Demographic and Clinical Characteristics of Mushroom Poisonings Presenting to Emergency Department

Acil Servise Başvuran Mantar Zehirlenmesi Olgularının Demografik ve Klinik Özellikleri

Mantar Zehirlenmeleri / Mushroom Poisonings

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Özet

Amaç: Mantar zehirlenmelerinin geniş klinik spektrumları ve ölümcül potansiyelleri açısından taşıdıkları önemin vurgulanması. Gereç ve Yöntem: İç Anadolu Bölgesinde bulunan iki büyük hastanenin acil servislerine 2010-2012 yılları arasında başvuran 135 mantar zehirlenme olgusu retrospektif olarak değerlendirildi. Bulgular: 135 hastanın 121'i (%89.6) erişkin, 14'ü pediyatrik yaş grubundaydı. Ortalama yaş 41.5±18.4 idi. Hastaların 126'sı yabani mantar yeme sonrasında zehirlenmişti. En sık gözlenen semptomlar bulantı, karın ağrısı, halsizlik ve kusmaydı. Hastalar en sık mantar yenmesinden sonraki ilk 6 saat içinde başvurdu. Olguların %50'si hospitalize edildi. Mortalite 3 olguda görüldü. Tartışma: Mantar zehirlenmesi halen önemli bir halk sağlığı sorunu olmaya devam etmektedir. Semptomların çeşitliliği ve non-spesifik olması nedeniyle tanısı zordur ve özellikle yağışlı havalarda acil serviste çalışan doktorlarının akıllarının bir köşesinde bulunmalıdır.

Anahtar Kelimeler

Mantar; Zehirlenme; Acil Servis

Abstract

Aim: To emphasize the significance of mushroom poisonings in terms of their wide clinical spectrum and potentially lethal feature. Material and Method: In this retrospective study 135 cases of mushroom poisoning admitted to emergency departments of two major hospitals in Central Anatolian Region between 2010 and 2012 were evaluated. Results: 121 (89.6%) of 135 patients were adult and 14 were in pediatric age group. The mean age was 41.5±18.4. A hundred twenty-six of patients had poisoned after consuming wild mushrooms. The most frequent symptoms were nause, abdominal pain, fatigue and vomiting. Patients were admitted most commonly within the 6 hours after ingestion. Fifty percent of patients were hospitalized. Mortality was seen in 3 patients. Discussion: Mushroom poisoning still remains as an important public health problem. Due to the diversity and non-specificity of symptoms diagnosis is confounding and should be kept in mind of emergency physicians especially in rainy weather.

Keywords

Mushroom; Poisoning; Emergency Department

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Introduction

Mushrooms are ideal nutritional sources that are particularly rich in protein and commonly consumed by the populations of lower socioeconomic status.[1] It is estimated that approximately 5000 mushroom species are found on Earth, of which only 200 to 300 are safe to consume. It is well-known that 50 to 100 mushroom species are toxic to humans while 10 are lethal.[2,3]

Mushroom poisoning commonly occurs by inadvertently consuming toxic species because of inability to distinguish them from non-toxic species. In addition, intentional consumption of toxic species for suicidal, homicidal purposes, or consuming some species containing psychoactive toxins may also lead to intoxications.[4,5,6]

Depending on the consumed species, consumed amount, season in which mushroom takes place, geographical location, cooking method, and individual's response mushroom intoxication is usually characterized by temporary gastrointestinal, allergic and central nervous system (CNS) symptoms. Ninety to ninety-five per cent of all lethal mushroom poisonings are caused by the Amanita species containing amatoxin causing hepatic and/or renal failure.[7,8,9]

The time period from consumption to onset of symptoms is of utmost importance since this period is major determinant of both therapeutic approach and prognosis.[9]

In this study we aimed to present demographic, clinical and prognostic features of patients presenting with suspected mushroom poisoning to emergency departments (ED) of 2 major Training and Research Hospitals in Central Anatolian Region.

Material and Method

The present study retrospectively examined a total of 135 patients who presented to Kayseri Training and Research Hospital and Ankara Numune Training and Research Hospital with suspected mushroom poisoning between 2010 and 2012. The patients were evaluated in terms of demographic features, seasonal factors, symptoms and time to symptom onset, laboratory data, duration of hospital stay, applied treatment, response to treatment and prognosis.

Data were presented as the mean (SD) or percentage. All data were analyzed with IBM SPSS software, version 19.0. Statistical comparisons were carried out with Chi-Square test for categorical data and Mann Whitney U test for independent continuous variables. A p value less than 0.05 was considered to be statistically significant.

