BC Centre for Disease Control
An agency of the Provincial Health Services Authority

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Consider wild mushroom ingestion in differential diagnosis of gastroenteritis

Failure to consider wild mushroom ingestion may lead to missed diagnosis with potentially severe patient consequences.

The *Amanita phalloides* (deathcap) mushroom has been found in urban locations in Victoria and Vancouver. It has also been reported on the Gulf Islands, and in agricultural and suburban areas of the lower mainland. It is becoming more common thus increasing the risk of exposure. *A. phalloides* may be mistaken for edible species by mushroom pickers lacking experience or familiarity with local species, or may be unintentionally ingested by children.

A. phalloides causes life-threatening liver toxicity, with onset of gastrointestinal symptoms that is often delayed 6 hours or longer. Symptoms may temporarily improve but liver damage may be ongoing, leading to return of gastrointestinal symptoms and eventually hepatorenal failure. During the acute gastrointestinal phase, if association with mushroom ingestion is not made, the patient may be erroneously diagnosed with gastroenteritis and discharged.

- More information:
  - BC Inter-Ministry Invasive Species Working Group death cap mushroom fact sheet (attached)
  - BC Drug and Poison Information Centre Poison management manual: mushrooms – cyclopeptide (attached)

Other *Amanita* species found in BC can cause gastrointestinal symptoms with renal and neurological toxicity (see Table). Other types of wild mushrooms may also cause gastrointestinal symptoms but are not usually life-threatening. Mushroom poisoning may be categorized by clinical syndrome. Obtain a careful history including: when mushrooms were ingested, how many meals, timing of symptoms, how many kinds of mushrooms were eaten, were mushrooms cooked or raw, who ate the mushrooms, and is anyone else ill.

- More information:
  - BC Drug and Poison Information Centre Poison management manual: mushroom poisoning - general (attached)

Contact the Poison Control Centre (1-800-567-8911) if you suspect mushroom poisoning.

Mushroom identification

Mushroom resources with good colour photos may be helpful in identifying some mushrooms with distinctive characteristics, but accurate identification requires an expert mycologist.

Useful data for identifying mushrooms:
Location and habitat: Residential, agricultural, park or forest; street address or location; growing near or under what type of tree, or on open lawn or field; type of substrate (grass, rotting log, fallen leaves, compost or soil, etc.).
Photographs: Cap (top and gills), stem, and base including the bulb or root if any; photo of the habitat -- if growing under trees, photos of the leaves may be useful if tree species is unknown.
Specimen: Wrap in foil or wax paper and place in paper bag or in rigid container. Do not place in plastic bag.
Contact the Poison Control Centre for names of local mycologists who can assist in identifying mushrooms.

Table: *Amanita* mushroom poisoning from BC species

Species	Toxicity	Look-alikes	Notes
Amanita phalloides (deathcap)	Liver toxicity	Young "button" stage Amanitas can be confused with puffballs; more mature *A. phalloides* can be confused with Asian straw mushrooms	Toxins: cyclopeptide toxins. Characterized by 3 phases: gastrointestinal toxicity 6-24 hours post-ingestion, followed by an asymptomatic false recovery, then delayed hepatorenal failure. Death may occur 6-16 days post ingestion from hepatic coma and renal failure. Aggressively maintain fluid, electrolyte, and acid-base status, and treat coagulopathy and encephalopathy. There are no proven antidotes but N-acetylcysteine, penicillin G and silibinin have been used. Early consultation with a transplant centre is advised for seriously ill patients.
Amanita smithiana	Renal failure	Resembles the edible "pine mushroom" (*Tricholoma magnivelare)*	Toxins: not confirmed. Gastrointestinal symptoms are usually the first symptoms (usually 4-11 hours post-ingestion; may occur sooner if eaten raw). Patients may not present for days until the onset of renal failure. Renal replacement is often required until adequate renal function returns (weeks to months).
Amanita muscaria, A. pantherina, A. gemmata	Neurological toxicity		Toxins: Ibotenic acid (excitatory) and muscimol (inhibitory). Onset of symptoms begins 15 minutes to 2 hours post-ingestion with possible CNS excitation, then drowsiness. Deep coma-like sleep may ensue. Treatment is symptomatic and supportive until effects resolve (24 hours).

MUSHROOM POISONING - GENERAL

Description

Mushroom poisonings can be grouped into categories according to type of symptoms and time to symptom onset. Refer to the following table. For further information, **see** specific mushroom poisoning monograph, e.g. MUSHROOMS-GASTROINTESTINAL IRRITANTS.

