



# Poisoning

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28.03.2020

# Definition of poison

It is a substance which interferes with normal body function after it's swallowed , inhaled , injected or absorbed from skin exposure

## Important points in history and examination

- (1) Name
- (2) Magnitude of exposure.
- (3) Time of exposure
- (4) Progression of symptoms
- (5) Medical history
- (6) Complete physical examination

# DISPOSITION AFTER TOXIN EXPOSURE

- ASYMPTOMATIC PATIENTS : OBSERVED 4 TO 8 HOURS AND ADMIT IF SYMPTOMS DEVELOPED .
- PATIENT WITH SEVERE TOXICITY → ICU.

## DETERMINE

- HOME SAFETY AND PROVIDE POISON PREVENTION EDUCATION.
- IF PATIENTS IS INTENTIONALLY POISONED AS A FORM OF PHYSICAL OR SEXUAL ABUSE .
- IF TOXIC EXPOSURE IS A SUICIDAL ATTEMPT → PSYCHIATRIC EVALUATION .

# Treatment

## Decontamination.

OCULAR AND DERMAL DECONTAMINATION

SYRUP OF IPECAC

ACTIVATED CHARCOAL

GASTRIC LAVAGE

WHOLE BOWEL IRRIGATION

## Enhanced elimination.

MULTIPLE-DOSE ACTIVATED CHARCOAL

URINARY ALKALINIZATION

EXTRACORPOREAL METHODS

ANTIDOTES



# Decontamination

## Ocular and Dermal Decontamination

- IMMEDIATE AND THOROUGH IRRIGATION WITH TAP WATER OR NORMAL SALINE MAY AMELIORATE INJURY FOLLOWING OCULAR AND DERMAL EXPOSURES.
- CONTACT LENSES SHOULD BE REMOVED AND IRRIGATION OF THE EYES PERFORMED RAPIDLY TO PREVENT OCULAR DAMAGE.
- THE PATIENT'S CLOTHING, JEWELRY, AND OTHER ITEMS SHOULD BE REMOVED FOR DERMAL DECONTAMINATION, AND CARE IS REQUIRED TO PROTECT HEALTHCARE STAFF FROM SECONDARY TOXIN EXPOSURE.



## **Syrup of ipecac**

potent emetic that is no longer recommended as a home

## ACTIVATED CHARCOAL

ADMINISTERED INTO THE GASTROINTESTINAL TRACT BINDS MOST TOXINS, DECREASING SYSTEMIC ABSORPTION.

### IT IS INEFFECTIVE FOR

- IRON .
- LITHIUM .
- CYANIDE.
- ALCOHOLS.
- CAUSTICS.

**DOSE** : 1 G/KG BY MOUTH OR VIA NASOGASTRIC TUBE .

50 - 100 G FOR ADOLESCENTS AND ADULTS.

**C/I** PROTRACTED VOMITING , DECREASED .



# GASTRIC LAVAGE

WITHIN 1 TO 2 HOURS AFTER INGESTION OF A TOXIN.

## COMPLICATIONS

- VISCERAL PERFORATION.
- TRACHEAL PLACEMENT OF THE OROGASTRIC LAVAGE TUBE.
- ASPIRATION OF VOMIT.

## CONTRAINDICATIONS

- PATIENTS WITH DEPRESSED MENTAL STATUS .
- INGESTION OF A CAUSTIC OR HYDROCARBON.

**A LARGE-BORE LAVAGE TUBE (PEDIATRICS, 16–28 FR)**

**IS PLACED OROGASTRICALLY WITH THE PATIENT IN THE LEFT LATERAL DECUBITUS POSITION TO OPTIMIZE GASTRIC DECONTAMINATION AND REDUCE THE RISK OF ASPIRATION. THE STOMACH IS LAVAGED WITH 50 TO 250 ML OF WATER OR SALINE UNTIL CLEAR OF RESIDUAL TOXIN.**

# WHOLE BOWEL IRRIGATION

REDUCES ABSORPTION BY DECREASING GASTROINTESTINAL TRANSIT TIME.

## POLYETHYLENE GLYCOL ELECTROLYTE SOLUTION

ADMINISTERED VIA NASOGASTRIC TUBE AT RATES OF 25 TO 40 ML/KG/HOUR .

## INDICATIONS

INGESTION OF DELAYED-RELEASE MEDICATIONS, SUBSTANCES NOT ADSORBED BY CHARCOAL, OR DRUG-FILLED PACKAGES IN DRUG SMUGGLERS (“BODY PACKERS”).

# ENHANCED ELIMINATION

# ENHANCED ELIMINATION



IT HASTEN THE EXCRETION OF TOXINS FROM THE SYSTEMIC CIRCULATION

AT A RATE GREATER THAN ENDOGENOUS CLEARANCE.