Results

A hundered and thirty-five patients were enrolled in the study. Of these, 121 (89.6%) were adult and 14 were in pediatric age group. Age range of patients was between 6 months and 82 years (41.5±18.4). In point of gender distribution 90 (66.7%) of patients were female and 45 (33.3%) were male. A hundred twenty-six (94.1%) patients had consumed wild mushrooms and 8 (5.9%) had bought cultivated mushrooms. All the other demographic characteristics of patients were listed in Table 1. Majority of the mushroom poisoning occured during the months of october (62.2%) and november (26.7%).

A hundred and three (76.3%) of patients were admitted to ED most commonly with the complaints of nause, abdominal pain, fatigue and vomiting within the 6 hours after ingestion of mushrooms. The remainder of thirty-two (23.7%) presented after 6 hours of ingestion and also had most commonly gastrointestinal symptoms. (Table 2). A patient was admitted with muscarinic symptoms such as hypotension, bradycardia, sweating, salivation and respiratory stress in the first 6 hours of ingestion. Rates of hospitalization of patients admitted within the 6 hours and after the 6 hours of mushroom ingestion were 35.9% and 50%, respectively. The difference between the two groups was statistically significant (P = .017).

Table 1. Demographic characteristics of patients

		n (%)
Age	Pediatric	14 (10.4)
	Adult	121 (89.6)
Geographical region	Urban	68 (50.4)
	Rural	67 (49.6)
Gender	Erkek	45 (33.3)
	Kadın	91 (66.7)
Mushroom species	Wild	125 (94.1)
	Cultured	8 (5.9)

Table 2. Symptoms of patients and their frequency

	First 6 hours of ingestion	After 6 hours of ingestion	Total n (%)	*p
Nause	93	26	119 (88.1)	0.285
Abdominal pain	69	20	89 (65.9)	0.640
Fatigue	62	22	84 (62.2)	0.383
Vomiting	58	13	71 (52.6)	0.121
Headache	26	9	35 (25.9)	0.745
Dizziness	22	10	32 (23.7)	0.250
Loss of consciousness	11	10	21 (15.6)	0.012
Diarrhea	14	5	19 (14.1)	1.000
Seizure	-	2	2 (1.5)	0.086

*p was calculated with Chi-square

The most common laboratory findings at the initial presentation of patients were leukocytosis (27.9%) and hyperglycemia (27.2%). Twenty-two (16.2%) of patients had aspartate transaminase (AST) and alanine transaminase (ALT) elevation. Other labaratory findings of patients were listed in Table 3. Potassium values were found to be higher in patients who were presented to emergency department after the 6 hours of mushroom ingestion and this relationship was found to be statistically significant (Z = -2440, P = .015).

Eighty-one (59.6%) of patients were discharged after observation in the ED, 50 (36.8%) were hospitalized and one of the patients was referred to another hospital for hepatic transplantation. Patients observed in the emergency department had conventional treatment of fluid resuscitation, stomach irrigation and active charcole admimistration. Fourty-two (84%) of hospitalized patients were treated with penisilin G in addition to conventional therapy.

Mortality was seen in three cases and mortality rate was 2.2%. One of them was the patient referred to another hospital for hepatic transplantation. The second one was brought to our ED by the emergency medical service (112) in an unconscious

Table 3. Labaratory findings of patients

	n (%)	First 6 hours of ingestion	After 6 hours of ingestion	Reference range	p*
Leukocytosis	38 (27.9)	25	13	4400-11300	0.680
Prolongation of PT	6 (4.4)	5	1	11.5-14.5	0.680
Prolongation of aPTT	3 (2.2)	2	1	24-36	0.693
Elevation of AST	22 (16.2)	14	8	>35	0.128
Elevation of ALT	22 (16.2)	15	7	>35	0.330
Elevation of BUN	5 (3.7)	2	3	10-50	0.053
Elevation of creatinine	8 (5.9)	5	3	0.7-1.2	0.495
Elevation of bilirubin	16 (11.8)	11	5	>0.9	0.451
Hyperkalemia	4 (2.9)	1	3	3.5-5.1	0.015
Hyperglicemia	37 (27.2)	31	6	70-109	0.210

