# Emergency room cases of mushroom poisoning

*Abdulkerim Yilmaz, MD, Sinan Gursoy, MD, Osman Varol, MD, Naim Nur, MD, Esin Ozyilkan, MD.*

## ABSTRACT

**Objective:** To describe the pattern of mushroom poisoning in adults.

**Methods:** We investigated patients presenting at the Emergency Room, Medical School Hospital, Cumhuriyet University, Turkey between 1999 and 2003 with complaints of mushroom poisoning. They were kept under observation in the emergency unit and Anesthesia Department intensive care unit. We evaluated the demographical features of the patients, laboratory data, vital signs, and applied treatment and results.

**Results:** Seventy-eight patients aged between 19-72 (40.27±16.09) were studied, 52 were females (67%), and 26 were males (33%). It was established that 90% (70) of the patients had picked wild mushrooms, and 67.9% (53) had done so in springtime. Methods of cooking were as follows: 30 patients (38.4%) grilled the mushrooms, 28 patients (35.8%) stir fried, and 20 patients (25.6%) consumed mushrooms uncooked. Onset of symptoms was mostly (43.5%) within the first hour of consumption. The most common (42.3%) symptoms were gastrointestinal complaints. Upon examining laboratory results, 17.9% revealed variations in hematological parameters, impairment in renal functions in 6 patients (7.6%), hematuria in 10 patients (12.8%) and 15.3% had primarily elevated liver enzymes. Seventy-four patients (97%) completely recovered and were discharged. However, 2 patients (2.8%) who consumed raw mushrooms died due to acute hepatic failure. There was a 20-year-old patient at week 31 of her first pregnancy who had consumed uncooked wild mushrooms. She developed gastrointestinal symptoms and mild loss of consciousness within the first half hour. She received quadruple therapy and completely recovered.

**Conclusion:** The results of the study indicated that the public, as well as the cultivators, were rather unconcerned and uneducated regarding this issue. Efforts aimed at overcoming this problem will decrease the number of mushroom poisoning cases, will help to save on treatment costs, and more importantly, reduce patient mortality.

**Saudi Med J 2006; Vol. 27 (6): 858-861**

Consumption of wild mushrooms and resulting mushroom poisoning cases present a significant health hazard in many countries. Mushroom poisoning cases, which are a public health problem, are frequently reported in Europe, in the United States, and in the Far East.[1-4] There have been frequent cases of mushroom poisoning also in Turkey in recent years.[5-7] A wide variety of wild mushrooms can be encountered in the fields and woods, particularly in spring and autumn. Poisoning caused by mushrooms picked in such areas generally leads to clinical manifestations ranging from gastrointestinal, neurogenic and psychogenic complaints, to hepatic and renal failure and even coma.[6,8,9] While the genus Amanita is the most common poisonous mushroom, lepiota is a less common type. They contain amatoxins, which are peptide toxins. Amatoxins, particularly following the gastrointestinal absorption of amanitin, may induce hepatic and renal damage.[4,10,11] Mushroom poisoning is associated with the type and quantity of mushrooms

From the Department of Internal Medicine (Yilmaz, Ozyilkan), Department of Anesthesia and Reanimation (Gursoy), Department of Emergency Medicine (Varol), and the Department of Public Health (Nur), Medical School, Cumhuriyet University, Sivas, *Turkey*.

Received 9th October 2005. Accepted for publication in final form 21st March 2006.

Address correspondence and reprint request to: Dr. Abdulkerim Yilmaz, Department of Internal Medicine, Medical School, Cumhuriyet University, Sivas 58140, *Turkey*. Tel. +90 (346) 2191300/21192200. Fax. +90 (346) 2191284. E-mail: kerim@cumhuriyet.edu.tr

consumed. Pathogenicity of mushrooms depends on its cyclopeptide toxins. Hepatic and renal damage may ensue depending on the type and quantity of mushroom consumed. Amatoxins are accountable for this occurrence.[4,11] Although severity and clinical presentation are associated with type and quantity of mushroom consumed; time overlapped for first reaching the patient and then to the hospital, efficacy of medical intervention consisting of gastric lavage and antidotes, first while still in the ambulance and later at the hospital, are of critical significance as well.[12] In this study, we performed a 4-year retrospective study of patients who applied to the emergency clinic of the university hospital due to mushroom poisoning.

