CASE REPORT

Acute Pancreatitis Following Mushroom Toxicity: a case report

MOHAMMADREZA MOHAMMADI[1], ARMAN HAKEMI[2,*], ANAHITA ALIZADEH GHAMSARI[2]

[1]School of Medicine, Tehran University of Medical Sciences, Tehran, Iran
[2]Medical Toxicology Research Center, Mashhad University of Medical Sciences, Mashhad, Iran

Abstract

Introduction: Mushroom poisoning remains a global concern, with over 5,000 species of poisonous mushrooms worldwide. *Amanita phalloides* is responsible for approximately 95% of fatal poisonings globally. Although most cases typically present with mild gastrointestinal symptoms, complications such as acute pancreatitis are rare yet critical, warranting a deeper exploration into their implications and management.
Case Presentation: We report a case of acute pancreatitis in a 45-year-old Iranian woman at Emam Reza Hospital, Mashhad, Iran, in November 2024 after possible consumption of Amanita mushrooms approximately 6 hours before her presentation to the medical facility. The patient, who had no significant medical or family history of pancreatic disease, presented with acute upper abdominal pain, nausea and vomiting. Examination and subsequent laboratory tests confirmed acute pancreatitis, characterized by markedly elevated amylase and lipase enzyme levels. She was treated with aggressive hydration and total parenteral nutrition, which resulted in remarkable clinical improvement within 6 days.
Discussion: While it is acknowledged that most cases of mushroom poisoning lead to mild gastroenteritis, the occurrence of acute pancreatitis as a secondary complication following mushroom ingestion remains strikingly underreported. This phenomenon, although rare, has recently garnered attention in clinical discourse, as evidenced by several case studies that have illuminated the relationship between certain mushroom toxins and pancreatic injury. It is vital for clinicians to consider pancreatitis as a potential complication in cases involving mushroom poisoning, thereby allowing for prompt diagnosis and management.
Conclusion: In light of the severe implications associated with acute pancreatitis secondary to mushroom poisoning, further research is imperative to elucidate the precise mechanisms linking mushroom toxins to pancreatic damage. This research would underscore the importance of clinician vigilance during diagnosis, as well as the necessity for public education aimed at preventing mushroom-related illnesses.

Keywords: Mushroom, Poisoning, Acute Pancreatitis, *Amanita phalloides*

How to cite this article: Mohammadi M, Hakemi A, Alizadeh Ghamsari A. Acute Pancreatitis Following Mushroom Toxicity: a case report. Asia Pac J Med Toxicol 2025; 14(1): 16-18.

INTRODUCTION

There are more than 5000 species of poisonous mushrooms worldwide. Of these, about 150 species are responsible for the majority of cases of mushroom poisoning [1]. While the prevalence of mushroom poisoning in Iran remains relatively low compared to Europe and the United States, there has been a recent increase in its incidence [2]. Among poisonous species, *Amanita phalloides*, a highly toxic and dangerous mushroom, is widely recognized and is considered to be responsible for approximately 95% of fatal mushroom poisonings worldwide. Mushroom poisoning causes a wide range of symptoms, from mild gastrointestinal distress to life-threatening organ failure. While most cases involve self-limiting gastrointestinal symptoms such as nausea, vomiting, diarrhea, and abdominal pain, severe complications can occur, including hepatic encephalopathy, renal failure, and even death [1]. Acute pancreatitis following mushroom poisoning is an extremely rare complication that has only been reported in the literature [3]. Our case report focuses on the unique presentation of acute pancreatitis in a patient with mushroom poisoning, emphasizing the need for vigilance and timely intervention to improve prognosis.

CASE REPORT

A 45-year-old Iranian woman with no significant past medical or surgical history presented to the emergency department of Emam Reza Hospital, Mashhad, Iran, in November 2024 with acute, severe upper abdominal pain radiating to the back, nausea, vomiting, and jaundice. She did not mention any constitutional symptoms, such as weight loss, fever, chills, fatigue, or specific cardiovascular, respiratory, neurological, musculoskeletal, hematological, or endocrinological conditions. She reported the possible consumption of Amanita mushrooms based on the described appearance characteristics, in a rural area about 6 hours earlier. Assessments indicated that the patient had undergone routine screening, including lipid profile testing and blood

*Correspondence to: Dr. Arman Hakemi, MD, Fellowship of Clinical toxicology, Emergency Medicine specialist, Medical Toxicology Research Center, Mashhad University of Medical Sciences, Mashhad, Iran.
Email: hakemia971@mums.ac.ir. Tel: +989153464197.