*p was calculated with Mann-Whitney-U test

state and was cardiovascular arrested during her admission. The 57-year-old patient without any known comorbid disorder did not answer the cardiopulmonary resuscitation (CPR) and accepted exitus 40 minutes later. Her initial labaratory findings were as follows; urea: 272 mg/dL, creatinine: 10 mg/dL, ALT: 45 U/L, AST: 71 U/L, amilase: 493 U/L, creatine kinase (CK): 2448 U/L, CK-MB: 214 U/L, laktate dehydrogenase (LDH): 1166 U/L, potassium: 8.23 mmol/L, pHST: 6.73, pO2: 68 mmHg, pCO2: 28 mmHg, consentration HCO3 (cHCO3): 4.3 mmol/L, baz excess (BE): -29.9 mmol/L, white blood cell (WBC): 31,27 10*3/µ, minimum corpuscular hemoglobin consantration (MCHC): 30.2, mean platelet volume (MPV): 13.6 fl, platelet redistribution width (PDW): 22.9%, protrombin time (PT): 17.8 sc and international normalized ratio (INR): 1.47.

The third one was the son of the second patient and admitted to our ED with the complaint of severe diarrhea 8 hours after his mother. 30-year-old patient was hypotensive (arteriel blood pressure: 80/50 mmHg) and tachycardic (110 beats/minute) in his presentation and his physical examination was otherwise normal. His labaratory findings were as follows: urea: 87 mg/dL, , WBC: 17,58 10*3/µ, MCHC: 31.1, red cell distrubution width (RDW): 15.7%, pHST: 7.21, cHCO3: 16.2 mmol/L, BE: -11.6 mmol/L. He was taken under observation in the ED and symptomatic fluid therapy was started. Sudden cariovascular collapse was developed at his fifth hour of observation. His labaratory findings during CPR were as follows: urea: 97 mg/dL, creatinine: 1.6 mg/dL, potassium: 5.3 mmol/L, CK-MB: 48 U/L, WBC: 14,35 10*3/µ, pHST: 7.10, cHCO3: 16.7 mmol/L, BE: -16.7 mmol/L. He was unresponsive to CPR and accepted exitus.

Discussion

It is estimated that approximately 5000 mushroom species are found on Earth but only 200 to 300 of them are safe to consume. It is well-known that 50 to 100 mushroom species are toxic to humans while 10 are lethal.[2,3] Türkiye possesses a rich mushroom flora as a result of favorable ecological conditions. About a total of 115 mushroom genera and approximately 280 species related to these genera are present in Türkiye and about 20 are toxic among these.[5,7,10]

Cases of mushroom poisoning usually occur during spring or autumn when rainfall is abundant. It has been reported that intoxications become frequent between July and October in USA.[3] A study from Japan reported that number of intoxications increase during fall.[11] Ergüven et al.[12] from our country reported that intoxications more commonly occur during autumn, while Ünlüoğlu et al.[7] reported that most cases occur during spring and early summer. A study from our region demonstrated that among patients who presented to emergency departments with mushroom poisoning 58.8% did so during summer, 28.6% during spring, and 7.5% during winter.[4] In our study, on the other hand, majority of the intoxications (88.3%) took place during autumn (Figure 1).

Mushroom intoxications can occur from consumption of wild mushrooms gathered from nature as well as from eating culture mushrooms in which

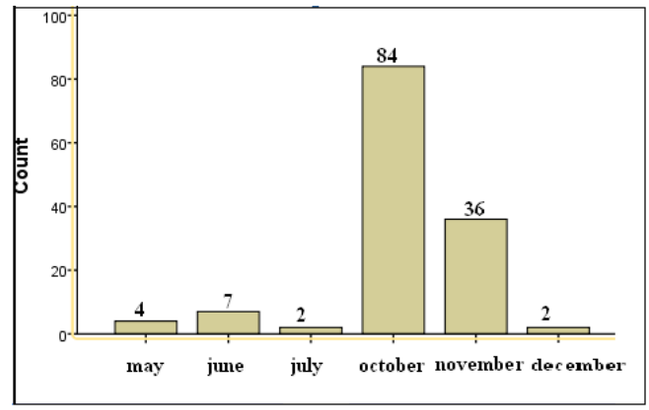


Figure 1. Distrubution of mushroom poisoning according to months

proteins are degraded into ptomaine and bacterial proliferation occurs as a result of prolonged storage. The latter form is characterized by a mild clinical picture predominantly with gastrointestinal symptoms.[10,13] In our study intoxication occurred as a result of consumption of wild mushrooms in 127 (94.1%) patients and culture mushrooms in 8 (5.9%) patients (Table 1).

