

Poisoning General Approach

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Definition



A **POISON** is a substance that causes illness or harm if someone eats, drinks, touches it, or breathes it in.



POISON PREVENTION NOW!

Substance Abuse

- The knowing misuse of any substance to produce a desired effect

Overdose

- Exposure to substance in excess amount resulting in toxic effects

- Poisoning: - acute
- chronic

- Dosis toxica minima: the minimum amount of toxic substance, which causes poisoning
- Dosis letalis minima: the minimum amount of toxic substance, which can cause death

Routes of Entry

- oral ingestion (PO)
- inhalation
- injection (IV, SC, IM, IO)
- absorption

History

- What?
- How much?
- How long?
- Multiple substances?
- Treatment attempted? How? Whose advice?
- Psychiatric history?
- History of suicide?

Symptoms

General:

- Sudden onset of CNS signs:
 - Seizures
 - Coma
 - Decreased LOC
 - Bizarre behavior
- Sudden onset of:
 - Abdominal pain
 - Nausea
 - Vomiting

Symptoms

- Sudden onset of unexplained illness
- Bizarre, incomplete, evasive history
- Trauma
(>50% of adult trauma alcohol-, drug-related)
- Pediatric patient with arrhythmias

General Management

- Support ABC's
 - Secure airway, intubate as needed
 - Ensure adequate oxygenation, ventilation
 - Maintain adequate circulation
 - Monitor ECG
 - Obtain vascular access
 - Manage hypotension initially with volume

General Management

- Keep patient calm
- Maintain normal body temperature
- Evaluate nature/toxicity of poison
 - Check container, package insert, poison center information
 - Treat the patient, not the poison

General Management

- Rule out
 - Trauma
 - Neurological disease
 - Metabolic disease
- Base general management on route of poison entry

Poison Entry

- oral ingestion (PO)
- inhalation
- injection (IV, SC, IM, IO)
- absorption

Ingested Poison

- accounts for 80% of all poisonings
- accidental or deliberate
- Activated charcoal
- ABCs and transport

- Objective - Remove from GI tract before absorption occurs
- Lavage:
 - Removes about 31% of substance
 - Helps get activated charcoal in patient, especially if patient is unconscious
 - Not helpful for sustained release tablets
 - Will not remove large tablets

Activated Charcoal

- Adsorbs compounds, prevents movement from GI tract
- Very effective at adsorbing substances
- Binds about 62% of toxin
- Dose
 - 5 - 10X estimated weight of ingested chemical

- Do not give until vomiting stops
- Do not give with
 - Cyanide
 - Methanol
 - Tylenol (±)
- Containers must be kept airtight
- Whole bowel irrigation
(iron, late presentation, large amount, enteric coated preparation) (0.5 to 1.5 liter/hr)

Inhaled Poisons

- Wide range of effects
- YOUR safety is 1st
- Move to fresh air immediately
- Provide airway support and rapid transport
- Bring contained agent or label to hospital

Injected Poisons

- Usually deliberate
- Impossible to remove or dilute
- Can be counteracted
- ABC's, high flow O₂, rapid transport

Absorbed Poisons

- Corrosives will damage the skin
- Some drugs are absorbed SubLingual (SL) or Per Rectum (PR)
- Treat chemical burns
- ABC's, O₂, rapid transport

- Objective: Remove poison from skin
- Liquid: Wash with copious amounts of water
- Powder: Brush off as much as possible, then wash with copious amounts of water
- Protect personnel from exposure
- Dilute / Irrigate / Wash
- Use soap, shampoo for hydrocarbons
- No need for chemical neutralization - heat produced by reaction could be harmful

Eye Irrigation

- Wash for 15 minutes
- Use only water or balanced salt solutions
- Remove contact lenses
- Wash from medial to lateral

