Serious Mushroom Poisonings in California Requiring Hospital Admission, 1990 Through 1994

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Hunting and consuming wild mushrooms is an increasingly popular avocation, as well as a means of subsistence for certain groups in the United States. Consuming wild mushrooms is relatively safe for persons with the necessary expert knowledge. Each year, however, tragic deaths or illnesses occur when unsuspecting persons ingest toxic mushrooms. We conjectured that there may be vulnerable subgroups that should be targeted for public service announcements and education about mushrooms. Therefore, we reviewed California's hospital discharge database from 1990 through 1994 to ascertain demographic characteristics and outcomes of "serious" mushroom exposures requiring a hospital admission. Children younger than 5 years have a higher rate of hospital admission for "serious" poisonings. Therefore, parents should be reminded of the need for supervising children in areas with wild mushrooms. We did not find evidence that poisoning rates differed significantly between ethnic or racial groups, but this finding may be limited by a failure of these groups to seek care after becoming ill. We discuss briefly the symptoms and treatment of cases of possibly lethal ingestion of amatoxin-containing species of mushrooms.

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In early February 1996, five northern Californians ingested poisonous mushrooms, resulting in one death and a lifesaving liver transplantation in another. In the fatal case, a migrant farm laborer became ill after eating mushrooms he picked from a field. He died three days later, never having sought medical attention. In the other case, a family picked wild mushrooms with a friend, who assured them that the mushrooms were safe to eat. Six to ten hours later, the family members—a woman, her two sons, and a daughter-began vomiting and having profuse watery diarrhea and crampy abdominal pains, and they sought medical attention. Over the next several days, three of the family members' liver function deteriorated but then improved. The young girl's health continued to deteriorate, and a partial liver transplantation was done. Her health and liver function rebounded after the procedure, allowing for the removal of the temporary transplant.

Background

These cases illustrate the possible risk associated with consuming wild mushrooms without appropriate expert knowledge. Most mushrooms are not poisonous. Of the 5,000 known species of mushrooms, fewer than 100 are poisonous to humans.^{1,2*} The origin of the word mushroom dates back to early Greek times and is best translated to the word "mucus."² Mushrooms were thought to have arisen from slimy, mucoid matter and were believed linked to frogs and toads, hence the name "toadstool." Whereas poisonous properties of wild mushrooms have been recognized since ancient times, most people consuming wild mushrooms have lived to tell about it. Euripides is credited with the earliest report of mushroom poisoning in 430 BC when, on a trip to Icarus, he recorded the fatal poisoning of a woman and her three children. Famous victims of mushroom poisoning include Pope Clement II, the widow of Tsar Alexis, and Charles VI of France.²

Today wild mushrooms are greatly prized as a culinary treat, and the popularity of mushroom hunting is growing throughout the world. Numerous guidebooks are available, and expert knowledge from mycologic clubs and societies enables novice collectors to forage with increased awareness of the dangers of toxic mushrooms. Despite this increased awareness, however, seri-

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^{*}See also the editorial by C.W. Pinson, MD, MBA, and Anne L. Bradley, MD, "A Primer for Clinicians on Mushroom Poisoning in the West," on pages 318–319 of this issue.



Figure 1.—The graph shows the number of admissions to California hospitals for mushroom poisoning by year during 1990 through 1994 (n = 146). They averaged 29.2 per year during this period.



Figure 2.—The number of admissions to California hospitals for mushroom poisoning for 1990 through 1994 (n = 146) is shown by month. Admissions for mushroom poisoning were more common in summer and early fall, peaking in August.

ous poisoning episodes continue to occur. This problem is especially common in Europe, where wild mushrooms are more widely collected and several hundred deaths occur annually.^{1,3}

A number of conditions are associated with mushroom poisoning. Many adults seem unaware of the risks of eating wild mushrooms. Inexperienced mushroom hunters may mistake the identity of a mushroom. Immigrants may come from areas where only edible look-alikes exist. Unsupervised infants, persons looking for hallucinogenic materials, and families who are told by "authorities" that the mushrooms are edible may be poisoned as well.⁴

Cases and Methods

We examined California's cases of "serious" mushroom poisonings requiring admission to a hospital. We elected to use the California Office of Statewide Health Planning and Development (OSHPD) Discharge Database.⁵ This database provided us with pertinent demo-



Figure 3.—The number of admissions to California hospitals for mushroom poisoning (rate per 10^s person-years shown above bars) during 1990 through 1994 (n = 146) is shown by selected counties of residence. The most hospital admissions for mushroom poisoning were among residents of Los Angeles County. When cases were adjusted by population, Humboldt County (northern California) had the highest hospital admission rate in the state, with 0.67 cases per 10^s person-years.

graphic information on mushroom poisoning victims and the outcome of cases.

