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AHC Media

# Mushroom Toxicity

*Several decades ago, two patients arrived in my emergency department with intense vomiting, bronchorrhea, and seizure-like activity. Their symptoms started about 30 minutes after consuming a mushroom stew. The wife considered herself to be an expert in mushroom identification and had picked several species of mushrooms, including a few very large ones with bright red tops. Both patients required intubation and several milligrams of atropine to handle their bronchial secretions. Several hours before, they had eaten a mushroom stew containing mushrooms they had picked that morning. The family brought in a plastic container of multiple different types of slimy, decaying mushrooms. A trained mycologist identified three different toxic species, including* Amanita muscaria, Amanita pantherina, *and Inocybe species. The patients made an uneventful recovery and they were warned against future foraging for mushrooms. So began my interest in mushroom toxicity.*

*— Sandra M. Schneider, MD, FACEP, Editor*

## Introduction

Toxicology can be confusing with the myriad of drugs and chemicals, each with their own toxicity and treatment. Add to that the large number of potential toxins from plants and mushrooms. This paper will attempt to simplify mushroom toxicity for the practicing emergency physician.

Of the more than 40,000 species of fungi known, only about 800 of these are toxic, and only a few are potentially fatal. In the United States and Europe, the most common deadly mushrooms belong to the *Amanita* family. Although mushrooms add texture and taste to food, they have little nutritional value. Commercially grown mushrooms are generally *Agaricus bisporus* (the large white mushroom) or shiitake (the brown, flat-capped mushroom popular in Chinese dishes). These mushrooms are grown under meticulous conditions with strict quality control. There are a few individuals who may have an allergic reaction to them, although true anaphylaxis is very rare.[1-3] In general, they are nontoxic.

This paper will discuss toxicity, the clinical scenario seen by emergency physicians — accidental ingestion of a lawn mushroom by a child, intentional ingestion to get "high" — and symptoms after eating foraged mushrooms.

## Case 1

A 3-year-old boy is brought to the emergency department after he was found chewing on a little brown mushroom in his front lawn. The parents immediately made him spit it out and then rinsed out his mouth. He is asymptomatic at this time. They brought the mushroom with them. They did not call poison control.

## Low-toxicity Mushrooms

Most lawn mushrooms are benign, although some species can be toxic. The local poison center can be very helpful in these cases. Personnel at the local poison center are generally familiar with the species of mushrooms found in the area. In addition, they often have a mycologist they can contact to assist

## Executive Summary

- Commercially grown *Agaricus bisporus* mushrooms bought in the grocery store are nontoxic.
- Several wild mushrooms cause gastrointestinal symptoms of nausea, vomiting, and diarrhea. If the symptoms begin shortly after eating (within 1-2 hours), the ingestion is benign.
- GI symptoms that are delayed (onset 4-24 hours) after ingestion suggest a more severe and possibly life-threatening ingestion. The most common ingestion of this type in the United States is *Amanita phalloides,* which grows primarily in the fall.
- Some mushrooms, for example *Amanita muscaria* and *Inocybe*, can cause a SLUDGE syndrome with increased salivation, lacrimation, GI upset, and bronchorrhea.

in identification of species that are questionable.

If the mushroom is available, consider sending a picture of it to the poison center, either by fax or mobile phone. There are specific areas the experts will want to see, so be sure to include these. The size and color of the mushroom cap are important. Many mushrooms have small dots on the top or even flakes of material adherent to the cap. These are warts, the remnant of a membrane or veil that once covered the mushroom. Warts are often seen on the *Amanita* species, some of which can be deadly.

Underneath the cap are the gills. Gills come in a variety of colors, but more important is the way they attach to the stem of the mushroom. Gills that end before the stem are called free gills — another characteristic of *Amanita* mushrooms. The shape of the stem is also important. *Amanita* species generally have stems that widen toward the ground and often end in a cup or rounded structure that holds up the mushroom. This cup is often left in the ground when the mushroom is harvested. The stem or stalk may have a membrane attached like a small skirt, called a ring. This, again, is the remnant of the veil and an important clue to identification. Describing these components, or better yet, photographing them, can greatly aid in identification of a mushroom.

