NEW ERA AGRICULTURE MAGAZINE

# Understanding Mushroom Poisoning: Risks and Remedies

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**Abstract: -**

There are thousands of different kinds of mushrooms in the world; some are edible, while others are toxic because they contain high levels of toxins. Edible mushrooms are a common food item with enticing flavor, taste, and nutritional value that are frequently cultivated at home and marketed commercially these days. The most common causes of mushroom poisoning are intentional searches for psychotropic mushrooms, inadvertent children ingestions, and mistaking a deadly mushroom for an edible species that bears striking similarities. Amanita Phalloides, also known as the "death cap," is the primary cause of deadly mushroom poisoning because of its high concentration of the powerful cytotoxin amatoxin. Fatal poisoning is typically accompanied by extremely severe symptoms that develop gradually and involve the liver, kidneys, brain, and hemolysis. The objective of this article is to provide knowledge and to prevent problems.



## Introduction:

Mushroom poisoning denotes harmful consequences resulting from the consumption of poisonous compounds found in mushrooms. The poisons identified are secondary metabolites synthesized through certain metabolic processes within the fungal cell. The majority of fatal poisonings globally are attributed to the Amanita phalloides mushroom, commonly known as the "death cap." Other hazardous mushroom species include Amanita verna, Amanita virosa, Gyromitra, Galerina, and Lepiota, all of which contain amatoxin, a strong cytotoxin. Consuming even a fragment of a mushroom from a toxic species can be lethal. The deadly dose of Amatoxin is around 10 mg, the quantity present in a single death cap mushroom. The defining pathological finding

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in deaths due to amatoxin-containing mushroom poisoning is acute massive necrosis of liver parenchyma. 1 Hemolysis and renal failure frequently occur alongside occasional pancreatitis. Mushroom poisoning typically occurs from the consumption of wild mushrooms, resulting from the misidentification of a poisonous species as an edible counterpart that closely resembles it. It is also observed that small infants, particularly toddlers in the 'grazing' phase, consume mushrooms located on the lawn. Adolescents purposefully use magic mushrooms for their hallucinatory effects. The clinical manifestations of mushroom poisoning primarily depend on the species of mushroom (degree of toxicity), quantity ingested, age (children exhibit more severe symptoms than adults due to lower body weight), onset time of symptoms (highly toxic mushrooms often present delayed and severe symptoms with hepatic, renal, hemolytic, and CNS involvement), geographic distribution (certain dangerous species may be more prevalent in specific areas, and some mushrooms that appear edible in one region may be lethal in another), and pre-existing hepatic and renal conditions.

**Clinical signs of amatoxin toxicity**

They are informed that this is the most lethal toxin. The manifestations transpire in various stages or phases.

1. A distinctive latent period of 6-24 hours following intake prior to the manifestation of symptoms.
2. Subsequently, abdominal cramping, nausea, vomiting, and severe watery diarrhea manifest, typically lasting for 24 hours; fluid losses may be substantial enough to induce profound dehydration, potentially leading to circulatory collapse. A remission period of symptoms lasts 1-3 days, during which the patient may be discharged if hospitalized. Ongoing liver damage is happening, as revealed by test examinations showing elevated serum aminotransferase levels and prothrombin time. Hepatic and renal damage become clinically evident and may advance to fulminant hepatic failure and renal failure. Mortality transpires within 3-7 days or recuperation occurs during 2-3 weeks.

**Adverse prognostic criteria in acute liver failure**

Prothrombin time exceeding 100 seconds Or Any three of the subsequent criteria:

i. Prothrombin time exceeds 50 seconds.
ii. Serum bilirubin level: >300 µmol/L (H"17.6 mg/dl)
iii. Serum Creatinine: >300 µmol/L (H 3.38 mg/dl)
iv. Duration from jaundice to encephalopathy is less than 7 days.

v. Age is either below 10 years or above 40 years, or Factor V level is less than 15% of normal with encephalopathy graded 3 or (severe confusion or coma).

These factors forecast a death rate exceeding 90%.