The OSHPD Discharge Database contains computerized records for all hospital admissions in California. Cases were selected for the years 1990 through 1994, if the primary E code was listed as mushroom poisoning (International Classification of Diseases, 9th revision, code E 865.5),6 regardless of the actual primary diagnosis code. We obtained demographic information, including the age, race, and sex of each patient. Hospital information included county of residence for each poisoning case and the month, day of week, and year of the admission. Outcome information included the length of hospital stay, the primary procedure done in the hospital, and the disposition of each patient. To check for victims of mushroom poisoning who died before being admitted to a hospital, we searched the California Death Registry⁷ for cause by mushroom poisoning (death code 865.5) from 1985 through 1994. We found only four cases, which occurred in March 1985, all in San Diego County. These were not included in our data.

Frequency counts for some of the OSHPD data were standardized into rates by calculating person-years for the population at risk (all Californians). These data were obtained from 1990 census information.⁸ Statistical

Demographic		nber (%)	Rate/10 ^s person-yr
Age, yr			
<5	26	(17.8)	0.22
5 to 18	25	(17.1)	0.09
19 to 44	56	(38.4)	0.08
45 to 64	22	(15.1)	0.09
≥65	17	(11.6)	0.11
Total	146	(100.0)	
Race			
White	87	(59.6)	0.1
Black	7	(4.8)	0.07
Hispanic	37	(25.3)	0.1
Native American	1	(0.7)	*
Asian	10	(6.9)	0.07
Unknown	4	(2.7)	
Total	146	(100.0)	
Sex			
Male	84	(57.5)	0.10
Female	62	(42.5)	0.08
Total	146	(100.0)	

TABLE 1.—Demographics of Persons in California With Mushroom

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TABLE 2.—Length of Stay, Principal Procedures Performed, and Disposition for Persons in California With Mushroom Poisoning Requiring Hospital Admission, 1990 Through 1994

Length of stay, days 0*
0*
1
2
3
4
5
6
7
8
9
Total
Principal procedures
Cardiac monitoring 14 (32.5)
Gastric lavage
Other diagnostic tests 11 (25.6)
Peritoneal lavage
Total
Discharge disposition
Routine discharge
Short-term care
Skilled nursing 1 (0.7)
Against medical advice
Home health services 1 (0.7)
Deceased 0 (0.0)
Total
*No overnight stay.

analysis was performed using the STATA statistical software packages.⁹

Results

There were 146 admissions for mushroom poisoning to California hospitals from 1990 through 1994, averaging more than 29 admissions per year and ranging from 17 in 1990 to 35 in 1992 (Figure 1). We were unable to determine the species of mushroom associated with each case. Most poisoning admissions were in the summer and fall months (Figure 2), peaking during August. The counties with the greatest number of hospital admissions for mushroom poisoning were Los Angeles (36), Riverside (14), San Bernardino (12), Orange (10), San Diego (9), Santa Clara (8), and Fresno (6) (Figure 3). When these county frequencies were divided by personyears, Humboldt County had the highest rate of admissions for mushroom poisoning, with 0.67 cases per 100,000 person-years, followed by Merced (0.45), Yolo (0.28), Riverside (0.24), Fresno (0.18), Marin (0.17), and Stanislaus (0.16).

Demographics for all cases admitted to a hospital for mushroom poisoning are shown in Table 1. The distribution of hospital admissions by age category indicates that children younger than 5 years had the highest rate of admission for poisoning, with 0.22 per 100,000 personyears. Adults aged 65 years or older had the next highest rate (0.11). The distribution of cases by race indicates that non-Hispanic whites had the greatest number of hospital admissions, followed by Hispanics, Asians, African Americans, and Native Americans. Four persons were not classified by race. Males were admitted to a hospital for mushroom poisoning more frequently than females by a ratio of 1.4 to 1.