Rarely, when serious ingestions are suspected, the poison center may want the actual specimen. It is important to place the mushroom in a paper bag, not a plastic one. Plastic accelerates the decomposition of mushrooms and will make the specimen much harder to identify. It is rare that specimens are required.

The most common reaction to mushrooms is nausea, vomiting, and diarrhea. It is very important to note the onset of these symptoms, as they relate to the time of the ingestion. Early onset of symptoms, within 1-2 hours, indicates a benign ingestion. The onset of symptoms after 4 hours and up to 24 hours can be ominous. Mushrooms that cause late onset of symptoms are nearly always intentionally ingested and will be discussed in case 3.

***Figure 1:*** Mushroom Cap with Warts

**The cap of a mushroom species photographed from above showing the characteristic "warts." These are adhered remnants of the veil or membrane that covered the mushroom when it emerged from the ground. At times these warts are washed off and white spots are present on the cap.**
**Image used with permission from Sandra Schneider, MD.**

While hundreds of mushrooms can cause early symptoms, the most common is *Chlorophyllum molybdites*, known as the green parasol. It grows primarily in the summer and resembles *Agaricus bisporus* — the white grocery store mushroom. Another mushroom often causing early onset of nausea and vomiting is the jack-o'lantern mushroom (*Omphalotus species*). This mushroom grows in clusters, often on stumps or at the base of trees. They are often bright orange and glow in the dark, even after they are picked. They are found throughout North America. In Europe, ingestion of this mushroom has been reported to cause some liver dysfunction.

While we often associate *Amanita* with deadly poisonings, several species within that family are edible (*Amanita caesarea*), and a few cause early onset of GI symptoms, which are self-limited. This latter group includes *Amanita brunnescens* and *Amanita flavoruberscens*. Most toxins that cause early onset of GI symptoms are heat labile. Host response to the toxins is quite variable, so there may be significant symptoms in some and little to none in others.

Symptomatic patients present with typical GI complaints of nausea, vomiting, and diarrhea. Abdominal cramping is common. Chills, headaches, and myalgias may occur. Symptoms generally remit within 6-12 hours. Treatment is supportive and is not dependent on the species ingested. Charcoal is often unnecessary, and there is little evidence to support its use. Intravenous fluids and standard antiemetics are useful.[4]

## Case Resolution

A picture of the mushroom was sent to experts at the local poison center, who confirmed it was not an *Amanita*. The child was sent home with reassurance. A call to the parents 12 hours later and again 24 hours later confirmed he was well. Most minor ingestions of lawn mushrooms by children are benign and cause no toxicity.

***Table 1:*** Identification of Mushrooms

- Call the local poison center.
- Photograph each mushroom (if available), including the cap from above, the gills underneath the cap (particularly where they attach to the stem), and the length of the stem.
- To transport the specimen(s), place in a paper bag. Do not use plastic.
- Note the time of ingestion and the time of onset of symptoms. Note the symptoms of others who may have ingested the same mushroom.

***Figure 2:*** Mushroom Characteristics

This mushroom displays characteristics of an *Amanita* mushroom. The cap has "warts." The stem or stalk widens as it gets near the ground. The stalk ends in a cup, which often remains in the ground after picking. Halfway up the stalk is a "ring," which is the remnant of the veil or membrane that covered the mushroom as it emerged from the ground.

Image used with permission from Sandra Schneider, MD.

**Table 2:** SLUDGE Syndrome

| |
|---|
| S — Salivation, bronchorrhea |
| L — Lacrimation |
| U — Urination, contracted bladder |
| G — GI upset with nausea, vomiting, diarrhea, abdominal cramping |
| D — Diaphoresis, defecation |
| E — Emesis |

## Case 2

A teenager is found by his parents to be acting strange. He admits to having ingested "magic mushrooms," which he is growing in his closet — the gift of a friend. The parents are concerned and bring him to the emergency department.

## Central Nervous System Toxic Mushrooms

Several mushroom species cause central nervous system (CNS) effects, but the most common are *Psilocybe*. These mushrooms have been grown and used for centuries[5] and can be purchased via the Internet. *Psilocybe* mushrooms are little, brown mushrooms with a cap that is 0.5-4 cm in diameter with grayish gills. When cut, the flesh may turn blue or green. Their caps become sticky or slippery when wet.

