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Mushroom poisoning: An updated review

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Abstract:

Mushrooms have been consumed frequently worldwide since ancient times. In addition to edible and harmless species, there are also poisonous species that cause a wide range of clinical syndromes, from simple gastrointestinal (GI) irritation to death. However, it is not possible to distinguish the poisonous species from some edible species morphologically. Therefore, the unintentional consumption of mushrooms is an important public health problem. Mushrooms can be categorized according to their toxins, such as cyclopeptides, gyromitrin, muscarine, coprine, orellanine, psilocybin, and GI irritants. Mushrooms containing cyclopeptide-amatoxin are responsible for more than 90% of deaths due to mushroom poisoning. *Amanita phalloides* is responsible for many fatal cases because of the toxicity of this species. This article reviews the clinical syndromes that may develop after the consumption of various poisonous mushroom species, the mechanisms of action of their toxins, and the current treatments applied.

Keywords:

Acute renal failure, liver injury, mushroom poisoning, mushroom species, toxicity

Introduction

The global significance of mushroom poisoning

Mushrooms are proven sources of diverse and bioactive secondary metabolites that exhibit a range of beneficial properties as therapeutic agents for various diseases.[1] Edible species constitute an ideal source of carbohydrates, dietary fiber, and protein. However, some poisonous mushrooms that should not be eaten are morphologically very similar to some harmless mushrooms. Because these mushrooms contain extremely potent toxins, they are associated with poisoning syndromes that require medical treatment.[2] However, in up to 80% of cases, the type of mushroom ingested has not been identified. Therefore, the diagnosis of mushroom poisoning should be based on the signs and symptoms present. The consumption of wild mushrooms can expose humans to toxic or lethal doses of poison, highlighting the importance of understanding mushroom toxicity.

Challenges in distinguishing poisonous mushrooms from nonpoisonous mushrooms

The consumption of wild mushrooms is common, especially in the countryside.[3] Spring is the best season for mushrooms to grow and be found, but it is also the season in which we most frequently encounter mushroom poisoning. Despite numerous warnings about the risks of serious and potentially fatal poisoning, many people still consume wild mushrooms due to their morphological similarity to edible species. For example, the phenotype of *Amanita phalloides*, which constitutes 90% of fatal mushroom poisonings, and the phenotype of

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Agaricus bisporus, a frequently consumed nonpoisonous species, are almost the same. This similarity increases the risk of poisoning from poisonous mushrooms and causes thousands of mushroom poisonings worldwide every year.[4]

Mushroom species, toxic substance specifications, and impact mechanism

Mushrooms can be categorized according to their toxins, such as cyclopeptides, gyromitrin, muscarine, coprine, orellanine, psilocybin, and gastrointestinal (GI) irritants [Table 1].

Amatoxin-containing mushroom poisoning

Mushrooms containing cyclopeptide-amatoxin are responsible for more than 90% of deaths due to mushroom poisoning.[3] *A. phalloides* is responsible for many fatal cases due to its toxicity.[5] Amatoxin poisoning generally has a poor prognosis because of the high risk of liver failure. There are no widely accepted guidelines for the treatment of patients with amatoxin poisoning, but the treatment involves supportive care with multiple combinations of medications, including antidote antibiotics and antioxidant treatments. Various antidotes are used, including benzylpenicillin, ceftazidime, silybin, and N-acetylcysteine (NAC). Liver transplantation has markedly increased the survival probability for patients poisoned with *A. phalloides* and remains the main treatment for patients with fulminant liver failure. The probability of survival depends on the degree of hepatic injury, the ability of residual liver cells to regenerate, and the management of complications that may occur during the course of poisoning.

Among these mushrooms, cyclopeptide-containing mushrooms are the most toxic species worldwide and are responsible for 90%–95% of human deaths from mushroom poisoning.[3] The main toxic substances are amatoxins found in the *Amanita*, *Lepiota*, and *Galerina* genera.[6] *Amanita* peptide toxins are divided into 3 subgroups: amatoxins, phallotoxins, and virotoxins. Phallotoxins constitute the subgroup that has the fastest toxic effect.[7]