General Treatment of Mushroom Poisoning

1. **Topical**: Contact dermatitis has occurred, usually in workers and mycologists.

2. **Ocular**: Mushroom spores can cause mechanical injury. Irrigate eyes with a gentle stream of tepid water for 15 minutes. Obtain ophthalmologic opinion if irritation persists. Chronic exposure to volatile hydrazines from the *Gyromitra* species during drying of mushrooms may cause eye irritation.

3. **Inhalation**: Inhalation of vapours during cooking of the *Gyromitra* species can cause CNS and gastrointestinal toxicity. *See* MUSHROOMS-MONOMETHYLHYDRAZINE.

4. Acute inhalation of mushroom spores can produce effects resembling bronchial pneumonia. Corticosteroids may be beneficial. Hypersensitivity pneumonitis has developed in commercial mushroom workers.

5. **Ingestion**: Administer activated charcoal in recent ingestions. Save vomitus for possible mushroom identification in suspected serious cases. **See** Storage/Identification of Mushrooms for procedure for storing fresh specimens or stomach contents for identification.

6. Obtain careful history regarding ingestion (time of ingestion, amount ingested, cooked or raw, how many types of mushrooms ingested, if anyone else has ingested mushrooms and is symptomatic, whether alcohol or drugs have been ingested, and whether mushrooms were consumed at more than one consecutive meal).

7. Determine time (post ingestion) of onset of initial symptoms, nature of symptoms and order of appearance.

8. **Refer to the following table to determine possible mushroom group, then refer to the specific mushroom poisoning monograph for that group** (e.g. MUSHROOMS-COPRINE).

9. A delay in symptom onset may indicate that a highly toxic mushroom species may be involved. If symptom onset is 4 or more hours post ingestion, every effort should be made to have mushrooms identified. However, a few serious mushroom poisonings can result in symptoms < 4 hours post exposure. **See** Storage/Identification of Mushrooms.

10. Treat patient symptomatically and provide supportive care. Ensure good urine output.

11. Monitor vital signs, CBC, blood gases, electrolytes, renal and hepatic function.

12. Do not use antidiarrheal or antispasmodic drugs to control gastrointestinal symptoms. These may delay removal of toxins.

13. Dialysis may be required for treatment of renal failure.

Storage/Identification of Mushrooms

1. Obtain one or more fresh specimens of suspect mushroom. Wrap in foil or wax paper and place in paper bag or in rigid container. Do not place in plastic bag. Store in refrigerator. Do not freeze.

2. Contact Poison Control Centre for name of local mycologist or botanist who can assist in identifying mushrooms.

3. Contact identifier by telephone and arrange for rapid transportation of specimen. Provide identifier with information regarding habitat if available (e.g. growing near what type of tree, growing in open fields, etc.). Instruct conveyor to keep package cool.

4. Mushroom textbooks with good colour plates may be helpful in identifying some mushrooms with distinctive characteristics, but accurate identification requires an expert mycologist or botanist.

