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Management of confirmed mushroom poisoning

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Abstract

During the autumn season, it is common for mushroom pickers to eat poisonous mushrooms. This is due to many reasons, including the ignorance of pickers. The most common is the misidentification of inedible species as edible mushrooms. The result is poisoning, which in many cases requires hospitalization.

This paper presents a case report of toadstool poisoning by a 62-year-old female patient. In addition, other relevant publications on intoxication, diagnostic methods and treatment are reviewed. The purpose of this paper is to present the management and treatment of a case of toadstool poisoning.

The effects of mushroom poisoning can range from mild disorders, mainly gastrointestinal to organ failure and even death. Significant elevations of liver enzymes and prothrombin time are important in laboratory tests in cases of intoxication. Treatment most often involves specific antidotes, such as Sylimarin, Acetylcysteine. The diagnosis in most cases is made on the basis of the clinical picture and a thorough interview with the patient, and the diagnosis is established by detecting toxins in a urine test. The prognosis is better if patients with toadstool poisoning are admitted to the hospital early. It is worth noting that gastrointestinal syndromes can also result from eating raw or inadequately cooked mushrooms, and by eating them excessively or too often.

Proper diagnosis and treatment of mushroom poisoning can save lives. It is very important to raise public awareness of mushroom poisoning, as well as early detection of potential signs of intoxication. Cooperation with the poison center and mycologists is very important, especially in high-risk cases.

Keywords: Wild mushroom intoxication, poisoning, hepatotoxicity, Amanita Phalloides

INTRODUCTION

It is estimated that there are about 5000 species of mushrooms worldwide of which only 200-300 have been declared safe for consumption. About 50-100 types of mushrooms are listed as harmful to human health, while the toxicity profile of the remaining species has not been thoroughly investigated [1,2]. Consumption of toxic mushrooms can cause a range of different clinical symptoms, starting with the most benign ones, which mainly include gastrointestinal disorders, and ending with multiple organ failure and even death [2,3,4,5]. The poisonous effects of fungi mainly include the presence of protoplasmic poisons (responsible for generalized cell destruction and organ failure), neurotoxins (causing neurological symptoms such as convulsions, hallucinations and coma) and gastrointestinal irritants, causing nausea, vomiting, abdominal cramps and diarrhea [6,7,8]. Although mushroom poisoning accounts for only a small proportion of the total number of emergency poisoning consultations [9], it is a significant seasonal and regional public health problem, with reported mortality rates of 8-12% [10, 11] and higher (up to 15-30%) for amatoxin-containing fungi [1,2].

Amanita phalloides, belonging to the section Phalloideae of the Amanita family of fungi, is the culprit of most deaths after eating mushrooms worldwide [12]. Amatoxin poisoning is caused by fungi assigned to three genera: Amanita, Galerina and Lepiota. However, Amanita phalloides is responsible for the majority of fatalities [13]. This extremely deadly fungus, and in fact the compound α-amanitin contained in it, is responsible for about 90% of deaths among fungal poisonings [14]. Although data on mushroom poisoning are not accurate due to a large proportion of underreported cases, amatoxin intoxication is a worldwide problem. Approximately 50-100 fatal cases per year are reported in Western Europe. Amatoxin poisoning is also known to occur in Asia, Africa, Central and South America or Australia [14,15].

