Clinical, Laboratory and Prognosis Evaluations of Our Mushroom Poisoning Cases

Mantar Zehirlenmesi Olgularımızın Klinik, Laboratuvar ve Prognostik Değerlendirilmeleri

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ABSTRACT

OBJECTIVE: Mushroom poisoning may cause diverse clinical presentations ranging from mild gastrointestinal symptoms to fulminant hepatic failure requiring liver transplantation. It may lead to high mortality if not intervened. Toxic wild mushrooms usually grow up in spring and autumn and poisoning by these mushrooms occur mostly in these seasons. The aim of this study was to evaluate demographics, clinical features and prognosis in a large mushroom poisoning case series.

MATERIAL and METHODS: In this study, the demographics, clinical and laboratory findings, treatment methods and prognosis of 84 mushroom poisoning cases were evaluated retrospectively from their medical records.

RESULTS: The mean age of the 84 cases (52 women, 32 men) was 39.8 ± 13.4 years. The main complaints upon admission were recorded as nausea-vomiting (80%), diarrhea (64%), abdominal pain (40%), and dizziness (20%). Twenty-five patients were applied hemoperfusion due to renal and hepatic failure. A case died of renal and hepatic failure. The mean of hospitalization was 6.3 ± 5.6 days. Alanine aminotransferase (ALT), aspartate aminotransferase (AST), prothrombin time (PT), INR, and urea values had decreased significantly at the time of hospital discharge compared to baseline values ($P < 0.001$); however, no statistically significant difference existed between baseline and discharge creatinine levels ($P > 0.05$).

CONCLUSION: In our study, it was observed that early hemoperfusion provided better prognosis by enhancing the efficacy of the treatment. However, the best method to reduce the mortality is to enlighten the community about the risks of mushroom poisonings.

KEY WORDS: Mushroom poisoning, Hemoperfusion, Clinical prognosis

ÖZ

AMAÇ: Mantar zehirlenmesi hafif gastrointestinal semptomlardan karaciğer transplantasyonu gerektiren fulminan hepatik yetmezliğe uzanan farklı klinik sergilenmeler ortaya koyabilir. Eğer müdahale edilmezse yüksek mortaliteye neden olabilir. Zehirli yabani mantarlar genellikle ilkbahar ve sonbaharda yetişir ve bu mantar zehirlenmeleri daha çok bu mevsimlerde olur. Bu çalışmanın amacı, geniş bir mantar zehirlenmesi olgu serisinde demografik, klinik özellikleri ve prognozu değerlendirmekti.

GEREÇ ve YÖNTEMLER: Bu çalışmada, 84 mantar zehirlenmesi olgusunun demografik, klinik ve laboratuvar bulguları, tedavi yöntemleri ve prognozu tıbbi kayıtlardan retrospektif olarak değerlendirildi.

BULGULAR: 84 olgunun (52 kadın, 32 erkek) ortalama yaşı 39,8 ± 13,4 yıl idi. Başvuru sırasında olan ana şikayetler bulantı-kusma (%80), diyare (%64), karın ağrısı (%40), ve sersemlik (%20) olarak kaydedildi. 25 hastaya renal ve hepatik yetmezlik nedeniyle hemoperfüzyon uygulandı. Bir vaka renal ve hepatik yetmezlik nedeniyle öldü. Ortalama hastaneye yatış süresi 6,3 ± 5,6 gündü. Alanin aminotransferaz (ALT), aspartat aminotransferaz (AST), protrombin zamanı (PT), INR ve üre değerleri hastaneden taburcu esnasında bazal değerlere göre anlamlı olarak azalmıştı. ($P < 0,001$); fakat bazal ve taburculuk kreatin değerleri arasında anlamlı istatistiksel farklılık mevcut değildi ($P > 0,05$).

SONUÇ: Bizim çalışmamızda, erken hemoperfüzyonun tedavi etkinliğini arttırarak daha iyi prognoz sunduğu gözlemlendi. Fakat mortaliteyi azaltmak için en iyi yöntem mantar zehirlenmesinin riskleri ile ilgili toplumu aydınlatmaktır.