Once a poisoning victim was admitted to a hospital, the primary procedures performed included gastric lavage, cardiac monitoring, other diagnostic procedures such as lumbar punctures and respiratory monitoring, and peritoneal dialysis (Table 2). No liver transplantations were done for poisoning victims during this time period. The typical length of stay was less than 4 days in more than 88% of the cases. The great majority (93%) of the patients improved and were routinely discharged, 3% left against medical advice, and none died.

Discussion

Hospital admissions for mushroom poisoning in California occur more frequently in the warmer months and reach their peak in August. In general, the typical peak season for wild mushrooms coincides with the rainy season. In California, this is usually from late October through March. The farther north along the West Coast, the season arrives progressively earlier and is compressed; the farther south, it starts correspondingly later and is more erratic.^{10(p25)} We are unsure of the reasons behind the discordance between the peak hospital admission rate and peak growth and were unable to review the individual cases to ascertain the offending mushroom. The California admissions peak for mushroom poisoning in summer may, in large part, be due to the mistaken ingestion of poisonous Agaricus species (which resemble other edible types), resulting in gastrointestinal symptoms and admission for dehydration.

The location of hospitalizations for mushroom poisoning reflects the great diversity of habitats where poisonous wild mushrooms may be found. More poisonings might be expected in the wetter northern California counties, but drier, more southerly counties such as San Diego and Orange have a substantial number of hospital admissions for poisoning as well. High rates in San Joaquin Valley counties of Fresno and Merced indicate that toxic wild mushrooms can be encountered throughout the state and not just along the coast. In addition, more urban counties like Los Angeles County have large numbers of admissions because they have a large population that may hunt for wild mushrooms. Because wild mushrooms are capable of growing on lawns and median strips, excursions into more rural areas may not be necessary for finding poisonous mushrooms.

Our data suggest that, compared with adults, young children—younger than 5 years—have more than twice the risk of being admitted to a hospital for eating a poisonous mushroom. One scenario may entail an unsupervised or poorly supervised child playing outdoors and eating a mushroom he or she pulls from the ground. In a survey of the American Association of Poison Control Center's 1989 National Data Collection System, it was found that 81% of reported cases of mushroom exposures occurred in those younger than 6 years.¹¹ Another explanation may be metabolic differences, with children being more vulnerable to mushroom toxins than adults. In addition, clinicians may be more apt to admit children to a hospital for the intravenous treatment of excessive fluid and electrolyte loss through vomiting and diarrhea, whereas adults are more likely to be discharged with instructions to increase their oral intake of fluids.

Poisonings have been reported to occur when immigrants coming from areas where only edible mushrooms exist encounter a poisonous look-alike in their new community.4 We had heard this story in reference to Asian communities who forage for wild mushrooms for subsistence. But our data do not reflect an increased prevalence of admissions for mushroom poisoning for any racial group. The lack of an elevation of risk in any ethnic community would seem to lessen the need for increased education targeted toward a specific racial group. This suggestion should be tempered with the understanding that our data may be limited because immigrant groups may also be less likely to seek care. despite becoming ill after eating toxic mushrooms, as the brief report of the farmworker in the initial paragraph illustrates.

The duration of hospital stays was short (<4 days) in most cases, reflecting the generally favorable prognosis after eating toxic mushrooms. Mushrooms produce different types of poisoning-that is, gastrointestinal irritation from unknown toxins; perspiration and lacrimation by muscarine; hallucinations from psilocybin; headache, sweating, and palpitations from coprine; and hepatotoxicity from amatoxin-that are called mycetisms.^{2,3} The overwhelming majority of patients with poisonous ingestions have a good prognosis, and the only treatment needed is inducing emesis and supportive care (hydration). The ingestion of mushrooms containing amatoxin (species of Amanita, Galerina, and Lepiota), however, is the exception to this rule. Of these, the most lethal is Amanita phalloides, which has been estimated to cause 90% to 95% of all mushroom poisoning deaths.^{1,2} Eating a single mushroom weighing about 50 grams (containing 5 mg of toxin) is potentially lethal.² Whereas most of the reported cases of A phalloides poisoning have occurred in Europe, cases have increasingly been reported in the United States, especially on the West Coast.1 Amanita mushrooms flourish from late summer to fall in temperate zones, have a proclivity for oak trees, and may grow on urban lawns. Amanita phalloides are large mushrooms, look appetizing, have no offensive taste to warn of their danger, and can be mistaken as an edible species, especially by amateur mycologists.^{1,12–14} Because of the unusually warm weather in northern California during fall and winter in 1995 and 1996, fruiting A phalloides mushrooms were observed well past their normal season.