Toxicity of the firefly is associated with two completely distinct groups of toxins: phallotoxins and amatoxins. The harmfulness of the former consists in damaging the cell membrane of enterocytes by binding the thiamidol sulphur atom located on the indole ring. Thus, they are responsible for the initial gastrointestinal symptoms such as nausea, vomiting and diarrhea that are present in almost all patients. Amatoxins, on the other hand, are bicyclic octapeptides, composed of at least nine different compounds [16]. Their main representative is α-amanitin, which is probably responsible for the toxic effect [17, 18]. The effect of amanitin directly interacts with the enzyme, RNA polymerase II, in eukaryotic cells and thereby inhibits transcription, causing a progressive decrease in mRNA, deficiency of protein synthesis and cell death. For this reason, metabolically active tissues such as gastrointestinal cells, hepatocytes and proximal ileal tubules of the kidney are disproportionately affected. These are also tissues dependent on a high rate of protein synthesis. The lethal dose of amanitin is very low: as little as 0.1 mg/kg body weight can be lethal in adults, and this amount can be absorbed even by eating a single mushroom. Other likely toxic mechanisms include the synergistic action of alpha-amanitin with endogenous cytokines (e.g. tumour necrosis factor) causing cell damage through induction of apoptosis [19]. Finally, post-mortem human and animal studies also indicate cell damage in the pancreas, adrenal glands and testes [20, 21].

DISCUSSION

We would like to present a case of a 62-year-old female patient who required hospitalization in the Clinical Toxicology and Cardiology Department of the WSS in Lublin due to toadstool poisoning. The patient had eaten 1 fried cap of the lamellar mushroom in the evening. The first symptoms appeared the following day in

the form of nausea, vomiting and abdominal pain. A few hours later, diarrhoea appeared. A urinalysis was performed, which showed the presence of amanitin. Laboratory tests showed a gradual increase in liver damage parameters with a peak in liver enzymes around 2 days after ingestion of the mushroom – as presented in Table 1. At that time, AST was 1780 U/L and ALT 2524 U/L. In addition, there was also an increase in other parameters specific for liver damage, such as a slight coagulopathy (INR 1.39, PLT 124 thou/ul), elevated bilirubin (1.88 mg/dl) and cholestatic enzymes (GGTP 82 U/L, ALP 104 U/L). After symptomatic treatment and specific antidotes (Sylimarin, Acetylcysteine), an improvement in the patient's general condition was observed along with a gradual normalization of liver parameters.

Table 1. Results of the patient's laboratory tests on consecutive days after the first symptoms of intoxication.

	1 day	2nd day	3rd day	4th day	5th day	6th day	7th day	8th day
PT (9,4-12,50 [S]		13,4	16,2	14,9	15,0	13,9	13,6	13,3
INR (0,8-1,15)		1,15	1,39	1,28	1,29	1,20	1,17	1,14
ALT (5-31) [U/l]	42	2524	1971	1155	822	660	514	411
AST (5-32) [U/l]	42	1780	1024	291	129	91	68	56

This case report presents severe intoxication with Amanita phalloides. Although the patient did not have multiple organ failure, in the literature, intoxications with ALT elevation of more than 850 U/l are considered severe [22]. Symptomatology in cases of toadstool poisoning is three-phase. After an asymptomatic latency period, severe diarrhea, vomiting, abdominal pain and nausea occur within 8-24 hours. The symptoms then pass and the liver deteriorates dramatically (increase in transaminases and decrease in clotting factors). The end-stage of poisoning, i.e. 3-5 days after ingestion of the fungus, may be multi-organ failure with cardiac and renal decompensation and acute liver dystrophy. The course of poisoning in the described patient was very similar. The patient was admitted to hospital the day after ingesting the mushroom with nausea, vomiting and abdominal pain. 2 days after eating the toadstool, there was a severe hepatic reaction with a transaminase output of more than 2000 U/L, an increase in bilirubin and INR. However, in this case, after conservative treatment was instituted, a systematic improvement was achieved, with no evidence of multiple organ failure. In the patient, the presence of amanitin was confirmed by urinalysis. Commonly, the toxin can be confirmed by radioimmunoassay in urine and serum and botanical classification by experts confirms the type of fungus.

