

## Rhabdomyolysis Induced by *Agaricus Bisporus*

Nazire Belgin Akilli<sup>1</sup>, Zerrin Defne Dünder<sup>2</sup>, Ramazan Köylü<sup>1</sup>, Yahya Kemal Günaydın<sup>1</sup>, Başar Cander<sup>2</sup>

<sup>1</sup>Department of Emergency Medicine, Konya Training and Research Hospital, Konya, Turkey

<sup>2</sup>Department of Emergency Medicine, Necmeddin Erbakan University Meram Faculty of Medicine, Konya, Turkey

### Abstract

Mushroom poisoning may present with a variety of clinical conditions, extending from simple food poisoning to life-threatening liver and renal failure. Rhabdomyolysis is a recently described syndrome that is observed within the clinical spectrum associated with mushroom poisoning. In this report, we present two patients-one presenting with a state of rhabdomyolysis and the other case with simple symptoms only-following consumption of cultivated mushroom together in the same meal. (*JAEM 2014; 13: 212-3*)

**Key words:** Mushroom, rhabdomyolysis, *agaricus bisporus*

### Introduction

Mushroom poisoning is a common environmental emergency. It presents with a wide clinical spectrum, extending from simple food poisoning to life-threatening liver and renal failure. Rhabdomyolysis is a recently described syndrome that is observed within the clinical spectrum associated with this type of poisoning. It was first reported by Bedry et al. for *Tricholoma equestre* (1). It is known that there are approximately 2000 edible mushroom species worldwide, and *Tricholoma equestre* is one of the edible mushrooms. In addition, there are 20 cultivated mushroom species in our country. The most commonly produced cultivated mushroom species is *Agaricus bisporus* (white mushroom), and the second one is *Pleurotus ostreatus*, including oyster mushroom (2).

In an experimental study on mice with edible mushroom, cultivated mushrooms were also shown to be myotoxic (3). On the other hand, there are no reports on rhabdomyolysis induced by cultivated mushroom among humans in the literature. In this report, we present two patients-one presenting with a state of rhabdomyolysis and the other case with simple symptoms only-following consumption of cultivated mushroom together in the same meal.

### Case Presentations

#### Case 1

A 52-year-old male patient with symptoms of nausea, vomiting, and loss of consciousness was brought to the emergency depart-

ment. The patient's wife stated that 6 hours prior to development of syncope in the patient, they had consumed approximately 0.5 kg of cultivated mushrooms, which they had bought from a bazaar. She also stated that her husband consumed most of the cooked mushrooms. There were no previous known diseases, no consumption of any other suspicious foods apart from the mushrooms during the previous 24 hours, no trauma, no fever, no diarrhea, and no injections in the patient's medical history.

Upon arrival at the emergency department, the patient was conscious. The vital signs and physical examination findings of all systems were normal. The initial blood analyses on admission were within normal limits. Hypoxia, hypercarbia, or acidosis was not determined in the arterial blood gases analysis. The carboxyhemoglobin level was measured, and urinary bedside toxicological screening of 11 parameters (Triage Tox Drug Screen®, Biosite Incorporated, San Diego, United States) was performed. No pathological findings were determined in either of the two tests. Twelve-lead electrocardiogram revealed normal sinus rhythm. Computerized brain tomography revealed no pathological findings.

The patient was monitored with a diagnosis of edible mushroom poisoning. Supportive treatment was initiated. A dose of 1 g/kg of activated charcoal was administered. It was thought that the toxicity might be induced by gyromitrin toxin, because the patient had neurological symptoms. Then, 25 mg/kg pyridoxine was administered. The symptoms of nausea and vomiting regressed, and the blood analyses 24 hours after the consumption of mushrooms showed AST: 70 U/L (5-34 U/L), ALT: 19 U/L (0-55 U/L), and CK: 8450

*This study was presented in International Symposium of Emergency Medicine, April 13-15, 2012, Baku, Azerbaijan.*



**Correspondence to:** Nazire Belgin Akilli; Department of Emergency Medicine, Konya Training and Research Hospital, Konya, Turkey  
Phone: +90 332 223 67 00 e-mail: drbelginakilli@hotmail.com

**Received:** 12.05.2014 **Accepted:** 23.05.2014

©Copyright 2014 by Emergency Physicians Association of Turkey - Available online at [www.akademikaciltip.com](http://www.akademikaciltip.com)  
DOI: 10.5152/jaem.2014.150

U/L (30-200 U/L). The results of the complete blood count, urea, creatinine, electrolyte levels, and coagulation profile were within normal limits. The patient's diagnosis was confirmed as rhabdomyolysis, and treatment with hydration, mannitol, and sodium carbonate was commenced. On Day 4 of hospitalization, the enzymes reached peak levels: CK: 31,065 U/L, AST: 684 U/L, ALT: 100 U/L, and LDH: 680 U/L. Urea, creatinine, bilirubin, and bleeding function tests followed a normal course. Upon regression of enzyme levels to normal values, the patient was discharged from the hospital on the 8th day of hospitalization.

## Case 2

The wife of the patient in Case 1, a 46-year-old female patient, was brought to the emergency room with similar symptoms. It was learned that she had only consumed 3-4 spoonfuls of the mushroom. Her vital signs and physical examination findings of the systems were normal. Blood and urinary analyses were within normal limits. Upon regression of the symptoms and the observation of no pathological findings in the blood analyses during the follow-up period, the patient was discharged from the hospital 48 hours after admission.

