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Amanita Phalloides Mushroom Poisoning: A Cluster of Four Fatalities

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ABSTRACT: A group of four illegal aliens had been without food for several days when they found a group of wild mushrooms growing in a field in Southern California. Each man consumed a meal of one to six fried mushrooms. Two days after eating the mushrooms, all four men developed abdominal pain, nausea, diarrhea, and intractable vomiting. Three days after consuming the mushrooms, all four were hospitalized and their clinical courses rapidly deteriorated to refractory hepatorenal failure and coma. Three of the victims died three days after admission to the hospital and the fourth died eight days after admission. The autopsy findings are presented and the mechanism of Amanita phalloides mushroom poisoning is discussed.

KEYWORDS: pathology and biology, poisoning, Amanita Phalloides mushrooms, death

Fatalities as a result of mushroom poisoning will occur as long as individuals collect and eat wild mushrooms. In 1985, the American Association of Poison Control Centers reported 7245 human mushroom poisoning incidents out of 900 513 exposures to all toxic agents [*1*]. Of these 7245 cases, 6000 involved children and only 18 had potentially life-threatening symptoms; 4 fatalities were reported. Deaths associated with Amanita phalloides mushrooms usually involve amateur mushroom collectors and their families who have mistakenly eaten these mushrooms. Probably the worst single incident of Amanita phalloides mushroom poisoning occurred near Poznan, Poland, in 1918 when 31 school children died after eating a dish prepared from these mushrooms at school [*2*]. Deaths from Amanita phalloides mushroom poisoning in the United States are rare and we report a cluster of four deaths associated with the consumption of a single meal of Amanita phalloides mushrooms.

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Case Reports

In March of 1985, four illegal aliens who had been without food for several days consumed fried wild mushrooms after picking them in a field in Southern California. They each ate between one and six mushrooms and, approximately one to two days after consuming the mushrooms, all four men developed gastrointestinal symptoms including nausea, vomiting, abdominal cramps, and watery diarrhea. They went to a local mission for the homeless where they were unable to eat. Their symptoms continued and they were taken to two local hospitals.

On admission, all four men appeared dehydrated and three were hypotensive (blood pressure less than 100/50). One man stated that he had developed "white stools." Three of the men were initially assessed as having gastroenteritis or acute hepatitis or both. The fourth man had been admitted to a separate hospital with the diagnosis of possible acute mushroom poisoning when he was able to identify the mushrooms he had consumed from a picture of Amanita phalloides. The hospital where the other three victims were being treated was contacted and the diagnosis and treatment were modified accordingly.

During their hospitalizations, each of the victims showed evidence of both hepatic and renal failure with serum glutamic-oxaloacetic transaminase (SGOT) values ranging from 6680 to 16 520 U/L; alkaline phosphatase from 144 to 590 IU/L; total bilirubin from 3.1 to 4.6 md/dL and blood urea nitrogen (BUN) from 6 to 59 mg/dL. Creatinine values ranged from 5.0 to 5.9 mg/dL. Three of the men died within three days of admission (five days after eating the mushrooms), and the fourth died eight days following admission (eleven days after eating the mushrooms).

At autopsy, each of the men demonstrated findings typical of hepatic failure (that is, anasarca, scleral icterus, serous effusions, and pulmonary edema), and their livers were grossly necrotic. The kidneys were pale, swollen, and in one case displayed multiple cortical infarctions (Fig. 1). The heart in each case demonstrated hemorrhage ranging from patchy petechiae to confluent subendocardial left ventricular intramuscular hemorrhage (Fig. 2). Two of the men demonstrated hemorrhagic gastritis, one had focal rectal ulcers, and the fourth had no gastrointestinal abnormalities. Microscopically, the liver pathology ranged from hepatic necrosis with preservation of hepatocytes and evidence of regeneration to massive hepa-

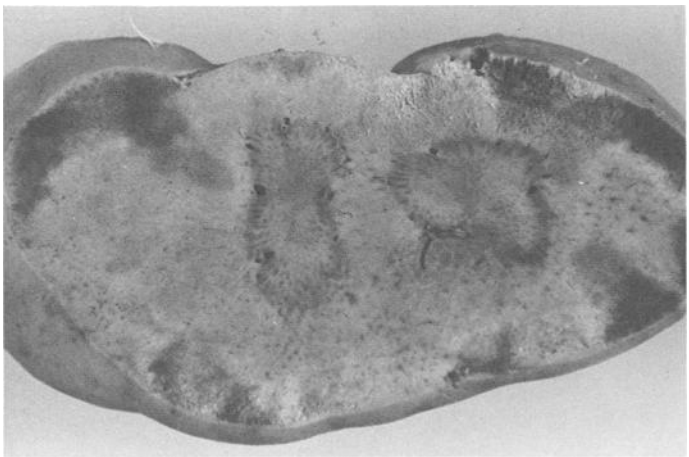

FIG. 1—*Pale, swollen kidney typical of renal failure displays multiple peripheral wedge-shaped infarctions* (dark areas).

