Abstract—Mushrooms are ubiquitous in nature. They are an important source of nutrition; however, certain varieties contain chemicals that can be highly toxic to humans. Industrially cultivated mushrooms are historically very safe, but foraging for mushrooms or accidental ingestion of mushrooms in the environment can result in serious illness and death. The emergency department is the most common site of presentation for patients suffering from acute mushroom poisoning. Although recognition can be facilitated by identification of a characteristic toxidrome, the presenting manifestations can be variable and have considerable overlap with more common and generally benign clinical syndromes. The goal of this two-part article is to review the knowledge base on this subject and provide information that will assist the clinician in the early consideration, diagnosis and treatment of mushroom poisoning. Part I, presented in this issue of the Journal, reviews the epidemiology and demographics of mushroom poisoning, the physical characteristics of the most toxic varieties, the classification of the toxic species, and an overview of the cyclopeptide-containing mushroom class. Part II, to be published in the next issue of the Journal, will be focused on the presentation of the other classes of toxic mushrooms along with an up-to-date review of the most recently identified poisonous varieties. © 2005 Elsevier Inc.

Keywords—mushroom; amanita; orellanine; indoles; monomethylhydrazine; cyclopeptide; isoxazoles; mycotoxins

INTRODUCTION

Mushrooms are ubiquitous. They can be found in our yards, in the woods, along the roadside, and prominently displayed on our grocery store shelves. Historically, store mushrooms were limited to the white button type, agaricus bispora; however, today, most markets may have more than six types of mushrooms including portabellas, shiitakes, morels, chanterelles, oysters, and others (1). In 1988, five million pounds of specialty mushrooms and 632 million pounds of agaricus mushrooms were produced (2). Beyond commercial production, the practice of mushroom hunting has long been common in Europe, and has become increasingly popular over the past decade in the United States (3,4).

Mushrooms are often thought of as plants, but they do not contain chlorophyll and are members of kingdom Fungi, which is in the domain Eukaryote. The word fungus is most likely derived from the Greek word sphongis, meaning sponge (5). However, as suggested by Lehmann and Khazan, the origin may be more appropriately derived from the Latin “fungus ago,” meaning “I make a corpse” (5). The term toadstool comes from the German word todesstuhl meaning death’s stool, and has been used inconsistently when referring to poisonous mushrooms. There are about 5000 mushroom species, of which approximately 50–100 are known to be poisonous to humans, whereas only 200 to 300 varieties have been clearly established to be safely edible (6–9).

Mushrooms are parasitic, surviving off of other living matter, saprophytic, requiring a decaying host, or mycorrhizal, living symbiotically with other plant matter. Although they can grow prolifically, strict environmental
and nutrient requirements limit growth and reproduction. Most mushrooms are mycorrhizal, and classified as either lignophilic, i.e., wood loving, or coprophilic, i.e., dung loving, requiring excreta or decaying organic matter to survive (10–12).

Mushrooms are high in protein and essential amino acids and tend to be fat free, cholesterol free, and low in calories (2). There are some data to suggest that mushrooms may improve the immune system, inhibit clotting, and lower cholesterol (13). Shiitake and Maitake mushrooms have been shown to reduce blood pressure in hypertensive rats as well as decrease cholesterol levels (14). Agaricus are reported to activate T lymphocytes and augment immune function in tumor-bearing mice (15).

Mycoxins refer to fungal compounds that poison other organisms (16). Euripides is credited with the first report of mushroom poisoning in 430 B.C., when he wrote of the tragic fatal poisoning of his wife and three children following mushroom ingestion (17). Serious mushroom poisoning is an uncommon occurrence in the United States. In 2002, the American Association of Poison Control Centers (AAPCC) reported 8722 cases of mushroom-related toxicity, which represented 0.6% of the total number of non-pharmaceutical toxic exposures in the preceding year (18). The actual number of mushroom poisoning events is probably substantially higher because of both underreporting and the failure to associate the protean clinical manifestations of mycoxins with mushroom ingestion.

