

Clinical Approach To Toxic Mushroom Ingestion

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Background: This review provides the physician with a clinical approach to the diagnosis and management of toxic mushroom ingestion. It reviews the recent literature concerning proper management of seven clinical profiles.

Methods: Using the key words "mushroom poisoning," "mushroom toxicology," "mycetism," "hallucinogenic mushroom ingestion," and "Amanita poisoning," the MEDLINE files were searched for articles pertinent to the practicing physician. Much of the original data were gathered at the Aspen Mushroom Conference held each summer throughout the 1970s at Aspen, Colorado, sponsored by Beth Israel Hospital and the Rocky Mountain Poison Center. Texts related to poisonous plants and specific writings concerning mushroom poisoning were also consulted; many of these texts are now out of print.

Results and Conclusions: The 100 or so toxic mushroom groups can be divided into seven clinical profiles, each of which requires a specific clinical approach. Two of the seven groups (amanitin and gyromitrin) have a delay in onset of symptoms of up to 6 hours following ingestion and provide essentially all the major morbidity and mortality associated with toxic mushroom ingestion. These two groups are the major focus of this review. Treatment of the potential mushroom ingestion, as well as guidelines for asking clinical questions, are included. These questions serve as a form of algorithm to assist the clinician in arriving at the correct toxic group. (J Am Board Fam Pract 1994; 7:31-7.)

The diagnosis and treatment of mushroom poisoning have advanced considerably since the early 1970s. The Colorado Mycological Society and the Rocky Mountain Poison Center in Denver have provided leadership in transforming a confusing array of symptoms and signs, treated frequently with atropine, into a systematic structure guided by patient history, physical examination, and laboratory assessment. The physician is provided a sound approach to assisting those who have mistakenly or by design consumed mushrooms that are causing uncomfortable or life-threatening problems.^{1,2}

Of the 5,000 to 10,000 species of mushrooms present in North America, about 100 are considered toxic and 100 are considered edible (only a minority have actually been tested for edibility). Toxic mushrooms can be grouped into seven categories, each with recognizable symptom complexes and respective treatment plans (Table 1).² Only two groups have life-threatening consequences; both of these will receive major atten-

tion in this review. The other five groups cause self-limited symptoms and signs; these ingestions pose no threat to immediate survival.

It is always important for the treating physician to have mushrooms identified, if at all possible. Consulting with local mycologists or becoming familiar with field guides and dichotomous keys might be necessary. Poisindex, available through MicroMedex, contains a fungal identification system and botanical terms.³ Frequently, however, mushrooms are not available.

This clinical review provides a systematic approach based on history and examination, allowing the physician to care for the patient without necessarily having the mushroom in hand or a mycologist to consult.

Although North Americans generally are mycophobic, wild mushroom ingestion is becoming more common as a result of increased availability of field guides, cookbooks, and other writings about wild mushrooms. The true incidence of mushroom poisoning is unknown (it is not a reportable illness). The American Association of Poison Control Centers maintains a national data bank of poisonous exposures. They estimate that mushroom encounters represent 0.6 percent of all human exposures to poison substances, with 7023 exposures in 1987 and 7834 exposures in 1988.³

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Table 1. Seven Toxin Groups of Commonly Ingested Mushrooms.

Group	Mushroom Toxin
1	Cyclopeptide (amanitin)
2	Gyromitrin (monomethylhydrazine)
3	Coprine (disulfiram-like reaction)
4	Muscarine
5	Ibotenic acid-muscimol (isoxazole)
6	Psilocybin
7	Gastrointestinal irritation

In 1989, more than 2000 patients required treatment, and more recent occurrences have claimed lives.⁴ The largest age group affected by potentially toxic mushroom ingestion (more than 50 percent) are 12- to 14-month-old infants. Most common is the potential ingestion, where mothers who find their children with little brown mushrooms in their mouths seek advice on management. First- and second-generation Americans of central and southern European origins are among the most often poisoned, along with recent Asian immigrants. California leads the country in mushroom poisonings, with Northeastern states close behind.⁵

Classification of Mushroom Poisonings

Individual mushroom toxicity varies depending on the time of year, location, soil characteristics, stage of life cycle (age), hybridization, and individual susceptibility of the patient. As a food substance, compared with raw fruits and vegetables, mushrooms are low in calories, high in protein and crude fat, low in carbohydrate, and high in total minerals. They are also high in phosphorus, iron, and B vitamins. The high-protein and iron-containing forms are difficult to digest. Large amounts of mushrooms would need to be eaten to obtain the minimum daily requirements of B vitamins (0.5 to 3.3 pounds).