TABLE Common Types of Mushroom Poisoning

Time of Symptom Onset	Symptoms	Mushroom Group
5 to 10 minutes after drinking ethanol, if mushroom was ingested in preceding 3-5 days	Flushing of face and neck, metallic taste, paresthesia of hands and feet, tachycardia, chest pain, hypotension. Nausea and vomiting may occur.	**See** MUSHROOMS - COPRINE
10 to 30 minutes	Euphoria, drowsiness, mood changes, laughter, anxiety, hallucinations, ataxia, muscle weakness.	**See** MUSHROOMS - PSILOCYBIN
15 minutes to 2 hours	Perspiration, salivation, lacrimation; blurred vision, miosis; bradycardia, hypotension; abdominal cramps, diarrhea; bronchorrhea, wheezing, dyspnea.	**See** MUSHROOMS - MUSCARINE
15 minutes to 2 hours	Drowsiness, confusion, dizziness, ataxia, delirium illusions, muscle twitching, deep sleep.	**See** MUSHROOMS - IBOTENIC ACID/MUSCIMOL
15 minutes to several hours (usually less than 4 hours)	Nausea, vomiting, abdominal cramps, diarrhea.	**See** MUSHROOMS - GASTROINTESTINAL IRRITANT
2 hours	Nausea, vomiting, diarrhea. Dyspnea, muscle pain, rhabdomyolysis may follow.	**See** MUSHROOMS - RHABDOMYOLYSIS *Russula subnigricans*
2 to 11 hours	Nausea, vomiting, possible diarrhea, abdominal pain; possible myalgia; delayed polyuria, oliguria, renal failure.	**See** MUSHROOMS - *AMANITA SMITHIANA*
5 to 12 hours	Bloating, nausea, vomiting, diarrhea, abdominal pain; headache, dizziness, fatigue, fever; jaundice; rarely hemolysis.	**See** MUSHROOMS - MONOMETHYLHYDRAZINE
6 to 12 hours (up to 36 hours)	Colicky abdominal pain, vomiting, watery diarrhea. Symptoms subside after approximately 1 day; about 72 hours post-ingestion gastrointestinal symptoms recur along with signs of impending hepatic failure.	**See** MUSHROOMS - CYCLOPEPTIDES
1-3 days	Intense burning pain in hands and feet, numbness, paresthesias.	**See** MUSHROOMS - ERYTHROMELALGIA
1-3 days	Fatigue, weakness, myalgia, dark urine, nausea, sweating, leg stiffness, rhabdomyolysis.	**See** MUSHROOMS - RHABDOMYOLYSIS *Tricholoma equestre*
36 hours to 21 days	Nausea, vomiting, abdominal pain; night sweats, rigors, chills, headache; delayed oliguria, polyuria, renal failure.	**See** MUSHROOMS - *CORTINARIUS*

Clinical manifestations and time of onset may be highly variable if a mixture of mushrooms has been ingested, if alcohol, drugs or other substances have been taken concomitantly, if mushrooms have been contaminated with pesticides or bacteria, or if patient has an allergic or idiosyncratic reaction from eating a particular type of mushroom. Time of symptom onset post ingestion may also be misjudged if a toxic mushroom is eaten in two or more consecutive meals. A careful history should be obtained and patient treated symptomatically.

INVASIVE SPECIES ALERT!

DEATH CAP MUSHROOM (*Amanita phalloides*)

NATIVE RANGE

Death cap mushrooms are native to Europe.

DESCRIPTION

- Death cap mushrooms emerge from the ground as white buttons (called primordia) about the size of small chicken eggs. At this stage, they can be mistaken for puffballs or straw mushrooms.
- If the primordia are cut in half from top to bottom, a very careful examination will reveal the cap, gills and stem of a tiny mushroom.
- As the fungus matures, the stem elongates and the white tissue enveloping the developing mushroom (universal veil) breaks, leaving a membranous white sac (volva) at the base of the stem that may require careful excavation to keep it intact for observation.
- As the stem elongates further and the cap expands, a second white tissue (partial veil) that had covered the gills breaks, leaving a skirt-like ring or veil on the stem.
- In the mature mushroom, the cap has a distinctive olive or green hue, although the cap overall can appear pale green, pale brown, pale yellow or sometimes white, with: white gills; a white stem (or tinged with the cap's colour); a white, skirt-like partial veil or ring on the stem; and a white, membranous, sac-like volva surrounding the base of the stem.
- The death cap mushroom usually fruits in the fall, but it can fruit in the summer when yards are watered.
- Visit the following link for more photos and descriptions: https://en.wikipedia.org/wiki/Amanita_phalloides

REPORT INVASIVE SPECIES

www.reportinvasives.ca

PRIMARY IMPACT

The death cap mushroom is **deadly poisonous** if eaten. It can be mistaken for edible puffballs when young or the Asian straw mushroom when older.

DEATH CAP MUSHROOM (*Amanita phalloides*)

BIOLOGY AND SPREAD

The death cap mushroom forms mutually beneficial symbioses called ectomycorrhizas with the fine roots of certain trees native to Europe. The death cap mushroom was likely unintentionally introduced many decades ago from Europe on the roots of horticultural trees. It is likely that the mushroom became established in bare root tree nurseries in North America and has since spread to urban areas on the roots of trees raised in these nurseries and then planted along streets and boulevards. Common host trees are hazelnut, hornbeam, beech, linden, sweet chestnut and oak.

In Victoria, the death cap mushroom has been found to associate with Garry oak roots. If it acclimates to the Garry oak, the death cap mushroom may move out of urban areas into native Garry oak woodlands.