Amatoxins are potent RNA polymerase inhibitors that block mRNA production and protein synthesis in liver and kidney cells. Moreover, amatoxin-induced liver injury involves p53- and caspase-3-dependent apoptosis. Toxophallin toxin isolated from *A. phalloides* has been identified as an L-amino acid oxidase related to oxidative stress. Oxidative stress, which contributes to massive necrosis, plays an important role in the development of severe hepatotoxicity. Approximately 60% of toxins are excreted into the bile and return to the liver via the hepato-intestinal circulation, resulting in toxic inflammation and hepatocyte necrosis.[8]

Nonamatoxin-containing mushroom poisoning

False lamb mushrooms containing gyromitrin may cause neurotoxic seizures by inhibiting glutamic acid decarboxylase activity, preventing gamma-aminobutyric acid formation, and causing pyridoxine deficiency. Like the cyclopeptide group, these mushrooms are also hepatotoxic. They may cause methemoglobinemia-like conditions in the clinic by causing oxidative stress.

Mushrooms such as *Clitocybe dealbata*, which are not very common and contain muscarine, cause cholinergic syndrome (salivation, lacrimation, urination, defecation, and GI symptoms).

Mushrooms containing coprine toxins inhibit disulfamide-like aldehyde dehydrogenase, causing a disulfiram-like reaction.

Mushrooms containing psilocybin toxin have a direct hallucinogenic effect.

Mushrooms containing orellanine toxin and allenic neurosin may present with renal failure.[9]

Several species of mushrooms cause acute gastroenteritis without any toxicity immediately after ingestion. *Chlorophyllum molybdites* is the molybdite that most commonly causes GI irritation.

Diagnostic Tests

There are few fungal toxins available for use in laboratory tests, and laboratory tests are not routinely used in diagnostic studies.[10] However, these toxins can be obtained through reference laboratories. Qualitative evidence of amatoxin in blood and urine may be valuable, as a confirmed diagnosis allows early initiation of aggressive treatment and possibly reduces mortality rates. For these reasons, the diagnosis should first be made on the basis of clinical findings, laboratory results, and anamnesis. The diagnosis of mushroom poisoning is based on three principles: identification of the ingested mushroom, time interval between mushroom ingestion and onset of symptoms, and confirmation by laboratory tests.[11] However, it is generally impossible to determine the type of mushroom ingested. Digestive symptoms are the most common.[12] This diagnosis should be suspected in patients with a history of severe GI symptoms (nausea, vomiting, diarrhea, and abdominal cramps) that occur several hours or a day after mushroom ingestion. A complete blood count, urinalysis, international normalized ratio (INR), and especially liver–kidney function tests should be routinely requested. Studies are being carried out on new markers (automatic magnetic bead-based chemiluminescence immunological test [CLIA]) for the early diagnosis of

phallotoxin-containing peptide-amatoxins in blood and urine. Studies on the detection of amatoxin in blood or urine (quadrupole-time-of-flight high-resolution mass spectrometry and ultra-performance liquid chromatography-mass spectrometry) have also been carried out.[13]

Acute liver failure, characterized by a sudden increase in aminotransferase enzymes and bilirubin, is the main pathophysiological feature of amatoxin poisoning.[14] In addition to the liver, the kidneys and central nervous system are affected. The overall severity of *A. phalloides* poisoning depends on the amount of toxin ingested and the time elapsed between the time of poisoning and the start of treatment.

The detection of amatoxin in blood or urine is not routinely used in many hospital laboratories, but amatoxin can be obtained through reference laboratories. However, these results should not delay the start of treatment. Amatoxins can also be detected in blood or gastric contents, but a urine test is preferred. Detection methods include polymerase chain reaction, high-performance liquid chromatography/mass spectrophotometry, lateral flow immunoassay, enzyme-linked immunoassay, and radioimmunoassay. The enzyme-linked immunosorbent assay (ELISA) test for amanitin in urine is the most technically simple and has high sensitivity. The ELISA test is most reliable for detecting amatoxins within 36 h of mushroom poisoning. Amatoxins are usually undetectable in the blood or urine four days after mushroom ingestion.

Clinical Findings – Differential Diagnosis

Mushrooms that cause symptoms more than 6 h after consumption cause serious and potentially fatal toxicity. The most well-known species of poisonous mushrooms are those that irritate the GI tract and generally do not cause life-threatening clinical findings.[12] However, the differential diagnosis is very difficult for clinicians.