Turning to treatment, a specific antidote for amatoxins is not available and therapy is only supportive. Among the treatments used to remove toxins is gastrointestinal lavage with charcoal or other absorptive perfusion. However, this method is only applicable in the initial phase of intoxication and the vast majority of patients are admitted at a relatively late stage of intoxication. Another method is rapidly initiated hemodialysis and simultaneous forced diuresis. Carbohemoperfusion can also be used. However, strict criteria must be met, such as time less than 48 hours after ingestion, biochemical signs of toxicity, intake of a potentially lethal dose and an increase in serum enzymes.

Pharmacologically, penicillin G and silibinin are used. Penicillin G administered intravenously at a dose of 1 000 000 IU/kg/d provides some protection against liver toxicity. The chemically modified silibinin, silibinin disodium dihydrosuccinate (Legalon), is administered intravenously at a dose of 20-50 mg/kg/day. In cases of disseminated intravascular coagulopathy and coagulation disorders, heparin, glutamate-pyruvate transaminase and antithrombin III should be given. At any stage, and especially if hepatic encephalopathy is suspected, liver transplantation should be considered. The main indications for liver transplantation are initially reduced PT (prothrombin time) and factor V levels (<10%). In one study, the majority of patients with PT values below 10% died, while patients with minimal values of 40% or more survived [23]. Serum transaminases had a lower correlation with survival rates and creatinine showed no correlation with survival rates. Six cases of patients treated with extracorporeal albumin dialysis are also reported in the literature [24]. One patient died, the other five did not develop multiple organ failure.

The case of our patient and the patients included in other studies show how important it is to know what mushrooms are being harvested, as the general increase in collecting activities, whether for food or medicinal purposes, increases the risk of poisoning. Mycologists recommend being particularly careful when collecting or

buying wild mushrooms for consumption. If mushrooms of unknown origin are possessed, samples should first be examined, identified and declared edible by an experienced mycologist [25].

The increasing problem of mushroom poisoning in countries where it has not previously been a major problem is also due to the current migration crisis. Refugees, due to the low socio-economic level and, above all, hunger, collect and eat mushrooms, disregarding the type and species of mushroom [26]. In 2016, the Azerbaijan Medical Association Journal posted an article about the deaths of six Syrian migrants after eating poisonous mushrooms identified as Amanita phalloides [27]. A similar situation was reported in Denmark in 2017. It concerned the death of two children and the hospitalization of nine other family members, probably from the Congo, after being poisoned by mushrooms of unknown origin [28]. The German media, on the other hand, indicate that cases of mushroom poisoning in Germany have steadily increased since the beginning of the refugee crisis, including five refugees from Eastern Europe receiving treatment after eating Amanita phalloides [29].

In addition, prompt diagnosis and referral of the patient to a specialized center to implement the correct procedure is extremely important. An article from 2022 presented the case of a 28-year-old man, following poisoning by a phalloides toadstool, whose diagnostic pathway was guided inappropriately and too slowly [30]. Because the correct diagnosis was made too late, the typical treatment of A. phalloides poisoning was ineffective. As a result, the patient developed life-threatening liver failure, requiring a liver transplant from a deceased donor. This case serves as a cautionary tale for clinicians who first come into contact with a patient poisoned by the fungus. In such situations, symptoms should not be ignored and the information provided by the patient during history-taking should not be downplayed, as this may prevent unnecessary medical interventions.

CONCLUSION

Fungal intoxications are not a very common occurrence and in the vast majority of cases they take the form of mild symptoms associated primarily with gastrointestinal involvement. However, cases of poisoning with potentially fatal mushrooms have been reported both in Poland and worldwide, with a fulminant course and development of very severe complications. The lack of knowledge of mushroom pickers and the failure to make an accurate medical diagnosis are two key reasons for the serious course of the disease. This is why it is so important to cooperate with the local poison center and mycologist to identify the fungi and decisions relating to the correct management of the patient, especially in high-risk cases [31]. In addition, preventive procedures should be developed, such as specialized training of medical staff and information material for the public (e.g. via websites or other mass media) on the correct handling of potentially life-threatening fungi, especially during the high-risk fungal poisoning season.

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