HABITAT

Presently in B.C., the death cap mushroom is known primarily from urban areas in Vancouver and Victoria. The mushroom fruits on the ground in the fall under suitable host trees and in the summer where lawns are watered. On Galiano Island, death cap mushrooms have fruited on the ground under a hazelnut tree that was planted decades ago when a farm was being developed there. In the Fraser Valley, death cap mushrooms have fruited in agricultural and suburban areas under old sweet chestnut and hazelnut plantings in Langley, Mission and Surrey.

WHAT SHOULD I DO IF I FIND ONE?

- Collect the whole mushrooms, bag them and dispose of them in the garbage.
- Wash your hands with soap and running water after handling the mushrooms.

B.C. Drug and Poison Information Centre:
1 800 567-8911

www.gov.bc.ca/invasive-species

BRITISH COLUMBIA

HOW CAN WE SLOW ITS SPREAD?

- Plant non-host trees on private property, boulevards or in parks.
- Collect, bag and dispose of these mushrooms in the garbage, preferably while they're still in the button stage. Although this step will not eradicate the fungus on the host tree's roots, it may slow this mushroom's spread via spore dispersal.
- Do not water known death cap mushroom sites on lawns during the summer.

August 2017

MUSHROOMS - CYCLOPEPTIDES

Description

This group accounts for almost all fatal mushroom poisonings.
Includes *Amanita phalloides* (death cap), *A. verna*, *A. virosa*, *A. bisporigera* and *A. ocreata*. Cyclopeptides are also found in other mushroom genera such as *Lepiota*, *Galerina* and *Conocybe*.
Poisonous *Lepiota* species include: *L. brunneoincarnata*, *L. castanea*, *L. chlorophyllum*, *L. helveola*, *L. josserandi*, and *L. subincarnata*. Poisonous *Galerina* species include: *G. autumnalis*, *G. marginata* and *G. venenata*.
Galerina species, *Conocybe filaris* and *Conocybe rugosa* are small brown mushrooms that have been mistaken for *Psilocybe* species (magic mushrooms). *Amanitas* have been eaten in error by those who confuse them with edible species.

Toxicity

Primary toxicity is centrilobular hepatic necrosis. Death may occur 6-16 days post ingestion from hepatic coma and renal failure. With intensive supportive care, mortality rate is 10-15% in adults and 30-50% in children. Mortality has decreased during the last decades possibly due to improved supportive care.
A small number of patients who survive moderate-severe poisoning develop chronic active hepatitis.

Mechanism of Toxicity

Principal toxins are cyclopeptides, amatoxins and phallotoxins. Phallotoxins are only slightly absorbed from gut. Toxic effects in human poisoning are caused by amatoxins which inhibit RNA-polymerase II enzyme, thus inhibiting protein synthesis and causing cell death. Cells with a rapid turnover are most affected. Upon ingestion, GI tract is first exposed and mucosal cells begin to degenerate. Liver is next organ to receive a high initial dose of amatoxins. Early damage occurs as protein synthesis is inhibited in hepatocytes; a centrilobular hepatic necrosis ensues and apoptosis may also be involved. Renal damage may be seen about 5 days post ingestion; initial dehydration and hypovolemia may worsen renal damage. Acute tubular necrosis has been seen on autopsy. Damage to other organs can also occur (pancreas, heart, marrow, colon).

Toxic Dose

Lethal dose of amatoxins in an adult is 5-7 mg or about 0.1 mg/kg or less of body weight; this amount can be present in one fresh *Amanita phalloides* cap of 60 g weight (fresh mushrooms contain about 94% water). Concentration of amatoxins in *Amanita phalloides* is 2.5-4.4 mg/g of dry weight, while *Galerina* and *Lepiota* species contain about 1.2 mg/g of dry weight. *Galerina* species are smaller than *Amanitas* and estimated lethal dose would require 100-150 g fresh mushrooms (10-20 caps). Concentrations vary widely. Amatoxins are not destroyed by drying, freezing or cooking.

