Turk J Intensive Care
DOI: 10.4274/tybd.galenos.2019.93695

CASE REPORT / *OLGU SUNUMU*

Ebru Karakoç,
Kemal Demirtaş,
Serdar Ekemen,
Ayşe Ayyıldız,
Birgül Yelken

Mushroom That Breaks Hearts: A Case Report

Kalpleri Kıran Mantar: Olgu sunumu

Received/Geliş Tarihi : 13.11.2018
Accepted/Kabul Tarihi : 10.04.2019

Ebru Karakoç, Kemal Demirtaş, Serdar Ekemen, Ayşe Ayyıldız, Birgül Yelken
Osmangazi University Faculty of Medicine, Department of Anesthesiology and Reanimation, Intensive Care Unit, Eskişehir, Turkey

Ebru Karakoç MD (✉),
Osmangazi University Faculty of Medicine, Department of Anesthesiology and Reanimation, Intensive Care Unit, Eskişehir, Turkey

E-mail : ebrukarakoc1983@gmail.com
Phone : +90 506 260 46 81
ORCID ID : orcid.org/0000-0002-2995-5893

ABSTRACT Because of the high mortality and morbidity, mushroom poisoning is a significant medical emergency that causes liver and kidney damage; however, its effect on cardiac functions has not been established yet. We aimed to present a 44-year-old woman who was poisoned by mushroom and complicated with hepatic, renal and cardiac toxicity. Patients who had mushroom poisoning should also be evaluated especially in terms of cardiac dysfunction with clinic signs, electrocardiography, cardiac enzyme tests and echocardiography.
Keywords: Mushroom poisoning, cardiotoxicity, amatoxin

ÖZ Yüksek mortalite ve morbidite nedeniyle mantar zehirlenmesi karaciğerde ve böbreklerde hasara neden olan önemli bir acil durumdur, ancak kardiyak fonksiyonlara olan etkisi henüz belirlenmemiştir.Kardiyotoksisite,hepatik toksisite,nefrotoksisite ile komplike olmuş 44 yaşında bir kadın mantar zehirlenmesi olgusunu sunmayı amaçladık.Mantar zehirlenmesi olan hastalar klinik belirtiler,elektrokardiyografi,ekokardiyografi,kardiyak enzimlerle kardiyak zehirlenme açısından da değerlendirilmelidir.
Anahtar Kelimeler: Mantar zehirlenmesi, kardiyotoksisite, amatoksin

Introduction

Because of the high mortality and morbidity of mushroom poisoning;it is a significiant medical emergency. Cultivated mushrooms can be eaten almost safely but environmental foraging for mushrooms can result in serious illness and death (1). Mushroom poisoning is almost diagnosed in spring and autumn in Turkey because of the rains that grow up mushrooms at forest and enviroment.

Amanita phalloides (A. phalloides) is responsible for the majority of the mortality and morbidities caused by mushroom poisoning. Poisoning with *Amanita phalloides* results in a mortality rate 20% in adults and 50% in children (2). One of the major problems for amanita poisoning is diagnosis. The characteristic symptoms due to amatoxin can be seen approximately about 15 hour after ingestion regardless of the dose (3). This latent period represents both the minimum time for uptake and enzyme inhibition by the toxin and for the hepatic protein depletion that initiate hepatic dysfunction. Amatoxins cause gastric distress and diarrhea 6–10 hour of ingestion. Following this period, there is an apparent recovery phase during that time the patient seems to improve clinically. However, hepatic and renal damages occur during this phase, marked by abnormal hepatic and renal function tests. Hepatic and renal injury becomes clinically apparent in the third stage. Organ dysfunction progresses with jaundice, right upper quadrant abdominal pain, weakness and decreased urinary output (4). It causes damage in liver, kidneys and rarely pancreas, causing encephalopathic coma, disseminated intravascular coagulation, hemorrhage, hypovolemic shock and death. However, its effect on cardiac functions has not

been established yet (5). In this case report, we aimed to present a 44-year-old female patient poisoned by mushroom complicated with multi-organ failure and heart failure.

Case Presentation

Forty-four year old, conscious open and hemodynamically stabile female patient assessed at emergency service with a history of mushroom eating two days ago and with symptoms of vomiting and nausea. She said that she picked up mushrooms from countryside. She had been diagnosed and being treated as familial mediterranean fever and hypotiroidia for more than two years. She was medicated with colchicine and L-thyroxine.