pressure assessment, 2 months prior, with results reported to be within the normal range. There was no history of recent trauma, infection, cigarette smoking, alcohol consumption, or taking any medications including herbal medicines. She also reported no family history of autoimmune disorders including pancreatis-related diseases. The patient was promptly admitted to the hospital and on examination was found to be awake, fully oriented, and cooperative with a heart rate of 78 beats per minute (bpm), blood pressure of 110/75 mmHg in the sitting position, respiratory rate of 16 breaths per minute and temperature of 37.1°C. The patient's body mass index was calculated to be 21.6, with a height of 161 cm and a weight of 56 kg. Abdominal examination showed epigastric tenderness without rebound or guarding. The ultrasound performed in the emergency department showed no abnormalities in the gallbladder, intrahepatic, or extrahepatic bile ducts, including dilatation, stones, or sludge, and the result was reported as normal. Initial laboratory tests were significant for markedly elevated amylase levels (380 U/L) and lipase (310 U/L). Her primary complete blood count, electrolytes, total and direct bilirubin, troponin levels, and the history of normal-range lipid profile tests from two months earlier helped to rule out other causes such as cardiovascular abnormalities and hypertriglyceridemia (Table 1). Liver test results on admission were SGOT of 41 U/L, SGPT of 63 U/L, and ALK-P of 163 U/L. Later spiral abdominopelvic computed tomography was done at the emergency department and showed signs of acute pancreatitis noted in the head with thickening of right pararenal fascia without any signs of perforation or necrosis (Figure 1). The patient was treated with aggressive intravenous hydration to compensate for dehydration caused by vomiting while being maintained on NPO (nothing by mouth) to preserve the pancreas. To eliminate toxins, after gastric lavage via a nasogastric tube, activated charcoal was initiated and continued at a dose of 50 grams every 6 hours. Penicillin G and N-acetylcysteine (NAC) were introduced as effective antidotes to the fungal toxins. NAC was given by continuous intravenous infusion over 24 hours at a total dose of 300 mg/kg, and penicillin G was infused at a dose of 1,000,000 U/kg per day [4,5]. Eight hours after admission, SGPT and SGOT started to rise, reaching 435 U/L and 211 U/L, respectively. After 24 hours of admission, because of the continuous rise in SGPT and SGOT (1200 mg/dl and 830 mg/dl respectively), aggressive intravenous hydration continued, and on the third day of admission, liver aminotransferases finally started to decrease. Also, on the third day of admission, follow-up laboratory tests showed a reduction in amylase (81 U/L) and lipase (28 U/L) (Table 1). Abdominal pain was significantly reduced and the patient was switched to oral intake without complications. The diagnosis of acute pancreatitis caused by mushroom poisoning was made based on a comprehensive evaluation, including a complete medical history, a thorough physical examination, and a diagnostic workup. Key laboratory tests

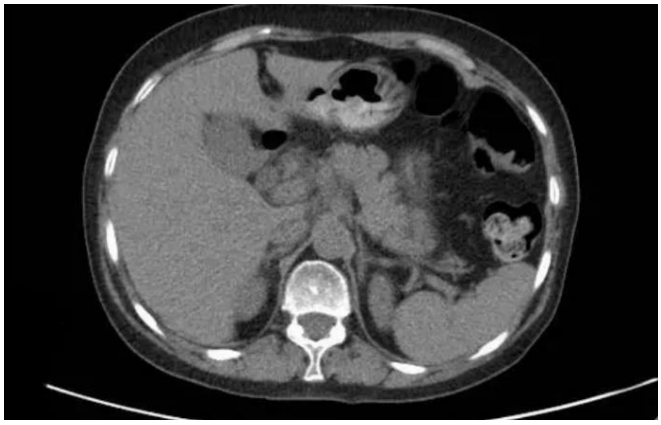

Figure 1. Axial computed tomography image of the patient

Table 1. Patient's laboratory tests

Laboratory Test	Normal Range	Primary Patient Value	8 Hours of Admission	24 Hours of Admission	72 Hours of Admission
Amylase	25 - 125 U/L	380	-	410	81
Lipase	10 - 140 U/L	310	-	360	28
Serum glucose	70 - 110 mg/dL	86	-	92	104
Serum calcium	8.5 - 10.5 mg/dL	9.1	-	8.9	9.2
Serum triglycerides	< 150 mg/dL	74	-	-	71
Serum bilirubin (total)	0.2 - 1.2 mg/dL	0.8	-	-	0.8
Serum bilirubin (direct)	< 0.3 mg/dL	0.2	-	-	0.3
ALT	7 - 56 U/L	63	435	1200	870
AST	5 - 40 U/L	41	211	830	530
ALP	44 -147 U/L	163	160	90	74
WBC count	38% - 52%	10000	-	9100	8500
Hemoglobin	12 – 15 g/dL	13.7	-	13.5	13.3
Platelet count	25 - 125 U/L	189000	-	210000	178000
Troponin	0 – 0.04 ng/mL	0.01	-	-	-

supporting the diagnosis included significantly elevated amylase and lipase levels. These findings, together with the patient's history of previous consumption of probable Amanita mushrooms, confirmed the diagnosis. We followed our patient for six months and there were no further episodes.

DISCUSSION

Mushroom poisoning varies worldwide, displaying regional differences in incidence. Although the incidence of mushroom poisoning in Iran is lower than in Europe, it can still be associated with significant mortality. Confusing poisonous mushrooms with edible ones is the most common cause of poisoning. Therefore, public awareness and education are essential to prevent accidental ingestion. The clinical consequences of mushroom poisoning range from mild, self-limiting gastroenteritis (nausea, vomiting, diarrhea) to severe complications, including fulminant hepatitis, renal failure, and even death. However, elevated pancreatic enzymes with clinical signs of acute pancreatitis in the context of mushroom consumption are rarely documented [6, 7].

