Editorials

A Primer for Clinicians on Mushroom Poisoning in the West

THE HISTORY OF mushrooms is surrounded by much mystery and superstition. This mystique is perhaps most commonly associated with the mushroom's poisonous reputation. Most mushroom species, however, are not poisonous; of the 5,000 known species of mushrooms, fewer than 100 are poisonous to humans. Poisonous mushrooms have no characteristic smell or taste and often resemble edible mushrooms. Cooking seldom destroys the deadly toxins. This poses a possibly lethal risk for novice enthusiasts of mushroom hunting.

Hunting and consuming of wild mushrooms have become increasingly popular, an epicurean delight for some, and for others, a means of subsistence. Unfortunately, errors in identification lead to many thousands of reported poisonings all over the world each year. Almost 10,000 mushroom exposures are reported annually to the American Association of Poison Control Centers, representing just under 1% of their calls.[1] Whereas severe mushroom poisonings fortunately are rare, about 100 to 200 fatalities per year due to mushroom poisoning occur in the United States and Europe. Moreover, 90% to 95% of deaths are caused by a single species, *Amanita phalloides*. This species is reported with increasing frequency in this country, especially along the West Coast.[2]

The prevention of mushroom poisoning by increased knowledge continues to be a reasonable goal. With caution, collecting and cooking wild mushrooms is relatively safe. Unfortunately, edible and nonedible mushrooms are frequently misidentified by mushroom collectors. As the saying goes, "There are old mushroom hunters and bold mushroom hunters, but there are no old, bold mushroom hunters." Thus, more emphasis on public education through service announcements could be warranted and cost effective if directed at appropriately targeted groups and at appropriate times of the year. The article in this issue by Jeffrey Jacobs, MD, MPH; Julie Von Behren, MPH; and Richard Kreutzer, MD, "Serious Mushroom Poisonings in California Requiring Hospital Admission, 1990 Through 1994," provides from their database some of this needed demographic targeting.[3]

One of the most interesting findings in this report is the increased incidence of hospitalization for mushroom poisoning in children younger than 5 years. This point certainly behooves parents to teach small children not to eat any mushroom they come across. Jacobs and coworkers report that certain counties in California, such as Los Angeles, Riverside, San Bernardino, Orange, San Diego, Santa Clara, and Fresno, have the most hospital admissions and that Humboldt, Merced, Yolo, Riverside, Fresno, Marin, and San Luis Obispo counties have the highest incidence. They also note that there is an increased incidence of poisoning in the months July through October. These geographic locations and times of year reflect not only natural growth patterns of mushrooms, but probably also the increased incidence of mushroom collecting because of the higher population concentrations in these locations and an increased focus on the out-of-doors during these months. We could conclude that educational efforts for poisoning prevention in California should occur beginning in the summer and in the above-mentioned counties. Mushroom collectors should know how to identify mushrooms with absolute certainty, and if there is even the slightest concern, leave it there!

If there is any problem in the methods of their study, it would be under identification of cases of serious mushroom poisoning because of incorrect diagnostic coding. Death due to mushroom poisoning may be hidden because fatalities may be recorded as hepatic failure, increased intracranial pressure, and the like. Also, the species involved unfortunately could not be determined from their database.

Because the total prevention of mushroom poisonings is a goal likely never to be reached, it is important for clinicians to focus on diagnosis and treatment. The differential diagnosis of mushroom poisoning includes food poisoning, gastroenteritis, infectious colitis, acetaminophen overdose, carbon tetrachloride ingestion, psychosomatic symptoms, or psychoses. As with any clinical problem, the diagnosis of mushroom poisoning must at least be considered in a patient with the appropriate symptoms.

Based on the classification of toxin and clinical symptoms, there are different types of mushroom poisoning, including syndromes caused by *A phalloides, Cortinarius orellanus, Gyromitra* species, *Amanita muscaria, Amanita pantherina*, psilocybin, gastrointestinal mushroom, *Coprinus quadrifidus*, and *Paxillus involutus*. Poisonings caused by *A phalloides, C orellanus*, or *Gyromitra* or *Paxillus* species are generally serious and often require intensive care treatment. Considerable differences in symptoms may be seen with the various mushroom species due to seasonal and location variance in the toxin content, variance in oral bioavailability, differences in metabolic activation, and differences in toxin susceptibility.[4] In general, these symptoms can include nausea, vomiting, abdominal cramping, diarrhea, chills, thirst, oliguria or anuria, weakness, dizziness, diaphoresis, hyperthermia, muscarinic symptoms, vertigo, bradycardia, hypotension, headache, euphoria, disorientation, hallucinations, and seizures.