ANAHTAR SÖZCÜKLER: Mantar zehirlenmesi, Hemoperfüzyon, Klinik prognoz

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INTRODUCTION

Other than nonpoisonous mushrooms which are used as food (*Cantharellus cibarius*, *Lactarius deliciosus*), there are 30-100 poisonous mushroom species that may be highly toxic to humans (1). Fatal mushroom poisonings occur with the ingestion of *Amanita* (*Amanita phalloides, Amanita verna, Amanita ocreta*) and *Gallerina* species (2). *Amanita* species mushroom (*Amanita phalloides*) poisonings are seen relatively more common. Cooking- and freezing-resistant amatoxins (alpha- and beta-amanitin) produced by *Amanita phalloides*, are responsible for 90% of the deaths due to severe hepatic, renal, and/or central nervous system injury (3,4). Poisoning symptoms are correlated with the mushroom type and amount ingested; while all age groups may be affected. Clinical findings of mushroom poisoning may be in a wide range from gastrointestinal to neurogenic and psychogenic symptoms and signs; and may lead to hepatic failure, renal failure, coma, and/or death (5,6). The aim of this study is to evaluate demographics, clinical features and prognosis in a large mushroom poisoning case series admitted to the internal medicine clinics of a State Hospital in Istanbul.

MATERIAL and METHODS

In our study, the patients admitted to the Internal Medicine Clinics of Istanbul Göztepe Training and Research Hospital between 01.01.2004 and 01.01.2007, were evaluated retrospectively. Age, gender, seasonal distribution of the admissions, symptoms, laboratory findings (alanine aminotransferase (ALT), aspartate aminotransferase (AST), prothrombin time (PT), INR, urea, creatinine), duration of hospitalization, treatments applied, and prognosis of the cases were evaluated. The normal serum levels of laboratory parameters were considered as follow: ALT < 40 IU/L, AST < 40 IU/L, urea < 35 mg/dL, creatinine < 0.95 mg/dL, INR < 1.2 and PT < 13 second. The patients were divided into three groups according to the treatment approaches applied:

Group 1 - patients who had received Penicillin G;

Group 2 - patients who had received symptomatic therapy; and

Group 3 - patients who had received penicillin G together with hemoperfusion.

Statistical analyses

For statistical analysis, SPSS (Statistical Package for Social Sciences) for Windows 15.0 was used. In statistical evaluations, besides descriptive (mean, standard deviation, frequency) statistics methods, comparison between groups for variables that show normal distribution was done by one-way ANOVA test; and the *post hoc* Tukey HSD test. The comparison of the parameters that show non-normal distribution in more than two groups and for determination of the group that due to difference were done by the Kruskal-Wallis and Mann-Whitney U test, respectively. For evaluations of repeated measures that show normal distribution, variance analysis for repeated measures and paired sample t test for determination the measure that due to difference were used. For evaluations of repeated measures that show non-normal distribution, Friedman test and Wilcoxon signed rank test was used when appropriate. Categorical variables were compared by Chi-Square test. The relationship between laboratory values, duration of hospitalization and treatments applied were analyzed. The results were evaluated in 95% confidence interval, at $P < 0.05$ significance level.

RESULTS

Demographics and initial complaints at admission

The mean age of total 84 cases (52 females (61.9%) and 32 males (38.1%)) was 39.8 ± 13.4 years. It was determined that 73.8% of the cases had been admitted in the autumn, and 16.7% in the winter.

The major symptoms at admission were nausea-vomiting (80%), diarrhea (64%), abdominal pain (40%), and dizziness (20%). The mean hospitalization duration was 6.3 ± 5.6 days.

Laboratory findings

When laboratory values of cases were evaluated, ALT values in 47 (56%), AST in 54 (64.3%), PT in 38 (45.2%), INR in 35 (41.7%), urea in 33 (39.3%), and creatinine in 32 (38.1%) were higher than upper normal limits. The hepatic and renal dysfunction and coagulation defects were observed 64.5% (n= 54), 39.3% (n=33) and 45.2% (n=38) respectively. Hepatic dysfunction (n=32, 91.4%) and renal dysfunction (n=21, 60%) were detected also in the patients with a coagulation defect (n=38). Duration of hospitalization for cases that had high ALT, AST, PT, INR, urea, and creatinine values at admission to the hospital was significantly longer than those had normal values ($P < 0.001$, Table I). Statistically significant relationships between INR increase and ALT, AST, urea increase ($P < 0.001$), or creatinine increase ($P < 0.05$) were also detected. Similarly, there was a significant relationship between PT increase and ALT, AST, urea increase ($P < 0.001$), or creatinine increase ($P < 0.05$) as well as between ALT / AST increases and urea increase ($P < 0.001$) and creatinine increase ($P < 0.05$). When the ALT, AST, PT, INR, and urea values at admission and at discharge were compared, the decreases were found to be significant ($P < 0.001$), whereas there was no detectable significant change in creatinine levels (Table II).