These mushrooms contain the toxin psilocybin, which inhibits the firing rate of serotonin-dependent neurons, similar to LSD. Their toxicity is preserved with drying. Ingestion of 10 mg of fresh mushrooms will cause euphoria. In doses above that, hallucinations and heightened imagination are seen. The onset of action is rapid, generally by 30 minutes,[6] and symptoms abate by 6 hours. Patients anticipate the reaction and, therefore, rarely present to the emergency department.

Serious side effects are rare. Patients often have tachycardia and hypertension.[7] Children who have ingested these mushrooms have developed fever and seizures.[8] There is a case report of myocardial infarction related to ingestion of this mushroom.[9] Flashbacks have been reported and lasted for several months.[10]

Treatment is supportive. Patients should be placed in a room with limited stimulation. Sedation with benzodiazepines may be needed for severe agitation. Seizures can occur and are best treated with diazepam (0.1 mg/kg IV in children, or 2-5 mg IV in adults, repeated as needed every 5 minutes).

There are other species of mushrooms that contain psilocybin, including some species of *Amanita*. These are rarely ingested.

Other CNS toxins found in mushrooms may cause symptoms, among them muscarine and isoxazole. Muscarine is found in a variety of mushrooms, including *Amanita muscaria*, the bright red capped mushroom with white warts often depicted on the cover of *Alice in Wonderland* books. Ironically, this mushroom contains less muscarine than many other species. Higher concentrations are seen in *Inocybe* species. *Inocybe* mushrooms are brown mushrooms with conical caps that can be 6 cm in diameter. The stalk is long and thin and often covered with fine brown or white hairs. These mushrooms commonly grow under conifers or hardwoods in the summer. Ingestion of muscarine stimulates postganglionic cholinergic receptors similar to acetylcholine. Patients develop visual hallucinations and ataxia, but the secretory effects often predominate. Ingestion leads to the classic SLUDGE syndrome, which refers to salivation, lacrimation, urination, GI upset, diaphoresis, and emesis. (*See Table 2.*) Other effects include constricted pupils and bradycardia, although tachycardia has been reported.

Symptoms begin within 30 minutes and last less than 24 hours. Treatment is supportive. Bronchorrhea can be severe, requiring intubation and frequent suctioning. Atropine can reduce the secretions (0.01 mg/kg IV every 5-10 minutes as needed), but should be reserved for life-threatening symptoms. There is no upper limit to the amount of atropine used if secretions are significant. Atropine should be used with caution, as it often will increase the CNS effects of the muscarine.

Several mushrooms contain isoxazole derivatives that can produce CNS effects, including visual changes and excitement. *Amanita muscaria* and *Amanita pantherina* are among the most common mushrooms involved. *Amanita muscaria* is distinctive by its bright red cap, whereas *Amanita pantherina*'s cap is brown and often darker at the rim. When an *Amanita pantherina* is cut, the exposed flesh will turn pink. These mushrooms grow throughout the summer into fall.

Ibotenic acid is a derivative of isoxazole, which, when dried, forms muscimol. Muscimol increases serotonin levels in the brain, causing CNS effects.[11] Symptoms generally begin within 30 minutes and last 2 hours. Headache can persist up to 48 hours. There is a case report of psychosis lasting 5 days.[12] Patients are ataxic and appear to be intoxicated. Visual disturbances and delirium are common.[13,14] Muscle twitching and seizures can be seen. Paralysis of ocular convergence is an interesting but rare effect.[13]

Treatment is largely symptomatic. Benzodiazepines are used for control of agitation or seizures. Phenobarbital has been used (0.5 mg/kg IV children; 30 mg IV adults). In patients who have ingested *Amanita muscaria*, sedation can lead to sudden apnea or flaccid paralysis.

## Case Resolution

The patient was placed in a quiet room. Two hours later, he appeared to be entirely normal. He refused mental health/substance abuse counseling and was discharged in the care of his parents.