Case Reports

Three adults and a 3-year-old ingested cooked *Amanita verna* and12 hours later experienced nausea, watery diarrhea and crampy abdominal pain. They were treated with IV fluids and an antiemetic. At 36 hours post ingestion, all were asymptomatic with normal vital signs. In the adults, ALT became elevated and peaked 48-72 hours post ingestion. INR was mildly elevated (1.33-1.89) within 48 hours of ingestion and began to correct within 96 hours (1.08-1.41). Renal function was not significantly affected. Adults received activated charcoal every 6 hours until coagulation parameters normalized; all recovered without sequelae. The 3-year-old was asymptomatic at 72 hours post ingestion, but developed an AST 7892 U/L, ALT 5326 U/L, INR 8.5. INR continued to rise despite 10 U fresh frozen plasma. On day 5, stage IV hepatic encephalopathy progressed rapidly and patient required mechanical ventilation. Pneumonia developed on day 7, and patient died in fulminant hepatic failure complicated by sepsis on day 11.

Pharmacokinetics

Studies in humans are limited. Absorption of amatoxins from gut and entry into liver are believed to be rapid, active processes. Amatoxins are excreted unchanged, principally by kidneys, and have been detected in urine as early as 2 hours post ingestion.
In a series of 23 patients, renal excretion of amatoxins of 0.06-8.5 mg/patient occurred over 6-72 hours post ingestion. Urinary excretion continued for 96 hours post ingestion and may have been due to enterohepatic recycling of amatoxins or further absorption from mushrooms in gut. By the time initial gastrointestinal symptoms have started (6-24 hours post ingestion), plasma amatoxins have fallen to low levels of about 2-4 ng/mL; therefore, only very small amounts are removed by charcoal hemoperfusion or hemodialysis at this time (in range of micrograms only). Amatoxins have been detected in plasma for at least 72 hours post ingestion but at very low concentrations. No correlation was found between serum concentrations and severity of poisoning. High levels are present in gastroduodenal aspirate, vomitus, and some toxin is also excreted in feces.

Clinical Effects

General: Cyclopeptide poisoning is characterized by 3 phases: onset of gastrointestinal toxicity in 6-24 hours post ingestion, followed by an asymptomatic false recovery and then delayed onset of hepatorenal failure. Death may occur 6-16 days post ingestion from hepatic coma and renal failure.

Phase 1: Gastroenteritis: 6-24 (mean 12 hours) hours post ingestion, lasting about 24 hours: Sudden onset of cramping abdominal pain, vomiting, watery diarrhea. Dehydration may result.

Phase 2: Latent: Apparent recovery occurs in most patients but asymptomatic liver damage is progressing. Serum aminotranferases rise rapidly after 24 hours post ingestion, peak 3-4 days post ingestion. INR increases about 48 hours post ingestion and peaks at 3-5 days.

Vitamin K may have little or no effect. Fibrinogen levels may also decrease.

Phase 3: Hepatorenal: By 72 hours post ingestion: Abdominal symptoms and diarrhea recur, along with indications of hepatic damage such as liver tenderness and enlargement, jaundice and hypoglycemia. Hypoglycemia may be due to hepatic failure as well as to release of insulin from a damaged pancreas.

CVS: Cardiac dysrhythmias have occurred rarely.

Neurologic: Confusion, delirium, convulsions and progressive coma (hepatic encephalopathy) may occur in severe cases.

Mixed sensory and motor polyneuropathy occurred in a few patients after *Lepiota* poisoning. Most recovered in one year, while one patient had progressive deterioration.

GI: Sudden onset of cramping abdominal pain, vomiting, watery diarrhea may occur 6-24 hours post ingestion; appears to resolve and then recur again later in phase 3. Ileus, toxic megacolon and colitis have occurred rarely.

Hepatic: Serum aminotranferases rise rapidly after 24 hours post ingestion, peak 3-4 days post ingestion. INR increases about 48 hours post ingestion and peaks at 3-5 days. Serum ammonia levels begin to rise between days 4-7. Liver tenderness. May rapidly progress to fulminant hepatic failure and death.

A small number of patients who survive moderate-severe poisoning develop chronic active hepatitis.

GU: Renal failure may be evident by days 5-7; blood urea may remain low due to lack of synthesis by the liver.

Fluids/Lytes/Acid-Base: Hypoglycemia. Hypocalcemia and hypophosphatemia and have occurred between days 4-7 post ingestion.

Metabolic acidosis may occur in severe cases.

Blood: Coagulopathy.

Pregnancy: In case reports of poisoning in pregnant women, fetal liver damage has not occurred at 13 weeks gestation or longer; however, a nine-week fetus had hepatocellular damage. In a series of cases with mild to moderate toxicity in the mother, birth weight was lower than controls but there was no difference in anomalies.