Treatment approaches

Of the 84 patients, 36 (42.8%) had penicillin G (Group 1), 23 (27.3%) had symptomatic treatment (Group 2), and 25 (29.7%) had penicillin G with hemoperfusion (Group 3). Twenty-five patients (29.8%) were applied hemoperfusion. Mean hemoperfusion duration was detected as 8.0 ± 4.1 days.

Table I: Hospitalization duration evaluation according to hepatic dysfunction, renal dysfunction and coagulation defect.

			Hospitalization Duration		P
			Mean±SD	Median	
Hepatic Dysfunction	ALT	High	8.93±6.32	8	0.001**
		Normal	3.05±1.08	3	
	AST	High	8.09±6.27	7.5	0.001**
		Normal	3.20±1.32	3	
Renal Dysfunction	Urea	High	9.21±5.63	8	0.001**
		Normal	4.49±4.76	3	
	Creatinine	High	9.53±7.06	8	0.001**
		Normal	4.38±3.20	3	
Coagulation Defect	INR	High	10.54±6.16	8	0.001**
		Normal	3.34±2.26	3	
	PT	High	9.87±6.35	8	0.001**
		Normal	3.43±2.30	3	

Mann-Whitney U Test ** $P < 0.001$

Duration of hospitalization for treatment groups were 3.2 ± 1.5, 4.1 ± 2.0 and 13.0 ± 6.0 for group 1, 2 and 3, respectively.

When improvement in laboratory values during hospital stay was compared by the treatment groups, no significant decreases were observed in Group 1 and 2, while statistically significant decreases were observed in Group 3 (penicillin G plus hemoperfusion) only ($P < 0.001$, Table III). The evaluations of pre-hemoperfusion, post-hemoperfusion and discharge measurements of ALT AST, PT, INR, urea, and creatinine in Group 3 are presented in Table IV.

The duration of hospitalization for group 3 was statistically significantly longer than the other groups ($P < 0.001$); while there was no significant difference between Group 1 and 2.

Only one of our patients died due to renal and hepatic failure.

DISCUSSION

Due to the suitable ecological conditions, Turkey has a very rich mushroom flora. Especially in spring and autumn, due to gathering and consuming as food by the poor socioeconomic communities, mushroom poisonings occur frequently (7,8,9). In Iran and Japan, it has also been reported that most of the mushroom poisonings occur in autumn months (10,11). In two studies from Turkey carried out in Eskişehir, it was reported that most of the cases were observed in May and June (71.8% and 86%, respectively) (12,13). In another study, Ecevit et al. reported that 81% of patients were admitted to the hospital in autumn in Izmir (14). Similar to the above mentioned studies, in our study, it is observed that 62 cases (73.8%) were admitted in autumn and 44% in November.

The most frequent symptoms in the patients admitted to the emergency service with mushroom poisonings are nausea, vomiting, abdominal pain, diarrhea, agitation, dizziness, unconsciousness, and encephalopathy (10,15). In our study cases, the most frequently observed symptoms were related to the gastrointestinal system.

The diagnosis can be made by history, analysis of mushroom samples, and clinical symptoms (12,15). The American Association of Poison Control Centers (AAPCC) showed that differentiation of toxin groups could not be made in approximately 90% of mushroom poisoning cases between 1975 and 1995 (12). In the same study, it was stated that 25% of the cases required treatment and that only 0.3% presented with severe toxicity. Since a toxin differentiation analysis could not have been made in our hospital, the cases had been managed based on the clinical and laboratory findings. In a patient suspected for mushroom poisoning, full blood count, plasma glucose level, renal and hepatic function tests, electrolytes, urine analysis, if necessary coagulation parameters, fibrinogen, artery blood gas analysis should be ordered, and if mushroom samples are available, mycological evaluation should be performed (16,17). It has been reported that hepatic and renal function tests should be periodically repeated in the first 36 hours after ingestion (12). As recommended, in our study, ALT, AST, PT, INR, urea, and creatinine levels had been measured.

Table II: The evaluation of AST, ALT, urea, creatinine, INR, and PT measurements at admission and discharge.