## Case 3

A Laotian family of three presents to the emergency department 10 hours after eating a stew that contained mushrooms picked earlier that day. The large white mushrooms looked identical to mushrooms they had picked and eaten many times in Laos. All three patients have severe nausea, vomiting, abdominal cramps, and watery diarrhea.

## Delayed GI Toxic Mushrooms

Ingestion of the mushroom *Amanita phalloides* and its two relatives *Amanita verna* and *Amanita virosa* account for 90-95% of all mushroom fatalities. These mushrooms are believed to be native to central and eastern Europe and were brought to the United States as spores in wooden furniture. *Amanita phalloides* has a large white or greenish cap with warts. The cap averages 4-16 cm in diameter. The stalk is thick, ending in a bulb-shaped structure that loosely attaches to a cup (often underground). The mushroom is quite similar to nontoxic species typically found in Laos and surrounding countries. Immigrants from these areas may consume these dangerous mushrooms by accident. Others may be picked accidentally by even experienced foragers. Some *Lepiota* species contain high concentrations of amatoxin as well. All of these mushrooms characteristically grow in the fall.

These mushrooms contain two primary toxin groups: amanitins and phalloidins. Phalloidin inhibits F-actin and disrupts membranes, leading to an efflux of calcium and potassium. In animals, phalloidin is rapidly fatal. Phalloidin does not appear to be absorbed or play a significant role in human toxicity.[15]

The exact mechanism of toxicity associated with amatoxins is unknown. These toxins are remarkably stable and are not destroyed by heating, freezing, or drying.[16] Amanitin is absorbed from the gut and transported to the liver, where it possibly undergoes some metabolism to a more toxic product.[17,18] It is known to bind to ribonucleic acid (RNA) polymerase II and inhibit formation of messenger RNA. Effects in the liver are seen within 15 hours of ingestion with swollen mitochondria and fatty degernation.[19,20]

Patients present 4-16 hours after ingestion with severe nausea, vomiting, watery diarrhea, and abdominal cramps. (*See Table 3.*) Those patients who develop symptoms between 4 and 10 hours are more likely to experience severe toxicity. The GI symptoms generally resolve within 24-36 hours, and patients appear well. However, like acetaminophen toxicity, hepatic damage develops between 24 and 48 hours after ingestion, and symptoms of hepatic failure become obvious by 72 hours. The severity of hepatic toxicity appears to be in part host-dependent and in part related to the amount of toxin ingested. Two individuals consuming

***Figure 3:*** Mushroom Gills

**The way that gills attach to the stalk can aid identification of a mushroom. This mushroom has "free gills," which means they end before the stalk begins. Other mushrooms, such as chanterelles, have gills that run part way down the stalk. Free gills are seen with *Amanita* species.**

**Image used with permission from Dr. Sandra Schneider.**

**Table 3:** Indications of Amatoxin Ingestion

| Suspect amatoxin ingestion if ... |
|---|
| • significant nausea, vomiting, diarrhea occurs 4-10 hours after ingestion of wild mushrooms; |
| • presenting patients are immigrant family members foraging for wild mushrooms; |
| • the mushrooms are harvested in the fall; |
| • resolution of symptoms occurs 12-24 hours later; |
| • elevation of AST, ALT often over 1000 IU/L; |
| • renal failure and coagulopathy occur in addition to hepatic failure |

**Table 4:** Treatment of Amatoxin Ingestion

| |
|---|
| • Monitor fluid, electrolytes, and glucose. Replace as necessary. |
| • Monitor liver transaminases and coagulation studies every 6 hours for at least 48 hours. Patients will likely need admission. |
| • Start treatment with silymarin, if available. |
| • Consider treatment with cimetidine, high-dose penicillin, or hyperbaric oxygen. |
| • Transfer to tertiary center if significant hepatic failure develops — transaminases > 1000 IU/L. |
| • Consider liver transplant if transaminases > 2000 IU/L or PT > 50 sec. |

the same meal can have marked differences in the severity of toxicity. Children often display more significant toxicity. Amatoxin is capable of massive, rapid hepatic destruction. Extremely high levels of liver transaminases are seen, although the exact level does not predict prognosis. While hepatic failure is most common, renal failure can develop with hepatic toxicity.[19]

Hypoglycemia can occur either early as part of the GI effects or later with hepatotoxicity. It likely arises from decreased hepatic gluconeogenesis, increases in insulin release, and pancreatic injury. Regardless of the mechanism, glucose should be monitored closely and supplemental glucose given as needed.