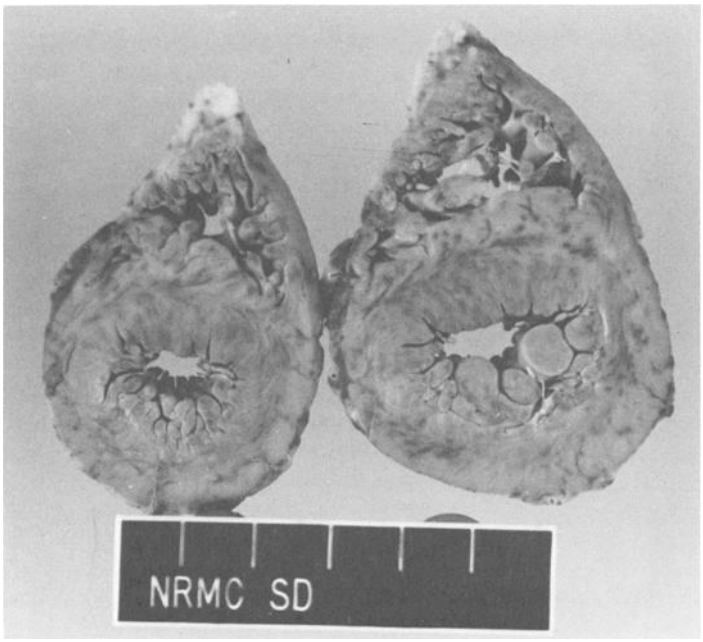

FIG. 2—*Cross sections of heart demonstrate multiple myocardial petechiae. Markers in scale represent 0.5 in. (1.3 cm).*

tocellular necrosis with preservation of only portal triads and vascular structures (Fig. 3). Granular casts were seen in the kidneys of all victims.

Discussion

Of the approximately 2000 known species of mushrooms, fewer than 50 are considered to be poisonous [*3*]. Of the poisonous mushrooms, Amanita phalloides is the most toxic and it is currently responsible for virtually all fatal mushroom poisonings in this country [*4*]. For many years the Amanita phalloides mushroom was primarily a species involved in fatal poisonings in Europe and this species has only been positively identified as growing wild in the United States since the 1970s. The mushroom is believed to have made the transatlantic migration as spores either carried by air currents or contained in the soil of plants imported into this country [*2*]. The typical Amanita phalloides mushroom (common name "death cap") has an olive green cap, white gills, a skirt-like ring on the stipe, and a volva. It reportedly has a pleasant taste.

California has had more reported serious and fatal cases of mushroom poisoning than any other state [*2*]. The Amanita phalloides has frequently been mistaken in California for a common edible mushroom, Amanita calyptroderm. The Amanita calyptroderm species is large with an orange to orange-brown cap that is partially covered with a patch on the top.

Various myths as to the toxicity of mushrooms have been promulgated over the years [*2*]. The more common myths are as follows:

1. A silver coin will darken when placed in water containing a poisonous mushroom but will retain its luster if the species is edible.
2. A mushroom is edible if its skin can be peeled off easily.
3. A mushroom is edible if it is eaten by animals.

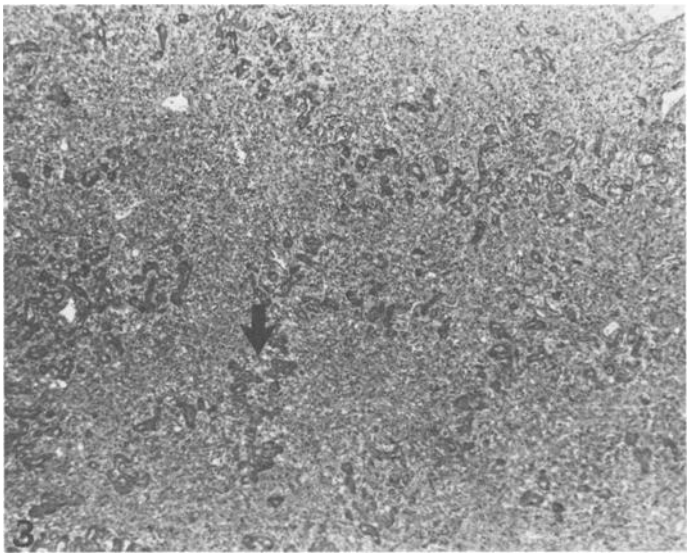

FIG. 3—*Photomicrograph shows massive hepatic necrosis with preservation of portal areas* (arrows) *(×75).*

