MUSHROOM POISONING

MUSHROOMS ARE A COMMON TOXIC EXPOSURE, MOST OCCURRING IN KIDS AND MOST TOXIDROMES BEING UNINTENTIONAL, WITH THE MAJORITY HAVING A BENIGN OUTCOME.

AVOIDANCE OF EATING WILD MUSHROOMS IS THE BEST MEANS OF PREVENTION, AS THERE ARE NO EASILY RECOGNISABLE DIFFERENCE BETWEEN NON-POISONOUS AND POISONOUS MUSHROOMS

MUSHROOM TOXINS ARE NOT HEAT LABILE AND SO ARE NOT DESTROYED OR DEACTIVATED BY COOKING

Table 214-1 Mushrooms: Symptoms, Toxicity, and Treatment

Symptoms	Mushrooms	Toxicity	Treatment
GI symptoms			
Onset <2 h	*Chlorophyllum molybdites*	Nausea, vomiting, diarrhea (occasionally bloody)	IV hydration
	Omphalotus illudens		Antiemetics
	Cantharellus cibarius		
	Amanita caesarea		
Onset 6–24 h	*Gyromitra esculenta*	Initial: nausea, vomiting, diarrhea	IV hydration, glucose; monitor AST, ALT, bilirubin, blood urea nitrogen, and creatinine levels, prothrombin time, partial thromboplastin time
	A. phalloides, *A. bisporigera*	Day 2: rise in AST, ALT levels	
		Day 3: hepatic failure	For *Amanita:* activated charcoal
			Consider penicillin G, 300,000–1,000,000 units/kg/d
			Silymarin, 20–40 milligrams/kg/d
			Consider cimetidine, 4–10 grams/d
			Consider hyperbaric oxygen therapy
Muscarinic syndrome	*Inocybe*	SLUDGE syndrome (*s*alivation, *l*acrimation, *u*rination, *d*efecation, *G*I hypermotility, and *e*mesis)	Supportive; atropine, 0.01 milligram/kg, repeated as needed for severe secretions
Onset <30 min	*Clitocybe*		
Central nervous system excitement	*A. muscaria*	Intoxication, dizziness, ataxia, visual disturbances, seizures, tachycardia, hypertension, warm dry skin, dry mouth, mydriasis (anticholinergic effects)	Supportive; sedation with diazepam, 0.1 milligram/kg IV for children; diazepam, 2–5 milligrams IV, or phenobarbital, 30 milligrams IV for adults
	A. pantherina		
Onset <30 min			
Hallucinations	*Psilocybe*	Visual hallucinations, ataxia	Supportive; sedation with diazepam, 0.1 milligram/kg or 5 milligrams IV for adults, or phenobarbital, 0.5 milligram/kg or 30–60 milligrams IV for adults
Onset <30 min	*Gymnopilus*		
Disulfiram reaction	*Coprinus*	Headache, flushing, tachycardia, hyperventilation, shortness of breath, palpitations	Supportive; IV hydration
2–72 h after mushroom, and <30 min after alcohol			β-Blockers for supraventricular tachycardia
			Norepinephrine for refractory hypotension

MUSHROOM TOXICITY IS DIVIDED INTO *EARLY (WITHIN 2 HOURS) AND DELAYED (6-20 HOURS) TOXICITY.* AS A GENERAL RULE, IF TOXICITY BEGINS WITHIN 2 HOURS, THE COURSE WILL BE BENIGN, IF SYMPTOMS OCCUR LATER, THE CLINICAL COURSE WILL BE MORE SERIOUS AND POTENTIALLY FATAL

EARLY ONSET GI SYMPTOMS:

- Most wild mushrooms cause mild GI irritation, most toxins are poorly described

CLINICAL FEATURES:

- Majority of mushroom-induced intoxications are mild and do not prompt visits to ED
- Chlorophyllum molybdites is an exception that may cause severe symptoms; acute onset of vomiting and diarrhoea <2 hours after ingesting the mushroom
    - Most commonly, symptoms are mild and self-limited. Causes dehydration and electrolyte imbalances.
    - May last 24 hours to days

TREATMENT:

- For toxic mushroom ingestions --> give ACTIVATED CHARCOAL 0.5-1G/KG, orally or by nasogastric tube
- Other treatment is largely supportive, and include IV fluid and electrolyte replacement when necessary

EARLY-ONSET NEUROLOGIC SYMPTOMS:

- Several classes of mushrooms cause neurologic symptoms:
    - HALLUCINOGENIC MUSHROOMS = “magic mushrooms”, ingested for mind-altering qualities. Contain PSILOCYBIN, effects similar to LSD
    - Isoxazole derivatives (ibotenic acic and muscimol) thought to mediate via GABA and anticholinergic activity

CLINICAL FEATURES:

- Symptoms typically develop within 2 hours of ingestion of hallucinogenic mushrooms
- Euphoria, a heightened imagination, a loss of the sense of time and visual distortions or hallucinations are common
- Symptoms generally last 4-6 hours
- Isoxazole-containing mushrooms produce symptoms within 30 minutes --> dizziness, mild intoxication, ataxia, muscular jerking, and anticholinergic symptoms (tachycardia, HT, warm and dry skin, dry orla mucosa, mydriasis)

TREATMENT:

- For hallucinogenic mushrooms [illegible] largely supportive. Place in dark/quiet room. Sedation with benzodiazepines as appropriate, preferred to phenothiazine derivatives, which lower the seizure threshold.
- For symptomatic isoxazole derivatives, ACTIVATED CHARCOAL, replace fluids and electrolytes as appropriate
- Consider PHYSOSTIGMINE FOR THOSE WITH SEVERE ANTICHOLINERGIC TOXIDROME