Treatment of amatoxin ingestion is largely supportive. Poison control should ideally be involved in all patients with symptoms. Consultation with a hepatologist and possibly a liver transplantation surgeon should occur early for patients who display hepatotoxicity. The course of this toxin is quite rapid, and transfer to a tertiary center should be entertained early.

Several treatments commonly used in the past have been shown to be less effective. Hemodialysis and charcoal perfusion were recommended in the past, but are not effective and may be detrimental.[20] Studies have shown that amatoxin is rapidly taken up by the liver within 5 hours after ingestion.[21] Likewise, plasmapheresis is not effective.[22] Thioctic acid and benzylpenicillin were recommended in the past, but in a large retrospective study they did not appear to have a significant benefit.[20,23]

Silymarin is found in the common milk thistle *Silybum marianum* and is sold in health food stores in the United States as an antioxidant. It is available intravenously in Europe and for some cases through the poison control system. It is thought to bind to plasma membranes inhibiting the penetration of amanitin.[24-28] In addition, it appears to be a free radical scavenger and stimulates RNA polymerase I.[29-31] In two retrospective studies, silymarin use was associated with decreased mortality.[23,32] Silymarin is given 5 mg/kg IV followed by 20 mg/kg/day for 6 days or until the liver transaminases return to normal. Oral silymarin is less well studied and is given in a dose of 1.4-4.2 g/day.

Several treatments showing success in animal models include hyperbaric oxygen[60] and cimetidine (dose 4-10 g/day IV).[17,23] The treatment of amatoxin ingestion is summarized in Table 4.

These patients can be extremely ill, and rapid changes should be anticipated. Most patients with hepatic failure will require intensive care, often at a tertiary center. Glucose levels should be monitored every 2-6 hours and replacement given as needed. Fluid and electrolytes should be monitored and replaced. Coagulopathy is frequent. Vitamin K and fresh frozen plasma are often indicated. In many cases, however, coagulopathy is severe and not corrected with treatment. Encephalopathy is an ominous sign and is treated with oral lactulose 30-45 mL every 6-8 hours. Neomycin can be used in patients who cannot tolerate oral lactulose. Standard hepatic failure treatment is followed.

Liver transplantation has been life-saving in many cases, with a survival rate of 60-80%.[23] The problem is the timing of the transplant. The natural history of amanitin toxicity is severe, progressive hepatic failure followed by death or complete (and relatively rapid) resolution of the hepatic failure. While certain factors appear to be predictive of outcome, individual patients have survived without obvious sequelae despite meeting the criteria for transplantation.[33,34] The MELD score appears to be predictive in patients with amatoxin hepatic failure.[35] Other authors recommend transplantation when the ALT or AST levels are over 2000 IU/L or the prothrombin time is greater than 50 seconds.[36] Since this is a rapidly progressive toxicity, it is recommended that patients be transferred to a transplant center before these levels are reached.

## Case Resolution

Two of the patients developed

elevations of liver transaminases without coagulopathy and were discharged home on day 7. The third member of the family developed severe hepatic failure with transaminase levels over 2000 IU/L and a coagulopathy that was uncorrectable. She underwent liver transplantation on day 5 and recovered without consequence. She remains on immunosuppressive therapy.

## Other Mushroom Toxicity

There are other mushrooms that cause significant toxicity. (*See Table 5.*) *Gyromitra* is a dark brown mushroom that has a wrinkled cap. It resembles the more edible morel mushroom. The *Gyromitra* grows primarily in the spring and is more common in Europe. It is even sold in the marketplace in parts of Europe. The toxin gyromitrin is heat labile, and the mushroom can be eaten if properly parboiled (boiling, discarding the water, boiling again, discarding the water). Similar to amatoxin, gyromitrin causes delayed symptoms.