The majority of toxic ingestions involve children under 6 years of age (18). In 2002, AAPCC reported the majority of mushroom exposures were in children under 6 years of age and almost 90% were in individuals under 19 years of age (18). Typically, a child will take one or two bites from a little brown mushroom (LBM) out of curiosity and experience only limited toxicity. Adult mushroom hunters who seek mushrooms for nourishment or culinary diversity may ingest one or more varieties of whole mushrooms. These hunters may be misguided by mushroom myths; for example, poisonous mushrooms will cause a silver coin placed in the utensil in which the mushrooms are cooked to tarnish, mushrooms that peel easily are edible, the presence or absence of insects on the mushroom may guide toxicity, or soaking mushrooms in salt or boiling water will render poisonous mushrooms innocuous (19). If the variety is misidentified, serious or even fatal toxicity may occur. Other high-risk groups are immigrants who may be accustomed to foraging for mushrooms in their country of origin. These individuals are prone to mistake a poisonous “look alike” with an edible variety from their homeland (20,21). Ingestions of hallucinogenic mushrooms are common on college campuses and can result in toxic responses from the hallucinogenic chemicals or from mycoxins present in misidentified mushrooms (22,23).

### GENERAL GUIDELINES IN DIAGNOSIS AND MANAGEMENT

History is the cornerstone of diagnosis. The first task is to link the clinical presentation with mushroom ingestion. In the most serious ingestions the association with mushrooms may be obscured due to the delay between symptom onset and the mushroom meal. Furthermore, not all patients will experience similar toxicity from a particular mushroom variety (24). The physician must consider the possibility of mushroom toxicity and query the patient directly. It is important to ascertain what type of mushroom the patient thought was eaten as well as a description of the mushroom, the environment from which it was harvested, and the preparation before ingestion.

When interviewing patients suspected of suffering from mushroom poisoning, a detailed history concerning the ingestion should be obtained. Key questions include the number of different types of mushrooms ingested. Storage before consumption should be questioned, as some poisoning may be due to contamination with bacteria or preformed toxins. How were mushrooms prepared, sautéed, boiled, or other? If boiled, was water ingested? Some toxins are destroyed by heat, whereas others are water soluble. The time frame between the mushroom ingestion and onset of symptoms can provide essential clues, as the most deadly toxins are generally associated with a 6-h or longer delay between consumption and symptom onset. Was alcohol consumed? Disulfiram-like mushrooms cause alcohol sensitivity. Is everyone who ate the mushrooms ill? If only one person is ill, consider food sensitivity or alternative diagnosis. Is there anyone ill who did not eat the mushrooms? Consider infectious causes. What type of mushroom did you think you were eating? Often the description of the mushroom, its color, the presence of specific features or a hand-drawn diagram will aid in species identification. Are there any similar mushrooms or remnants remaining for identification? One should also obtain a detailed past medical history, especially in regards to liver disease and chronic alcohol use (24,25).

Figure 1 is a diagram of a mushroom that demonstrates characteristic features that are commonly, but not exclusively, present in toxic varieties. If whole mushrooms or mushroom fragments are available, they should be saved for possible identification. Mushroom material should be wrapped in wax paper and stored in a paper bag in a refrigerator. Spore prints can be a useful adjunct to species identification and can be made by placing the
mushroom cap on a white piece of paper and covering it to prevent drafts. If a cap is not available, it also may be possible to retrieve spores from gastric contents. Spores can be obtained by centrifuging gastric aspirate that has been filtered through cheesecloth (7). Proper mushroom identification, spore print analysis or spore identification under light microscopy will require consultation with an experienced mycologist.

In lieu of mushroom identification, recognition of a characteristic mushroom toxidrome can expedite diagnosis and therapeutic decision-making. Clinically, mushroom poisoning is divided into several toxidromes based on the principle toxins present in the variety ingested. The principle toxin groups are: cyclopeptide, orellanine, monomethylhydrazine, disulfiram-like, hallucinogenic indoles, muscarine, isoxazole, GI-specific irritants (Table 1). Although almost all toxic varieties fall into one of these eight categories, there can be overlap. In addition, newly recognized toxic mushroom components with unique clinical features require creation of a ninth category that, for lack of a better term will be simply called miscellaneous. Group designation is based on the toxins present in greatest quantity; however, some mushrooms contain more than one harmful chemical. The name of the mushroom may be misleading. A prominent example is Amanita muscaria, named after the toxin muscarine, which is present in only trivial quantities, but categorized in the toxic group isoxazole, because it contains large

