



Letter to the Editor

Mushroom Poisoning in Iran: Causative Species, Clinical Syndromes, Diagnosis, and Management

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Dear Editor

Poisonous mushrooms comprise numerous species with diverse clinical manifestations and may be difficult to distinguish from edible mushrooms by appearance alone. Therefore, classification according to toxin, clinical syndrome, and time to symptom onset is useful for diagnosis, management, and prognosis. Figure 1 shows some of the poisonous mushrooms discussed in this letter, several of which resemble edible species [1-3].

Gastrointestinal syndrome

Gastrointestinal toxins typically cause symptoms within 30 minutes to 3 hours, including nausea, vomiting, abdominal pain, diarrhea, and lethargy. Treatment is supportive, with fluid and electrolyte replacement and symptomatic use of antiemetics or antispasmodics [2, 3]. Species reported from Iran include *Agaricus arvensis*, *A. silvicola*, *A. xanthodermus*, *Chalciporus piperatus*, *Rubroboletus satanas*, *Entoloma sinuatum*, *Hebeloma crustuliniforme*, *H. bulbiferum*, *Hypholoma fasciculare*, *Lepiota cristata*, *Leucocoprinus leucothites*, *Lactifluus glaucescens*, *Omphalotus olearius*, *Russula emetica*, *Scleroderma citrinum*, and

Tricholoma sulphureum [3, 4].

Muscarinic syndrome

Muscarine-containing mushrooms produce predominantly peripheral muscarinic effects because muscarine neither crosses the blood-brain barrier nor significantly stimulates nicotinic receptors. Symptoms usually begin within 30 minutes to 2 hours and include salivation, lacrimation, sweating, miosis, nausea, vomiting, diarrhea, abdominal cramps, bradycardia, hypotension, bronchospasm, and increased bronchial secretions. Treatment is mainly supportive; moderate or severe poisoning may require intravenous atropine, repeated until muscarinic manifestations resolve [2, 3]. Reported species from Iran include *Leucocybe candicans*, *Clitocybe phyllophila*, *C. dealbata*, *C. ericeticola*, *Entoloma rhodopolium*, *Inosperma erubescens*, *Inocybe dulcamara*, *I. fastigiata*, *I. geophylla*, and *Mycena pura* [4].

Phalloides syndrome

Cyclopeptide-containing mushrooms reported from Iran include *Amanita phalloides*, *A. verna*, *A. virosa*, *Lepiota brunneoincarnata*, and *Galerina marginata* [4-7]. Their major clinically important toxins are amatoxins, which inhibit RNA polymerase II and protein synthesis, causing severe gastrointestinal and

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Figure 1. Some poisonous mushrooms that are difficult to distinguish from edible mushrooms based on appearance. A–F: Examples of mushrooms containing gastrointestinal toxins, which cause gastrointestinal poisoning. G–I: Examples of mushrooms containing muscarine, ingestion of which causes the muscarinic syndrome. J–L: Three *Amanita* species—*A. phalloides*, *A. virosa*, and *A. verna*—which contain amatoxins and cause phalloides syndrome. Image credits: A, Piernario Maculan (86926214); B, Mary Ellen (57484376); C, T. Pedro Hafermann (87255539); D, Jo Ga (247091652); E, moorebuesing (18742664); F, Bethany Teigen (139419517); G, Liubov Ilminska (137795315); H, Liubov Ilminska (137814185); I, Sigrid Jakob (105185098); J, Anne Parsons (329393834); K, Солей (394621124); L, Danny Cicchetti (48049610). All photographs: CC0 1.0, via iNaturalist.

hepatotoxic effects. Amatoxin poisoning typically progresses through an initial gastrointestinal phase (4–24 hours), an apparent recovery phase (12–36 hours), and a subsequent hepatotoxic phase (2–4 days) that may progress to acute liver failure, encephalopathy, coagulopathy, hypoglycemia, renal failure, seizures, and coma. Mortality is approximately 15%–30% [2, 3].

A delayed onset of gastrointestinal symptoms, particularly 4–6 hours or more after ingestion, should raise suspicion for amatoxin poisoning. However, when several mushroom species are ingested, gastrointestinal symptoms may begin earlier because of rapidly acting toxins from other mushrooms. Persistent symptoms

should therefore not exclude concomitant amatoxin poisoning. *Hypholoma fasciculare*, although generally associated with gastrointestinal toxicity, has also been reported to cause phalloides syndrome [1, 6].

The Meixner test may provide rapid presumptive evidence of amatoxins when the mushroom is available, although false-positive results can occur, particularly with *Psilocybe* species [2, 3]. Treatment consists primarily of aggressive supportive care, with multiple-dose activated charcoal, intravenous penicillin G, silymarin, and N-acetylcysteine used in appropriate patients; severe cases may require management of acute

liver failure [1, 2, 8].