**Diagnosis of mushroom poisoning**

1. A beneficial history of mushroom consumption is evident from the clinical symptoms. Symptoms may be moderate and initial when the mushroom species is less toxic, the quantity swallowed is minimal, and a single species is consumed; nevertheless, when multiple species of mushrooms are ingested, early symptoms do not preclude the possibility of delayed and fatal sequelae from a highly toxic species. Cases of poisoning from very toxic mushrooms typically have a delayed start of symptoms, frequently involving hepatic, hemolytic, renal, and central nervous system complications.
2. If a specimen of the consumed mushroom is located, analysis for amatoxins should be conducted using a Maixner test.
3. By applying a drop of liquid from the specimen onto a paper composed of wood pulp, such as newspaper, allowing it to dry, and subsequently placing a drop of 10-12 N HCl on this location. After many minutes, a blue color will manifest if amatoxin is present. An skilled mycologist can analyze and detect spores in gastrointestinal contents. Three A regional toxicology center may be notified.
4. High performance liquid chromatography for the identification of amatoxin in plasma, feces, urine, or vomit should be conducted if facilities are available.

**Differential diagnosis**

1. Gastroenteritis
2. Plant poisoning from different species
3. Hypovolumic shock and
4. Other causes of acute liver and renal failure

**Management**

1. Pre-hospital care: Implement supportive measures as necessary, such as intravenous access and oxygen administration, and induce emesis. Should a mushroom specimen be accessible, enclose it in a dry paper bag (avoid moisture and refrigeration) for identification by a mycologist.
2. Emergency department care - administer aggressive treatment to a patient with suspected mushroom consumption, as the death rate associated with swallowed amatoxin is approximately 20%.
3. Mitigation of amatoxin absorption. Four Administer gastric lavage if the patient has not yet vomited. Decontamination should be conducted within one hour of intake.

Due to the delayed presentation, the success of this surgery remains unknown, as patients typically feel sick and seek medical assistance after a delay of 12 hours or more. Administer repeated oral doses of activated charcoal for any recent consumption of an unidentified or potentially dangerous mushroom. Amatoxins seem to engage in entero-hepatic circulation; repeated administration of activated charcoal and laxatives may disrupt this cycle and mitigate toxicity. Gastroduodenal aspiration may be performed to eliminate toxins excreted in bile, thereby disrupting this cycle and mitigating toxicity. The cornerstone of treatment comprises vigorous intravenous fluids and electrolytes to rectify and sustain proper hydration and urine output, alongside intense supportive care for hepatic failure.

**Discussion**

An insufficient database exists to assess global exposure and mortality resulting from mushroom poisoning. Patients with severe hepatitis due to mushroom poisoning are considered to have a bad prognosis and often require liver transplantation for survival. However, with prompt and assertive multidisciplinary intervention, such patients exhibit enhanced outcomes and may circumvent the necessity for liver transplantation. A retrospective research conducted in San Francisco, USA, involving 8 hospitalized patients over a 5-year period, revealed that all patients lived; however, 3 suffered encephalopathy, and 1 experienced acute renal failure necessitating hemodialysis. A retrospective analysis conducted over 15 years involving 105 patients from Florence, Italy documented 7 fatalities, occurring solely in 2 patients who were admitted 60 hours post-mushroom ingestion. The remaining patients recovered fully without sequelae, all having received treatment within 36 hours of ingestion, and none required liver transplantation. A 20 year retrospective analysis of amatoxin poisoning treatment in 2108 hospitalized patients from North America and Europe revealed minimal efficacy of Penicillin G, thioctic acid, or steroids, while demonstrating the effectiveness of silybin, N-acetyl cysteine, and detoxification procedures.

**References**

1. Jules L D. Toxic and Drug Induced Hepatitis. Dennis L. Kasper , Anthony S. Fauci , Dan L. Longo et al. Harrison's Principles of Internal Medicine: Mc Graw- Hill. 17th ed. Newyork: McGraw-Hill; 2008: 1949-51. Jones A L, Karalliedde L.Poisoning. Davidson's Principles & Practice of Medicine. 20th ed. New

Delhi: Churchill Livingstone Elsevier; 2006: 952-3.

2. Kent R. Poisoning. Stephen J, Maxine A. Current Medical Diagnosis & Treatment. 47th ed. Newyork: McGraw-Hill Lange; 2008: 1378-9.
3. Litovitz T L, Smilkstein M, Felberg L et al. 1996 annual report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. *Am J Emerg Med.* 1997; **15**: 447-500.