Mushroom poisoning is diagnosed with identification and toxicological analysis of the mushroom species and, more importantly, the clinical picture of the patient. However, the species and toxins cannot be determined in more than 95% and 90% of the cases, respectively. Thus, patient history is of paramount importance. Although the history of mushroom consumption may be obscured due to the delay between the consumption and symptom onset in most severe intoxications and not all patients experience similar toxic symptoms in a particular mushroom species, the clinician should first link the clinical presentation with mushroom ingestion.[2,14] It is very important to remember that time from mushroom ingestion to symptom onset may be delayed. This point has been reported in many reports on mushroom poisoning as an important predictor for a severe or fatal outcome in acute poisoning.[9]

Depending on the time from mushroom consumption to symptom onset, mushroom intoxications were classified as early (< 6 hours) and late (6-24 hours). Symptoms emerging at the early period suggest ingestion of low toxicity mushrooms, while late-onset symptoms are indicative of intoxication with mushrooms having a more severe clinical course characterized by liver and renal failure, or even death.[4,5,7,15] A mushroom species may con-

tain a single toxin or multiple toxins of varying amounts.[14] In our study a late symptom onset in the younger of the two patients in whom CPR was performed, a fatal course of the disease in both patients, finding out from their relatives that both patients were in a low socioeconomic level and consumed mushrooms indicate the importance of the relationship between the diagnosis of mushroom poisoning with patient history and clinical presentation and the importance of time to symptom onset for prediction of prognosis for clinicians as emphasized in the literature.

Despite a variable clinical picture depending on the mushroom's particular toxin content, gastrointestinal symptoms commonly arise irrespective of mushroom type. Previous studies have shown that patients most commonly present with early-onset gastrointestinal syndrome following a mushroom ingestion.[2,7,16] Alsoin our study the patients presented mostly within the first 6 hours and most commonly with nausea, vomiting, and abdominal pain (Table 2).

In mushroom poisoning cases laboratory tests should be ordered depending on clinical signs and the type of a particular toxin (hepatotoxic, nephrotoxic, rhabdomyolitic, hemolytic, etc.). Serum metabolic profile (creatine phosphokinase, electrolytes, glucose, renal and hepatic function tests), complete blood count, prothrombin time (PT), and activated partial thromboplastin time (aPTT) usually suffice in most cases.[17,18] The most common laboratory abnormalities are abnormalities in hematological parameters and transaminase elevations.[4,5] In our study leukocytosis, hyperglycemia, and transaminase elevation were the most common laboratory abnormalities (Table 3). Serum creatine kinase levels may also increase. This condition should suggest rhabdomyolytic mushrooms (Tricholoma equestre and Russula subnigricans). Since there is no evidence to suggest any intentional intoxication, rhabdomyolysis should be considered as a result of mushroom intoxication.[19,20,21] We also determined that those levels were high in our 2 patients in whom CPR was performed.

The main objects in management of patients with mushroom poisoning include decontamination, fluid replacement, elimination of the ingested toxic content, application of some specific antidotes against certain types of mushroom toxins, and liver transplantation in case of fulminant liver failure.[22] Seizure activities as a result of intoxications with mushroom species containing Gyromitrin are treated with a standard anticonvulsant agent (like lorazepam) and additional pyridoxine; methemoglobinemia due to the same toxin with methylene blue; intoxications secondary to mushroom species containing muscarine are treated with atropine and glycopyrrolate; intoxications due to species containing amatoxin are treated with penicillin G, N-acetylcysteine, and silibinin.[20] In our study, 1 patient who presented with muscarinic symptoms was administered atropine at a dose of 0.02 mg/kg as an antidote and the patient's symptoms were improved. Patients were monitored at the emergency department and they received aggressive fluid therapy followed by decontamination with active charcoal. Penicillin G treatment was given to patients who were admitted with suspected amatoxin intoxication.

Mushroom poisoning still remains as an important public health problem. It is a common cause of emergency department presentations during months having abundant rainfall. The diagnosis is challenging owing to diversity and non-specificity of symptoms. In conditions when analyses towards toxins and certain species are not available it is of paramount importance to suspect a mushroom intoxication by combining information from patient history, symptoms, and time from symptom onset.

Competing interests

The authors declare that they have no competing interests.