After she was admitted to intensive care unit vital signs were normal at first evaluation; blood pressure 130/80 mmHg, pulse rate was 88 beats/min, body temperature 36.8 °C. The patients laboratory findings (i.e., complete blood test, arterial blood gas analyses, electrolytes, international normalized ratio, prothrombin time, activated partial thromboplastin time) were in normal ranges except for the renal and liver function tests. In biochemical tests; blood urea nitrogen and creatinine levels were extremely higher than upper limits of our labarotory test ranges. Her liver enzymes were minimal higher than normal ranges. Her urine output was decreased for two days and she was treated for acute renal failure by hemodialysis and L-thyroxine was added to her medications. After 24 hour hospitalization at ICU the urine output was still not enough and a second hemodialysis applied with ultrafiltration theraphy. At the third day of the treatment plasmapheresis was applied against amatoxin and suspected other potent toxins that may cause mushroom poisinings fatal complications by the same time the liver enzymes were decreased but the creatinine levels were continuously increased. So that three more hemodialysis sessions was applied. At the fifth day of the stay in ICU, hypoksemia and severe swelling resistant to ultrafiltration therapy was evaluated with bedside echocardiogram (ECO). Bedside ECO revealed a global left ventricular hypokinesia with ejection fraction 20%, end-diastolic diameter of 5.9 cm, and systolic pulmonary artery pressure of 40 mmHg. (figure 1) The admission electrocardiogram (ECG) showed sinus tachycardia. The cardiac enzymes were normal ranges (creatine kinase MB 2.73 ng/mL and cardiac troponin I 0.02 ng/mL). Oxygen therapy was administrated by oxygen mask to gain saturation levels upper than 90%. Urine output started at sixth day and loop diuretics were added to medications. At the seventh day creatinine level started to decrease unless liver enzymes started to increase again. So we performed three more plasmapheresis sessions. After the plasmeferesis sessions liver enzymes were decreased. Following days the oksigen requirement was recovered and oxygen therapy was administrated by nasal cannula, liver enzymes and creatinin levels decreased to acceptable ranges.(table 1) At twentieth day she was evaluated with control ECO and ejection fraction measured was 44%, end-diastolic diameter of 4.9 cm, and systolic pulmonary artery pressure of 25 mmHg (figure 2), than at the fifteenth day patient discharged from the ICU, adviced to be controlled by endocrinology and rheumatology clinics. According to rheumatology clinic records, liver enzymes and creatinin levels are still in normal ranges and after a year follow up she has no complaints.

Discussion

The most widespread results of mushroom poisoning are kidney and liver toxicity. Most of the cases of fatal mushroom poisoning in the world consist A. phalloides and it is responsible for 90% of the deaths of mushroom poisoning (6). There are three main groups of toxins in these mushrooms: amatoxins, phallotoxins, and virotoxins. However, the common responsible toxins from the fatal poisoning are amatoxins.

The most common manifestations of severe amatoxin poisoning are hepatic damage and renal failure alike, our patient was presented with renal failure and hepatic damage (7).There are few reports about cardiac dysfunction with mushroom poisoning in spite of significant reports defining

Table 1. Laboratory data

Variable	Normal range	Days							
		1	3	5	7	9	11	13	15
Blood urea nitrogen	(6.0 – 20 mg/dL)	24	10,4	56,3	64,9	58,6	41,8	18,5	10
Creatinine	(0.6 – 1.2 mg/dL)	4,64	2,74	5,57	5,29	4,75	2,54	1,21	0,88
Alanine aminotransferase	(1 – 41 U/L)	134	34	578	310	273	142	83	21
Aspartate aminotransferase	(1 – 38 U/L)	48	22	800	361	124	64	57	20

amatoxin induced liver and renal injury in the literature.

In litareture, Forró and Mándly reported that cardiac functions were depressed during post-operative period in three patients who underwent liver transplantation due to A. phalloides poisoning (8). Unverir et al. reported a 56-year-old patient who had ingested amanita phalloides mushroom with elevated levels of cardiac enzymes in addition to liver and renal dysfunction despite normal echocardiographic and electrocardiographic findings (1). Similarly, our patient had normal cardiac enzymes and electrocardiographic findings but she had cardiac failure in addition to mushroom poisoning due to renal failure and hapatic damage.

Aygul and colleagues reported a case that developed cardiogenic shock in addition to liver and kidney toxicity due to ingestion of A. phalloides mushroom in a twentyfour year old patient (9). Patient had cardiac enzymes were within normal levels (creatine kinase MB fraction 2.14 ng/mL and troponin I 0.01 ng/mL). This case had sinus tachycardia in electrocardiogram and global left ventricular hypokinesia, dilated left ventricular diameter with ejection fraction of 24% in echocardiography. This patient successful treatment with intra-aortic balloon counterpulsation. Control echocardiography one month later presented normal systolic left ventricular functions.In a similar fashion, our patient had cardiac enzymes were within normal levels (creatine kinase MB 2.73 ng/mL and cardiac troponin I 0.02 ng/mL), sinus tachycardia in electrocardiogram and global left ventricular hypokinesia, dilated left ventricular diameter with ejection fraction of 20% in echocardiography. In addition, our patient had normal systolic left ventricular functions with ejection fraction 44% in echocardiogram at twentieth day of her treatment and she discharged from the ICU at fifteenth day of the stay.