The time at which symptoms start is very important in the differential diagnosis of mushroom poisoning. Patients usually begin to show GI symptoms within the first 2–3 h. Symptoms of deadly mushrooms usually appear after 6–8 h, with GI symptoms such as nausea, vomiting, diarrhea, and abdominal heaviness. Although there may be laboratory evidence of hepatotoxicity developing 24–36 h after ingestion, there may be a quiescent interval during which the patient typically improves in terms of symptoms. Progressive acute liver and kidney failure after a delay of 24–72 h may cause nephropathy, coagulopathy, seizures, hepatic coma, encephalopathy, brain edema, and mortality.[10]

The degree of liver damage is directly proportional to the amount of mushrooms consumed. In the differential diagnosis, gastroenteritis, pancreatitis, hepatitis, acute renal failure, hallucinogenic poisoning, anticholinergic-cholinergic poisoning, disulfiram toxicity, and food poisoning should be considered. *Cortinarius orellanus* may cause late acute renal failure with its orellanine toxin, and *Amanita smithiana* may cause late acute renal failure with its allenic norleucine toxin. Many types of mushrooms can cause GI irritation.

The most important feature in the differential diagnosis is the onset of clinical symptoms. *C. molybdites* and *Boletus* species, which are the species that most commonly cause GI irritation, cause symptoms of abdominal pain, diarrhea, nausea, and vomiting within 1–3 h, whereas other mushroom species that can cause fatal toxicity cause clinical symptoms after more than 6 h. An exception is the *A. smithiana* species; late findings, 24 h after the onset of GI symptoms in the first 6 h, may lead to acute renal failure.[9]

Notably, in some cases, the GI symptoms caused by mushrooms are not the result of those that are poisonous but of others that are microbially contaminated, raw or undercooked, eaten in large quantities, or eaten too often.

Treatment

The general management of amatoxin poisoning is as follows:

- Volume replacement therapy
- Preventing amatoxin absorption
- Elimination of absorbed amatoxins
- Potential antidote treatment against liver damage
- Liver transplantation.[14,15]

When patients are hospitalized, they experience dehydration due to vomiting and diarrhea, which needs to be compensated for, including electrolytes. Since the toxin is eliminated primarily through the kidneys, patients should be monitored for adequate urine production during this period.

Management of amatoxin-containing mushroom poisoning

When *A. phalloides* is taken orally, amatoxins are rapidly absorbed in the GI tract. Therefore, gastric lavage and activated charcoal at a dose of 0.5–1 g/kg or up to a maximum of 50 g should be used immediately to effectively reduce the absorption of amatoxins from the GI tract.[16] However, owing to the asymptomatic lag phase between mushroom ingestion and symptom onset, patients are usually admitted to the hospital only after serious GI disorders. Therefore, the effectiveness of gastric lavage is significantly reduced. Although

the effectiveness of activated charcoal mostly depends on the time between ingestion and treatment, it can be applied routinely, as it not only potentially reduces toxin absorption but also interrupts the enterohepatic circulation of amatoxins.[16]

Three drugs are available as antidotes: penicillin G, silibinin, and NAC. While benzylpenicillin and silibinin inhibit the uptake of amatoxin by liver cells, Vitamin C, cimetidine, and NAC are antioxidants that stop the lipid peroxidation caused by amatoxin, thus causing cell membrane instability.[17]

