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Case report

Elevated cardiac enzymes due to mushroom poisoning

Sema Avcı, Eren Usul, Nezih Kavak, Fatih Büyükcam, Engin Deniz Arslan, Selim Genç, Seda Özkan

Department of Emergency Medicine, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara, Turkey

Summary. Mushroom poisoning is an important reason of plant toxicity. Wild mushrooms that gathered from pastures and forests can be dangerous for human health. The clinical outcomes and symptoms of mushroom toxicity vary from mild gastrointestinal symptoms to acute multiple organ failure. Toxic effects to kidney and liver of amatoxin are common but cardiotoxic effects are unusual. In this case, we reported the cardiotoxic effect of amatoxin with the elevated troponin-I without any additional finding in electrocardiography, echocardiography and angiography

Key words: cardiac enzyme, mushroom, poisoning, toxicity, troponin

Case Report

A 55-year-old-man admitted to emergency department with dyspnea and palpitation seven hours after an unknown type of mushroom ingestion. The patient stated that the mushrooms are gathered from Ilgaz Mountains National Park. The patient told that visual hallucinations started two hours after ingestion. The patient's vital signs were as follows; blood pressure, 100/60 mmHg; body temperature, 36.7˚C; hearth rate, 115 beats/min. On physical examination, the patient was oriented, alert and conscious. There was no chronic disease or medication in history. Laboratory results were as follows; alanine aminotransferase (ALT): 24U/L, aspartate aminotransferase (AST): 32U/L, troponin-I: 1.929 ng/mL (Siemens ADVIA Centaur® Troponin I Ultra), creatine kinase: 68 U/L, CK-MB: 26 U/L (Siemens ADVIA Centaur® CK-MB). The electrocardiogram (ECG) revealed sinus tachycardia. Transthoracic echocardiography revealed no cardiac wall motion abnormality; cardiac functions were normal; ejection fraction was 60% and systolic pulmonary arterial pressure was calculated as 30 mmHg. There was no pathology in thoracic radiography. The patient hospitalized and followed in the coronary intensive care unit. There wasn't any pathology in coronary angiography that was electively performed. On follow up; cardiac enzymes are decreased progressively. Troponin-I level was decreased to 0.613 ng/mL level at the 24th hour of hospitalization. Patient was called for control after a week and there was no symptom or complication on examination.

Discussion

The clinical outcomes and symptoms of mushroom poisoning vary from mild gastrointestinal problems to acute multiple organ failure (1). Their toxins are split into seven parts as amatoxins (cyclopeptides), orellanus (cortinarius species), gyromitrin (monometylhydrazine), muscarine, ibotenic acid, psilocybin and coprine (disulfiramlike) (1). Amanita group known as cytotoxic and harmful for kidney and liver; orellanine is harmful for kidneys (2). Amatoxin accelerates protein synthesis and cell cycle; in this way that damages particularly hepatocytes, epithelial cells, mucosa in the intestinal lumen and renal tubular epithelium (3). Also it is estimated that Amanita group are responsible for death from mushroom toxicity (1).

Toxic effects to kidney and liver of amatoxin are common but cardiotoxic effects are unusual (3). A small number of study about cardiotoxic effects of amatoxin manifested that phalloidin is a calcium sensitizer thus it enhances calcium sensitivity; so it causes extreme force on cardiac muscle (3). Lima et al. (2) mentioned that Genus Psilocybe ingestion may cause sympathomimetic symptoms, tachycardia, hypertension, neurological symptoms and rarely myocardial infarction in their study about poisonous mushrooms. In the same study, they referred a cytolytic pore protein which called ostreolysin that was isolated from Genus Pleuroutus leads to cardiac ischemia, tachycardia, hypoxia, blood pressure increase and hyperkalemia (2).

Herein we reported the cardiotoxic effect of amatoxin; we showed the elevated troponin-I level without any additional finding in electrocardiography, echocardiography and angiography. Since troponin-I is a specific biomarker of acute myocardial injury, we can say that mushroom toxicity could be a cause of cardiac toxicity. Plasma half life of troponin-I is nearly one day in acute myocardial infarction in patients with normal renal function (4). But in intoxications, half life of troponin-I can change; O'Brien PJ reported the half life of troponin-I as 6 hours in isoproterenol-induced cardiotoxicity in rat samples (5). So we can consider that the three times of decrease in troponin-I levels in 24 hours could be a result of possible variance of troponin-I levels in intoxications.

In conclusion, emergency physicians should be awake about cardiotoxic effects of mushroom poisoning so electrocardiogram and cardiac enzymes should be evaluated.

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Correspondance:
Sema Avcı, MD
Department of Emergency Medicine,
Dışkapı Yıldırım Beyazıt Training and Research Hospital,
Ankara, Turkey
Phone: +90 312 596 2000
Fax: +90 312 318 6690
E-mail: semaakhan@hotmail.com