4. A mushroom growing on wood is edible.
5. Boiling mushrooms in water with (or without) salt will remove all poisons.

All of the above statements are myths as to the toxic nature of the various species of wild mushrooms. Even experienced mycologists may have difficulty in the proper discrimination of toxic versus nontoxic species and have been known to make fatal errors.

The toxins of Amanita phalloides are all cyclopeptides and divided into amatoxins and phallotoxins. They are heat stable and are ten times deadlier than cyanides [*2*]. The total concentration of these toxins has been determined to be 0.2 to 0.4 mg per gram of fresh mushroom tissue [*5*]. Phallotoxins have an almost immediate effect and act by binding to cellular membranes. They are responsible for the initial gastrointestinal symptoms following mushroom ingestion. Amatoxins are slower acting and display an 18 to 24-h latent period. They work by inhibiting the transcription of deoxyribonucleic acid (DNA) to ribonucleic acid (RNA) by inhibition of nuclear RNA polymerase I. As a consequence, protein synthesis in blocked followed by necrosis of the affected cells. The gastrointestinal epithelium, hepatocytes, and renal tubular epithelium are the predominate cell types affected. This decrease in protein synthesis also results in the decreased production of the coagulation Factor V, VII, and VIII leading to hemorrhage. Amatoxin is also found in the Galerinas mushroom which may also contain toxic levels [*2*]. This species of mushroom is small and dingy brown with long, thin brittle skins.

The disease process that occurs after eating Amanita phalloides mushrooms is divided into three stages. The first stage begins abruptly 6 to 24 h after ingestion and is characterized by nausea and vomiting, severe abdominal cramps, and profuse watery diarrhea. There are profound fluid and electrolyte losses which frequently lead to fever, dehydration, tachycardia, and hypotension. The second or refractory stage demonstrates spontaneous clinical improvement lasting from 12 to 24 h. The third stage is characterized by severe hepatocellular and renal tubular damage which is evidenced by rapidly increasing levels of hepatic transaminases as well as elevated levels of BUN and creatinine. The massive hepatocellular necrosis and acute renal failure cause profound metabolic derangements, which terminate in

coma. Death usually occurs four to seven days after consumption of the poisonous mushrooms.

Various therapeutic modalities have been tried as treatment for amatoxin poisoning. Since symptoms do not begin until several hours after ingestion, emesis is rarely beneficial. Hemodialysis may be useful in treating the renal failure. Intensive supportive measures are needed. Thioctic acid has been proposed as a treatment for amatoxin poisoning. It was introduced as a treatment in 1959 by Kubick and is generally administered intravenously at a dose of 400 mg/day divided into four doses [5]. This regimen lasts for thirteen days and the only reported adverse effect is hypoglycemia. This treatment has recently lost favor, and the San Francisco Bay Regional Poison Center stopped recommending its use in 1984 [6]. Activated charcoal is sometimes used to try to bind the amatoxin. Penicillin, phenylbutazone, and chloramphenical have also been used in treatment. These agents work by competing with binding sites on serum proteins thereby causing more toxin to be free for renal excretion.

There are no chemical or serologic tests for mycotoxins that will detect these substances in human or animal tissues. The diagnosis of mushroom poisoning is largely based upon the history of mushroom consumption by the victims as well as recognition of the offending fungus by the mushroom collector. Early in the course of the disease, diagnostic spores may be present in the gastric and intestinal contents, but, as a result of vomiting and diarrhea, spores will only be present for a few hours. Gastric contents, mushroom samples (if available), and stool specimens should be submitted for identification of spores [7]. If mushrooms are submitted, they should be freshly picked and include the cap, stem, and base. The specimen should be wrapped loosely in tissue paper and never placed in a plastic bag.

Conclusion

In conclusion, although Amanita phalloides mushroom poisoning is rare, it must be considered in the differential diagnosis of gastroenteritis in combination with acute heptitis and renal failure. In those areas where poisonous mushrooms grow, public education should stress the dangers of consuming wild mushrooms. Medical examiners must remain alert to the possibility of Amanita phalloides mushroom poisoning and its subsequent morbidity and mortality.

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