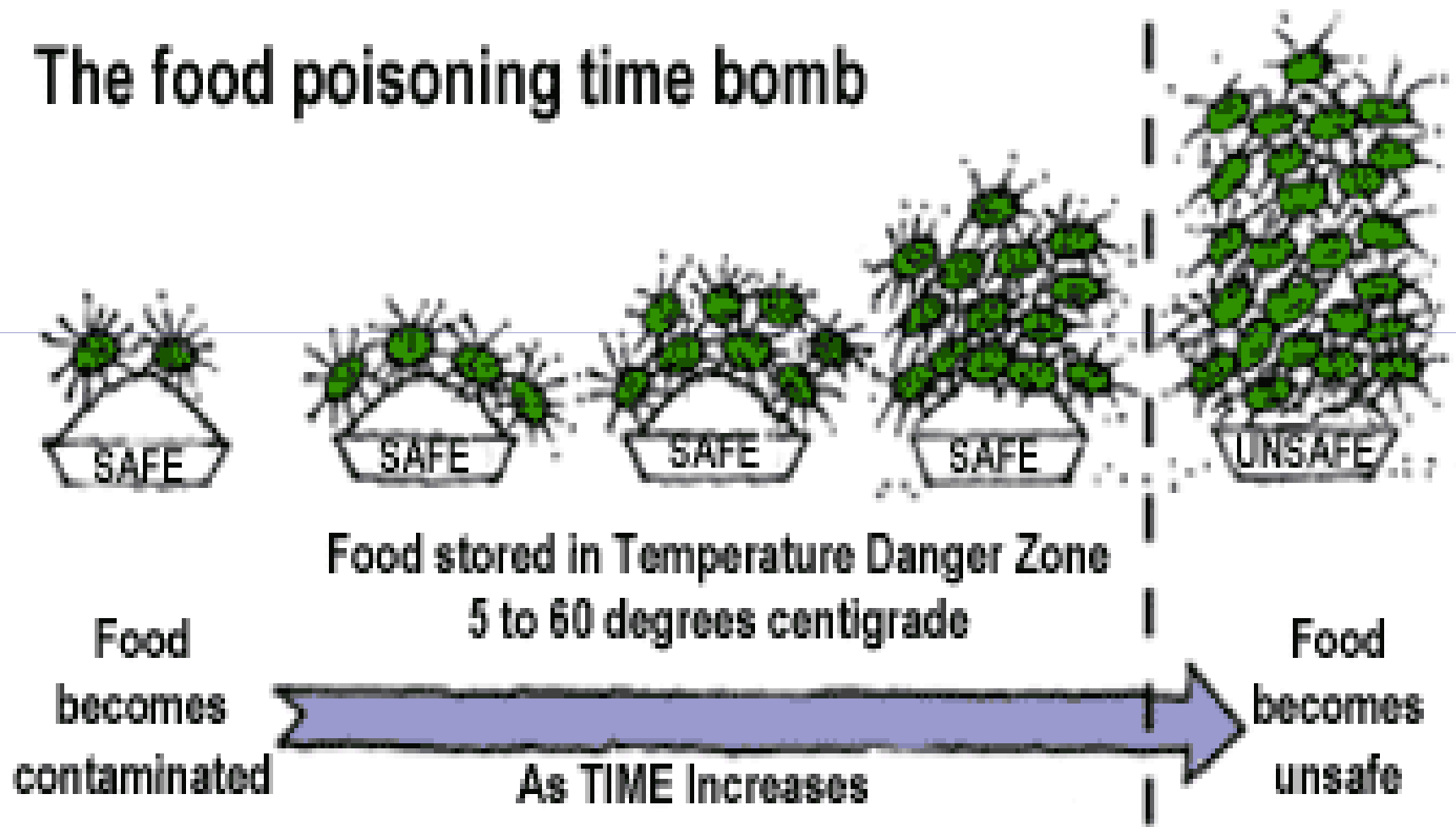
Food Poisoning

Food Poisoning

- Single meal
- Early symptoms
- Intoxications rather than infections
- Severity
 - # of organisms
 - the status of the host