**Methods.** This present study, retrospectively investigated patients who applied to the emergency room of the Cumhuriyet University Medical School Hospital due to mushroom poisoning between 1999 and 2003. The patient records from the emergency room and intensive care unit were examined. The following data were recorded: patients' age, gender, season and location of mushrooms picked, method of cooking, symptoms, onset time of symptoms, laboratory findings, site of observation, monitorization period, type of treatment administered, and results of therapy. It was not possible to determine the type of mushrooms consumed. Methods of therapy were classified as follows: Dual therapy, such as stomach irrigation and administration of activated charcoal; triple therapy, such as adding atropine to dual therapy, quadruple therapy, such as adding crystallized penicillin to the triple regimen.

**Results.** Seventy-eight patients aged between 19-72 (40.27 ± 16.09) were studied, 52 were females (67%), and 26 were males (33%). Thirty-eight patients (51.3%) were observed in the emergency care unit, and 36 patients (48.6%) were observed in the intensive care unit. The remaining 4 patients did not comply with therapy and left the hospital upon expressing their desire to do so. It was established that 53 patients (67.9%) had consumed the mushrooms in the spring, whereas 25 patients (32.1%) had done so in the autumn. Seventy patients (90%) had picked the mushrooms in the wild (hills, riverbanks, fields), and 10 patients had bought cultivated mushrooms. Methods of cooking were as follows: 30 patients (38.4%) grilled the mushrooms, 28 patients (35.8%) stir fried, and 20 patients (25.6%) consumed mushrooms uncooked. Two of the patients who developed allergic reactions additionally received antihistamines. Of the 74 patients admitted and given therapy, 72 (97%) completely recovered and were discharged (**Table 1**). However, 2 patients who consumed raw mushrooms died due to acute hepatic failure. There was a 20-year-old patient at week 31 of her first pregnancy who had consumed uncooked wild mushrooms. She had developed gastrointestinal symptoms and mild loss of consciousness within the first half hour. She received quadruple therapy and completely recovered.

**Discussion.** Mushroom poisoning presents a major health risk in Turkey. Mushroom picking and consumption is a common habit in the rural areas following rainy seasons. Some studies, although in limited numbers, have stated the frequency and outcomes of mushroom picking in Turkey.[5,6] Epidemiological studies carried out in Europe, USA, and Japan on mushroom poisoning reported data regarding its prevalence and critical outcomes.[2,9,13,14] There are approximately 2000 species of wild mushrooms, with only about 50 of them having toxic consequences.[3] Amatoxins, which account for 90% of fatal mushroom poisonings, have their most significant impact on the liver.[4,15] Amatoxin uptake by hepatocytes is followed by excretion in bile, and then it is reabsorbed in the enterohepatic circulation.

**Table 1 -** Clinical and laboratory results of patients.

| Variables | n | (%) |
|---|---|---|
| ***Symptoms*** | | |
| Gastrointestinal | 33 | (42.3) |
| Loss of consciousness | 23 | (29.4) |
| Cholinergic/anticholinergic | 8 | (10.2) |
| Hallucination/delirium | 8 | (10.2) |
| Coma | 4 | (5.1) |
| Allergic reaction | 2 | (2.6) |
| ***Laboratory results*** | | |
| Hematological | 14 | (17.9) |
| Hepatic | 12 | (15.3) |
| Urinary | 10 | (12.8) |
| Renal | 6 | (7.6) |
| Hypoglycemia | 4 | (5.1) |
| ***Therapy*** | | |
| Dual | 26 | (35.1) |
| Triple | 20 | (27) |
| Quadruple | 28 | (37.8) |
| ***Length of hospital stay (day)*** | | |
| 1 | 27 | (34.6) |
| 2 | 16 | (20.5) |
| 4 | 24 | (30.7) |
| >4 | 7 | (8.9) |
| ***Rejection of therapy*** | 4 | (5.1) |
| ***Outcome*** | | |
| Recovery | 72 | (97.2) |
| Exitus | 2 | (2.8) |