		Admission	Discharge	P
		Mean±SD	Mean±SD	
Hepatic Dysfunction	+ALT (Median)	213.00±658.58 (33) (min:40,max:5491)	47.88±49.32 (30) (min:40,max:271)	0.001**
	+AST (Median)	182.03±500.60 (30.5) (min:40,max:3500)	146.75±1097.05 (25) (min:40,max:10800)	0.001**
Renal Dysfunction	++Urea	35.69±18.11 (min:35,max:120)	28.55±9.67 (min:35,max:53)	0.001**
	++Creatinine	0.99±0.46 (min:0.8,max:3.7)	0.92±0.26 (min:0.8,max:1.98)	0.127
Coagulation Defect	+INR (Median)	1.31±0.64 (1.1) (min:1.2,max:2.75)	1.12±0.44 (1.0) (min:1.2,:max:4.8)	0.001**
	+PT (Median)	15.14±6.28 (13.3) (min:13.5,max:27.1)	13.40±5.47 (12.7) (min:13.5,max:21)	0.001**

+Wilcoxon signed rank test ++ Paired Sample t test
** $P < 0.001$

A relationship between mortality and prothrombin time has been demonstrated by Pajoumand et al. (10). Furthermore, Krenova et al. have confirmed that prognosis and early hepatic or renal recovery by the time of discharge depended mostly on the decrease in prothrombin index, and on the increase of serum transaminase and bilirubin levels. In addition, an increase in serum creatinine levels has been reported to be a negative factor for organ recovery (18). In our study, when ALT, AST, PT, INR, and urea values at admission and at discharge were compared, the decreases were found to be significant ($P < 0.001$), whereas there was no change in creatinine levels ($P > 0.05$).

Specific treatments in the past decades consisted of detoxification procedures (toxin removal with bile, forced diuresis, extracorporeal purification) and administration of various drugs (19). In mushroom poisonings, nasogastric tubing and gastric lavage are recommended initially, and then the administration of active charcoal (starting dose, 1 g/kg, and maintenance dose, 0.5 g/kg) in the next 2-4 hour period to prevent toxins from entering the enterohepatic circulation (12,15,4,20). Mushroom toxins may be neutralized by antidote binding. For amatoxin, penicillin G (300,000 – 1,000,000 U/kg/day, i.v.) and silibin (20-50 mg/hg/day, p.o.) have been used as an antidote. It is thought that ß-lactam antibiotics (benzylpenicillin, ceftazidim) prevent amatoxin from entering into hepatocytes, and facilitate its elimination via the kidney preventing its binding to plasma proteins, especially when combined with steroids. Silibin (milk thistle) decreases amatoxin entering into cells by preventing competitively its binding to the hepatocyte membrane receptor. Conjugating with its toxic metabolite, N-acetylcysteine (loading dose, 140 mg/kg; maintenance dose 70 mg/kg, p.o. every 4 hours), increases toxin elimination (21,22). Enjalbert et al. published a review concerning clinical symptoms and treatment of 2108 hospitalized patients with amatoxin poisoning over the last 20 years (19). They concluded that benzylpenicillin either alone or in combination with other drugs, was the most frequently used chemotherapy but showed little efficacy. Silibinin and N –acetylcysteine were the most effective therapeutic models; however, no benefit was found after use of thioctic acid (23). It has been reported that hemoperfusion, hemofiltration, plasmapheresis, hemodialysis, and albumin dialysis may be effective especially when applied in the early period (first 24 hours) after mushroom ingestion (2,4). The beneficial effect of hemoperfusion is to clear plasma not only from amanitin, but also from neurotoxic substances such as methionine, tryptophan, and phenylalanine. Hepatic encephalopathy improves with hemoperfusion in 75% of patients, but the effects of neurotoxic substances on mortality and life span have not been determined (24,25). In a study on in vitro elimination of alpha-amanitin

Table III: The evaluation of peak and discharge measurements of ALT, AST, urea, creatinine, INR, and PT according to treatment groups.