- MULTIPLE-DOSE ACTIVATED CHARCOAL
- URINARY ALKALINIZATION
- EXTRACORPOREAL METHODS
- ANTIDOTES

## **MULTIPLE-DOSE ACTIVATED CHARCOAL**

- (1) DISRUPTING ENTEROHEPATIC RECIRCULATION OF THE TOXIN.  
PHENOBARBITAL, QUINIDINE, THEOPHYLLINE, CARBAMAZEPINE,  
AND DAPSONE AND SALICYLATES.
- (2) UTILIZING THE INTESTINAL MUCOSA AS A DIALYSIS MEMBRANE  
("GUT DIALYSIS") TO DRAW THE TOXIN FROM THE BLOOD STREAM  
INTO THE INTESTINAL LUMEN.
- (3) BINDING ANY TOXIN PRESENT IN THE GUT.

### **DOSE**

0.5 - 1 G/KG BY MOUTH OR VIA NASOGASTRIC TUBE EVERY 4 HOURS.  
IT SHOULD BE DISCONTINUED IF ILEUS DEVELOPS.

### **COMPLICATIONS**

CHARCOAL ASPIRATION, FORMATION OF CHARCOAL BEZOARS, AND  
INTESTINAL OBSTRUCTION.



# URINARY ALKALINIZATION

ENHANCES THE ELIMINATION OF DRUGS THAT ARE WEAK ACIDS (SALICYLATES) BY TRAPPING THE IONIZED DRUG IN THE RENAL TUBULE, WHERE IT IS SUBSEQUENTLY EXCRETED IN THE URINE.

## SODIUM BICARBONATE

1 TO 2 MEQ/KG IV FOLLOWED BY A CONTINUOUS INFUSION TITRATED TO A URINARY PH OF 7.0 TO 8.0.

# EXTRACORPOREAL METHODS

- HEMODIALYSIS.
- CONTINUOUS HEMOFILTRATION.
- HEMOPERFUSION.

## USES

- SEVERE CASES.
- IMPAIRMENT OF THE NATURAL MECHANISM OF ELIMINATION.
- PATIENTS DETERIORATING DESPITE MAXIMAL SUPPORTIVE CARE.
- PATIENTS WITH TOXINS WITH DELAYED EFFECTS.
- IF A BLOOD LEVEL OF THE TOXIN IS ASSOCIATED WITH SEVERE TOXICITY OR DEATH.

## HEMODIALYSIS :

SALICYLATES, THEOPHYLLINE, METHANOL, BARBITURATES, ETHYLENE GLYCOL, AND LITHIUM.

# Laboratory evaluation

## **(1) Blood concentration**

Salicylates , Acetaminophen , Iron , Digoxin , Anticonvulsants,  
Lithium ,Theophylline, Methanol , CO .....etc

## **(2) Urine for drug screening**

**(3) Other investigations** (Bl sugar , LFTs , RFTs , CBC ,  
Radiological studies .....etc. )



# Acetaminophen

**Acute toxic dose for child  $\leq 12$  y > 200 mg / kg**

## Clinical manifestations ( 4 stages )

### Stage I (1/2- 24 h )

Anorexia, nausea , vomiting , malaise , pallor , diaphoresis

### Stage II (24-48 h) quiescent phase

Resolution of the above; right upper quadrant abdominal pain and tenderness ; elevated bilirubin , prothrombin time , hepatic enzymes ; oligouria

### Stage III (72-96 h) period of maximal hepatotoxicity.

Peak liver function abnormalities ; anorexia , nausea , vomiting , malaise may reappear. In severe cases fulminant hepatic failure is manifested by coagulopathy, encephalopathy, and coma. Fatalities typically occur between 3 and 5 days because of multiorgan failure, hemorrhage, respiratory failure, sepsis, and cerebral edema.

### Stage IV (4 days- 2 weeks)

Resolution of hepatic dysfunction or complete liver failure

## Laboratory investigations

serum acetaminophen level, liver transaminases, electrolytes, BUN, creatinine, and PT.

## Treatment As soon as possible

\* Activated charcoal

\* N-acetylcysteine (NAC)

started within 8 to 10 hours of drug ingestion

**21-hour protocol:** loading dose of 150 mg/kg administered over 1 hour, followed by an additional 50 mg/kg over the next 4 hours, and a subsequent 100 mg/kg over the next 16 hours. Repeated 100 mg/kg doses over 16 hours can be administered if the patient continues to have significantly elevated liver transaminases.