FOOD/SEAFOOD POISONING TIME BOMB

The food poisoning time bomb



Pre-formed Toxins

- Symptoms appear within 12 hrs
- Vomiting > diarrhea
- No fever
- Agents:
 - *Cl. botulinum*
 - *S. aureus*
 - *Bacillus cereus*



Toxin Production “In Vivo”

- Onset 12-24 hours post-ingestion
- Abdominal cramps, watery diarrhea
- Fever, vomiting uncommon
- Symptoms usually disappear in 24 hours
 - *Cl. perfringens* - 10,000 cases/year
 - *Bacillus cereus*

Clostridium botulinum

- Anaerobic, sporulated gram-positive rod
- Spores get into food or wounds. Home canning a notorious source of intoxication
- Consumption of honey a risk for infants

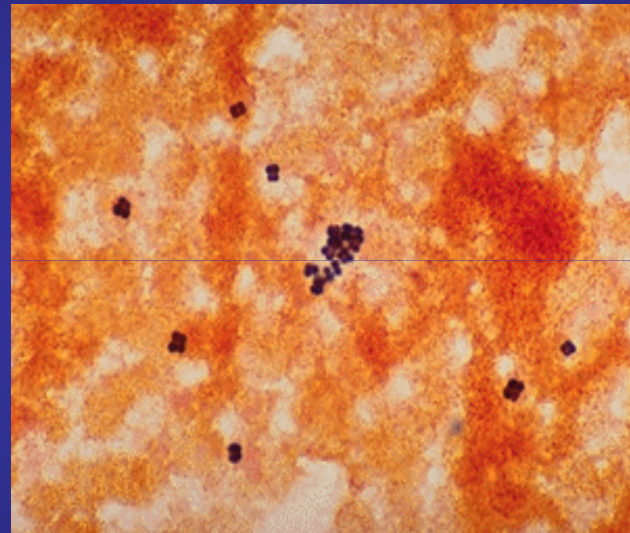


Clinical presentations

- **Classical:** Food-borne (sausages, meats, canned food). Occasionally preserved fish (type E toxin). **Infant botulism:** Production of toxin in GI tract. Associated with honey
- Progressive disease -irreversible binding of neurotoxin. Weakness, dizziness, blurred vision, dilated pupils, dry-mouth, constipation, abdominal pain, bilateral descending weakness of peripheral muscles
- Mortality 10%. Death due to respiratory paralysis

S. aureus

- Gram-positive cocci in clusters
- One of the most common pathogens
- Six-enterotoxins (A-F)
- Ingestion of toxin-contaminated food
- Enterotoxin resistant to hydrolysis and heat-stable at 100°C



S. aureus

- Implicated foods: ham, potato salad, salted pork, custard-filled pastries, ice cream, mayonnaise, egg salad, sausages
- Food usually kept at room temperature
- Human-human transmission (nasopharynx carriers)