<table>
<thead>
<tr>
<th>Toxic Group</th>
<th>Mushroom</th>
</tr>
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<tbody>
<tr>
<td>Cyclopeptides</td>
<td>A bisporigera, suballicia, tenuifolia; A brunescens; A ocreata; A Phalloides; A Verna; A Virosa; Conocybeilaris; Galerina autumnalis; Galerina marginata; Galerina venenata; Lepiota helveola</td>
</tr>
<tr>
<td>Orellanine</td>
<td>A smithiana; C splendidens; Cortinarius (orellanus, rainieriensis, limonis, rubellus); Cortinarius speciosissimus</td>
</tr>
<tr>
<td>Monomethylhydrazine</td>
<td>G brunnea, G caroliniana, G fastigiata, G gigas, G infolia, Gyromitra esculenta, Helvella lacunose, Helvella elastica, Paxina species, Sarcosphaera coronaria</td>
</tr>
<tr>
<td>Disulfiram-like reaction</td>
<td>Clitocybe claviceps; Coprinus atramentarius</td>
</tr>
<tr>
<td>Muscarine</td>
<td>Boletus spp; Clitocybe (cerussata, illudens, rivulosa); Clitocybe dealbata; Inocybe (fastigiata, lilacina, patouillardii, pudica, rimosus); Inocybe geophylla</td>
</tr>
<tr>
<td>Hallucinogenic indoles</td>
<td>Conocybe cyanopus; Gymnopilus (aeruginosus, spectabilis, validipes); Panaeolus (foenisecii, subbaltatus); Psilocybe (baeocystis, caerulescens, A19cyanescens, mexicana, pelliculosa, semilanceata, silvatica); Psilocybe cubensis; Stropharia species; Strotopharin</td>
</tr>
<tr>
<td>Isoxazoles</td>
<td>A muscaria; A pantherina; Amanita (cokeri, solitaria, strobiliformis, cothurnata, gemmata, citrina); Panaeolus campanulatus</td>
</tr>
<tr>
<td>GI irritants</td>
<td>Boletus (miniato-olivaceus, satanas, eastwoodii); Chlorophyllum molybdites; Lactarius (torminosus, glaucescens); Russula emetica; Tricholoma (pardinum, venenatum)</td>
</tr>
</tbody>
</table>

References: (11,117,196,197).
has decreased in the last decade secondary to more effective supportive measures. The fatality rate in most reports is higher in children under 10 years old (6,29,30). Curiously, co-ingestion of alcohol with A. phalloides may improve outcome (31). In North America, Amanita virosa, which is found in the Northeast and Pacific Northwest, may be a more common cause of cyclopeptide poisonings (27,32,33). Cyclopeptides are also found in lower concentrations in other, but not all, Amanita species, as well as in some Galerina, Cortinarius, and Lepiota species, see Table 1 (34). The quantity of cyclopeptide toxin in 15–20 Galerina caps is equivalent to that in one A. phalloides cap. A phalloides are most abundant in the late summer and autumn (35). In North America, these mushrooms are generally found in the cool coastal regions on the west coast, the northeast, and the mid-Atlantic coast (3,32). Amanitas tend to grow in moist forests in association with deciduous trees such as oak and chestnut; however, they also have been found in urban lawns (20,36,37). Amanita can be identified by their green cap, white gills, white spore print, the presence of an annulus (“ring of death”) and the volva (“death cup”), see Figure 1. Cap color is not an absolute, as it may vary from an off-white to a green, depending on weather, soil, and age.

Cyclopeptides consist of two principal hepatotoxic peptides: amatoxins and phallotoxins. In animal studies, phallotoxin, a cyclic heptapeptide, is not absorbed from the intestine and therefore is not felt to play a significant role in human toxicity (38). There have been nine amatoxins identified, as well as in some Galerina, Cortinarius, and Lepiota species, see Table 1 (34). The quantity of cyclopeptide toxin in 15–20 Galerina caps is equivalent to that in one A. phalloides cap. A phalloides are most abundant in the late summer and autumn (35). In North America, these mushrooms are generally found in the cool coastal regions on the west coast, the northeast, and the mid-Atlantic coast (3,32). Amanitas tend to grow in moist forests in association with deciduous trees such as oak and chestnut; however, they also have been found in urban lawns (20,36,37). Amanita can be identified by their green cap, white gills, white spore print, the presence of an annulus (“ring of death”) and the volva (“death cup”), see Figure 1. Cap color is not an absolute, as it may vary from an off-white to a green, depending on weather, soil, and age.

