

① Salicylates poisoning (Aspirin)

- Acute toxic dose $\rightarrow > 150 \text{ mg/kg}$

- More significant toxic dose $> 300 \text{ mg/kg}$

- Fatal toxicity $> 500 \text{ mg/kg}$

- Resp. Alkalosis, high anion gap MA.

- Serum salicylate level should be closely monitored every 2-3 hrs)

- Initial treatment \rightarrow gastric decontamination with activated charcoal.

Focus on \rightarrow aggressive volume resuscitation
 \rightarrow initiation of NaHCO_3 (for symptoms)

- The primary mode of therapy for

Salicylate toxicity \Rightarrow Urinary alkalization.

- give glucose if pt altered mental status \Rightarrow CNS hypoglycemia!

- in severe case \Rightarrow hemodialysis

→ its toxicity ⇒ Mcc of acute liver failure in US!

② Paracetamol (Acetaminophen)

- The single acute toxic dose of APAP

→ > 200 mg/kg

In the asymptomatic patient, if the AST is normal and the APAP is $<10 \mu\text{g/mL}$, then no therapy is indicated. A normal AST and an elevated APAP warrants NAC dosing for at least long enough for the drug to metabolize while the AST remains normal.

An elevated AST puts the patient in the "hepatic injury" category. A patient presenting with symptoms (i.e., right upper quadrant pain, vomiting, jaundice) should be empirically started on NAC pending lab results.

- ↑ AST, ALT, Coagulation profile, KFT, APAP

- Do APAP level within 4hrs from ingestion!

treatment based on where level falls on

(Rumack Matthew nomogram)

① prophylaxis!

Any pt with serum APAP level within probable hepatotoxicity ⇒ treat with NAC.
N-Acetyl-Cysteine. ←

* pt with Normal AST, documented APAP unknown time of ingestion ⇒ treatment should ensue until level is non detectable.

② injury ⇒ ↑ AST/ALT ⇒ treat!

* Foreign body ingestion

Disk Battery ingestion.

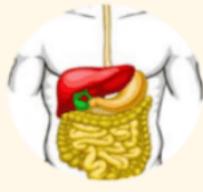


CLINICAL PICTURE

Witnessed Ingestion



- Drooling
- Coughing
- Stridor
- Shortness of breath



- Vomiting
- Difficulty in swallowing
- Painful swallowing

Unwitnessed Ingestion

Presents with complications



- Haemoptysis
- Chest pain
- Cough & Stridor
- Hoarseness
- Sore throat



- Haematemesis
- Melena
- Abdominal pain
- Weight loss



- Fever
- Neck Pain & Stiffness (spondylodiscitis)

** High index of suspicion for battery ingestion in children (toddlers) presenting with acute onset haematemesis.

<https://bit.ly/DETBE-ESPGHAN>



* Don't force pt to vomit!

* Confirm battery passage by daily inspection of all stool.

POISON	ANTIDOTE
Acetaminophen	N-Acetylcysteine
Benzodiazepine	Flumazenil
β-Blocking agents	Glucagon
Carbon monoxide	Oxygen
Cyclic antidepressants	Sodium bicarbonate
Iron (< 20 mg/kg)	Deferoxamine

POISON	ANTIDOTE
Lead	Edetate calcium disodium (EDTA) BAL (British anti-Lewisite [dimercaprol]) Succimer (2,3-dimercaptosuccinic acid ([DMSA])
Nitrites/ methemoglobinemia*	Methylene blue
Opiates	Naloxone
Organophosphates	Atropine Pralidoxime (2 PAM; Protopam)

Corrosive ingestion.

Acidic agents	Alkaline agents
PH<7 → if <2 → ↑ Risk of injury	PH>7
Pungent odor and noxious taste	Tastless, odorless (larger amount)
Coagulation necrosis => formation of coagulum layer limit the depth of injury	liquefaction necrosis => direct extension, deeper injuries
Less esophageal injury	Common → tasteless
More gastric injury	In stomach, partial neutralization by gastric acid may result limited

more esophageal injury
more abs with stricture.

Management

- ABC
- diluting with water or milk
- Remove contaminated clothing and rinse the affected skin
- Keep NPO
- IV fluids administer Gastric acid suppression with intravenous PPI
- Adequate pain relief with intravenous narcotics
- Airway evaluation - laryngoscopy
- R/O perforation - Plain films of chest and abdomen
- Observation for Clinical signs of perforation, mediastinitis, or peritonitis
- Broad spectrum antibiotics - given for patients with Grade 3 caustic injury or high suspicion for esophageal perforation.
- Endoscope