Amatoxins disrupt RNA polymerase II, with primary toxicity affecting the gastrointestinal mucosa, liver, and kidneys. Hepatotoxicity leading to liver failure is the most significant effect. Although elevated pancreatic enzymes are rarely reported in association with mushroom ingestion, a definitive mechanism for pancreatic injury remains elusive. A review of the literature found two cases of mushroom poisoning by *Lactarius volemus* in Turkey in 2016, which resulted in acute pancreatitis without severe complications [3, 8, 9].

In another case, a 43-year-old woman with a history of hepatitis B carrier status presented to the emergency department with gastrointestinal symptoms following the consumption of amatoxin-containing mushrooms. The patient was found to have fulminant hepatic failure with significant elevation of pancreatic enzymes consistent with acute pancreatitis. These cases support the notion that mushroom poisoning may lead to pancreatic toxicity, although the mechanism causing such an effect has not been investigated [10, 11].

CONCLUSION

Mushroom poisoning remains a global problem, with *Amanita phalloides* being a major culprit. While most cases involve gastrointestinal symptoms, our report reveals a rare complication: acute pancreatitis. Further research is needed to investigate the association between mushroom poisoning and pancreatitis, particularly to understand the possible underlying mechanisms based on reported related toxins such as amatoxins. Clinicians should also be vigilant and consider pancreatitis in cases of mushroom poisoning. Public awareness and education are essential to prevent accidental ingestion. Timely intervention and appropriate management strategies may improve outcomes.

Ethical approval

This case report was conducted under the ethical standards of the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Ethical approval was granted by the Ethics Committee of Mashhad University of Medical Sciences, Mashhad, Iran.

Availability of supporting data

Due to the sensitive and confidential nature of the data associated with this case report, detailed clinical data, including laboratory results and patient history, are not publicly available. However, de-identified data that support the findings of this study are available from the corresponding author upon reasonable request. Requests for access to these data will be reviewed by the corresponding author and will require compliance with applicable privacy regulations and approval by the institutional review board.

Conflicts of interest: The authors declare that there are no conflicts of interest regarding the research, authorship, and/or publication of this article.

Funding and Support: The authors declare that no funds, grants, or other support were received during the preparation of this manuscript.

REFERENCES

1. Wennig R, Eyer F, Schaper A, Zilker T, Andresen-Streichert H. Mushroom Poisoning. Dtsch Arztebl Int. Oct 16 2020;117(42):701-8. doi:10.3238/arztebl.2020.0701
2. 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. Sep 26 2022;23(1):72. doi:10.1186/s40360-022-00614-1
3. Karahan S, Erden A, Cetinkaya A, Avci D, Ortakoyluoglu AD, Karagoz H, et al. Acute Pancreatitis Caused By Mushroom Poisoning: A Report of Two Cases. J Investig Med High Impact Case Rep. Jan-Mar 2016;4(1):2324709615627474. doi:10.1177/2324709615627474
4. Caré W, Bruneau C, Rapior S, Langrand J, Le Roux G, Vodovar D. [Amatoxin-containing mushroom poisoning: An update]. Rev Med Interne. Nov 8 2023;Syndrome phalloïdien: mise au point. doi:10.1016/j.revmed.2023.10.459
5. Enjalbert F, Rapior S, Nouguier-Soulé J, Guillon S, Amouroux N, Cabot C. Treatment of amatoxin poisoning: 20-year retrospective analysis. J Toxicol Clin Toxicol. 2002;40(6):715-57. doi:10.1081/clt-120014646
6. Soltaninejad K. Outbreak of Mushroom Poisoning in Iran: April-May, 2018. Int J Occup Environ Med. Jul 2018;9(3):152-56. doi:10.15171/ijoem.2018.1380
7. Dadpour B, Tajoddini S, Rajabi M, Afshari R. Mushroom Poisoning in the Northeast of Iran; a Retrospective 6-Year Epidemiologic Study. Emerg (Tehran). 2017;5(1):e23.
8. Barbosa I, Domingues C, Ramos F, Barbosa RM. Analytical methods for amatoxins: A comprehensive review. J Pharm Biomed Anal. Aug 5 2023;232:115421. doi:10.1016/j.jpba.2023.115421
9. Karlson-Stiber C, Persson H. Cytotoxic fungi--an overview. Toxicon. Sep 15 2003;42(4):339-49. doi:10.1016/s0041-0101(03)00238-1
10. Mottram AR, Lazio MP, Bryant SM. Lepiota subincarnata J.E. Lange induced fulminant hepatic failure presenting with pancreatitis. J Med Toxicol. Jun 2010;6(2):155-7. doi:10.1007/s13181-010-0062-1
11. Silakhori S, Dadpour B, Najaf Najafi M. A 3-Year Survey of Mushroom-Poisoned Patients: Clinical Features, Management and Outcomes. Asia Pac J Med Toxicol, 2018; 7(2): 33-37. doi: 10.22038/apjmt.2018.1134