Mushroom poisonings can be grouped into seven types, two with delayed onset of symptoms, and five with immediate onset. Those with delayed onset of symptoms will be discussed first, as they represent the greater portion of fatal ingestions. This classification is based upon ingestion of a single mushroom type, as a mixed ingestion (more than one type) could provoke both immediate and delayed symptoms. Group numbering represents the most accepted arrangement of poisonings (Table 1).

Group 1: Cyclopeptide (*Amanitin*) Poisoning

Poisoning by the genus *Amanita* accounts for 90 to 95 percent of fatal ingestions in North America, and it ranks third worldwide in incidence of oral chemical intoxications.⁵ A single mushroom cap contains enough toxin to be lethal. Of the three common *Amanita* species (*A. phalloides*, *A. verna*, and *A. virosa*), *A. phalloides* causes more than 90 percent of fatalities. Figure 1 provides basic anatomical features for identification. Most poisonings by these mushrooms could be prevented by recognizing these characteristics.

A little brown mushroom of the genus *Galerina* also contains amanitin. This wood-loving mushroom is quite common in the fall. Fifteen to 20 *Galerina* can provide an adult a lethal dose. Children and hallucinogenic mushroom seekers are the most likely ones to ingest this mushroom.

Modern chemical work has shown amanitin to be an eight-amino acid thermostable cyclopeptide, one of the most toxic materials known. Amanitin is absorbed directly through the intestine with enterohepatic circulation lasting up to 48 hours. It is weakly bound to serum proteins,

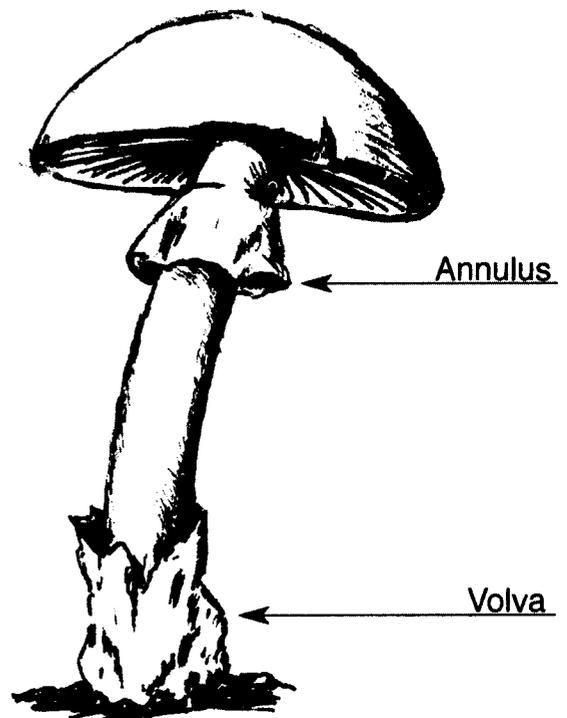


Figure 1. Death Cap, *Amanita phalloides*. Drawing by Frank Batcha, MD.

and it acts by inhibiting messenger RNA production and, therefore, protein synthesis.⁵

The organs most severely affected are those most active in protein synthesis (gastrointestinal epithelium, liver, kidneys, and pancreas). The 6- to 12-hour delay in onset of symptoms (range 2 to 36 hours) relates to depletion of protein stores. There might be a direct neurotoxic effect as well. Characteristic microscopic changes occur in the hepatocellular nucleus and nucleolus and in the convoluted renal tubules. In severe cases there is massive hepatic necrosis with centrilobular hemorrhage. Most of the renal damage could be due to hepatorenal syndrome or hypovolemia and might not reflect direct toxic injury.⁵