EARLY ONSET MUSCARINIC SYMPTOMS:

- Muscarine was the first mushroom toxin to be identified
- Inocybe and clitcybe genera are most commonly implicated
    - Typically found under conifers and hardwoods

Clitocybe candicans

PATHOPHYSIOLOGY:

- Muscarine is a PARASYMPATHOMIMETIC COMPOUND that is heat stable and acetylcholine-like
- It is not degraded by cholinesterase and therefore has a long duration of action
- Acetylcholine receptors on the heart, apocrine glands and smooth muscle are activated by muscarine

CLINICAL FEATURES:

- Characterised by SLUDGE syndrome:
    - **S** alivation
    - **L** acrimation
    - **U** rination
    - **D** efection
    - **G** I hypermotility
    - **E** mesis
- In addition --> diaphoresis from apocrine stimulation
- Muscle fasciculations
- Bradycardia and bronchorrhoea
- TYPICALLY PRESENT WITHIN 30 MINUTES OF INGESTION, SPONTANEOUSLY RESOLVE IN 4-12 HOURS
- TREATMENT:
- Most cases are mild and self-limited
- Emesis makes activated charcoal difficult
- ATROPINE IS THE ANTIDOTE FOR MUSCARINIC SYMPTOMS, given to those with severe symptoms, especially refractory bradycardia and hypotension, or those with diaphoresis, bronchorrhoea and oral secretions
    - Repeat dose of 1mg as needed, large doses may be needed

DELAYED ONSET GI SYMPTOMS:

- Two different genera (GYROMITRA AND AMANITA) cause significant toxicity, which characteristically presents several hours after ingestion
- Amanita genus responsible for 95% of deaths related to mushrooms
- AMANITA PHALLOIDES IS MOST TOXIC

Amanita phalloides

PATHOPHYSIOLOGY:

- Gyromitrin is a volatile heat-labile toxin that is responsible for symptoms
- Amanita phalloides contains several PHALLOTOXINS AND AMOTOXINS --> absorbed rapidly through the intestinal mucosa carried to the liver and UNDERGO ENTEROHEPATIC RECIRCULATION, thus resulting in prolonged toxin exposure after infection
    - Amanitin has greatest impact on cells with rapid protein synthesis and turnover (e.g. GI mucosa, hepatocytes and renal tubular epithelium

CLINICAL FEATURES:

- Distinctive features of GYROMITRIN-CONTAINING MUSHROOM TOXICITY ARE INTENSE GI SYMPTOMS/SIGNS (NAUSEA, VOMITING, WATERY DIARRHOEA) DEVELOPING 6-24 HOURS, but most typically 6-8 hours after ingestion
    - Hypovolaemia is common and in severe cases, hepatic failure is evident on day three. HYPOGLYCAEMIA MAY COEXIST
- Those who ingest AMATOXIN-CONTAINING MUSHROOMS also have delayed onset of GI symptoms (6-24 hours)
    - Typically the later the onset of GI symptoms, the milder the disease

    - Gastroenteritis is often intense, requiring fluid and electrolyte replacement
    - FOUR STAGES:
        - First: latent, absence of any signs
        - Second: abdominal pain, nausea, vomiting and diarrhoea (both stools and vomitus can become bloody). *Misdiagnosed as gastro.*
        - Third: convalescent phase --> patient feels and looks better, but levels of liver enzymes begin to rise, heralding onset of liver damage
        - Fourth: 2-4 days post ingestion, transaminase levels rise dramatically and liver/renal function deteriorates. Hyperbilirubinaemia, coagulopathy, hypoglycaemia, acidosis, hepatic encephalopathy and hepatorenal syndrome are all noted
- Mortality rate from GYROMITRA ingestion is ~15-35% and is generally attributed to hepatic failure, renal failure or fluid/electrolyte disorders
- Historically, amatoxin liver failure had mortality rates as high as 50%, but improved care for hepatic failure and availability of liver transplant have improved rates to 10-15%
- Diagnosis relies on clinical suspicion, identification of species of mushroom relies on trained mycologist

TREATMENT:

- If a patient presents with severe vomiting and diarrhoea within a few hours of mushroom ingestion, ADMINISTRATION OF ACTIVATED CHARCOAL IS INDICATED.
    - Repeated doses of charcoal for at least 24 hours, particularly in presence of amatoxin due to enterohepatic circulation
- Monitor fluid, electrolyte status, as well as glucose levels
    - *HYPOGLYCAEMIA IS A MAJOR CAUSE OF EARLY DEATH*
- Monitor closely for 24-48 hours for appearance of hepatic or renal failure --> low protein diet and standard supportive therapy for hepatic failure
- FFP, vitamin K used for coagulation issues
- In severe cases, preparation for liver transplantation is made
    - No firm criteria; consider in progressive coagulopathy, encephalopathy, renal failure in spite of maximal medical therapy
- GYROMITRIN SPECIFIC TREATMENT --> neurologic symptoms can be treated with high-dose pyridoxine for regeneration or GABA (25mg/kg IV up to 25g/day)
- AMATOXIN-SPECIFIC TREATMENT --> HIGH-DOSE PENICILLIN advocated, as it blocks uptake of amatoxin into the liver by its shared active transport (huge doses of penicillin G (300,000 units/kg/day).)
    - SILYMARIN; acts a free radical scavenger and may interrupt enterohepatic circulation
    - CIMETIDINE; in huge doses (10g/day) is effective in animals