Benzylpenicillin is one of the most commonly used drugs in the treatment of *A. phalloides* poisoning. Benzylpenicillin has been shown to significantly increase the viability of hepatocytes exposed to amatoxins and reduce the expression of apoptosis markers. Silibinin is obtained from milk thistle and is the pharmacologically active substance in the silymarin complex. Silibinin stabilizes the membranes of liver cells and has been shown to exert a strong protective effect against α-amanitin-induced toxicity in human hepatocytes.[18] The entry of toxins into cells is effectively prevented. It has a pronounced antioxidant effect: lipid peroxidation is inhibited, and the synthesis of inflammatory substances is reduced. It helps regenerate liver cells, accelerate protein synthesis, and regenerate damaged membranes. Benzylpenicillin together with silymarin inhibits the elevation of plasma aminotransferases (alanine aminotransferase and aspartate aminotransferase) and alkaline phosphatase in dogs poisoned by *A. phalloides*. Especially when silymarin is not available, a high dose of 300,000-1 million U/kg/day (maximum 40 million U/day) intravenous (IV) infusion is recommended as a continuous infusion.[19] Benzylpenicillin doses are recommended at 1 MU/kg/day and 0.5 MU/kg/day by CIAV (Portuguese Poisoning Information Center) and TOXBASE (www.toxibase.org), respectively. However, benzylpenicillin is not recommended by the New Zealand National Poisons Center because of its safety and allergy potential.[14] It has also been reported that co-administration of silymarin is not superior to silymarin alone. Silibinin is more effective when given within 24 h of ingestion. It is recommended that silymarin be given to poison patients intravenously at a loading dose of 5 mg/kg over 1 h, followed by a continuous dose of 20 mg/kg per day until liver function and the INR return to normal.[14] NAC functions not only as a "scavenger" reducing agent of free radicals but also as a glutathione precursor when endogenous stores are depleted. NAC may not have been recommended by the CIAV, possibly because of the high incidence of anaphylactic reactions and its elevation of the INR, a marker of clinical outcome in patients with *A. phalloides* syndrome.[20] However, a dose of 150 mg/kg intravenously over 15 min, followed by 50 mg/kg over 4 h, and then 100 mg/kg over 16 h are recommended by the New Zealand National Poisons Center (www.toxinz.com). Although the results in humans are not yet clear, the use of Vitamin C and cimetidine in combination with silibinin and acetylcysteine in patients with mushroom poisoning and evidence of hepatotoxicity is recommended, as they do not have significant side effect profiles.[19] Until clinical improvement is achieved, 300 mg of IV cimetidine every 8 h and 3 g of IV Vitamin C per day are recommended.[19]

Extracorporeal methods can be applied to patients admitted to the hospital during the latent period to remove toxins.[14] The plasmapheresis method is considered an effective treatment for *A. phalloides* poisoning. In general, plasmapheresis combined with supportive treatment removes amatoxins and their metabolic waste from the blood and provides albumin, immunoglobulins, clotting factors, fibrinolytic proteins, and mineral salts to maintain the internal environment for hepatocyte regeneration. Hemodialysis and hemoperfusion were previously recommended for patients poisoned by *A. phalloides*. However, recent detailed studies have shown that the effects of these treatment methods are negligible because toxins are only detected very early in the plasma and are present for a very short period of time.[14]

Management of mushroom poisoning, with the exception of amatoxin-containing mushrooms

The general approach to poisoning by mushrooms other than mushrooms containing amatoxin is to initiate general supportive care and implement specific treatments for this syndrome. These are important steps in the initial treatment of poisoning by mushrooms. Supportive treatments and GI decontamination with activated charcoal are sufficient for the appropriate treatment of most patients with mushroom poisoning.[21] Vomiting can be safely treated with an antiemetic (e.g., ondansetron 0.15 mg/kg intravenously) and does not increase the amount of toxin absorbed. These fungi can cause a variety of conditions.

Fungi that cause gastroenteritis without liver failure generally do not cause mortality, and treatment is supportive.

Mushrooms containing psilocybin toxin can cause hallucinogenic symptoms within 30 min to 2 h; death is generally rare, and symptoms resolve within 12 h. Benzodiazepines can be used for agitation during treatment. In addition, the treatment to be applied is supportive treatment.[22]

Symptoms of mushroom ingestion that cause central nervous system depression or stimulation begin within

Table 1: Toxins of mushroom species and associated clinical findings

Clinical finding	Toxins
Acute gastroenteritis without liver failure	GI irritants
Hallucinogenic	Psilocybin, psilocin
CNS excitation and depression	Ibotenic acid, muscimol
Cholinergic	Muscarine
Disulfiram-like reaction	Coprine
Gastroenteritis and delayed onset renal failure	Allenic norleucine
Liver toxicity	Cyclopeptides: Amatoxins, phallotoxins
Seizures, delayed gastroenteritis, and liver toxicity	Gyromitrin
Delayed renal failure	Orellanine, orellanine, *Cortinarius*
Delayed rhabdomyolysis	Unknown
Erythromelalgia	Acromelic acid
Delayed encephalopathy	Polyporic acid (causes violet-colored urine)
Immune-mediate hemolytic anemia	Antibodies to *Paxillus involutus*
Shiitake dermatitis	Lentinan
Allergic bronchoalveolitis	Allergic reaction to spores of *Lycoperdon* species

GI: Gastrointestinal, CNS: Central nervous system

30 min to 2 h, and death is rare. Symptoms usually resolve within 24 h. Treatment is supportive, and benzodiazepine is used for agitation.[22]

After the ingestion of mushrooms containing muscarine toxin, cholinergic syndrome symptoms (vomiting, diarrhea, bradycardia, bronchorrhea, bronchospasm, salivation, lacrimation, etc.) occur within 30 min to 2 h. Death is rare, and symptoms usually resolve within 12 h. Treatment for cholinergic syndrome should be applied. Atropine (0.02 mg/kg initial dose) and supportive treatment are administered until the bronchial secretions dry.

