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Case Report

Early hemodiafiltration (HDF) is life-saving in cases with mushroom poisoning

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The consumption of food or water contaminated by pathogenic microorganisms, toxins, or chemical substances can cause food borne diseases, which may occur sporadically or epidemically. Although most food borne diseases cause gastrointestinal symptoms, are self-limiting, and have a rapid recovery, they can also lead to clinical situations with high mortality and morbidity, such as serious mushroom poisoning, which requires hospitalization and intensive care. Herein we present the clinical courses, laboratory results, and treatment of 3 cases with mushroom poisoning who developed renal failure to emphasize the importance of renal replacement treatment in mushroom poisoning. We aimed to evaluate methods used in the epidemiology, diagnosis, treatment, protection, and prevention of food and mushroom poisoning in children. Awareness should be raised that mushrooms can be toxic, and that if symptoms of poisoning do occur, individuals should be treated as early as possible. HDF treatment should also be applied as soon as possible in order to reduce mortality rates.

Keywords: Mushroom poisoning, children, hemodiafiltration

INTRODUCTION

The consumption of naturally grown mushrooms is quite common in our country, especially among those with low socioeconimic status who are living in rural areas. Because of this, hundreds of people are admitted to hospitals with mushroom poisoning every year, and many lose their lives due to the complications (Erguven et al., 2007).

The ecological conditions of our country are suitable for the rich growth of mushroom flora, (Onat, 1996), and death rates due to wild mushroom consumption increase particularly in the spring and autumn. In some regions, wild mushrooms are even sold at bazaars. Because there is a large supply of wild mushrooms, they are inexpensive, and therefore, are widely consumed. Because the toxins enter the enterohepatic circulation upon consumption and are absorbed by the kidneys, mushroom poisoning is highly toxic (Onat, 1996). Amatoxins are responsible for gastrointestinal and neurological symptoms, as well as hepatic and renal failure. The gastrointestinal symptoms due to mushroom poisoning are mostly mild. Mushrooms with low toxicity often cause vomiting in the early period, while more toxic mushrooms lead to vomiting approximately 6 hours after their consumption. Symptoms with late initiation are 90-95% fatal. Herein we aimed to present the clinical courses, laboratory results, and treatment of 3 cases with mushroom poisoning who developed renal failure (Kayaalp, 1993; Karlson-Stiber and Persson, 2003) in order to emphasize the importance of renal replacement treatment in mushroom poisoning.

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Table 1. Biochemical Changes for case 1

Day	1	2	3	4	5	6	7	8
Urea (mg/dL)	78	50	128	105	97	119	45	94
Cr (mg/dL)	4.1	2.5	4.9	3.9	3.7	4.8	4.3	4.9
Uric acid (mg/dL)	6.3	4.4	8.2	6.5	3.2	4.3	2.8	3.2
K (mmol/L)	5.4	4.4	4.8	4.8	4.2	3.4	3.4	3.3
P (mg/dL)	6.1	5.8	6.8	6.3	5.8	4.4	4.1	4.2
AST (mg/dL)	506	249	123	69	35	22	18	20
ALT (mg/dL)	808	372	246	185	129	77	59	48
LDH (mg/dL)	3160	2800	2638	2278	1815	1271	1012	731
CK-MB (ng/mL)		87		38	15			
Troponin (mg/mL)		0.101		0.161	0.169	0.2		
CK (IU/L)	614	3131	1861	715	260	128	74	68
Myoglobin (ng/mg)			2229	1068				

Table 2. Biochemical changes for case 2

Day	1	2	3	4	5	6	7	8
Urea (mg/dL)	53	44	37	66	108	139	134	142
Cr (mg/dL)	4.3	3.5	3.3	5.7	8.4	8.6	5.8	4.6
Uric acid (mg/dL)	6.7	2.6	2.5	3.2	4.4	5.7	7	7.9
K (mmol/L)	5.3	3.6	3.03	3.4	2.9	3.1	3.1	3.1
P (mg/dL)	6.4	4	3.4	3.3	4.2	5.3	6.8	7.1
AST (mg/dL)	87	55	28	29	27	21	16	15
ALT (mg/dL)	553	207	59	83	76	61	50	41
LDH (mg/dL)	817	517	317	261	178	121	88	72