The presenting symptoms are nonspecific and cholera-like (Table 2). A latent phase should not be misconstrued as apparent recovery. Liver, renal, glucose, and coagulation tests will provide an indication of impending difficulty. Hepatic and renal injury are next apparent, with possible progression to fulminant hepatic failure. The differential diagnosis includes other toxins (heavy metals, drugs, botulism), viral gastroenteritis, and food poisoning (*Vibrio*, *Staphylococcus*, or *Salmonella* organisms).

Mainstays of treatment are outlined in Table 3. Intensive care, including management of dehydration, liver support, and maintenance of renal function can reduce mortality from 50 percent to as low as 5 percent. The gastrointestinal phase (2 to 3 days) results in dehydration, hypovolemia, and acid-base electrolyte disturbances. Renal failure can supervene and might be prevented with adequate fluids. Forced diuresis facilitates renal excretion of adsorbed amatoxins. Activated charcoal is given for 72 hours to interrupt the enterohepatic cycle. Prothrombin time can be indicative

Table 2. Signs and Symptoms of Cyclopeptide (Group 1) Poisoning.

Phase	Signs and Symptoms
1	Delayed 6–24 hours postingestion Nausea and vomiting Sharp abdominal pain Bloody diarrhea
2	Improvement 12–48 hours
3	Abdominal pain Hepatic failure with jaundice, bleeding Renal failure Convulsions, coma, death

Table 3. Treatment of Cyclopeptide (Group 1) Poisoning.

Treatment	Measures
General	Reduce absorption using 30–100 g of activated charcoal in 8 oz water, repeated every 6 h for 72 h Provide adequate urine output with fluids, furosemide Monitor intake and output of fluids
Specific	Order and follow: liver panel, glucose, blood urea nitrogen, creatinine, protime, activated thromboplastin time, and fibrinogen For hepatic support — lactulose, neomycin, decreased dietary protein, vitamin K, thiamine For renal support — fluids, furosemide, dialysis, penicillin G 300,000–1,000,000 U/kg/d, silibinin 20–50 mg/kg/d

of outcome, and abnormalities should be treated with vitamin K, fresh frozen plasma, and cryoprecipitate. Hypoglycemia, frequently severe, represents a grave prognostic sign, reflecting massive hepatic necrosis.⁵

A number of specific therapies have been suggested, but not proved, by controlled clinical trials. Thioctic acid was first used for treatment in the United States in 1970. It acts as a coenzyme, exerting an effect at the level of the Krebs cycle. Hypoglycemia accompanies its use and must be treated with concomitant administration of intravenous glucose. This unproven therapy could be tried until a better treatment is found.

Penicillin in high doses could help to promote renal excretion, but its mechanism of action is as yet unknown. Good clinical evidence supports its use, especially when combined with silibinin, a flavolignone that interrupts the enterohepatic recirculation of amanitin and inhibits the penetration of amanitin into hepatic cells.^{6,7}

Corticosteroids have been used to treat acute toxic hepatitis. Intravenous insulin and growth hormone have been suggested to induce liver cell proliferation. Plasma exchange has also been recommended.

Because the toxin is absorbed directly through the intestine, with enterohepatic distribution, focus should be on gastrointestinal aspiration, neutralization of the toxin, and forced diuresis. A duodenal tube should be inserted, with administration of charcoal through the distal port and removal of gastroduodenal fluid from the proximal port.

Criteria are now being developed for orthotopic liver transplantation (Table 4). A number of patients with fulminant hepatic failure can be saved with this procedure. Patients in whom hepatic encephalopathy develops should be sent to a center that can provide liver transplantation as an option.⁷⁻¹¹

Group 2: Gyromitrin (Monomethylhydrazine)

Poisoning

Poisonings by the genus *Gyromitra* (false morels) are responsible for the second group of fatal ingestions, have delayed onset of symptoms, and account for the rest of the fatalities from mushroom poisonings in the United States. As one of the commercial mushrooms in Europe, *Gyromitra* causes 24 percent of mushroom fatalities there. Since 1900 only about 20 poisonings have been reported in the United States, but there has been a 50 percent mortality rate.

