) complications
- GRADING:
  - 0 → NORMAL, 1 → mucosal oedema, hyperaemia
  - IIA → superficial ulcers/bleeding
  - IIB → deep focal or circumferential ulcers
  - IIIA → focal necrosis
  - IIIB → extensive necrosis

MANAGEMENT:

- **TIME CRITICAL EMERGENCY**
- Early life threat is AIRWAY COMPROMISE → intervene early
- NO NASOGASTRIC UNTIL AFTER ENDOSCOPY
- Keep patient NBM
- Urgent surgical intervention is required if full thickness necrosis or perforation
- Broad spectrum antibiotics if there is evidence of perforation
- DO NOT INDUCE VOMITING, ADMINISTER CHARCOAL OR ATTEMPT PH NEUTRALISATION
- Patients who are asymptomatic and tolerating oral fluids at four hours are cleared for discharge
- Symptomatic patients → endoscopy within 24 hours
- NO ROLE FOR STEROIDS → may ↑ mortality following grade III injury

HYDROFLUORIC ACID

FOUND IN CAR WHEEL CLEANERS, RUST REMOVING SOLUTIONS AND IN PREPARATIONS FOR GLASS ETCHING

ACCIDENTAL DERMAL EXPOSURE IS COMMON AND TOXICITY MAY RANGE FROM MINOR DERMAL INJURY TO LIFE-THREATENING SYSTEMIC COMPLICATIONS

INGESTION OF HF IS POTENTIALLY LETHAL

RISK ASSESSMENT:

- Any dermal exposure may lead to delayed severe pain and tissue injury
- Systemic life-threatening FLUOROSIS is associated with ingestion or extensive dermal exposure:
    - Dermal exposure with 100% HF to 2.5% BSA
        - 70% solution to 8% BSA
        - 23% to 11% BSA
        - Ingestion of ≥ 100mL of low concentration solution or any volume of higher concentration

TOXIC MECHANISM:

- Fluoride ions bind directly with CALCIUM AND MAGNESIUM, as well as interfering with cellular potassium channels to cause cell dysfunction and death
- Systemic toxicity and ventricular dysrhythmias are due to ↓Ca, ↑K, ↓Mg and acidosis

TOXICOKINETICS:

- HF penetrates deeply into tissues to release fluoride ions

CLINICAL FEATURES:

- DERMAL EXPOSURE:
    - Skin contact with HF ≤50% not immediately painful and may go unnoticed for hours
    - Gradual onset of severe and unremitting pain without obvious erythema
    - Pallor and blanching appear after several hours
    - Blistering or tissue loss is delayed many hours or days
    - Very large exposures result in SYSTEMIC FLUOROSIS
- INHALATIONAL EXPOSURE:
    - Immediate onset of mucosal irritation with potential for pulmonary injury
- INGESTION:
    - Low concentrations are minimally corrosive to GIT
    - Patients may experience vomiting, dysphagia, abdominal pain
    - CARDIAC ARREST from systemic fluorosis may occur without warning 30 minutes to six hours post ingestion
- SYSTEMIC EFFECTS:

- ↓Ca and ↓Mg manifest as tetany and QT prolongation → ventricular arrhythmia/death

INVESTIGATIONS:

- Serial ECG → extent of QT prolongation
- Serum calcium (or ionized) and magnesium
- Endoscopy to delineate extent of mucosal injury

MANAGEMENT:

- TIME-CRITICAL EMERGENCY
- Have IV calcium at the bedside
- In event of ventricular dysrhythmia:
  - Standard APLS
  - IV calcium gluconate (60mL) or chloride 20mL → give every five minutes until ROSC
    - Large doses are required
  - Give bicarbonate and magnesium
- Decontamination:
  - Dermal → remove clothes and irrigate thoroughly with water
  - Ocular → water irrigation
  - Ingestion → do not induce vomiting
- ANTIDOTES:
  - Calcium chloride/gluconate → parenterally to treat hypocalcaemia or calcium gluconate GEL to all symptomatic patients following dermal exposure → administer until pain resolves
    - If pain is refractory → IV/intra-arterial calcium
    - Pain is out of proportion to local signs and frequently requires parenteral opioids until calcium can be effectively delivered

HYDROGEN PEROXIDE:

AN OXIDISING AGENT, WHICH WHEN INGESTED CAN CAUSE SERIOUS TOXICITY AND DEATH DUE TO CORROSIVE EFFECTS AND FROM GAS EMBOLISM CAUSED BY RELEASE OF OXYGEN GAS

RISK ASSESSMENT:

- Ingestion of >30mL of 3% causes more significant GI injury and may result in gas embolism
- Ingested of concentrated (>10%) → life threatening corrosive injury and venous/arterial gas embolism
- Exposure to the eye causes permanent corneal injury

TOXIC MECHANISM:

- Causes toxicity by three mechanism:
  - Direct corrosive injury
  - Oxygen gas formation and gas embolism → can also cause mechanical distension and rupture of hollow viscous
  - Lipid peroxidation

TOXICOKINETICS:

- H_2O_2 rapidly metabolized to oxygen and water

CLINICAL FEATURES:

- INGESTION:
  - Severe corrosive injury manifested by blistering of mouth and oropharnyx → laryngospasm, stridor, cyanosis and respiratory arrest
  - Painful gastric distension
  - ↑HR, lethargy, confusion and coma → cardiac arrest within minute
  - Cerebral gas embolism → progressive neurological disturbance
- DERMAL:
  - Inflammation, blistering and skin necrosis
- OCULAR:
  - Subepithelial corneal and conjunctival bubbles → ulceration and perforation

MANAGEMENT:

- Early airway management if threatened
- High flow oxygen
- Hyperbaric oxygen if treatin cerebral gas embolism
- Immediate eye irrigation with copious amounts of saline for at least 15 minutes