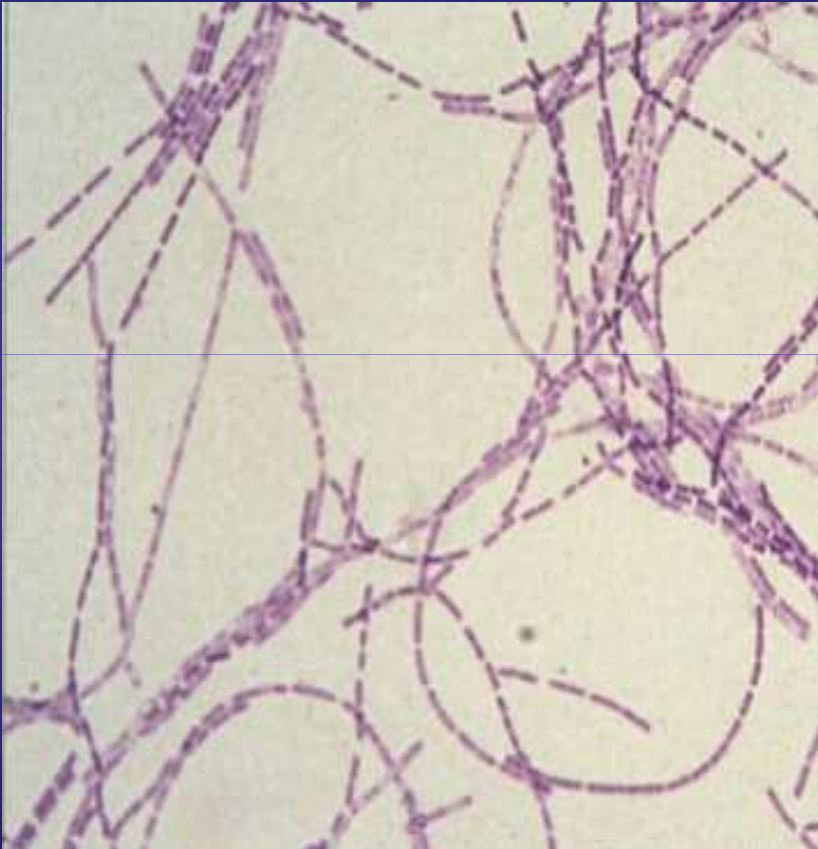
S. aureus: Pathogenicity

- Mechanism of action of enterotoxin: CNS
- Acute onset (1-6 hrs)
- Severe vomiting, abdominal pain and nausea, no diarrhea
- Symptoms disappear within 24 hours



All people are believed to be susceptible to this type of bacterial intoxication; *however*, intensity of symptoms may vary.

Bacillus cereus



Gram stain



Culture on blood agar

B. cereus enterotoxins

- Heat-stable enterotoxin - Emetic form, toxin is preformed in food. Mechanism unknown
- Heat-labile enterotoxin - Diarrheal form similar to ETEC and *V. cholerae*. Toxin formed in the GI tract. Stimulates production of adenylate cyclase cyclic AMP

B. cereus: Symptoms

	Emetic form	Diarrheal form
Implicated food	Rice	Meat, vegetables
Incubation period	<6 hours (mean: 2 hrs)	>6 hours (mean: 9 hrs)
Symptoms	Vomiting, nausea, abdominal cramps	Diarrhea, nausea, abdominal cramps
Duration	8-10 hours (mean: 9 hrs)	20-36 hours (mean: 24 hrs)
Enterotoxin	Heat-stable	Heat-labile

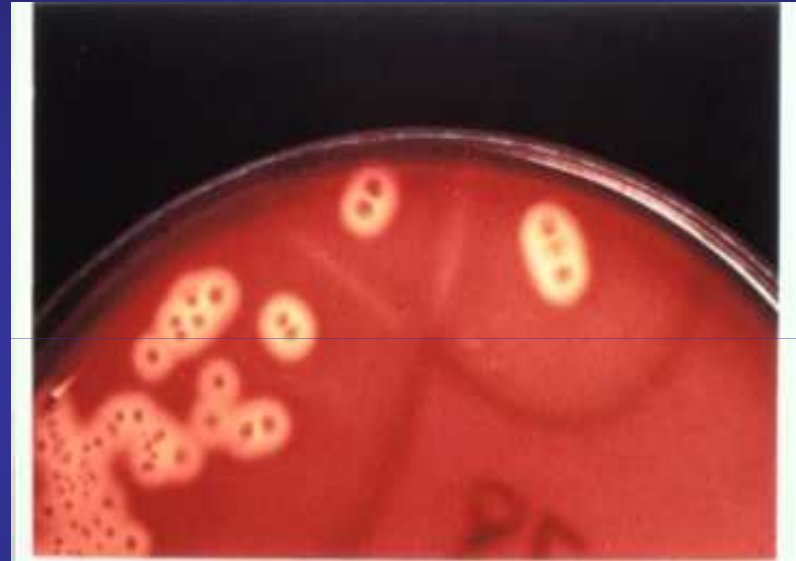
Clostridium perfringens

- Anaerobic Gram-positive sporulated bacillus
- Most frequently isolated clostridium
- 3rd most common cause of food-poisoning
- Spores rarely seen in vivo or in culture
- 4 major toxins (A-E)
- Food poisoning - strains A, C, D enterotoxin disrupts ion transport in ileum