Most patients present with gastrointestinal symptoms. It is important to establish the latency period of symptoms after ingestion as this can help determine which of the various syndromes are most likely. For example, of all fatal mushroom poisonings, 90% to 95% are attributable to *A phalloides*, with symptoms that range from those of gastroenteritis to fulminant hepatic failure. Symptoms of gastroenteritis begin 6 to 24 hours after the initial ingestion of the mushroom, characterized by severe, crampy abdomi-

nal pain; nausea; vomiting; and watery diarrhea. Hepatic failure can be seen as early as 48 hours after ingestion. In fatal cases, death occurs 6 to 16 days after ingestion.

The diagnosis of mushroom poisoning is based on careful history taking primarily, the identification of leftover mushrooms, chemical analyses, or both. It is important to elicit the first onset of symptoms and any symptoms that others who shared in the ingestion might have had. If possible, it is helpful to know the species involved, including the amount and type of preparation.

Toxins from mushroom remnants can be identified by chemical analysis using thin-layer chromatography, high-performance liquid chromatography, or gas chromatography and mass spectrometry. In those patients with suspected *A phalloides* poisoning, body fluids can be analyzed for α-amanitine by a recently developed radioimmunoassay.[4] Measuring alkaline phosphatase isoenzyme levels may be helpful. Unfortunately, positive identification is often not made; in less than 5% of the cases is the mushroom species actually identified.[1] Whether positively identified or not, when a clinician suspects the presence of mushroom poisoning, he or she should contact the local poison control center. The poison control center staff can provide much-needed, appropriate, and detailed information on treatment.

The initial laboratory evaluation should include prothrombin time, platelet count, complete blood count, blood gas, and serum glucose, electrolytes, phosphate, creatinine, urea, alanine and aspartate aminotransferase, lactate dehydrogenase, and alkaline phosphatase levels. If available, factor analysis, fibrinogen, and lactate levels may be helpful.

Because of the possible development of fulminant hepatic failure, clinicians must be watchful for rising hepatic enzyme levels, especially in those patients with *A phalloides* poisoning. Throughout the patient's course, basic laboratory measurements should be observed at selected intervals and even as long as a few months after the event.

Appropriate routine treatment includes hydration and electrolyte replacement, primary and secondary detoxification, and sometimes specific antidotes. For example, specific therapy for *A phalloides* ingestion includes the administration of silibinin and penicillin G. Because delay following ingestion until treatment is common, the efficacy of both primary and secondary detoxification is limited. Nevertheless, induced emesis and gastric lavage are indicated. Because of extensive enterohepatic circulation of α-amanitine and other toxins, the repeated administration of activated charcoal is recommended. Secondary detoxification with forced diuresis and saline catharsis is also indicated. Hemodialysis and plasmapheresis are less efficient. Supportive measures can include oxygen, cardiac monitoring, and the administration of atropine sulfate if muscarinic effects are present.

If the patient progresses to hepatic dysfunction, digestive tract decontamination, the repletion of clotting factors, and protein intake restriction are appropriate initial steps. In patients exhibiting hepatic dysfunction, immediate consultation with a hepatologist and preferably with a liver transplantation unit is mandatory. In the most serious cases, early transfer to a liver transplantation center is important to identify appropriate candidates and to begin the search for a donor organ. Liver transplantation offers definitive therapy for selected severely poisoned patients. In addition, auxiliary transplantation has been recently shown in a few cases to be an excellent option because this transplant can be removed and the patient taken off immunosuppression after the native liver regenerates. A poor outcome is more likely if the decision for liver transplantation is delayed too long.

On the other hand, we certainly would not wish to pursue this form of therapy for patients who would recover spontaneously. Reasonable prognostic indicators are available from somewhat limited data, and criteria for indications of liver transplantation have been developed: Indications for transplantation include primarily a markedly prolonged prothrombin time that is only partially correctable and a constellation of findings that can include metabolic acidosis, hypoglycemia, hypofibrinogenemia, reduced factor levels, and an increased serum ammonia level following a pronounced elevation in serum aminotransferase levels. Clinicians should not wait for hepatic failure to advance to hepatic encephalopathy, azotemia, or jaundice.[5]

In summary, the article by Jacobs and colleagues helps to emphasize important points about mushroom poisoning, including prevention, diagnosis, and treatment. Mortality from mushroom poisoning has decreased in recent decades due to the vast improvements in critical care treatment, poison control center information, and the availability of liver transplantation. Historically, *A phalloides* syndrome has carried a mortality of greater than 50%, but with improved treatment in recent years, this has dropped to less than 20%. The cases of the five patients mentioned in the introduction to this article demonstrate the wide spectrum of outcomes of mushroom poisonings, with three recovering in a few days, one requiring liver transplantation, and one dying without appropriate therapy.[3]

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