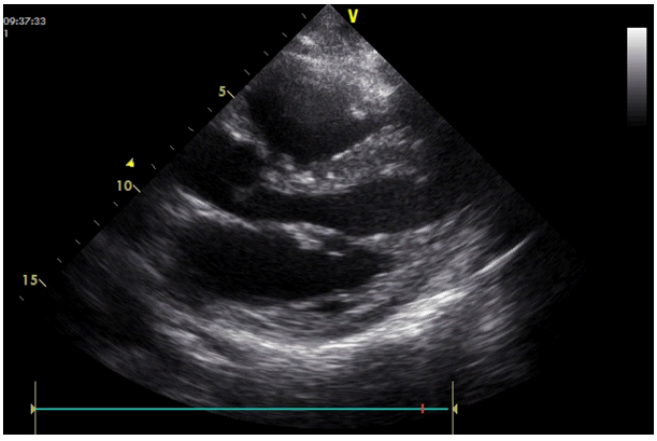

Figure 1. A global left ventricular hypokinesia with ejection fraction 20

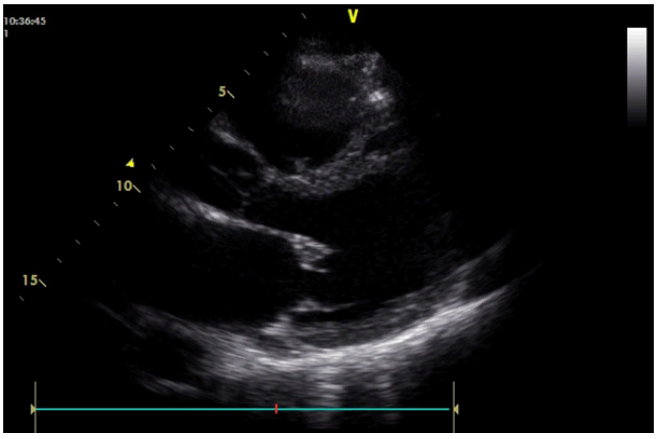

Figure 2. A normal systolic left ventricular functions with ejection fraction 44

Conclusion

In conclusion, mushroom poisoning constantly causes renal and hepatic failure but it influence cardiac functions rarely. This case that we presented manifested mushroom poisoning cause abnormal cardiac functions. Patients who had mushroom poisoning should also be evaluated especially in terms of cardiac dysfunction with clinic signs, electrocardiogram, cardiac enzyme tests and echocardiography.

Acknowledgment: I would like to thank to Prof. Dr. Birgül Yelken for her support to write this case.

Source of Finance: During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.

Conflıct of Interest: No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise,working conditions, share holding and similar situations in any firm.

Authorship Contributions: Idea/Concept: Birgül Yelken;Design: Ayşe Ayyıldız; Control/Supervision:Serdar Ekemen; Data Collection and/or Processing: Ayşe Ayyıldız, ; Analysis and/or Interpretation:Ebru KarakoçLiterature Review: Ayşe Ayyıldız

Writing the Article;Ebru Karakoç Critical Review: Kemal DemirtaşReferences and Fundings: Ebru KarakoçMaterials: Kemal Demirtaş, Ebru Karakoç.

References

1. Renal and hepatic injury with elevated cardiac enzymes in Amanita phalloides poisoning: a case report, Pinar Unverir, Burak Cem Soner, Erhan Dedeoglu, Ozgur Karcioglu, Kaya Boztok and Yesim Tuncok, Human & Experimental Toxicology (2007) 26: 757–761
2. Jander S, Bischoff J, Woodcock BG. Plasmapheresis in the treatment of Amanita phalloides poisoning: II. A review and recommendations. Ther Apher 2000; 4:308–12.
3. Williams PL, James RC, Roberts SM. Principles of toxicology: environmental and industrial applications, second edition. John Wiley & Sons, Inc. 2000.
4. Goldfrank LR, Mushrooms. In Goldfrank LR, Flomenbaum NE, Lewin NA, Howland MA, Hoffman RS eds. Goldfrank's toxicologic emergencies. McGraw-Hill, 2002.
5. Escudie´ , L., Francoz, C., Vinel, J.P., Moucari, R., Cournot, M., Paradis, V., Sauvanet, A., Belghiti, J., Valla, D., Bernuau, J., Durand, F., 2007. Amanita phalloides poisoning: reassessment of prognostic factors and indications for emergency liver transplantation. J. Hepatol. 46 (3),466–473
6. Klein, A.S., Hart, J., Brems, J.J., Goldstein, L., Lewin, K., Busuttil, R.W., 1989. Amanita poisoning: treatment and the role of liver transplantation. Am. J. Med. 86 (2), 187–193.
7. Vetter, J., 1998. Toxins of Amanita phalloides. Toxicon 36 (1), 13–24.
8. Forró , M., Mándli, T., 2003. Liver transplantation after Amanita phalloides poisoning from the viewpoint of anesthesia and intensive care based on three cases. Orv. Hetil. 144 (6), 269–273.
9. Aygul N, Duzenli MA, Ozdemir K, Altunkeser BB. A case report of an unusual complication of Amanita phalloides poisoning: Development of cardiogenic shock and its successful treatment with intra-aortic balloon counterpulsation. Toxicon. 2010;55(2–3):630–632