Clostridium perfringens



In tissue



In culture

C. perfringens: Clinical Presentation

- Short incubation period (12-24h) and short clinical course
- Watery diarrhea, intense abdominal cramps, no fever, nausea, or vomiting
- Few deaths reported
- Enteritis necroticans (rare)

Food Poisoning Diagnosis

- Clinical
- Interview with patient(s)
- Culture of food items
- Detection of enterotoxin in food or stool

Treatment

- Symptomatic treatment
- Anti-motility drugs for diarrhea (if present)
- For botulism
 - Intensive supportive treatment
 - Be ready to do CPR
 - Induce vomiting if
 - Ingestion is recent
 - No seizures
 - No loss of consciousness
 - Gastric lavages - to remove toxin
 - Polyvalent antitoxin (trivalent A,B,E)
- Antibiotics not useful

Prevention

- Prevention by proper refrigeration of foods after cooking and before serving
- Food should be boiled for 30 min to inactivate toxin
- Do not leave food at room temperature
- People with skin infections should not prepare food
- Proper canning. Botulism toxin can be destroyed in food by heating it at 80° for 20 min.

Summary: Clinical and Epidemiologic Features of Food Poisoning

Etiology	Incubation Period (hr)	Clinical Findings	Characteristic Foods
<i>B. cereus</i>	9	Diarrhea, cramps	Meat, sauces
<i>B. cereus</i>	1-6 (emetic form)	Vomiting, diarrhea	Rice
<i>S. aureus</i>	1-6	Vomiting	Hams, custards, potato salads, mayonnaise, meats
<i>Cl. botulinum</i>	12-24	Neuromuscular paralysis	Canned vegetables, meats, honey
<i>C. perfringens</i>	8-24	Watery diarrhea	Meat, poultry

Acid and alkali poisoning

Acids and Alkalis

- Corrosives - denotes an acidic substance
- Caustics - denotes an alkaline substance
- Cause tissue injury by a chemical reaction - accept (base) or donate (acid) a proton.
- Severity of tissue injury is determined by:
 - the substances pH,
 - concentration,
 - duration of contact and
 - volume of contact.

Acids and Alkalis

- Ingestion is an extremely common event
- alkali drain cleaners and acidic toilet bowl cleaners are responsible for most fatalities
- 10% of caustic ingestions result in severe injury requiring treatment
- between 1-2% result in stricture formation
- 80% of ingestions are in children > 5 years
- most adult ingestions are intentional

Acids

- Mechanism of action - denature proteins
 - coagulative necrosis
 - cell morphology not grossly altered, destruction of enzymatic proteins
 - acid burns cause formation of tough leathery eschar or coagulum (which sloughs in 3-4 days)
 - little to no systemic absorption
 - stomach is most commonly involved organ

Acids

- Management - any ingestion constitutes a medical emergency
- skin or eye contact -
 - flush with copious amounts of water.
Remove contact lens and jewelry
 - wash skin with mild soap
 - do not apply topical ointments, creams, or dressings

Acids

- Ingestion
 - serious solid ingestions are rare as the particles usually stick to the oropharynx, but liquid exposures can be severe
 - burning is so severe patient probably can not swallow
 - do not give carbonated beverages
 - do not give water, water + acid explosive release of steam
 - exothermic reaction
 - do not induce vomiting, gastric lavage contraindicated!
 - maintain airway