This large, fleshy mushroom family is ubiquitous throughout the United States and is especially numerous in the spring. Although it can be confused with the common morels (*Morchella*), it looks more like a brain on a stem, without the pitted appearance so characteristic of morels. *Gyromitra esculenta* (Figure 2) is the prototype poisoner in this group. Mushroom handbooks present variable recommendations, and toxicologic studies indicate a markedly varied amount of toxin among species, even within the same species at different locations.^{12,13} The toxin gyromitrin is hydrolyzed in the body to produce monomethylhydrazine (MMH), a common rocket fuel. Clinical illness from ingesting the mushroom or from exposure while handling the fuel is the same.^{1,2,14}

Those who insist on eating *Gyromitra* (it has, in fact, a low incidence of poisoning) should adhere to parboiling the mushrooms for 15 minutes



Figure 2. False Morel, *Gyromitra esculenta*. Drawing by Frank Batcha, MD.

twice, each time discarding the used water and avoiding the fumes. The mushrooms should then be sauteed again before eating. They should never be consumed raw. Because of severe illness associated with eating this mushroom, and because parboiling is not 100 percent effective, it is best to avoid eating all mushrooms of this genus.

The presenting symptoms of gyromitrin poisoning are somewhat similar to those of *Amanita* poisoning (Table 5). The toxin interferes with normal utilization of vitamin B₆, is a gastrointestinal irritant, is a hemotoxin and a hepatotoxin, and is quite toxic to the central nervous system. Methemoglobin can be detected early in the blood and is an indicator of MMH exposure. Liver enzymes rise, blood glucose falls, and free hemoglobin can be detected in the serum.

Mainstays of treatment begin with general measures similar to those outlined earlier for *Amanita* poisoning (Table 3). Of special note is the use of pyridoxine, 25 mg/kg intravenously, daily, to help reverse the inhibition of enzyme reactions that use pyridoxal phosphate as a cofactor. Hepatic and renal laboratory values are followed and tracked accordingly, as toxic hepatitis could occur and renal injury might result from hemolysis.¹⁴

Group 3: Coprine Poisoning

Poisoning by the common and edible inky cap mushroom (*Coprinus atramentarius*) occurs only with an associated ingestion of ethyl alcohol and is similar to that produced by the ingestion of

Table 4. Criteria for Orthotopic Liver Transplantation.*

Stage II encephalopathy
Protime greater than 2 times normal despite treatment
Acidosis and hypoglycemia requiring glucose
Serum bilirubin greater than 25 mg/dL and/or marked aspartate aminotransferase and alanine aminotransferase elevations
Gastrointestinal hemorrhage

*Individuals meeting these criteria should be in a transplantation center but might not require transplant.

Table 5. Signs and Symptoms of Gyromitrin (Group 2) Clinical Presentation.

Phase	Signs and Symptoms
1	Nausea, vomiting, watery diarrhea Lower abdominal cramps and distension Weakness, lassitude, headache, cyanosis
2	Fever, jaundice Confusion, ataxia, convulsions, coma

alcohol and disulfiram. Investigators were unable to find disulfiram in the mushroom but did isolate coprine, a cyclopropanone derivative.^{4,15,16} Reports of *Coprinus*-alcohol reactions are common in the literature, antedating the discovery of the syndrome and the subsequent isolation of coprine. With blockage of acetaldehyde dehydrogenase, acetaldehyde accumulates in the serum and causes its vasomotor effects through the beta receptors of the autonomic nervous system. Flushing of face and neck, nausea, vomiting, and sweating occur and can be followed by chest pain and palpitations. *Coprinus comatus* (shaggy mane) does not produce this reaction. Nausea and vomiting can be associated with eating morels, the chicken mushroom (*Polyphorus sulphureus*), and a few other species while drinking alcohol; however, this reaction does not appear to be related to acetaldehyde accumulation, and ingesting these mushroom varieties does not produce the syndrome as described.

