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Mushroom poisoning: Case series

Pinar Baydar Yücel; Veysel Karani Belen; Alper Taşkin; Mustafa Polat; Ali Karakus*

Hatay Mustafa Kemal University, Faculty of Medicine, Department of Emergency Medicine, Hatay, Turkey.

***Corresponding Author: Prof. Dr. Ali karakus**
Emergency Medicine Specialist, Mustafa Kemal University, Tayfur Ata Sökmen Faculty of Medicine, Department of Emergency Medicine- Hatay, Turkey.
Phone: 0.326.2291000, 0.505.2540433;
Fax: 03262455654;
Email: drkarakus@yahoo.com

Abstract

Mushroom poisoning is quite common due to the unconscious collection and consumption of wild mushrooms. Mushroom poisonings show clinical changes according to the mushroom species. Clinical findings; gastrointestinal side effects range from neurogenic, psychogenic, cholinergic findings to organ failure and even death. After eating several different types of mushrooms they collected from the plateau, a family consting of mother, father and son had mushroom poisoing.

Keywords: Mushroom poisoning; Seizure; Bradycardia; Death.

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Introduction

Mushroom poisoning is quite common in our country as a result of unconsciously collecting and eating mushrooms. In some studies conducted in our country, it is seen that 9.3-10.9% of all intoxications are caused by mushroom poisoning. It is thought to be [1]. Toxicity; It varies according to the amount of mushroom eaten, age, season, geographical region and cooking method [3]. Clinical symptoms and findings in mushroom poisoning can be seen as variable, ranging from Gastrointestinal (GI), neurogenic and psychogenic findings to liver failure, kidney failure, coma and death [4,5].

Case 1

The 81-year-old male patient has no known additional disease, it was stated that he ate less than other family members. Nausea, vomiting, abdominal pain were present. His general condition was good, he was conscious, he was cooperative, his neurological examination was not rebound in defense, other systemic examinations were normal. His blood pressure was 100/60 mmHg, heart rate was 70/ min, temperature was 36°C, oxygen saturation was 96%, ECG was in normal sinus rhythm. The patient was monitored and followed up in the emergency observation room. Symptomatic treatment, hydration, gastric lavage with nasogastric tube, activated charcoal administration (1 g/kg), urinary output was followed by inserting a catheter into the bladder. Repeated doses of activated charcoal were administered (0.5 g/kg). In the laboratory results; glucose 111 mg/dl (74 -106), BUN 18 mg/dl (6-20) creatinine 0.8 mg/dl (0.5-1.1), AST34U/L (0-40), ALT17U/L (0-49), PT-INR 1.08 sec (0.8-1.1), APTT 24.5 sec (26-35), Wbc 9k (4-10k), hb 13.4 g/dl (11-15), platelet 155k (150-450k), potassium(K) 3.6 mmol/l (3-5), sodium(Na) 142 mmol/l (136-145), venous blood gas pH 7.36 (7.35-7.45), $HC0_3$ 25 mmol/l (21-27), lactate 0.8 (0.5-1.6). The patient, whose follow-up was natural, was hospitalized and followed up, and at the end of the 3rd day, he signed a treatment rejection form and left the hospital.

Case 2

A 59-year-old male patient was taken to an external center with neurogenic symptoms such as dizziness, seizures, and convulsions. When he had seizures and contractions, he was se-

dated with 5 mg benzodiazepam at the hospital he came to. His general condition is moderate to poor, his consciousness is sedated, his neurological examination could not be evaluated completely, other systemic examinations are normal. His blood pressure was 120/80 mmHg, heart rate was 96/min, fever was 36°C, oxygen saturation was 96%, respiratory rate was 24, his ECG was in normal sinus rhythm and fingertip blood glucose was 129 mg/dl. Laboratory results; glucose 108 mg/dl, BUN 21 mg/dl, creatinine 1.01 mg/dl, AST24 U/L, ALT 22 U/L, PT-INR 1.07 sec, APTT 27 sec, Wbc 9 .67 bin, hb 14.7 g/dl, platelet 238 thousand, K4 mmol/l, Na 141 mmol/l, venous blood gas pH 7,4, $HC0_3$ 28.5 mmol/l, lactate 1.5 mmol/l. The patient was monitored, he was followed up in the emergency resuscitation room, symptomatic treatment, hydration, activated charcoal application by inserting a nasogastric tube, and urine output was followed by inserting a catheter into the bladder. Diazem 5 mg IV was administered to the patient who had seizures again during the follow-ups. The control laboratory results were within normal limits. The patient was admitted to the intensive care unit, the seizures stopped and the patient was admitted to the service within 10 hours. The patient, whose follow-up was natural, left the hospital by signing the treatment rejection form at the end of the 3rd day.