Acids

- Clinical concerns:
 - perforation may occur after the third to fourth day as eschar sloughs
 - gastric outlet obstruction may develop over a 2-4 week period
 - upper GIT hemorrhage

Acids

- Common acid containing sources
 - toilet bowl cleaners
 - automotive batteries
 - rust removal products
 - metal cleaning products
 - drain cleaning products

Alkalis

- More common cause of toxicity than acids
- Most common alkaline agents causing toxicity
 - Ammonia
 - Sodium hydroxide
 - both of these are found in household cleaners and drain cleaners
- Most cases are in children under the age of five years

Alkalis

- Mechanism of action
 - tissue injury causes by liquefactive/colliquative necrosis (saponification of fats and solubilization of proteins)
 - cell death occurs from emulsification and disruption of cell membranes
 - OH ion reacts with tissue collagen causing it to swell and shorten
 - most severe injured tissues are the squamous epithelial cells of the oropharynx and esophagus (the most commonly involved organ)
 - alkali burns to the skin are yellow, soapy and soft

Alkalis

- Clinical concerns
 - tissue edema - leading to possible airway obstruction
 - erythema
 - ulceration
 - necrosis of tissues with possible stricture formation (depends on depth of burn)
 - perforation can occur

Alkalis

- Management of toxicity
 - eye and skin contamination - flush with copious amounts of water
 - ingestion - any ingestion constitutes a medical emergency
 - do not induce vomiting
 - give demulcent (milk, water or egg whites)

Alkalis

- Common base containing sources
 - drain cleaning products
 - ammonia - containing products
 - oven cleaning products
 - swimming pool cleaning products
 - automatic dishwasher detergent
 - hair relaxers
 - bleaches
 - cement

Alkalis

- Ammonia - NH_3 - at room temp is a highly H_2O soluble, colorless, irritant gas with a pungent odor.
- A component of many household cleaning products - (glass cleaners, toilet bowl cleaners, metal polishes, etc.)
- highly alkaline and corrosive
 - household ammonia - not as corrosive

Alkalis

- Most common mechanism is exposure to anhydrous ammonia - liquid or gas
 - $\text{NH}_3 + \text{H}_2\text{O} \rightarrow \text{NH}_4\text{OH}$
 - this reaction is exothermic - causes significant thermal injury
- Ammonium hydroxide also causes severe alkaline burns
- Ingestion of household solutions is usually accidental and occurs in young children, adult ingestions are usually suicide attempts

Alkalis

- Typical household ammonia
 - contain 3-10% ammonia hydroxide
 - pH less than 12.5 - caustic burns usually only seen with pH's >12.5 so household products do not typically lead to significant burns
 - patients present with oropharyngeal and epigastric pain
 - may cause aspiration pneumonitis
 - kids may bite smelling salts - 20% ammonia - can cause esophageal burns and mild respiratory symptoms

Alkalis

- Bleach - sodium hypochlorite - NaOCl
 - taste terrible and spontaneously vomited
 - never mix bleach with acid or alkaline cleaning agents - release of chlorine gas
 - good to apply topically for any bites