Here, it binds with RNA polymerase II and inhibits messenger RNA structure, revealing its toxic effect on the liver.[16,17] The most critical outcome of mushroom poisoning is acute hepatic failure. The best approach for such patients is liver transplantation. It was reported in a study carried out in Turkey by Unluoglu et al[7] that the death of 4 patients were due to acute hepatic failure, and as the patients were considered to be in terminal stage regarding transport steps, transplantation had not been possible. Pawlowska et al[14] reported liver transplantation for 3 patients following Amanita Phalloides poisoning. Braussard et al[1] reported liver transplantation for some patients who had developed hepatic failure because of mushroom poisoning. We had 2 patients in our study that developed fulminant hepatic failure within 4 days of mushroom consumption. The patients died. Liver transplantation could not be performed. A study carried out in USA reported that 21% of the mushroom poisoning subjects were contaminated with toxic species.[18] Food poisoning associated with mushroom consumption indicates that bacteria and toxins remain in commercial products. This shows that sanitation and hygienic conditions are not complied with in mushroom production.[19-21] Of the cases investigated in our study, 10% had consumed commercially cultivated mushrooms. This result demonstrated a lack of compliance to rules in mushroom production in Turkey as well. Mushroom farms should be subjected to regular inspections; and rigorous measures should be taken to prevent wild mushroom spores entering such zones in order to avoid toxic and contamination risks. A study performed in Japan reported that most mushroom poisoning cases took place in the autumn.[2] Unluoglu et al[7] stated in their study carried out in Turkey, that a great deal of the mushroom poisoning cases occurred in the spring or early summer. Mushroom poisoning in spring consisted of 67.9% of the cases in our study. Mushroom poisoning cases can be reduced by warning the public during rainy seasons, and providing detailed information regarding the issue. A considerable percentage of the cases in our study (25.6%) had consumed raw mushrooms. In fact, 2 of the patients who died had done so. The most common method of cooking were grilling and stir-frying in our study. Although the toxin (amatoxin) included by Genus Amanita, Lepiota ve Galerina could not be destroyed by any cooking methods. The toxin (gyromitrin) including by Gyromitra species is soluble in water and it is volatile. The effective cooking and drying of this fungus for a long time might have decreased the risk of poison.[22] Gastrointestinal complaints have generally been caused by Amanita phalloides, Galerina, Lepiota, Pholiolina, Clitocybe and by Inocybe spp.[23] Gastrointestinal complaints constituted a large part (42.3%) of the symptoms in our study. They appeared within the first hour (43.5%). Unluoglu et al,[24] in their study in which 64 pediatric patients have been examined, found the symptoms of the gastrointestinal system to be approximately 70.6% in all age groups. Pajoum et al[25] also reported that the most common symptoms have arisen from gastrointestinal systems. Similarly, Jaeger et al[26] reported that they have observed gastrointestinal symptoms in all of the 45 patients examined. *Hapalopilus rutilans* and *Gyromitra spp* are the fungi which cause neurological problems,[22,27] while Amanita phalloides induced hepatic encephalopathy gives rise to non-neurological problems.[10,11] In our study, loss of consciousness of various degrees was observed in 23 (29.4%) of the patients. Pajoum et al[25] reported loss of consciousness in 28%, and 4% convulsions of 37 patients aged between 12-65. Unluoglu et al[24] reported 5 cases of confusion, 3 dizziness and 2 coma in 64 pediatric cases. Of the 9 cases Benjamin[28] identified to have been poisoned by Amanita pantherina and Amanita muscaria, 4 had seizures or myoclonic twitching. The most important impact of fungal poisoning is on the liver. Amatoxins of the species of Amanita cause the increase of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels in the serum through inhibiting RNA polymerase II in hepatocytes.[16,17] For example, Pajoumand et al[25] have shown an increase in ALT and AST levels in 17 of 25 patients, and Covic et al[29] have reported increased levels of ALT in all of the 6 patients aged between 7 and 16. In another study examining 6317 cases,[30] an increase in transaminase levels has been reported for 0.5% of the patients. In the present study, 15.3% of the cases were found to have increased levels of this enzyme.