Coprine-alcohol poisoning is self-limited (2 to 4 hours) and might not require treatment, but general measures (as with *Amanita*) should be instituted. Cardiac arrhythmias can occur and can be treated orally or intravenously with propranolol; hypotension is treated with isotonic fluids.

Group 4: Muscarine Poisoning

Muscarine, a parasympathetic stimulant, was the first chemical toxin found in mushrooms and was once thought to be the primary toxic ingredient in most mushroom poisonings. Atropine, useful against this toxin, was considered the antidote for all mushroom intoxications. In fact, atropine should only be used for this toxin group; its use could increase the severity of poisonings from other mushroom groups (e.g., hallucinogenic *Amanita*).

Muscarine is found in the little brown *Inocybe* and *Clitocybe* mushrooms that reside around shrubs and in grassy areas. They are ingested by small children or are misidentified by mushroom hunters searching for hallucinogenic mushrooms.

Deaths have been reported in Europe but none in the United States.¹⁷

Muscarine poisoning produces an onset of symptoms within 30 to 120 minutes that lasts up to 2 hours if untreated. The ensuing perspiration-salivation-lacrimation syndrome with miosis does not occur with any other mushroom poisoning. Nausea, vomiting, and abdominal pain can occur, and wheezing and bradycardia might produce shortness of breath and hypotension. Along with general treatment measures as outlined for *Amanita*, intravenous fluids and atropine can be given for those severely intoxicated. The dose of atropine ranges from 0.5 to 1 mg given intravenously for adults, repeated frequently, and 0.05 mg/kg in children. Treatment is titrated to control output of secretions.

Group 5: Ibotenic Acid — Muscimol Poisoning (Hallucinogenic Amanitas)

The genus *Amanita* also contains several mushrooms that are widely known for their hallucinogenic properties. Anatomically they are identical to their lethal relatives, except for a brightly colored cuticle on top of the cap. The species *Amanita muscaria* has a bright red or yellow cuticle. This cuticle and the area immediately beneath contain the psychoactive isoxazoles ibotenic acid and muscimol. *Amanita pantherina* (the panther mushroom), which is a brown-capped species, contains two to four times as much toxin as *Amanita muscaria* and has caused deaths in the Pacific Northwest. Toxin content varies with species, time of year, latitude, and local environmental conditions.¹⁸

Muscimol is considerably more active than ibotenic acid, which is metabolically converted to muscimol after ingestion. Drying these mushrooms increases the muscimol content. Muscimol is excreted unchanged in the urine and can be cleared from the body before maximum central nervous system excitation occurs. Drinking the urine of those who ingested the mushroom transmits the excitatory activity. These chemicals appear to act as false neurotransmitters in the γ -aminobutyric acid system.

Thirty to 60 minutes following ingestion (e.g., smoking dried mushrooms, drinking tea, eating soup, swallowing small pieces whole) inebriation begins with a drunken appearance, ataxia, and muscle cramps. Sleep following ingestion is quite deep, and the victim might not be arousable.

Symptoms persist for up to 8 hours with the clinical response depending on the amount of toxin ingested. Physical hyperexcitability with euphoria, agitation, visual disturbances, and hallucinations can be quite profound and could require chlorpromazine for management. Seizures might require diazepam. The experience for the individual depends on whether the toxin was ingested knowingly or accidentally. Accidental ingestions tend to be quite distressful and are seldom repeated. The variables of set (expectations and personality) and setting (physical and social environment) in which the drug is taken in large part determine the outcome of the intoxication.¹⁹ Atropine is contraindicated. Ibotenic acid and muscimol are available commercially.