Mushroom poisoning

Mushroom toxins

4 categories of mushroom toxins according to their physiological effects

1. Protoplasmic poisons
2. Neurotoxins
3. Gastrointestinal irritants
4. Disulfiram-like toxins

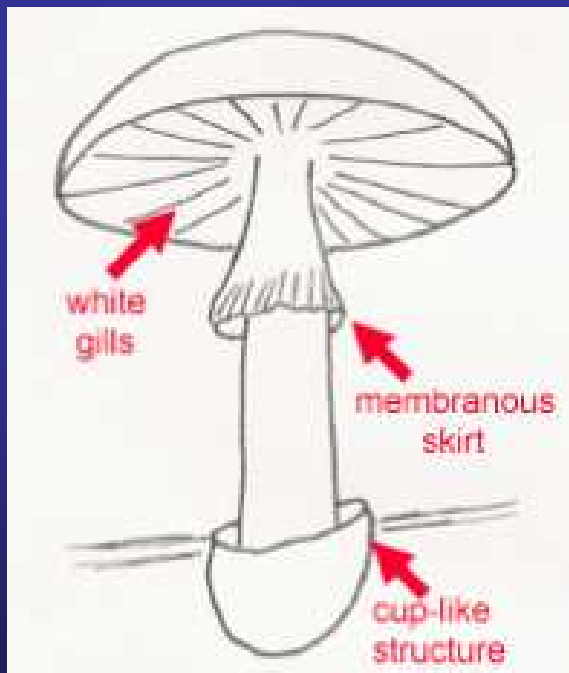
Protoplasmic poisons

Poisons that result in generalised destruction of cells, followed by organ failure

- Amatoxins
- Hydrazines
- Orellanine

Amatoxins

- Amanita phalloides
- Death cap



Amatoxin



Death cap



Destroying Angel

False morel (hydrazin)



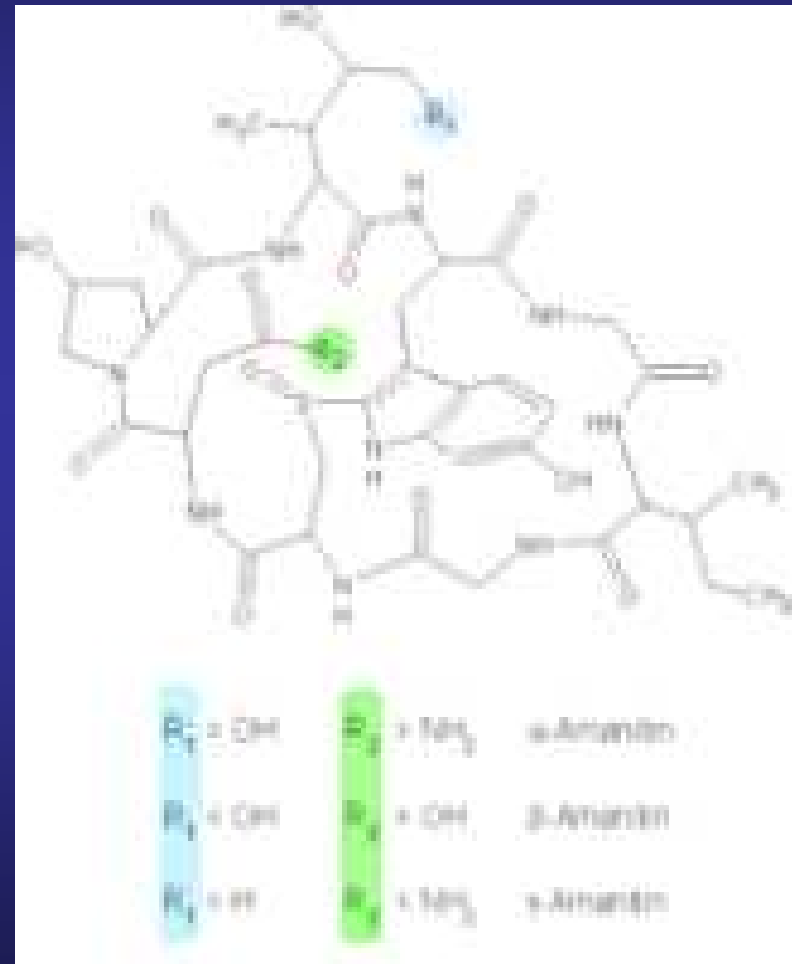
False morel (hydrazin)



Cortinarius orellanus (Orellanine)



- Called amanitins or Amatoxins
- α , β , γ forms
- Three genera of mushrooms contain the toxins
- *Amanita*, *Galerina*, *Lepiota*
- also contain phallotoxins, virotoxins and phallolysins
- Heat stabile
- Toxicity: 0.1 mg/kg BodyWeight