The toxin, once ingested, is hydrolysed to N-methyl-n-formyhydrazine, which is a component of rocket fuel. It appears to inhibit pyridoxal phosphate, which interferes with many enzyme systems.[37,38] Neurotransmitter levels fall in the brain, which can lead to seizures.[39] In the liver, two reactive intermediates are created which produce local hepatic destruction.[40]

Symptoms of gyromitrin toxicity are delayed 5-10 hours after ingestion and, like amatoxin, initially are nausea, vomiting, and diarrhea. In addition, patients may have dizziness and muscle cramps. Hepatic failure is seen, although it tends to be less severe than that seen with amatoxin.

Treatment is symptomatic. Fluid and electrolytes should be monitored and replaced. Glucose may need to be replaced as well. Pyridoxine 25 mg/kg up to 20 g/day has been used to control seizures.[38,40,41] However, doses this high may cause peripheral neuropathy. While hepatic failure is generally not as severe as with amatoxin, liver transaminases and coagulation studies should be monitored several times a day. Like amatoxin hepatotoxicity, timing of liver transplantation is uncertain.

**Table 5:** Other Mushroom-related Toxicity

| Mushroom | Toxicity |
|---|---|
| *Gyromitra esculenta* | Delayed nausea, vomiting, hepatotoxicity |
| *Cortinarius orellanus* | Delayed renal failure |
| *Amanita smithiana* | Delayed renal failure |
| *Coprinus* sp. | Delayed disulfiram reaction to alcohol |

While most mushroom toxicity has been known for centuries, more recently delayed renal failure from the ingestion of *Cortinarius orellanus* and *Amanita smithiana* has been described.[42,43] *Cortinarius* is more common in Europe, while *Amanita smithiana* is found in the Pacific Northwest.

The toxins found in these mushrooms are structurally similar to paraquat and diquat. The mechanism of action is unknown. The toxins appear to be heat stabile. Individuals who ingest these mushrooms have a very long delay until symptoms occur — often as long as 20 days. Individuals consuming the same dish may develop severe, mild, or even no obvious toxicity.[44]

There is no effective treatment. Most patients present long after the ingestion with unexplained acute renal failure. The history of mushroom ingestion is often only obtained when a cluster of cases arises. Patients are treated like any patient with acute renal failure, with attention to fluid and electrolyte balance and appropriate treatment of hyperkalemia. Patients with significant renal failure and those with uncorrectable hyperkalemia will require hemodialysis. While some patients will experience the return of normal renal function within a few months, others will require dialysis or renal transplantation.

One of the more curious mushroom toxicities, and one that is likely overlooked, is caused by the mushroom *Coprinus*. This is a common lawn mushroom that grows in most parts of the United States. It has a 2-8 cm cylindrical white cap on a thin 4-5 cm stalk. As the mushroom matures, the rim turns black and liquefies, giving the mushroom its common name "inky cap." The mushroom contains a toxin, coprine, which mimics the action of disulfiram. Patients who ingest the mushroom are asymptomatic, even if they ingest alcohol at the same time as the mushroom. However, for 1-3 days after ingestion, the toxin will cause an accumulation of acetaldehyde if the patient ingests alcohol. Acetaldehyde accumulation causes flushing, headache, diaphoresis, tachycardia, and GI symptoms of nausea and vomiting. These symptoms start 15-30 minutes after ingestion of alcohol. In severe cases, chest pain, shortness of breath, and orthostatic hypotension may develop. In general, symptoms are gone within 6 hours. Treatment is entirely supportive. Fluids and electrolytes may need to be replaced. Beta-blocking agents may be used for severe tachycardia.