References

1. Eren Ş. H, Oğuztürk H, Korkmaz İ, Varol O. 2004 Yılı içerisinde Cumhuriyet Üniversitesi Tip Fakültesi Acil Tip Anabilim Dali'na başvuran mantar zehirlenmesi vakalarinin değerlendirilmesi. CÜ Tıp Fakültesi Dergisi 2005;27(1):15–8.
2. Chew KS, Mohidin MA, Ahmad MZ, Nizam TH, Kamauzaman T. Early onset muscarinic manifestations after wild mushroom ingestion. Int J Emerg Med 2008;1:205–8.
3. Kintziger K. W, Mulay P, Watkins S, Schauben J, Weisman R, Lewis-Younger C et al. Wild mushroom exposures in Florida, 2003–2007. Public Health Reports 2011;126(6):844–52.
4. Eren ŞH, Demirel Y, Ugurlu S, Korkmaz İ, Aktas C, Kukul Güven FM. Mushroom poisoning retrospective analysis of 294 cases. Clinics 2010;65(5):41-6.
5. Yelken B, Erkan A. Mantar zehirlenmeleri: 104 olgunun değerlendirmesi. Journal of the Turkish Society of Intensive Care 2009;7(1):12-6.
6. Lima ADL, Costa R, Fortes M, Garbi Novaes RC, Percário S. Poisonous mushrooms; a review of the most common intoxications. Nutr Hosp 2012;27(2):402-8.
7. Ünlüoğlu I, Tayfur M. Mushroom poisoning: an analysis of the data between 1996 and 2000. European Journal of Emergency Medicine 2003;10:23–6.
8. Brayer AF, Schneider SM, Cevik AA. Chapter 214. Mushroom Poisoning. In: Tintinalli JE, Kelen GD, Stapczynski JS, eds. Tintinalli's Emergency Medicine: A Comprehensive Study Guide. 7th ed. New York: McGraw-Hill; 2011.
9. Schenk-Jaeger KM, Rauber-Lüthy C, Bodmer M, Kupferschmidt H, Kullak-Ublick GA, Ceschi A. Mushroom poisoning: A study on circumstances of exposure and patterns of toxicity. European Journal of Internal Medicine 2012;23:85–91.
10. Akpolat Ergin N. Mantar zehirlenmeleri ve tedavide genel yaklaşım. Türk Hij Den Biyol Derg 2000;57(2):109–18.
11. Ishihara Y, Yamaura Y. Descriptive epidemiology of mushroom poisoning in Japan. Nippon Eiseigaku Zasshi 1992;46:1071-8.
12. Ergüven M, Caki S, Deveci M. Mantar zehirlenmesi: 28 vakanın değerlendirilmesi. Turkish Pediatric Journal 2004;47:249-53.
13. Akdur O, Durukan P, Özkan S, Avşaroğulları L, İkizceli İ. Evaluation of mushroom poisoning patients admitted to the emergency department in a day. Erciyes Medical Journal 2007;29(5):381-6.
14. Berger KJ, Guss DA. Mycotoxıns revısited.The Journal of Emergency Medicine 2005;28(1):53–62.
15. Koyuncu F, Girişgin S, Cander B. Aynı mantar zehirlenmesinde iki farkli klinik (iki olgu sunumu). AKATOS 2011;2(3):20-2.
16. Ecevıt Ç, Hızarcıoglu M, Akgün Gerçek P, Gerçek H, Kayserili E, Gülez P ve ark. Acil servise başvuran mantar zehirlenmelerinin retrospektif olarak incelenmesi. ADÜ Tıp Fakültesi Dergisi 2004;5(3):11–4.
17. Diaz JH. Syndromic diagnosis and management of confirmed mushroom poisonings. Crit Care Med 2005;33(2):427-36.
18. Levine M, Ruha AM, Graeme K, Brooks DE, Canning J, Curry SC. Toxicology in the ICU: Part 3: Natural Toxins 2011;140(5):1357-70.
19. Chodorowski Z, Waldman W, Sein Anand J. Acute poisoning with Tricholoma equestre. Przegl Lek 2002;59(4-5):386-7.
20. Anand JS, Chwaluk P, Sut M. Acute poisoning with Tricholoma equestre. Przegl Lek 2009;66(6):339-40.
21. Bedry R, Audrımont IB, Deffıeux G, Creppy EE, Pomies JP, Ragnaud JM et al. Wild-Mushroom intoxication as a cause of rhabdomyolysis. N Engl J Med 2001;345(11):798-802.
22. Santi L, Maggioli C, Mastroroberto M, Tufoni M, Napoli L, Caraceni P. Acute liver failure caused by amanita phalloides poisoning. International Journal of Hepatology 2012;2012;487480; doi:10.1155/2012/487480.