Cyclopeptides consist of two principal hepatotoxic peptides: amatoxins and phallotoxins. In animal studies, phallotoxin, a cyclic heptapeptide, is not absorbed from the intestine and therefore is not felt to play a significant role in human toxicity (38). There have been nine amatoxins identified, but α-amanitine (amanitin) appears the most physiologically active. Amanitin is a thermostable bicyclic octapeptide that inhibits RNA polymerase II and therefore DNA transcription, resulting in arrest of protein synthesis and cell necrosis (27,39–41). Amanitin is transported by a non-specific transport system into hepatocytes, and is associated with a pattern of centrilobular necrosis (4,42–44). Amanitin is also filtered by the glomerulus and reabsorbed by the renal tubules, resulting in acute tubular necrosis (45). Metabolically active tissues dependent on high rates of protein synthesis such as the cells of the gastrointestinal tract, hepatocytes, and the proximal convoluted tubules of kidney are disproportionately affected (46,47). In animal and human post-mortem studies, cellular damage also has been found in the pancreas, adrenal glands, and testes (48,49). Amatoxins are not significantly protein bound and are cleared from plasma within 48 h of ingestion (50,51). Amanitins are predominately excreted in the urine, with a smaller quantity found in the bile, where significant enterohepatic circulation has been identified (27,34,52–54). A study in a dog model found that 85% of amanitin can be recovered in the urine within 6 h of ingestion (55).

Amanitin can be measured in the blood or urine by high performance liquid chromatography, high-performance thin-layer chromatography (HPLC), or radioimmunoassay (38,56–58). Unfortunately, these assays are not generally available to clinicians and are used most often in research settings. The Meixner Test is a screening assay for cyclopeptide poisoning that the clinician can perform (59,60). A drop of fresh mushroom pulp, methanol extract from dried mushrooms, or gastric material is placed on a high lignin-containing paper (e.g., newspaper) and allowed to dry. A drop of 10-N or 12-N hydrochloric acid is then applied to catalyze the reaction of the amatoxin with the lignin in the paper (61). The area will turn blue within 1 to 2 min if amatoxins are present. This reaction may be delayed if the amatoxin concentration is low, so a reading at 30 min should be made. A control should be placed on a blank piece of paper because on occasion the paper itself may cause a false positive. The utility of this test is limited by false positive results. This may result from the presence of psilocybin, exposure to sunlight, excessive heat, or several other mushrooms varieties (61–63). One investigator found 19% of non-Amanita gilled mushrooms tested gave a positive newspaper test, whereas another demonstrated false negative results (61,63).

The cyclopeptide toxidrome has four stages (Table 2). The first or latent stage lasts approximately 6 to 24 h post-ingestion and is characterized by the absence of any signs or symptoms (34). Stage two is heralded by the acute onset of intense cramping abdominal pain, nausea, vomiting, and severe secretory diarrhea (53). Both diarrhea and emesis may become grossly bloody. This gastroenteritic phase may be severe enough to result in acid-base disturbances, electrolyte abnormalities, hypoglycemia, dehydration, and hypotension. Physical findings at this stage may include epigastric tenderness and hepatomegaly (64,65). Liver function tests are usually normal at this point of the illness. If the association with toxic mushrooms is not made, these patients may be erroneously diagnosed with gastroenteritis and discharged home. This second stage lasts 12 to 24 h and is followed by a third, convalescent phase lasting 12–24 h. In the third stage, the patient feels and looks better, possibly leading the clinician and patient into a sense of false security. Despite clinical improvement, liver enzymes AST, ALT, and bilirubin begin to rise during this period (66). Renal function may also begin to deteriorate during this stage. The fourth and final stage begins 2 to 4 days post-ingestion. During this time, which spans 4 to 7 days, the transaminases rise dramatically and liver and renal function deteriorate, resulting in hyperbilirubinemia, coagulopathy, hypoglycemia, acidosis, hepatic encephalopathy, and hepatorenal syndrome (67). Multiorgan failure, disseminated intravascular coagulation
DIC), mesenteric thrombosis, convulsions, and death may result within 6 to 16 days post-ingestion (65). Mortality rates in adults are approximately 10%–30%. About 20% will go on to develop a picture of chronic active hepatitis, and the remainder will have clinical resolution within a week or so (4). Liver tests may not return to normal for several weeks to months (34,68).