\* Liver transplantation in severely affected patients

# Salicylates poisoning

The acute toxic dose  $\geq$  150 mg/ kg

- ▶ Affects most organ systems
- ▶ ↓ platelets adhesiveness
- ▶ ↑ pulmonary capillary permeability

## Clinical manifestations

- Nausea and vomiting (gastric irritation )
- Hyperventilation and hyperapnea ( stimulation of respiratory center ) → respiratory alkalosis
- Dehydration and progressive metabolic acidosis .
- CNS manifestations indicate serious toxicity ( Agitations , restlessness , confusion , coma due to cerebral edema )
- Pulmonary edema and hemorrhage
- Hyperglycemia or hypoglycemia
- Hepatotoxicity after chronic exposure or very large acute ingestion

## Causes of Death

( pulmonary edema and respiratory failure , cerebral edema , hemorrhage , severe electrolyte imbalance , or cardiovascular collapse )

## Laboratory investigations

- ▶ Serial serum level : level  $\geq 70$ mg/dl is life threatening
- ▶ Urine PH and volume hourly
- ▶ Blood gasses
- ▶ Blood glucose , serum electrolytes .
- ▶ Clotting studies
- ▶ LFTs

## Treatment

- (1) Activated charcoal – May need multiple doses.
- (2) Rehydration and correction of electrolyte abnormalities
- (3)  $\uparrow$  urinary excretion by  $\uparrow$  urine PH to change the non ionized salicylates to ionized one which is excreted in the urine without being reabsorbed again in the kidney .
- (4) Dialysis in severe cases ( serum level  $\geq 90$  mg / dl , change in neurologic status , respiratory or cardiovascular instability , refractory metabolic acidosis , severe hypokalemia , or renal failure )

# **WARFARIN** : RODENTICIDE

INHIBIT VITAMIN K, A COFACTOR REQUIRED FOR THE SYNTHESIS OF COAGULATION FACTORS II, VII, IX, AND X.

## **TYPICAL CLINICAL SCENARIO**

A TODDLER WHO HAS INGESTED A HOUSE RODENTICIDE.

THE ANTICOAGULANT EFFECT WILL NOT BE SEEN UNTIL THE CIRCULATING CLOTTING FACTORS HAVE BEEN DEGRADED, WHICH MAY TAKE 1 - 2 DAYS.

ECCHYMOSES, BLEEDING GUMS, SUBCONJUNCTIVAL HEMORRHAGE, HEMATEMESIS, MELENA, OR HEMATURIA.

**IN SEVERE CASES**, INTRACRANIAL HEMORRHAGE OR GASTROINTESTINAL BLEEDING.

# WARFARIN POISONING

WHEREAS COAGULOPATHY FROM WARFARIN POISONING OFTEN PERSISTS FOR A LONG TIME..

## LABORATORY MONITORING

**PT**

A NORMAL PT 48 HOURS AFTER INGESTION RULES OUT POISONING.

**INTERNATIONAL NORMALIZED RATIO (INR).**

**CBC**

# WARFARIN POISONING

## TREATMENT

REPEATED DOSES OF VITAMIN K1 (PHYTONADIONE)

FOR PROLONGED COAGULOPATHY, ALLOWS REGENERATION OF CLOTTING FACTORS, BUT PEAK EFFECT MAY TAKE 24 HOURS OR LONGER.

## HEMORRHAGE

**FRESH-FROZEN PLASMA.**

**PACKED RED BLOOD CELLS.**

# Iron poisoning

toxic dose (more than 60 mg/kg elemental iron)



## Clinical manifestations

- 30min-6hr      nausea , vomiting , diarrhea , abdominal pain hematemesis , bloody diarrhea and hypovolemic shock in serious poisoning
- 6-12 hr        GIT symptoms subside
- 12hr-48hr    Drowsiness – hypotension -hypoglycemia-metabolic acidosis-coma
- 2-4 wk        Gastric ulcer – pyloric stenosis – intestinal strictures

## Lab investigations

- (1) **Serum iron** 4 hr after ingestion , level > 500 microgram /dl indicate a significant toxicity  
(indication for using antidote)
- (2) **Blood gasses**
- (3) **Blood sugar**
- (4) **LFTs**
- (5) **Coagulation study**



## Treatment

- Supportive , symptomatic care
- Induce vomiting ( ipecac ) .
- Gastric lavage
- Whole bowel irrigation
- **Surgery if there is a mass of iron in stomach**
- **Desferoxamine ( Dysferal )**
  
- **Charcoal is useless**

# CARBAMAZEPINE

NYSTAGMUS, ATAXIA, DYSARTHRIA, MYOCLONUS, AND DYSTONIA, AND AT HIGHER LEVELS (ABOVE 40 MG/DL) RESULTS IN COMA, RESPIRATORY DEPRESSION, AND SEIZURES.

**CARDIOVASCULAR DISTURBANCES** INCLUDE TACHYCARDIA, HYPOTENSION, MYOCARDIAL DEPRESSION, AND CARDIAC CONDUCTION ABNORMALITIES, SUCH AS PROLONGATION OF THE QRS COMPLEX AND QT INTERVAL.