Case 3

An 83-year-old female patient has known hypertension. He was taken to an external center with dizziness and was brought to our emergency department because his condition did not improve. He was conscious, confused, and cooperative, his general condition was poor, his Glaskow Coma Scale (GCS) 10, pupillarysochoric, miotic light reflexes decreased, the body was moist, salivation increased, lung sounds were rhonchi and rall. Blood pressure 90/50 mmHg, heart rate 40/min, fever 36°C, oxygen saturation 96%, respiratory rate was 24. ECG was sinus bradycardia, fingertip blood glucose was 115 mg/dl. In laboratory results, glucose 116 mg/dl, BUN 24 mg/dl, creatinine 0.81 mg/dl, AST 23 U/L, ALT 16 U/L, PT-INR 1 sec, APTT 27 sec, Wbc 10.12 bin , hb12.9 mg/dl, platelet 88 bin, K 4.5 mmol/l, Na 143 mmol/l, venous blood gas pH 7.39, $HC0_3$ 25.4 mmol/l, lactat 0.8 mmol/l.L The patient was monitored, he was followed up in the emergency resuscitation room, symptomatic treatment, atropine 1 mg, hydration, activated charcoal by inserting a nasogastric tube, and urine output was followed by inserting a catheter into the bladder. Upon worsening of consciousness, GCS and convulsions, the patient was sedated with benzodiazepam and endotracheal intubation was performed. Connected to mechanical fan. After a single dose of atropine, his bradycardia and hypotension improved and his secretions decreased. The patient, who was hospitalized in the intensive care unit due to the lack of improvement in consciousness, continued with symptomatic treatments, atropine was administered again due to a decrease in the pulse rate.

Discussion

It is estimated that there are about 5000 mushroom species in the world, 200-300 of them can be safely eaten, and 30-100 species are thought to be poisonous [6]. In fungi; hepatotoxic fungi; (Cyclopeptide, Amanitaphalloides and some Lepiota species.) Neurotoxic fungi (false lamb mushroom), cholinergic agonists (Clitocybedealbalta), disulfiram reaction triggering (Corpinus spp.), hallucinogenic fungi (Psilocybeve conocybe) gastrointestinal irritating fungi, e.g. Chlordietesmophyllum, many gastrointestinal irritating fungi; lactarius) nephrotoxic (cortinarius) and rhabdomyolysis-inducing fungi (tricholomaequestre).

In mushroom poisoning, it causes various toxidromas depending on the toxin found in the edible species. The main toxin groups are; cyclopeptides, orelanins, Mono Methyl Hydrazines (MMH), disulfiram analogues, hallucinogenic indoles, muscarines, isoxazoles, Gastrointestinal (GI) specific irritants [7]. Muscarine-containing fungal poisoning (Inocybe species (I.patouillardii I.fastigiata, I.geophylla).

The onset of symptoms in Clitocybe species (C.dealbata, C.rividosa, C.candicans, C.clavipes, Mycena puta.) takes a few minutes to 2 hours, but mostly occurs in 15-30 minutes. Symptoms; Excessive sweating, salivation and tearing are the main symptoms. In addition, nausea, vomiting, diarrhea, visual disturbance, hypotension, bradycardibronchospasm and increased bronchial secretion are observed. Even without treatment in mild poisoning, the patient begins to recover. The antidote to muscarine is atropine. 1-2 mg of atropine can be done until the patient dries up. Mortality in these poisonings is 5% [5]. It was seen more frequently per hour. In our case 3, 83 years old female patient, muscarinic symptoms developed within the first 30 minutes and their vitals were within normal limits after atropine, and their secretions decreased. Our patient died on the 9th hour. It has not been determined exactly which type of mushrooms the patients ate.

However, deaths due to muscarinic mushroom poisonings such as Inocybe, Clitocybe, Rubinoboletus have been reported in Europe, Australia and Turkey [8]. The lethal dose of muscarin for humans is not known for certain, but is estimated to be between 40 mg and 495 mg (equivalent to 150 g of fresh Inocybe mushrooms) [9].

In the treatment, gastric lavage and activated charcoal application are recommended within the first hour. Symptomatic treatment is recommended in gastrointestinal findings with nausea and vomiting. Repeated doses of activated charcoal can be applied in amatoxin toxicity, N-acetylcysteine acid can be applied for antioxidant treatment, Sibilin (5 mg/kg/IV loading dose and 20 mg/kg/day maintenance dose until liver function tests return to normal) as an amatoxin inhibitor, if not Sliymarin 50-100 mg/kg (max single dose 2 g) every 8 hours or Penicillin G 300000-1000000 U/kg/day is recommended. Benzodiazepam can be used for sedation in neurological symptoms. Atropine 0.5-1 mg/kg is recommended for muscarinic symptoms until secretions dry up and hypotension and bradycardia are resolved.

Conclusion

In conclusion; It should not be forgotten that eating the same mushroom species will cause different symptoms in different individuals. It has been observed that especially mixing and eating several different mushroom species and eating them in large amounts worsen the click. Since it is not always known which toxin the fungus contains, patients should be recognized and treated according to their symptoms. Although most cases of mushroom poisoning resolve spontaneously after the effect of the poison, it should be kept in mind that some cases will

result in death.

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