## Summary

While about 800 mushrooms cause toxicity, there are only a few common toxicities seen. Most children found chewing on a lawn mushroom will be asymptomatic but should be observed for toxicity. Individuals who develop the early onset of nausea, vomiting, and diarrhea within 2 hours of ingestion can be treated symptomatically and released. An exception to this is a patient who ingests several different kinds of wild mushrooms who may have ingested

***Figure 4:*** Mushroom Veil

**Some mushrooms emerge from the ground covered with a membrane or veil. As the mushroom grows, the membrane breaks, leaving a residual ring on the stalk and warts on the cap. These features are seen on *Amanita* mushrooms, as well as other species.**
**Image used with permission from Dr. Sandra Schneider.**

an *Amanita* as well as other more benign mushrooms. *Amanita phalloides* (and relatives *A. verna* and *A. virosa*) are responsible for the majority of deaths associated with toxic mushroom ingestion. These mushrooms grow primarily in the fall. Patients who ingest these mushrooms have a delayed onset of nausea, vomiting, and diarrhea 4-10 hours after ingestion. They may develop significant hepatic failure and occasionally renal failure requiring transplantation. There is no specific treatment, although silymarin has been used with some success. Experimental therapy includes cimetidine and hyperbaric oxygen.

Some mushrooms that cause hallucinations and visual distortion are consumed intentionally. These patients are rarely seen in the emergency department, as the effects are expected. When such patients do present, sedation is the recommended treatment.

Mushrooms of the *Coprinus* species cause a delayed disulfiram reaction if the patient later consumes alcohol. Patients present with flushing, tachycardia, diaphoresis, nausea, and vomiting.

Finally, mushrooms found in the Pacific Northwest and in Europe may cause the delayed onset of acute renal failure. The cause is generally considered when a cluster of renal failure victims is identified. Treatment is supportive, and some patients return to normal renal function.

In any case of mushroom ingestion, it is important to contact the local poison control center to assist in identifying the mushroom and recommending treatment. The time of ingestion to the time of GI symptoms is extremely important to determine. If hepatic failure is present and severe, transfer to a tertiary center with transplant capabilities should be considered.

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## Physician CME Questions

1. A family of 14 presents to the ED with nausea, vomiting, and diarrhea. All family members ate the same stew, which contained large white mushrooms picked from the local park. All family members began vomiting within 30 minutes after ingestion of the stew. What is the recommended treatment?
   A. hemodialysis of all 14
   B. activated charcoal
   C. fluid and electrolyte replacement
   D. immediate transfer to a tertiary center

2. A 50-year-old male presents in acute renal failure without obvious cause. When told of his condition, he relates that his friend was recently diagnosed with renal failure as well. The two of them went hiking in Oregon and Washington two weeks ago. The best explanation of his renal failure is:
   A. ingestion of *Amanita phalloides*
   B. ingestion of *Amanita smithiana*
   C. ingestion of *Coprinus* sp. followed by alcohol consumption
   D. ingestion of *Agaricus bisporus*

3. Atropine is used with caution in patients who have ingested *Amanita muscaria* because:
   A. It can cause sudden apnea or flaccid paralysis.
   B. It can cause renal failure.
   C. It can cause significant bradycardia.
   D. It can increase bronchial secretions.

4. A teenager is found by his parents to be hallucinating and acting strange. They find an open packet of mushrooms in his room. The best treatment for this patient is:
   A. activated charcoal
   B. antipsychotics
   C. a quiet, low stimulus room
   D. silymarin orally

5. A patient presents with the onset of intense nausea, vomiting, and diarrhea 10 hours after ingesting a meal containing large, white wild mushrooms picked from a local park. All labs are normal, with the exception of mild dehydration. In addition to fluid replacement, you should:
   A. Admit the patient and monitor liver transaminases and coagulation studies.
   B. Discharge the patient after the symptoms subside.
   C. Transfer the patient for hyperbaric oxygen treatments.
   D. Start treatment with thioctic acid as soon as possible.

6. A patient ingested *Gyromitra* mushrooms purchased on a

**Emergency Medicine Reports**

*CME Objectives*

*Upon completion of this educational activity, participants should be able to:*

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

recent trip to Poland. She added the mushrooms to a stew without first parboiling them. She has developed intense nausea, vomiting, and diarrhea and, upon presentation to the ED, has several grand mal seizures. Appropriate treatment includes:

A. silymarin
B. pyridoxine
C. hyperbaric oxygen
D. thioctic acid

7. In North America, *Amanita phalloides* grows primarily in the:

A. summer
B. fall
C. spring
D. year round

8. The disulfiram reaction associated with *Coprinus* sp. typically presents:

A. with alcohol consumption at the same time as the mushroom
B. with alcohol consumption 1 week after ingestion of the mushroom
C. with alcohol consumption any time 1-3 days after ingestion of the mushroom
D. with alcohol consumption 1-3 hours after ingestion of the mushroom

9. Four patients present in rapid succession, all with diaphoresis, nausea, vomiting, and significant bronchorrhea after eating a mushroom stew. The possible mushroom involved is:

A. *Amanita phalloides*
B. shiitake mushrooms
C. *Gyromitra esculenta*
D. *Amanita muscaria*

10. A parent brings in the mushroom that her child picked from the ground and was found eating. The mushroom is large and white. The poison center wants photos sent. Which parts of the mushroom are particularly important to document?

A. the cap: color and warts
B. the stalk: ring and end
C. the gills, especially as they enter the stalk
D. all of the above

## In Future Issues

Cervical Spine Fractures

## CME Instructions

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:

1. Read and study the activity, using the provided references for further research.
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Emergency Medicine Reports
The Practical Journal for Emergency Physicians

# Mushroom Toxicity

## Identification of Mushrooms

- Call the local poison center.
- Photograph each mushroom (if available), including the cap from above, the gills underneath the cap (particularly where they attach to the stem), and the length of the stem.
- To transport the specimen(s), place in a paper bag. Do not use plastic.
- Note the time of ingestion and the time of onset of symptoms. Note the symptoms of others who may have ingested the same mushroom.

## SLUDGE Syndrome

S — Salivation, bronchorrhea
L — Lacrimation
U — Urination, contracted bladder
G — GI upset with nausea, vomiting, diarrhea, abdominal cramping
D — Diaphoresis, defecation
E — Emesis

## Indications of Amatoxin Ingestion

Suspect amatoxin ingestion if ...

- significant nausea, vomiting, diarrhea occurs 4-10 hours after ingestion of wild mushrooms;
- presenting patients are immigrant family members foraging for wild mushrooms;
- the mushrooms are harvested in the fall;
- resolution of symptoms occurs 12-24 hours later;
- elevation of AST, ALT often over 1000 IU/L;
- renal failure and coagulopathy occur in addition to hepatic failure

## Treatment of Amatoxin Ingestion

- Monitor fluid, electrolytes, and glucose. Replace as necessary.
- Monitor liver transaminases and coagulation studies every 6 hours for at least 48 hours. Patients will likely need admission.
- Start treatment with silymarin, if available.
- Consider treatment with cimetidine, high-dose penicillin, or hyperbaric oxygen.
- Transfer to tertiary center if significant hepatic failure develops — transaminases > 1000 IU/L.
- Consider liver transplant if transaminases > 2000 IU/L or PT > 50 sec.

## Mushroom Cap with Warts

The cap of a mushroom species photographed from above showing the characteristic “warts.” These are adhered remnants of the veil or membrane that covered the mushroom when it emerged from the ground. At times these warts are washed off and white spots are present on the cap.
Image used with permission from Sandra Schneider, MD.

## Mushroom Characteristics

This mushroom displays characteristics of an *Amanita* mushroom. The cap has "warts." The stem or stalk widens as it gets near the ground. The stalk ends in a cup, which often remains in the ground after picking. Halfway up the stalk is a "ring," which is the remnant of the veil or membrane that covered the mushroom as it emerged from the ground.

Image used with permission from Sandra Schneider, MD.

## Mushroom Gills

The way that gills attach to the stalk can aid identification of a mushroom. This mushroom has "free gills," which means they end before the stalk begins. Other mushrooms, such as chanterelles, have gills that run part way down the stalk. Free gills are seen with *Amanita* species.

Image used with permission from Dr. Sandra Schneider.

## Other Mushroom-related Toxicity

| Mushroom | Toxicity |
|---|---|
| *Gyromitra esculenta* | Delayed nausea, vomiting, hepatotoxicity |
| *Cortinarius orellanus* | Delayed renal failure |
| *Amanita smithiana* | Delayed renal failure |
| *Coprinus* sp. | Delayed disulfiram reaction to alcohol |

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