CASE REPORTS

Case 1: A 16 year-old male patient was admitted to our emergency service with complaints of nausea and vomiting. We were informed that he had eaten wild mushrooms collected from nature 8 hours prior to his admission. The patient was admitted to the nephrology service because his blood urea nitrogen (BUN) level was 53 mg/dl, his creatinine (Cr) level was 4.3 mg/dl, and his glomerular filtration rate (eGFR) was 9 ml/min. The case was evaluated as having renal failure due to mushroom poisoning. Hemodiafiltration (HDF) treatment was applied to the patient at a 100 cc/min pump rate for 72 hours. His daily blood biochemistry values are shown in Table 1. Diuresis began on the 3rd day, and therefore, HDF treatment was terminated. The kidney functions returned to normal on the 15th day. There was no permanent liver or renal impairment in the patient.

Case 2: An 11 year-old male patient was admitted to our emergency service with the complaints of nausea and vomiting. He had eaten mushrooms collected from the forest 10 hours prior to eating dinner. The patient was admitted to the nephrology because his BUN level was 32 mg/dl, his Cr level was 3.5 mg/dl, and his eGFR was 15 ml/min/1.73m^2. The case was evaluated as having renal failure due to mushroom poisoning, and HDF treatment was applied at 100 cc/min pump speed for 20 hours. Treatment was terminated after 20 hours for technical reasons, and therefore, intermittent hemodialysis (HD) at 200 cc/min pump speed was begun. In total, 20 hours of HDF and HD (2 times for 3 hours) were applied every other day. The patient's prognosis was anuric for the first 5 days. Diuresis began on the 5th day of his admission, and no renal replacement treatment was required. His daily biochemical values are listed in table 2. No permanent liver or renal impairment developed in the patient.

Case 3: An 8 year-old female patient (the sister of Case 2) was admitted to our emergency service with the complaints of nausea and vomiting. She had eaten mushrooms collected from nature 8 hours prior to eating dinner. The patient was admitted to the nephrology service because her BUN level was 78 mg/dl her Cr level was 4.1 mg/dl, and her eGFR was 16 ml/min/1.73m^2. The case was evaluated as having renal failure due to mushroom poisoning. Therefore, 200 cc/min pump speed intermittent HD was applied. Excess HD was applied for 8 days. Her daily biochemical values are presented in

Table 3. Biochemical changes for case 3

Day	1	2	3	4	5	6	7	8	9	10
Urea (mg/dL)	32	105	105	92	113	49	82	116	136	122
Cr (mg/dL)	3.5	5.2	6.5	7.7	8.8	8.9	6.1	6	4	2.3
Uric acid (mg/dL)	5.7	6.9	5.2		5.1	5.9	3.7	4.5	4.6	3.3
P (mg/dL)	6.4	7.1	6.5	5.2	6.3	6.1	6.7	7.3	7.3	5.3
K (mmol/L)	5.5	5.2	3.4	3.8	4.5	4.4	4.6	3.9	3.7	3.3
AST (mg/dL)	75	163	36	25	21	20	20	17	18	17
ALT (mg/dL)	215	500	206	139	105	88	64	61	54	43
LDH (mg/dL)	1957	1700	1400	1157	940	850	740	692	647	550

table 3. The patient's cardiac enzymes were checked due to the development of tachycardia. Echocardiograph revealed that the ejection fraction damage in left ventricular systolic functions was 35%. On the 33rd day, this value was 65%. Since the patient had proteinurea, a renal biopsy was performed. Stickiness was observed in the Bowman's capsule in two glomeruli in the biopsy, which was interpreted as acute interstitial nephritis. No permanent liver or renal impairment developed in the patient.

DISCUSSION

This study evaluated the role of dialysis (high-efficiency and high-flux) and other techniques used in the management of mushroom poisoning, including HDF.

Ecological conditions in our country are conducive to rich mushroom flora (Unluoglu and Tayfur, 2003), and death rates due to wild mushroom consumption increase in the spring and autumn, when collected mushrooms are often sold in bazaars in some regions (Unluoglu et al., 2004). Because wild mushrooms cost little to nothing to eat, they are widely consumed in our country, leading to incidences of mushroom poisoning (Mat, 1997; Deniz and Saygun, 2008).