- Amatoxins inhibit protein synthesis
- Clinical signs:
 - GI upset: 6 – 24 hours post ingestion
 - Adominal pain, vomiting, profuse watery diarrhea
- Signs may resolve before onset of more severe liver involvement
 - Liver failure: 48 to 96 hours post ingestion
- Hypoglycemia, coagulopathy, encephalopathy, coma
- Renal failure and pancreatitis are also possible
- Death typically 6 to 16 days post ingestion

Diagnosis

- History and compatible clinical signs
- ID genus/species of mushroom
 - visual examination
 - DNA fingerprinting
- detection of amanitin
- Detection of amanitin in biological samples
 - relatively rapidly eliminated via kidneys
- ELISA

Treatment

- ICU
- Decontamination
 - Activated charcoal – multiple doses
 - Hemoperfusion/hemodialysis
- Symptomatic and supportive care
 - Protect liver: silymarin, penicillin, N-acetylcysteine, others
 - Treat bleeding
 - Fluid and electrolyte support
- Liver transplantation – use of biomarkers (such as Factor V) to guide decision

2. Hydrazines

Clinical features resemble Amanita poisoning, but less severe

Latent period of 6-10 hours

Followed by sudden onset of abdominal discomfort, vomiting, diarrhoea, severe headache

Primarily affect the liver

Haemolysis and CNS disturbance are rare

Mortality rate 2-4%

Treatment: supportive

Pyridoxine may be given for CNS symptoms

3. Orellanine

Toxins activated in liver, accumulates in kidney

Extremely long latent period of 3 to 14 days

Polydipsia and polyuria are the first symptoms

Followed by nausea, headache, muscular pains, chills
spasms and LOC

Renal tubular necrosis & renal failure in severe case

Death occurs several weeks after acute poisoning

Mortality rate ~ 15%

Recovery may require several months

Treatment: supportive

Neurotoxins

- Compounds that cause neurological symptoms
 1. Muscarine
 2. Ibotenic acid/muscimol
 3. Psilocybin

1. Muscarine

Cholinergic symptoms

Onset: 15-30 mins, duration: 2 hrs

Profuse sweating, salivation, lacrimation common

Abdominal pain, vomiting, diarrhoea

No CNS symptoms

Deaths, due to cardiac or respiratory failure, are rare

Treatment: activated charcoal, supportive

Atropine for moderate or severe symptoms

Muscarine



Fly Agaric





2. Ibotenic acid/Muscimol

Both toxins produce same effects

Muscimol is 5 times more potent than ibotenic acid

Onset: within 1-2 hrs, duration: few hrs

CNS depression: drowsiness, dizziness, deep sleep

Followed by CNS excitement: hyperactivity, excitability, tremor, illusions and delirium

Drowsiness may alternate with excitement

Prognosis is good and fatality is rare in adults

Children in large ingestion: convulsion and coma

Treatment: supportive



Panthercap



3. Psilocybin

Syndrome similar to alcohol intoxication

CNS symptoms: euphoria, hallucination

Sympathomimetic effects

Intentionally eaten for their psychotropic effects in religious ceremonies of certain American tribes

Onset: 10-30 mins, duration: 2 hrs

Rarely fatal in adults

May cause fever, convulsion, coma and death in small children if large dose is taken

Treatment

1. Activated charcoal
2. Rest in quiet and safe environment
3. Avoidance of external stimuli
4. IV benzodiazepines if necessary

Psilococybe cubensis



Panaeolus copelandia



Gastrointestinal irritants

Numerous mushrooms contains toxins that causes nausea, vomiting, diarrhoea and abdominal cramps

Chemistry of toxin virtually unknown

Symptoms similar to the protoplasmic poisons

Chief diagnostic difference: rapid onset

Symptoms may last for several days

Major concerns: dehydration & electrolyte imbalance

Fatalities are rare except in debilitated and extreme of age

Treatment: supportive