Orellanine containing species of Cortinarius and amatoxin containing species of Amanita can cause renal problems.[22] Leathem et al[31] reported renal failure in 3 of 4 cases with fungal poisoning while Alves et al[32] reported renal dysfunction in only one of the 4 cases. In another study in Switzerland, chronic renal failure was reported for 9 of the 22 patients.[33] In the present study, 7.6% of the patients have been identified with renal dysfunction. While 34.6% of our patients were discharged after 24 hours, 8.9% required a hospitalization period over 4 days. The patients (n=36) who were admitted to the intensive care unit had more serious clinical presentations, and most of them received irrigation, activated charcoal, atropine and penicillin. While 2 of those patients died, the remaining 34 were discharged upon recovery. Mushroom poisoning cases are frequent in our

region, particularly in the spring and the autumn when mushrooms start to grow. Although cases resulting in fatal consequences are few, any symptom regarding food poisoning should be taken seriously in those periods and patients should be monitored. Our results have shown that the public is rather unconcerned and uneducated. Efforts aimed at improving this condition will decrease the number of mushroom poisoning cases, help to save on treatment costs, and more importantly reduce patient mortality.

## References


1. Broussard CN, Aggarwal A, Lacey SR, Post AB, Gramlich T, Henderson JM, et al. Mushroom Poisoning-From Diarrhea to Liver Transplantation. ***AJG*** 2001; 96: 3195-3198.
2. Ishihara Y, Yamaura Y. Descriptive epidemiology of mushroom poisoning in Japan. ***Nippon Eiseigaku Zasshi*** 1992; 46: 1071-1078.
3. Klein AS, Hart J, Brems JJ, Goldstein L, Lewin K, Busuttil RW. Amanita Poisoning: Treatment and the role of Liver Transplantation. ***Am J Med*** 1989; 86: 187-193.
4. Barbato MP. Poisoning From Accidental Ingestion of Mushrooms. ***Med J Aust*** 1993; 158: 842-847.
5. Aji DY, Caliskan S, Nayir A, Mat A, Can B, Yasar Z, et al. Haemoperfusion in Amanita Phalloides Poisoning. ***J Trop Pediatr*** 1995; 41: 371-374.
6. Paydas S, Kocak R, Erturk F, Erken E, Zaksu HS, Gurcay A. Poisoning Due to Amatoxin-Containing Lepiota Species. ***Brit J Clin Prac*** 1990; 44: 450-453.
7. Unluoglu I, Tayfur M. Mushroom Poisoning: An analysis of the data between 1996 and 2000. ***Eur J Emerg Med*** 2003; 10: 23-26.
8. Anonymous. Editorial, Mushroom Poisoning. ***Lancet*** 1980; 2: 351-352.
9. Perez-Moreno J, Ferrera-Cerrato R. A Review of Mushroom Poisoning in Mexico. ***Food Addit Contam*** 1995; 12: 355-360.
10. Ramirez P, Parrilla P, Sanchez Bueno F, Robles R, Pons JA, Bixquert V, et al. Fulminant Hepatic Failure After Lepiota Mushroom Poisoning. ***J Hepatol*** 1993; 19: 51-54.
11. Wieland T. Poisonous Principles of Mushroom of the Genus Amanita. Four-carbon amines acting on the central nervous system and cell-destroying cyclic peptides are produced. ***Science*** 1968; 159: 946-952.
12. Timar L, Czeizel AE. Birth Weight and Congenital Anomalies Following Poisonous Mushroom Intoxication during Pregnancy. ***Reproduc Toxicol*** 1997; 11: 861-866.
