Mushroom Poisoning

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Abstract

Wild mushrooms that grow in forests and meadows, are of various types, and it is common for the local population to consume them. Nevertheless, mushrooms are one of the most common toxic exposures, with over 12000 mushroom exposures reported to poison centers in 1996, or roughly 5 for every 100000 population. Despite these measures, the species is unknown in >90% of digestion. The symptoms of mushrooms digestion range from asymptomatic to fatal. We should learn more about identifying most common mushroom species. Besides, treating in patients with mushroom intoxication should depend on the patient's clinical conditions and vital signs.*(Ann Disaster Med. 2004;3 Suppl 1:S8-S11)*

Key words: Mushroom; Amatoxins; Plants Intoxication

Introduction

Wild mushrooms that grow in forests and meadows, are of various types, and it is common for the local population to consume them. Nevertheless, mushrooms are one of the most common toxic exposures, with over 12000 mushroom exposures reported to poison centers in 1996, or roughly 5 for every 100000 population. While most mushroom ingestions do not cause a clinically significant toxidrome, the lethal potentials of a select few make mushroom toxicity an important subject. Ingestion is the most common route of entry, but intravenous injections of mushroom toxins and inhalations of mushroom spores have been reported. The symptoms and signs of mushroom poisoning range from mild gastrointestinal symptoms to organ failure and death. Toxicity may also vary based on the amount ingested, the age of the mushroom, the season, the geographic location, and the way in which the mushroom has been prepared prior to ingestion. Eating poisonous mushrooms can cause various types of reactions, such as allergic gastroenteritis, psychological relaxation and fatal liver intoxication[1-3]. The pathogenicity of these mushrooms depends on the cyclopeptide toxins[4]. Despite these measures, the species is unknown in > 90% of digestion. However, Amantia species are responsible for the vast majority of deaths[5-7].

Mushroom toxidromes are classified according to toxins and clinical presentations. Mushroom toxins have been divided into the following 7 main categories:

- Amatoxins (cyclopeptides)
- Orellanus (Cortinarius species)

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- Gyromitrin(monomethylhydrazine)
- Muscarine
- Ibotenic acid
- Psilocybin
- Coprine(disulfiramlike)

Mushrooms of the genera Amanita and Lepiota contain amatoxins, which are thermostable and bicyclic peptide toxins. Amanitin phalloides syndrome or Mycetismus choleriformis accounts for 90-95% of all fatalities from mushroom poisoning in North America. This discussion follows a clinical format because the offending mushroom is frequently unavailable for identification and poisoning may occur from a single species or a combination of different species. Trestrail's data indicate that the mushroom was available for identification in only 3.4% of exposures. Amatoxins, especially amanitin, are absorbed by the gut and degrade the cells of liver and kidneys[8-12].

Management

There are nine general groupings useful for clinical management. These groups of toxin can be divided into early toxicity (within 1 hour after ingestion) and delayed toxicity (6 hour to 20 days). The groups causing early onset of symptoms include Coprine, GI toxin, Ibotenic acid, muscimol, Muscarine and Psilocybin. The other groups causing late onset of symptoms include Cyclopeptides, Orellanus and Gyromitrin.

Treatment

Asymptomatic patients

- When suspecting mushroom poisoning, try to get the specimen as possible as you can, and then contact a regional poison control center.
- Give the patients 0.5-1.0 g/kg of activated charcoal orally and intravenous fluid to prevent dehydration or electrolytes imbalance
- Monitor patients in the emergency department for more than four hours. If patients remain asymptomatic, you can discharge them with adequate instructions. Advise patients to contact the hospital immediately if they become symptomatic or have any discomfort. If the mushroom is identified as potentially toxic or the patient becomes symptomatic, admission to the hospital is recommended.

Symptomatic patients

- The basic elements of supportive care are critical in the evaluation and management of the poisoned patients. The priority must beasfollowing: airwayàbreathingàcirculation.
- Obtain the mushroom specimen as possible as you can.
- Consider gastric lavage with activated charcoal every 2-6 hours.
- Cardiopulmonary monitoring should be available.
- Closely monitor fluid, electrolytes, and glucose status and correct them. Rehydrate with isotonic fluids. Forced diuresis is not recommended.
- If amanitin ingestion is suspected or proven, careful attention to clotting studies and renal and hepatic profiles is important. Early consultation with a medical toxicologist is recommended.
- Intensive unit care may be necessary when the patient's condition is poor.

Special consideration

Amatoxin

Amatoxins within the mushrooms are the most common fatal conditions in mushroom poisonings. It has a latent period of 6-12 hours after digestion. At the period, the patient may have GI symptoms. Hepatic and renal failure may be encountered. Deaths may occur in 3-7 days. Mortality rates range from 10-60%. Some drugs may be useful according to animal experiments, but only anecdotal support is available for humans.

1. High doses of penicillin (300,000 to 1,000, 000 U/kg/day) are required to decrease toxicity.
2. Vitamin K (if coagulopathy is present)
3. Silybinin (water-soluble milk thistle extract, not available in the US)
4. Hyperbaric oxygen
5. High-dose cimetidine, vitamin C, zinc, and thiol compounds are useful in animal models.

Neurological symptoms

Psilocybin or psilocin toxins are neuroactive chemicals similar to lysergic acid diethylamide (LSD).

Anti-cholinergic symptoms

The symptoms include tachycardia, hypertension, warm, dry skin and mucous membranes, and mydriasis. When patients have anti-cholinergic symptoms, physostigmine may be considered.

Muscarinic symptoms

Muscarinic symptoms are characterized by the "SLUDGE" syndrome. The "SLUDGE" are as following: salivation, lacrimation, urination, defecation, GI hypermotility, and emesis. Atropine can be used for bradycardia and hypotension. Oxygen and inhaled beta-agonists are also helpful in treating patients with increased pulmonary secretions and bronchospasm.

Renal failure

Nephrotoxins in mushrooms are norleucine and chlorocrotylgycine. Patients digesting nephrotoxin-contained are usually asymptoms. Supportive hemodialysis may be required in 30-50% patients. The others recover without sequalae.

Accompanying alcohol digestion

A kind of mushroom, Coprinus genus, contains coprine which is chemically related to disulfiram. The toxin inhibits alcohol dehydrogenase 2 hours after digestion, and the effect may last up to 72 hours. When the patients digest alcohol with this kind of mushroom, the major symptoms are facial flushing, headache, tachycardia, nausea and vomiting.

Conclusion

The species of mushroom are numerous. There are various clinical presentations depending on the ingested species.

Most ingested species remain unknown. Treatments include gastric decontamination (activated charcoal), observation of 12 to 24 hours for delayed-onset symptoms (which may indicate serious toxicity), laboratory studies, intravenous fluid hydration and supportive care. In general, most cases of the mushroom poisonings are mild to moderate gastrointestinal upset. There are no rules available about treating mushroom intoxication or identifying mushroom toxin in the emergency department, so

the diagnosis and treatment must be based on the history of ingestion and associated clinical presentations.

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