Group 6: Psilocybin Poisoning ("Magic Mushrooms")

A second group of hallucinogenic mushrooms comes from the three genera *Psilocybe*, *Panaeolus*, and *Gymnopilus*. Mushrooms of the *Psilocybe* group stain blue when handled. These small dung-dependent mushrooms contain psilocybin, a lysergic acid diethylamide-like tryptophane derivative. These mushrooms, like the *Amanita muscaria* group, have been eaten recreationally for hundreds of years, with Mexican and Central American use of these drugs well described.²⁰ Field guides especially for these genera are available.²¹⁻²³

In the Pacific Northwest, this group of mushrooms is avidly consumed for its hallucinogenic properties. Psilocybin effects are dose related and consist of nausea, anxiety, weakness, and hyperkinetic compulsive movements. They are also often associated with laughter, euphoria, hallucinations, and impaired time-space perception. When unpleasant experiences or accidental ingestions occur, medical help might be sought. Protective measures might be necessary for dangerous hyperkinetic physical behavior, and chlorpromazine helps with hallucinations. Children can develop seizures, hyperpyrexia, and death from ingestion. Diazepam and cooling measures should be used. Avoid aspirin.

Group 7: Gastrointestinal Irritants

Most mushroom poisonings involve a simple 24-hour flu-like illness with vomiting and diarrhea. Onset occurs within 30 minutes to 2 hours and could result in dehydration. Mushroom identification should be attempted to rule out groups 1 and 2 poisonings.

Treatment guidelines for gastroenteritis should be followed. Some mushrooms, such as *Chlorophyllum molybdites*, can cause a protracted gastrointestinal illness lasting many days, requiring hospitalization and careful fluid and electrolyte management.

Differential Diagnosis of Mushroom Intoxication

Based on the above information, the attending physician should be able to make an accurate assessment of the patient's clinical problem and institute appropriate treatment with or without the mushroom or a mycologist at hand.^{24,25} Ask the following questions:

1. *When were the mushrooms eaten and when did the symptoms first occur?* Those ingestions producing symptoms within 2 hours are rarely serious, whereas those with a 6-hour or longer latent period (groups 1 and 2) are frequently life threatening.
2. *Was more than one kind of mushroom ingested?* More than one kind of mushroom could eliminate the latent period characteristic of groups 1 and 2 poisonings, providing a false sense of security about the seriousness of the intoxication.
3. *What were the initial symptoms?* Early-onset gastroenteritis, perspiration-salivation-lacrimation syndrome, hallucinations, inebriation, and a reaction associated with alcohol all suggest a diagnosis. Delayed onset of a cholera-like illness or abdominal pain with headache suggests the more serious groups 1 and 2 poisonings.
4. *Was an alcoholic beverage consumed within 72 hours after a mushroom meal?* It is possible with a group 3 poisoning to have delay in onset of symptoms for many hours or days between consumption of mushrooms and consumption of alcohol, although symptoms occur rapidly once alcohol is consumed. Confusion with the prolonged latent period could cause undue alarm regarding question 1.
5. *Did everyone who ate the mushrooms become sick?* If only one individual became ill, it could be food sensitivity rather than poisoning. Consider other illnesses or poisonings rather than mushroom poisoning.
6. *Did anyone who did not eat mushrooms become sick?* Consider food poisoning or other illnesses. Nontoxic mushrooms that were cooked, refrigerated, or canned can spoil.

Treatment of the Potential Ingestion

The most frequent clinical problem relating to mushrooms facing the primary care or emergency department physician is potential ingestion.³ Children are frequently found with little brown mushrooms in their mouths or hands, and the parent brings the child in for reassurance. The child is almost always without symptoms, unless ipecac was given at home with subsequent emesis. Some little brown mushrooms (e.g., *Galerina*, *Inocybe*, *Clitocybe*, *Psilocybe*) can be life threatening to children. Evacuation of the child's stomach contents should be carried out (preferably with ipecac), and a slurry of 20 to 50 g of activated charcoal in water should be given. A specimen of the mushroom should be kept and stored in alcohol until the child is free of illness. Identification of toxic ingestion and confirmatory signs and symptoms should follow adjusted adult treatment protocols.

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