## Discussion

Mushroom poisoning may present with a variety of clinical conditions. In general, they are stratified as early-, late-, and delayed-onset cases, based on the development of symptoms. A total of 14 syndromes have been described in these 3 groups. Furthermore, 4 new syndromes have recently been described: namely, nephrotoxicity, erythromelalgia, delayed neurotoxicity, and rhabdomyolysis (4).

The symptoms of early-onset mushroom poisoning occur within a period of less than 2 hours after the consumption of mushroom. Early-onset poisonings are usually associated with *Chlorophyllum molybdites*, *Omphalotus illudens*, and *Cantharellus cibarius* species. On the other hand, the symptoms of late-onset mushroom poisoning occur within 6 to 24 hours after consumption. Late-onset poisonings are usually associated with *Gyromitrin* and *Amanita phalloides* species (5).

The first published study of these poisoning cases was a series of patients developing rhabdomyolysis induced by mushroom, reported Bedry et al. In this study, an increase in AST and ALT levels without any findings of hepatic damage was detected, and no electrolyte imbalance was observed, in spite of severe rhabdomyolysis; there were no signs of renal failure (1). Later, in Poland, 3 cases of rhabdomyolysis induced by mushroom were reported (6).

In the literature, there are two experimental studies on mice using *Tricholoma flavovirens* extracts and one study using *Agaricus bisporus* extracts. In these trials, rhabdomyolysis was observed (1, 3, 7). To the best of our knowledge, based on the literature, this experimental study is the only trial indicating the development of rhabdomyolysis induced by *A. bisporus*.

The case presented in this report is the first case in the literature presenting with rhabdomyolysis caused by the consumption of *A. bisporus*, accompanied by elevated liver enzyme levels. In fact, the clinical states observed in our cases have common features with the symptoms and signs developing after the consumption of *T. flavovirens*. Following the development of the initial symptoms, elevation of CK reached a level of 31,065 U/L, and AST and ALT levels also in-

creased without any signs of renal failure or electrolyte imbalance, but hepatic functions were preserved.

The identification of the consumed mushrooms is complicated and time-consuming. Therefore, in all cases with a pre-diagnosis of mushroom poisoning, treatment should be initiated based on the symptoms of the patient, rather than identification of the mushroom (5, 8). Similarly, in our cases, development of disease in both the husband and wife following consumption of the same meal and the lack of other suspicious foods apart from the cultivated mushroom and no drug ingestion led us to initiate treatment for mushroom poisoning. Upon monitorization, rhabdomyolysis developed in one case, while the simple symptoms present on admission regressed in the other patient. We believe that this diversity in clinical states was due to the lower amount of mushroom consumed by the wife, compared to the husband. Moreover, we believe that individual sensitivity, which has been emphasized in various studies, also played a role in the development of rhabdomyolysis.

## Conclusion

Rhabdomyolysis is a recently described clinical syndrome of mushroom poisoning. It should be considered that poisoning may also be induced by edible mushrooms.

**Informed Consent:** Written informed consent wasn't obtained from patients who participated in this case, since we couldn't reach the patients.

**Peer-review:** Externally peer-reviewed.

**Author Contributions:** Concept - N.B.A., Z.D.D., R.K., Y.K.G., B.C.; Design - N.B.A., Z.D.D., R.K., Y.K.G., B.C.; Supervision - N.B.A., Z.D.D., R.K., Y.K.G., B.C.; Materials - N.B.A., R.K.; Data Collection and/or Processing - N.B.A., R.K.; Analysis and/or Interpretation - N.B.A., Z.D.D.; Literature Review - R.K., Y.K.G.; Writer - N.B.A.; Critical Review - B.C.

**Conflict of Interest:** The authors declared no conflict of interest.

**Financial Disclosure:** The authors declared that this study has received no financial support.

## References

1. Bedry R, Baudrimont I, Deffieux G, Creppy EE, Pomies JP, Ragnaud JM, et al. Wild-mushroom intoxication as a cause of rhabdomyolysis. *N Engl J Med* 2001; 345: 798-802. [\[CrossRef\]](#)
2. Şen S, Yalçın M. Dünya ve Türkiye'de kültür mantarcılığı ve geliştirilmesi. III. Ulusal Karadeniz Ormancılık Kongresi 2010; 3: 1208-16.
3. Nieminen P, Kärjä V, Mustonen AM. Myo- and hepatotoxic effects of cultivated mushrooms in mice. *Food Chem Toxicol* 2009; 47: 70-4. [\[CrossRef\]](#)
4. Diaz JH. Syndromic diagnosis and management of confirmed mushroom poisonings. *Crit Care Med* 2005; 33: 427-36. [\[CrossRef\]](#)
5. Brayer AF. Mushroom Poisoning. In: Judith E. Tintinalli, Editor. *Tintinalli's Emergency Medicine*. 7th ed. New York: McGraw-Hill, 2011. p.1394-8.
6. Chodorowski Z, Waldman W, Sein Anand J. Acute poisoning with *Tricholoma equestre*. *Przegl Lek* 2002; 59: 386-7.
7. Nieminen P, Mustonen AM, Kirsi M. Increased plasma creatine kinase activities triggered by edible wild mushrooms. *Food Chem Toxicol* 2005; 43: 133-8. [\[CrossRef\]](#)
8. Broussard CN, Aggarwal A, Lacey SR, Post AB, Gramlich T, Henderson M, et al. Mushroom poisoning-from diarrhea to liver to transplantation. *Am J Gastroenterol* 2001; 96: 3195-8.