13. Trestrail JH. Mushroom Poisoning in the United States - An Analysis of 1989 United States Poison Center Data. ***J Toxicol Clin Toxicol*** 1991; 29: 459-465.
14. Pawlowska J, Pawlak J, Kaminski A, Jankowska I, Hevelke P, Teisseyre M, et al. Liver Transplantation in Three Family Members After Amanita Phalloides Mushroom Poisoning. ***Transplant Proc*** 2002; 34: 3313-3314.
15. Mullins ME, Horowitz BZ. The Futility of Haemoperfusion and Hemodialysis in Amanita Phalloides Poisoning. ***Vet Hum Toxicol*** 2000; 42: 90-91.
16. Cole FM. A Puppy Death and Amanita Phalloides. ***Aust Vet J*** 1993; 70: 271-272.
17. Kroncke KD, Fricert G, Meier PJ, Gerok W, Wieland T, Kurz G. Alpha-Amanitin into Hepatocytes. ***J Biol Chem*** 1986; 261: 12562-12567.
18. Gecan JS, Cichowicz SM. Toxic Mushroom Contamination of Wild Mushrooms in Commercial Distribution. ***J Food Protec*** 1993; 56: 730-734.
19. Martin ST, Beelman RR. Growth and Enterotoxin Production of Staphylococcus aureus in Fresh Packaged Mushrooms (Agaricus bisporus). ***J Food Protec*** 1996; 59: 819-826.
20. Anderson JE, Beelman RR, Doores S. Persistance of serological and Biological Activities of Staphylococcal Enterotoxin A in Canned Mushrooms. ***J Food Protec*** 1996; 52: 1292-1299.
21. Karlson-Stiber C, Persson H. Cytotoxic fungi – an overview. ***Toxicon*** 2003; 42: 339-349.
22. Koppel C. Clinical symptomatology and management of mushroom poisoning. ***Toxicon*** 1993; 31: 1513-1540.
23. Unluoglu I, Cevik AA, Bor Ozcan, Tayfur M, Sahin A. Mushroom poisoning in children in central anatolia. ***Vet Hum Toxicol*** 2004; 46: 134-137.
24. Pajoumand A, Shadnia S, Efricheh H, Mandegary A, Hassanian-Moghadam H, Abdollahi M. A retrospective study of mushroom poisoning in Iran. ***Hum Exp Toxicol*** 2005; 24: 609-613.
25. Jaeger A, Jehl F, Flesch F, Sauder P, Kopferschmitt J. Kinetics of amatoxins in human poisoning: therapeutic implications. ***J Toxicol Clin Toxicol*** 1993; 31: 63-80.
26. Saviuc P, Flesch F. Acute higher fungi mushroom poisoning and its treatment. ***Presse Med*** 2003; 32: 1427-1435.
27. Benjamin DR. Mushroom poisoning in infants and children: the Amanita pantherina /muscaria group. ***J Toxicol Clin Toxicol*** 1992; 30: 13-22.
28. Covic A, Goldsmith DJ, Gusbeth-Tatomir P, Volovat C, Dimitriu AG, Cristogel F, et al. Successful use of molecular absorbent regenerating system (MARS) dialysis for the treatment of fulminant hepatic failure in children accidentally poisoned by toxic mushroom ingestion. ***Liver Int*** 2003; 23: 21-27.
29. Nordt SP. 5-Year analysis of mushroom exposures in California. ***West J Med*** 2000; 173: 314-317.
30. Leathem AM, Purssell RA, Chan VR, Kroeger PD. Renal failure caused by mushroom poisoning. ***J Toxicol Clin Toxicol*** 1997; 35: 67-75.
31. Alves A, Ferreira MG, Paulo J, Franc A¸ Carvalho A. Mushroom poisoning with Amanita phalloides - a report of four cases. ***Eur J Intern Med*** 2001; 12: 64-66.
32. Holmdahl J, Blohme I. Renal transplantation after Cortinarius speciosissimus poisoning. ***Nephrol Dial Transplant*** 1995; 10: 1920-1922.