		Treatment			P
		Group I	Group II	Group III	
		Mean±SD	Mean±SD	Mean±SD	
⁺ALT	Peak	212.64±910.24 (34) (min:15,max:5491)	37.82±26.67 (29) (min:15,max:134)	2056.52±2295.36 (1120) (min:45,max:9000)	0.001**
	Discharge	30.83±16.75 (27.5) (min:13,max:88)	29.56±20.45 (24) (min:9,max:98)	89.28±71.26 (68) (min:15,max:248)	0.001**
⁺AST	Peak	139.72±431.70 (32.5) (min:11,max:2459)	30.17±13.70 (27) (min:10,max:76)	2306.52±2626.62 (1120) (min:86,max:10080)	0.001**
	Discharge	22.63±9.87 (21.5) (min:6,max:51)	22.87±10.73 (20) (min:8,max:44)	439.44±2008.71 (33) (min:5,max:10080)	0.001**
⁺⁺Urea	Peak	38.40±17.47 (min:18,max:114)	34.67±13.33 (min:16,max:63)	61.18±28.59 (min:23,max:135)	0.001**
	Discharge	30.18±9.95 (min:12,max:54)	23.70±9.07 (min:10,max:39)	30.66±8.52 (min:17.4,max:99)	0.016*
⁺⁺Creatinine	Peak	1.02±0.30 (min:0.67,max:1.98)	1.02±0.21 (min:0.67,max:1.7)	1.42±0.72 (min:0.8,max:3.7)	0.002**
	Discharge	0.85±0.28 (min:0.47,max:1.98)	0.89±0.14 (min:0.5,max:1.1)	1.04±0.28 (min:0.6,max:1.42)	0.016*
⁺INR	Peak	1.15±0.24 (1.10) (min:0.967,max:2)	1.35±0.47 (1.20) (min:1,max:2.967)	3.01±2.24 (1.90) (min:1.1,max:9.5)	0.001**
	Discharge	1.05±0.10 (1.00) (min:0.9,max:1.43)	1.08±0.09 (1.00) (min:1,max:1.3)	1.27±0.78 (1.00) (min:0.8,max:2.2)	0.425
⁺PT	Peak	13.79±2.57 (13.0) (min:11.2,max:21.6)	16.08±5.65 (13.5) (min:11.5,max:35.5)	32.01±21.64 (22.8) (min:12.5,max:94.8)	0.001**
	Discharge	12.74±1.33 (12.7) (min:10.8,max:17.3)	12.94±1.33 (13) (min:9.6,max:14.9)	14.79±9.81 (12.3) (min:7.2,max:17.3)	0.633

⁺Kruskal Wallis Variance Analysis Test ⁺⁺ One-way ANOVA Test
* P < 0.05
** P < 0.001

with four different methods, it was detected that the most rapid detoxification was in hemoperfusion with resin, followed by hemofiltration, hemodialysis, and the slowest method of carbon hemoperfusion (26). Early hemoperfusion, either alone or combined with hemodialysis or plasmapheresis substantially decreases mortality due to hepatic and renal failure (27). In our study, 36 (42.8%) patients received penicillin G, 23 (27.3%) patients had symptomatic treatment, and 25 (29.7%) patients received penicillin G with hemoperfusion, in line with the recommendations.

Table IV: The evaluations of pre-hemoperfusion, post-hemoperfusion and discharge measurements of ALT AST, PT, INR, urea, and creatinine in hemoperfused patients.

	Hemoperfusion Group			**P**
	Pre	**Post**	**Discharge**	
	Mean±SD	**Mean±SD**	**Mean±SD**	
+ALT	772.80±988.77 (350) (min:33,max:3370)	1280.40±1643.30 (461) (min:54,max:5985)	89.28±71.26 (68) (min:15,max:271)	0.001**
+AST	938.80±1309.30 (275) (min:41,max:4250)	1480.72±1914.25 (710) (min:62,max:8362)	439.44±2008.71 (33) (min:5,max:184)	0.001**
++Urea	44.54±23.50 (min:23,max:116)	42.16±23.11 (min:14,max:95)	30.67±8.52 (min:10,max:50)	0.004**
++Creatinine	1.24±0.75 (min:0.7,max:3.7)	0.94±0.30 (min:0.2,max:1.7)	1.04±0.28 (min:0.6,max:1.42)	0.011*
+INR	2.32±2.07 (1.46) (min:1,max:9.5)	1.98±1.50 (1.43) (min:0.8,max:4.8)	1.27±0.78 (1) (min:0.8,max:4.8)	0.001**
+PT	23.73±19.66 (16.7) (min:11.7,max:94.8)	21.47±15.72 (16.2) (min:7.2,max:58.2)	14.79±9.82 (12.3) (min:7.2,max:21)	0.001**

+Friedman test ++ Repeated Measures ANOVA Test
* P < 0.05
** P < 0.001

Mushroom poisoning has high mortality rate, and is responsible of 50% of deaths caused by poisonings of plant origin (28). Oldrige et al. reported that annual deaths from mushroom poisonings in the UK were less than 2 (29). In 1975-1987, the number of deaths in 2785 mushroom poisoning cases was 15 in Germany. In Turkey, the number of deaths among 1315 mushroom poisoning cases was 44 in 1970-1975; and death rates were reported to be 2.8% for children and 2.5% for adults (30,31,32,33). The mortality rate in our study was detected as 1.2%. The relatively low mortality rate might be due to the application of the appropriate treatment approach as early as possible.

In our study, it has been observed that early hemoperfusion provided better prognosis by enhancing the efficacy of the treatment. In conclusion, mushroom poisonings may have a higher mortality rate if treatment is not started early. However, the best method to reduce the mortality is to enlighten the community about the risks of mushroom poisonings.

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