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Mushroom That Break Hearts: A Case Report

Kalpleri Kiran Mantar: Olgu Sunumu

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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 significant 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 environment.

Amanita phalloides (*A. phalloides*) is responsible for the majority of the mortality and morbidities caused by mushroom poisoning. Poisoning with *A. 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 Report

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 levothyroxine (L-thyroxine).

After she was admitted to intensive care unit (ICU) 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 laboratory 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 therapy. At the third day of the treatment plasmapheresis was applied against amatoxin and suspected other potent toxins that may cause mushroom poisonings 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

(ECHO). Bedside ECHO 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 1: 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 oxygen 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 ECHO 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.

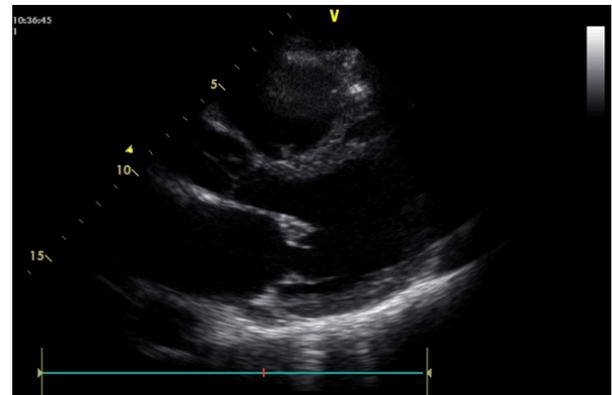


Figure 1. A global left ventricular hypokinesia with ejection fraction 20

Table 1. Laboratory data		Days							
Variable	Normal range	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

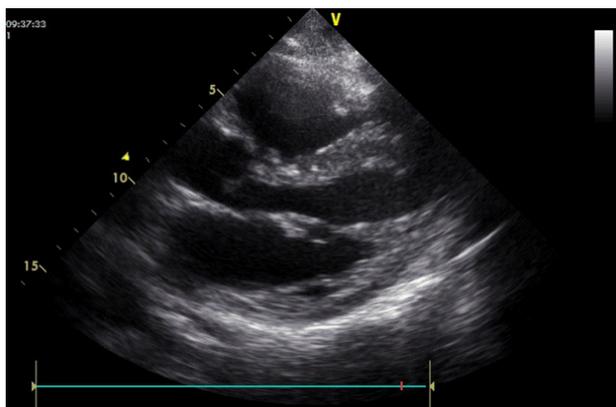


Figure 2. A normal systolic left ventricular functions with ejection fraction 44

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 amatoxin induced liver and renal injury in the literature.

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

Aygul et al. (9) 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.

Patient had cardiac enzymes were within normal levels (creatine kinase MB fraction: 2.14 ng/mL and troponin 1: 0.01 ng/mL). This case had sinus tachycardia in ECG 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 1: 0.02 ng/mL), sinus tachycardia in ECG 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 ECHO at twentieth day of her treatment and she discharged from the ICU at fifteenth day of the stay.

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, ECG, cardiac enzyme tests and echocardiography.

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Ethics

Informed Consent: The patient informed consent is received verbally.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: E.K., K.D., B.Y., Concept: B.Y., Design: A.A., Data Collection or Processing: A.A., Analysis or Interpretation: E.K., Literature Search: S.E., Writing: E.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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