Mushrooms containing coprine toxins may cause disulfiram-like reactions (flushing, headache, tachycardia, chest pain, anxiety, etc.) when taken within 3 days after alcohol consumption.[23] Symptoms begin within 30 min to 2 h and usually resolve within 6 h, and death is rare. The treatment is supportive therapy.[21]

After consuming mushrooms containing allenic norleucine toxin, GI symptoms may begin within 30 min to 3 h and cause renal failure after 12–24 h.[24] Mortality is rare, and kidney functions recover completely in most patients, but dialysis may be needed in the acute period.[25]

Mushrooms containing orellanine toxin also cause delayed renal failure when consumed, but symptoms occur later than those associated with allenic norleucine.[26] Renal failure may occur after 3–20 days, and its course is worse. Hemodialysis may be required for treatment. End-stage renal failure may develop in 11% of patients and may require a kidney transplant.

Gastroenteritis, seizures, methemoglobinemia, and liver toxicity may develop 4–10 h after the consumption of mushrooms containing gyromitrin toxin. Mortality rates ranging from 0 to 10% have been reported. Activated charcoal can be given for treatment,[16] benzodiazepines and pyridoxine (70 mg/kg, maximum dose: 5 mg) are recommended for seizures, IV methylene blue (1–2 mg/kg over at least 5 min) is recommended for methemoglobinemia, and IV fluid resuscitation is recommended for GI symptoms.

Rhabdomyolysis may develop 24–72 h after *Tricholoma equestre*-type mushrooms are consumed.[27] Twenty-five percent of these cases may result in mortality. The treatment is rhabdomyolysis (IV fluid replacement-hyperkalemia treatment-hemodialysis).

When the *Clitocybe acromelalga* mushroom, which contains acromelic acid toxin, is consumed, symptoms of severe burning and hyperalgesia in the extremities may develop, along with erythema and edema after 24 h. Mortality is rare, and treatment consists of supportive care and pain management.[28]

Delayed encephalopathy may develop 12–24 h after the consumption of some mushroom species (*Pleurocybella porrigens* and *Hapalopilus rutilans*).[29] While these mushroom species can be fatal in patients with renal failure, mortality is rare in otherwise healthy patients. Treatment is supportive therapy.

Dermatitis symptoms due to the lentinan toxin present in raw or undercooked shiitake mushrooms may occur 2 h to 5 days after consumption. No deaths related to these mushrooms have been reported thus far, and the treatment is symptom management; in severe cases, antihistamines and systemic corticosteroids are used.[30]

Conclusion

Mushroom poisoning can cause many clinical syndromes, depending on the type of mushroom ingested. The most frequently consumed poisonous species are more likely to present with GI symptoms and acute liver injury. With early diagnosis and early initiation of treatment, permanent damage and mortality can be prevented. Unfortunately, tests that allow the early detection of toxins (amatoxin) of high-mortality mushrooms, such as *A. phalloides*, in the blood and urine are not widely used in hospitals or emergency services. Moreover, there are no clear treatment instructions in toxicology

books. Therefore, these clinical syndromes should be well understood, the need for antidote treatment should be decided without delay on the basis of anamnesis and clinical and laboratory findings, and the necessary supportive treatment should be started. The most commonly used antidotes are penicillin G, silibinin, and NAC. If necessary, extracorporeal methods should be included in treatment without delay in patients who do not respond to antidote and supportive treatment. Plasmapheresis is accepted as an effective treatment approach for *A. phalloides* poisoning. Although all medical support and treatment, patients who are expected to develop acute liver failure should be provided to organ transplant units.

Author contribution statement

ÖT: Conceptualization, methodology, investigation, resources, data curation, and writing – original draft. AAA: Conceptualization, methodology, investigation, resources, data curation, writing – original draft, review and editing, and supervision. All authors approved the last version of the manuscript.

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