Prognosis: Signs and symptoms which may correlate with a poor prognosis include: Onset of initial symptoms < 8 hours post ingestion; peak transaminases > 5000 U/L with peak at 2 days; prothrombin time > 100 s at 96 hours post ingestion; hypoglycemia; decreased fibrinogen; metabolic acidosis; elevated ammonia levels by 4-6 days; bleeding tendency; persisting renal failure.

Treatment

1. **Ingestion**: Administer multiple dose aqueous activated charcoal (0.5 g/kg orally every 2 hours) for at least 24 hours. It may interrupt enterohepatic recycling of toxin and reduce toxicity. An antiemetic such as metoclopramide or ondansetron may be required if vomiting continues.

2. Replace and maintain fluids and electrolytes. Ensure moderately enhanced urine output for first 24-48 hours (100-200 mL/hr has been suggested).

3. Monitor electrolytes, glucose, creatinine, urea, liver enzymes, INR, calcium, phosphate. Blood gases should be monitored in severe cases. Amatoxins have been identified in body fluids by HPLC and radioimmunoassay but these tests are not generally available.

4. **Antidotes**: There are no proven antidotes but n-acetylcysteine, penicillin G and silibinin have been used. They may limit hepatic damage and are reasonably safe.
   - ***N-acetylcysteine:*** antioxidant properties may decrease progression of hepatic encephalopathy, nephropathy and coagulopathy. **See** N-ACETYLCYSTEINE antidote monograph. Dose as for acetaminophen toxicity and continue 3rd infusion until at least the 3rd day post ingestion in patients without hepatitis and longer in patients with hepatic damage.
   - ***High dose penicillin G:*** administer as a continuous IV infusion for 3 days.
     Adults: 0.5-1 million units/kg/day
     Children: 1 million units/kg/day.
     Penicillin may reduce re-excretion of amatoxins into bile; no controlled trials have been done. Caution: Large doses can cause seizures and increase serum sodium level (penicillin G sodium).
     Confirm patient is not penicillin allergic.
   - ***Silibinin*****:** 20-50 mg/kg/day IV for 48-96 hours has been used in Europe but controlled trials have not been done (extract of milk thistle; not available for IV use in North America). Oral preparations are available but may not be useful due to activated charcoal adsorption.

5. Aggressive symptomatic and supportive care may be required in severe cases including IV dextrose, crystalloids, fresh frozen plasma, blood products. IV vitamin K may only partially improve INR.

6. Liver transplantation has been successful in patients with fulminant hepatic failure. An early consult with a liver transplant centre should be considered in seriously ill patients as hepatic failure may progress rapidly. Patients who have had liver transplantation appear to have the same survival rates as other liver transplant patients.

7. Hemodialysis is not useful for toxin removal unless performed in first few hours post ingestion; may be required in patients with renal failure.

8. *Weiland/Meixner test for amatoxins*: Squeeze onedrop of juice from a fresh mushroom cap onto a piece of newspaper or filter paper. Circle spot in pencil and allow it to dry at room temperature or with

gentle low heat. Sunlight or high heat may give a false positive. Apply one drop of concentrated hydrochloric acid (8-12N HCl) to dried spot. Apply one drop of acid to another area of paper as a negative control (some newspaper turns blue with acid alone). If amatoxin is present, a blue colour will appear in 15-20 minutes (up to 1 hour) in test spot, assuming that control spot does not turn blue. Test is very sensitive for amatoxins but false positives may occur with other substances such as psilocin and 5-substituted tryptamines. False negatives may occur with low amanitin levels.

Key Points

- ✓ This group accounts for almost all fatal mushroom poisonings.
- ✓ Primary toxicity is centrilobular hepatic necrosis. Death may occur 6-16 days post ingestion from hepatic coma and renal failure.
- ✓ During acute gastrointestinal phase, if association with a mushroom ingestion is not made, patient may be erroneously diagnosed with gastroenteritis and discharged.
- ✓ Aggressive treatment of fluid and electrolyte abnormalities, acid-base disturbances, coagulopathy and encephalopathy are paramount.
- ✓ There are no proven antidotes but N-acetylcysteine, penicillin G and silibinin have been used.
- ✓ Acute hepatic failure from amatoxins may be partially differentiated from that due to acetaminophen overdose or infectious hepatitis by preceding history of acute gastroenteritis.
- ✓ If a patient has ingested more than one species of mushroom, an early onset of gastrointestinal upset may occur but does not rule out presence of amatoxin mushrooms in the mix.