## MONITORING

**SERUM CARBAMAZEPINE LEVEL EVERY 4 TO 6 HOURS** UNTIL A DOWNWARD TREND IS ESTABLISHED.

SERUM SODIUM FOR HYPONATREMIA.

ELECTROCARDIOGRAM

**TREATMENT** : SUPPORTIVE. ENHANCED ELIMINATION PROCEDURES MAY BE CONSIDERED IN PATIENTS WITH SEVERE TOXICITY THAT IS NOT RESPONSIVE TO STANDARD SUPPORTIVE CARE.

# VALPROIC ACID

CONFUSION , LETHARGY , COMA, CEREBRAL EDEMA, AND RESPIRATORY FAILURE.

## SEVERE TOXICITY

HYPOTENSION, METABOLIC ACIDOSIS, HYPERNATREMIA, HYPOCALCEMIA, PANCREATITIS, HEPATIC INJURY, HYPERAMMONEMIA, RENAL INSUFFICIENCY, AND BONE MARROW SUPPRESSION.

## ELEVATED AMMONIA LEVELS

ENCEPHALOPATHY ( ALTERED MENTAL STATUS, FOCAL NEUROLOGIC ABNORMALITIES, AND SEIZURES.)

## LAB MONITORING :

SERIAL VALPROATE LEVELS SHOULD BE OBTAINED UNTIL A DOWNWARD TREND IS ESTABLISHED AND THE PATIENT'S SYMPTOMS ARE IMPROVING. BLOOD GAS ANALYSIS, COMPLETE BLOOD COUNT, SERUM ELECTROLYTES, GLUCOSE, BUN, CREATININE, CALCIUM, LIVER TRANSAMINASES, BILIRUBIN, PT, LIPASE, AMYLASE, LACTATE, AMMONIA, AND CARNITINE.

# VALPROIC ACID

## TREATMENT

- **CARNITINE**  
IF HYPERAMMONEMIA, HEPATOTOXICITY, OR CARNITINE DEFICIENCY IS PRESENT.
- **ENHANCED ELIMINATION** IN SEVERE TOXICITY.
- **ASSISTED VENTILATION.**

# Organophosphorus poisoning

Insecticide

Cholinesterase enzyme inhibitor

## Clinical picture

- Muscarinic signs ( DUMBBLES )

**D**iarrhea

**U**rination

**M**eiosis

**B**radycardia

**B**ronchospasm

**L**acrimation

**E**mesis

**S**alivation

- Nicotinic signs

Muscle weakness

Fasciculation

Tremor

Hypoventilation

Hypertension

tachycardia

Dysrhythmia

- Severe manifestations

Coma , seizures , shock , arrhythmia , respiratory failure

## Lab investigations

Measurements of enzyme concentration , symptoms appear if the enzyme is  $< 25\%$  of normal

## Treatment

- (1) Decontamination
- (2) Supportive care
- (3) Antidote
  - (a) Atropine
  - (b) Pralidoxime

# Alkali and acids

Acids , alkali , and oxidizing agents as bleach

Acids → local tissues necrosis → penetration

**Alkali → transmural necrosis → Risk of perforation**

... the severity of the chemical burn produced depends on the PH , conc. Of the agent , and the time of contact .

Agents with PH < 2 or > 12 are most likely to produce injury .

## Clinical manifestations

Oral burn dysphagia , pain , esophageal stricture , drooling , vomiting , pyloric scar , pyloric obstruction

Skin and eye contact is associated with significant tissue damage

## Treatment

- (1) Flushing the skin and eye by water
- (2) Removal of the contaminated clothes
- (3) Withhold oral fluids and solid intake
- (4) Endoscopy

**\*\* Using charcoal , Gastric lavage or emesis are all absolutely contraindicated**

## Hydrocarbons

- Aspiration of Hydrocarbons into the lungs can lead to serious, even life threatening toxicity .

Ingestion → cough and gag → Aspiration

Vomiting → Aspiration

- Hydrocarbons are weakly absorbed and cause GIT mucosal irritation , Renal and bone marrow toxicity , Hepatic toxicity
- Local skin contact → irritation and chemical burn



## Clinical manifestation

- × Transient mild CNS depression.
- × Cough.
- × Mild respiratory symptoms - my progress to chemical pneumonitis and even to respiratory failure
- × Fever - persist up to 10 days after ingestion

## Investigations

X ray - can be positive very early after ingestion (2-3h post ingestion)

2-3wk xray (pneumatocele)

Leukocytosis

## Treatment

- Supportive care (O2 and bronchodilator may needed) – Ventilator in some.
- NO emesis or gastric lavage NO antibiotic or steroid.

Thank you