Coprinus syndrome

Coprine inhibits aldehyde dehydrogenase and may cause a disulfiram-like reaction when alcohol is consumed during or within 48–72 hours after ingestion, resulting in flushing, tachycardia, nausea, and vomiting. *Coprinopsis atramentaria* (formerly *Coprinus atramentarius*) and *Clitocybe clavipes* have been reported from Iran. Treatment is supportive [2, 6].

Pantherina (mycoatropine) syndrome

This syndrome is caused by ibotenic acid and muscimol and has been reported with *Amanita pantherina* and *A. gemmata* in Iran [2-4]. Clinical manifestations include drowsiness, confusion, hallucinations, delirium, agitation, altered consciousness, and, particularly in children, myoclonus and seizures. Management is supportive, with benzodiazepines used for significant agitation or restlessness [2, 3]. *Amanita muscaria*, although not reported in Iran, produces the same syndrome [3].

Orellanus syndrome

Cortinarius species causing orellanus syndrome have been reported from Iran [2-4]. Symptoms are characteristically delayed, usually beginning 24–36 hours after ingestion, with headache, chills, thirst, anorexia, nausea, vomiting, and flank or abdominal pain. Acute kidney injury may develop days to weeks later, accompanied by oliguria, hematuria, leukocyturia, and proteinuria. Treatment is supportive, and severe renal failure may require hemodialysis [2-4].

Paxillus syndrome

Paxillus involutus is the only species reported to cause this syndrome in Iran. Symptoms generally begin within 1–2 hours and may include abdominal bloating, nausea, vomiting, oliguria or anuria, hemoglobinuria, and immune-mediated hemolytic anemia [3, 4].

Rhabdomyolysis syndrome

Tricholoma equestre is the only species reported to cause rhabdomyolysis syndrome in Iran and has been reported from the Nahar Khoran region of Golestan Province. Symptoms usually develop within 24–72 hours and include fatigue, generalized weakness, and myalgia, followed several days later by dark urine associated with rhabdomyolysis and myoglobinuria [3, 4].

Conclusion

Recognition of the toxin-specific syndrome and, particularly, the interval between ingestion and symptom onset can substantially improve the early diagnosis and management of mushroom poisoning in Iran.

Conflicts of Interest

The author reports there are no competing interests to declare.

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References

- [1] Soltaninejad K. Outbreak of mushroom poisoning in Iran: April-May, 2018. *Int J Occup Environ Med*. 2018;9(3):152-6. [DOI: 10.15171/ijoem.2018.1380]
- [2] Goldfrank LR. Mushrooms. In: Nelson LS, Howland MA, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS, editors. *Goldfrank's Toxicologic Emergencies*. 11th ed. New York: McGraw-Hill; 2019. p. 1581-96.
- [3] Sanaei-Zadeh H. *Diagnosis and treatment of acute poisoning*. 4th ed. Shiraz: Moalefan Farhikhteh; 2023. [in Persian]
- [4] Asef MR, Shayan M. *Poisonous mushrooms of Iran*. Tehran: Iranshenasi Publication; 2009. [in Persian] [Link]
- [5] Janatolmakan M, Ganji MR, Ahmadi-Jouybari T, Rezaeian S, Ghowsi M, Khatony A. Demographic, clinical, and laboratory findings of mushroom-poisoned patients in Kermanshah province, west of Iran. *BMC Pharmacol Toxicol*. 2022;23(1):72. [DOI: 10.1186/s40360-022-00614-1]
- [6] Loni E, Sadeghizadeh-Yazdi J, Vatanichian M. The most important poisonous mushrooms identified in Iran. *J Food Qual Hazards Control*. 2025;12(2):82-3. [DOI: 10.18502/jfqhc.12.2.18856]
- [7] Caré W, Bruneau C, Rapior S, Langrand J, Le Roux G, Vodovar D. Amatoxin-containing mushroom poisoning: an update. *Rev Med Interne*. 2024;45(7):423-30. [DOI: 10.1016/j.revmed.2024.07.001]

[10.1016/j.revmed.2023.10.459](https://doi.org/10.1016/j.revmed.2023.10.459)

[8] Ahishali E, Boynuegri B, Ozpolat E, Surmeli H, Dolapcioglu C, Dabak R, et al. Approach to

mushroom intoxication and treatment: can we decrease mortality? Clin Res Hepatol Gastroenterol. 2012;36(2):139-45. [DOI: [10.1016/j.clinre.2011.11.004](https://doi.org/10.1016/j.clinre.2011.11.004)]