Amanita phalloides is one of the most important toxic mushrooms, as it contains phallotoxins and amatoxins. These toxins pose great risk to the gastrointestinal and renal tubular cells, which have rapid cell cycles (Splendiani et al., 2000). Although we were unable to determine the amanitin levels of the 3 cases described herein, their laboratry and clinical results indicated that the toxic effects originated from Amanita phalloides.

In amatoxin poisoning, the clinical findings are observed in 3 stages. The gastrointestinal stage begins 6-8 hours after eating the mushroom, and includes nausea, vomiting, and stomach ache. During the latency stage, which occurs within 12-24 hours, there is an increase in non-symptomatic liver enzymes. Renal failure occurs due to nephrotoxicity and hepateronal syndromes, which are caused by amanite in the renal tubulars (Bhutta, 2004; Broussard et al., 2001). In accordance with the literature, the clinical findings in our cases occurred 6-8 hours after mushroom ingestion. Vomitting was the first complaint observed in all three cases, and all were admitted to the hospital during the hepatorenal stage. Diarrhea and renal tubular effects were not observed in our cases, which is different from what is described in the literature.

In cases of mushroom poisoning, the main purpose of treatment is to remove the toxins from the body (Kayaalp, 1993). In the conventional treatment of mushroom poisoning, the intestines should be decontaminatied and liquid electrolytes should be given. Gastric lavage and active coal can also be administered to prevent the absorption of toxins into the gastrointestinal system and prevent its effects on the liver and kidneys. In addition, penicillin G and silibinin can be used to prevent liver toxicity, as they prevent the attachment of amanite to serum proteins and allow free toxins to be discharged through the kidneys (Splendiani et al., 2002). It has been reported that silibinin treatment is lifesaving in the early period (Saviuc and Flesh, 2003). In the cases presented herein, stomach lavage was not performed and silibinin was not given because they were admitted to the hospital more than 8 hours after mushroom poisoning. However, active coal (1gr/kg) and penicillin (300,000 IU/kg/day) were applied to all 3 subjects.

The most effective treatment for mushroom poisoning is the removal of exogen toxins by extracorporal techniques (e.g., hemoperfusion, HD, HDF, and plasmapheresis). Hemoperfusion is the first option in mushroom poisoning and has been determined to increase life expectancy (Parish and Doering, 1986). We planned to use hemoperfusion for the cases presented herein. However, we did not, because the cases arrived at the hospital too long after exposure. Other treatment options include plasmapheresis, HDF, and HD. HD was performed subsequent to HDF as renal replacement therapy in the first two cases. Intermittent HD treatment was performed in the 3rd case due to technical problems. The 3rd case had the the most severe clinical prognosis of the three cases, even though all three arrived at the hospital within the same time frame after eating the mushrooms and had similar renal funtion test results. The

first two cases had a full recovery after 15 days, while the third case recovered after 28 days. In addition, the third case had cardiac involvement accompanied by renal and liver involvement. We believe that the technical problems with the treatment were responsible for the caes's clinical prognosis, but we also believe that the high levels of toxin also had an effect. Considering that the 2nd and 3rd cases were brother and sister, we believe that the mushroom species they consumed should be the same. The only difference between the 3rd case and the other two was the type of renal replacement therapy.

Although all renal replacement therapies (RRT) are considered to be equally effective, the clinical recovery period was shorter in the 2 cases who received treatment with HDF. No permanent liver or renal impairment developed in these patients.

Literature indicates that HD and HDF treatments are as effective as hemoperfusion treatment in mushroom poisoning. Treatment should begin when poisoning symptoms occur, and early treatment is more important then the type of RRT used. Moreover, the best treatment results were obtained from patients who were treated within the first 36-48 hours of ingestion, and these precautions reduced the mortality rates to below 10% (Saviuc and Flesh, 2003).

The current case report series aimed to raise concern about the lack of knowledge regarding mushroom consumption in our society. We emphasize that the consumption of wild mushrooms has greater effects on children, and cases with poisoning should be admitted to the hospital immediately after ingestion. In addition, HDF treatment should be begun as soon as possible. Due to the possibility of poisoning, we believe that people should avoid the consumption of wild mushrooms. Awareness should be raised that mushrooms can be toxic, and that if symptoms of poisoning do occur, individuals should be treated as early as possible. HDF treatment should also be applied as soon as possible. We believe that all of these actions are very important to the reduction of mortality rates.

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