Treatment for cyclopeptide poisoning is supportive. Due to the severity and potential lethality of illness, all patients should be admitted to the hospital and placed in an intensive care setting. Therapy should focus on replacing electrolytes, fluids, administering glucose, and correcting coagulopathy (7,32,69). Patients most commonly present late during stage II of the toxidrome, thereby obviating the utility of gastric lavage. In scenarios of early presentation, gastric emptying may improve outcome (53,70). Liver tests may not return to normal for several weeks to months (34,68).

Treatment for cyclopeptide poisoning is supportive. Due to the severity and potential lethality of illness, all patients should be admitted to the hospital and placed in an intensive care setting. Therapy should focus on replacing electrolytes, fluids, administering glucose, and correcting coagulopathy (7,32,69). Patients most commonly present late during stage II of the toxidrome, thereby obviating the utility of gastric lavage. In scenarios of early presentation, gastric emptying may improve outcome (53,70). Liver tests may not return to normal for several weeks to months (34,68).

The lethality of cyclopeptide poisoning coupled with the lack of effective therapy has promulgated an extraordinary gamut of unsubstantiated treatments; see Table 3 (75).

The most often cited treatments are benzyl penicillin G (300,000–1,000,000 units/kg/day i.v.) and silibinin (26,29,32,69). Benzyl penicillin was shown to be effective when administered to dogs 5 and 24 h after ingestion of A. phalloides (76). Although the mechanism by which penicillin protects hepatocytes is still unknown, it is hypothe-

### Table 2. Toxidrome Stages

<table>
<thead>
<tr>
<th>Mushrooms</th>
<th>Onset</th>
<th>Clinical presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyclopeptides</td>
<td>Stage 1—Latent: 0–24 h</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td></td>
<td>Stage 2—Gastroenteritis: 6–24 h</td>
<td>N/V/Profuse diarrhea (cholera-like), abdominal pain, bloody diarrhea, hematuria</td>
</tr>
<tr>
<td></td>
<td>Stage 3—Apparent convalescence: 24–72 h</td>
<td>Asymptomatic, hepatic enzymes rising</td>
</tr>
<tr>
<td></td>
<td>Stage 4—Hepatic: 4–9 days</td>
<td>Hepatic and renal failure, cardiomyopathy, encephalopathy, convulsions, coma, death</td>
</tr>
<tr>
<td>Monomethylhydrazine (MMH)</td>
<td>6–24 h</td>
<td>N/V/D, abdominal cramps, delirium, seizures, coma, methemoglobinemia</td>
</tr>
<tr>
<td>Disulfiram-like reaction</td>
<td>&lt;30’ after ingestion EtOH, or up to 72 h post-mushroom-ingestion</td>
<td>Disulfiram reaction: HA, N/V, flushing of face and trunk, hypotension, tachycardia, chest pain, apprehension</td>
</tr>
<tr>
<td>Muscarine</td>
<td>0.5–2 h</td>
<td>Cholinergic syndrome: lacrimation, bradycardia, miosis, diaphoresis, salivation, bronchorrhea, bronchospasm</td>
</tr>
<tr>
<td>Muscimol</td>
<td>0.5–2 h</td>
<td>Lethargy, stupor, alternating mania, delirium, hallucinations</td>
</tr>
<tr>
<td>Hallucinogenic Indoles</td>
<td>0.5–3 h</td>
<td>Hallucinations, unmotivated laughter, Euphoria, agitation, compulsive behavior, dysphoria, fever/seizures in children</td>
</tr>
<tr>
<td>Orellanine</td>
<td>0.5–2 h</td>
<td>N/V/D, anorexia, abdominal cramps, ARF, thirst, decreased UOP</td>
</tr>
<tr>
<td>GI-specific irritants</td>
<td>0.5–2 h</td>
<td>N/V/D, abdominal cramps</td>
</tr>
</tbody>
</table>


N = nausea; V = vomiting; D = diarrhea; HA = headache; N/V, ARF = acute renal failure; UOP = urine output.
sized that penicillin inhibits hepatocyte uptake of amatoxins. Kröncke et al. demonstrated that penicillin does not inhibit hepatocyte uptake of amanitin and proposed that penicillin exerts its effect via a yet-to-be elucidated intracellular mechanism (43). Silibinin is a water-soluble preparation of silymarin; a flavolignone extracted from the milk thistle plant *Silybum marianum* (77). Silymarin has been shown in animal studies to reduce hepatocyte uptake of amatoxin (78). In a study of 205 patients with *Amanita* poisoning there were 46 fatalities, however, there were no deaths among the 16 patients who received silibinin (29). Silibinin is available in Europe and used in doses of 20 to 50 mg/kg/day (29). If used, administration should be as soon after ingestion as feasible (29). Although penicillin and silibinin treatments appear to be well tolerated and are supported by many case series, there are no prospective controlled trials utilizing these agents (77,79).

Hemoperfusion may have a role in preventing cerebral edema in the setting of fulminant hepatic failure, however, it has not been successful in the treatment of cyclopeptide poisoning (80,81). Although amatoxins are not highly protein bound, plasma exchange trials, including charcoal hemoperfusion, hemodialysis, and exchange transfusions have not been shown to be effective (82–84). Lack of efficacy may be related to rapid tissue uptake and low plasma concentrations of amatoxin (53). Thiocyst acid (α-lipoic acid) is a coenzyme for alphaketohydrogenase that is believed to be a free radical scavenger (85). Case reports have suggested some benefit, however, controlled trials are lacking (55,86). Controlled animal studies of thiocyst acid have failed to demonstrate efficacy (79). The drug may cause hypoglycemia and in a single human trial was associated with a higher mortality rate (29,87). Amatoxin-specific monoclonal antibodies had great theoretic promise; unfortunately, their use has been associated with increased amanitin toxicity (88). Temporary porcine liver hemoperfusion has been tried without success (4). Among some of the other proposed remedies are kultukin, an extract from the Indian herbal root *Picrothiza kurroa*, cimetidine, forced diuresis, furosemide, corticosteroids, mannitol, ethanol, ceftazidime, N-acetylcysteine, Cytochrome C, vitamin C, bowel sterilization, and hyperbaric oxygen (26,29,34,45,52,66,89–95). Beyond isolated case reports, none of these therapies has been clearly demonstrated to be effective. There are case reports of good outcomes with empiric treatment with Bastien’s regimen, which includes ingestion of a concoction of organic substances, see Table 3 (96–98). In 1981, Dr. Bastien ate 70 gm of *A. phalloides*, what should have been a fatal dose, and recovered after self-treatment with his regimen (95). Other cyclopeptide-containing mushrooms are listed in Table 1.

### Table 3. Proposed (Unproven) Treatments for Cyclopeptides

<table>
<thead>
<tr>
<th>Treatment</th>
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<tbody>
<tr>
<td>Activated charcoal</td>
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<tr>
<td>Amantadine</td>
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<tr>
<td>Bastien’s regimen: carrot broth as only nutrition, nifuroxazide (1200 mg/day), dihydrostreptomycin, Vitamin C (3 gm/day i.v.) for 3 days</td>
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<tr>
<td>Benzylpenicillin</td>
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<td>Bile salts</td>
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<tr>
<td>Chloramphenicol</td>
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<tr>
<td>Cimetidine</td>
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<tr>
<td>Cytochrome C</td>
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<tr>
<td>D-penicillamine</td>
</tr>
<tr>
<td>Eating burnt domestic fowl dung (8)</td>
</tr>
<tr>
<td>Furosemide</td>
</tr>
<tr>
<td>Hyperbaric oxygen therapy</td>
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<tr>
<td>Liver transplant</td>
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<td>Monoclonal antibody</td>
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<tr>
<td>N-acetylcysteine</td>
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<tr>
<td>Organic therapy (rabbit meat, rabbit brains, rabbit stomach) (3)</td>
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<tr>
<td>Peritoneal dialysis</td>
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<tr>
<td>Phenylbutazone</td>
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<td>Plasmapheresis</td>
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<td>Silibinin</td>
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<tr>
<td>Sulfamethoxazole</td>
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<tr>
<td>Steroids</td>
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<tr>
<td>Thioctic acid</td>
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<tr>
<td>Vitamin C</td>
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<td>Zinc</td>
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</table>

References: (29,55,79–98).

### SUMMARY

Prevention and education are the quintessence of care. Remembering the toxidromes is more important than recalling specifics about individual mushrooms. There are thousands of species of mushrooms and, although identifying the exact mushroom might be helpful, it is not necessary for effective management. Non-lethal mushrooms generally cause symptoms within 6 h. The caveat is that patients often ingest more than one type of mushroom, so that symptom onset within 6 h does not rule out a co-ingestion of a potentially lethal variety. Cyclopeptide-containing mushrooms are the most toxic varieties encountered and are responsible for the majority of recognized and reported fatalities, and therefore the most important to recognize at the earliest stages of presentation.

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