Amatoxins are heat-stable, cyclic octapeptides that preferentially affect rapidly dividing cells of the liver and gastrointestinal tract. Kidney, pancreatic, testicular, and lymphocytic cells are also affected. Amatoxins interfere with DNA transcription by inhibiting the synthesis of messenger RNA. Protein synthesis is disrupted, resulting in cell necrosis and death. Studies in dogs have shown that urinary elimination of this toxin is rapid, with 90% of an administered dose being eliminated within five hours. Hepatic uptake occurs through the bile salt transport system. There is substantial biliary excretion with an enterohepatic recirculation of toxin.^{1,15}

The onset of symptoms is the most helpful clue as to whether A phalloides poisoning has occurred. Poisoning should be presumed to be due to amatoxin if there is a latency period of 6 to 24 hours following ingestion (mean, 10 to 12 hours), in contrast to an earlier onset for other mushroom poisonings. The gastrointestinal phase follows the latent period and is characterized by crampy abdominal pain, nausea, vomiting, and profuse watery diarrhea. This phase generally lasts for 12 to 24 hours and can be complicated by severe dehydration and electrolyte disturbances. Another latent period ensues at this point in which the patient feels better, but clinical measures usually show evidence of hepatic injury. Finally, the hepatic phase begins 48 to 96 hours after ingestion and is heralded by rising aminotransferase levels, coagulopathy, and jaundice. Encephalopathy, renal failure, and death from liver failure may ensue within two weeks.1,2,16

Formerly, fatality rates of more than 50% were common following the ingestion of *A phalloides*. Improved treatment procedures, aggressive management, and the development of liver transplantation have reduced the overall death rate to about 20%. The amount of mushroom ingested is the main prognostic indicator. Thus prompt intervention—gastric lavage and emesis—may have an important effect on limiting the amount of toxin absorbed and reducing mortality.¹⁵

The general principles for the treatment of amatoxin poisoning call for prompt and aggressive measures to reduce amatoxin absorption. Because removing even the smallest amount of amatoxin may improve the prognosis, inducing vomiting and gastric lavage with activated charcoal are recommended to remove any unabsorbed toxin from the gastrointestinal tract and to interrupt the enterohepatic cycle.12,13 Extensive fluid loss from vomiting and diarrhea require aggressive rehydration with intravenous fluids.^{1,13} In addition, because most of the amatoxin is excreted in large quantities in the urine during the first days following ingestion, it is imperative to maintain an adequate diuresis (100 to 200 ml per hour).¹⁷ The value of extracorporeal techniques such as hemoperfusion and dialysis is limited unless they are used sooner than 36 hours after ingestion.1 This may be due to the rapid elimination of amatoxin from plasma, usually before 48 hours.¹⁷ Finally, although there is no specific antidote for amatoxin, the intravenous administration of high doses of penicillin and silibinin (the active ingredient in milk thistle) is recommended, as it is thought that they competitively inhibit amatoxin uptake by hepatocytes.^{1,2,15} It is essential to consult with a poison control center and liver transplantation unit early in the management of a patient with serious mushroom poisoning because hepatic failure can progress rapidly and organ procurement may require several days.¹ In our study, monitoring and gastric lavage were the primary procedures reported. This suggests that observation and symptomatic treatment were typically all that was required in these cases, again emphasizing the relatively favorable prognosis in most cases of poisonous mushroom ingestion. In addition, no liver transplantations were done for mushroom poisoning during the 1990 through 1994 time period.

Conclusion

The hunting and eating of wild mushrooms are increasing in popularity throughout the United States. Although most mushroom species are safe to eat, expert knowledge is essential to avoid the small risk of death and disability from mushroom toxins. Undersupervised voung children are particularly at risk of suffering adverse effects from the ingestion of poisonous mushrooms. Parents should be warned explicitly about allowing unsupervised children free rein of their backyards, where mushrooms may be growing. Ethnic communities should be encouraged to seek information and assistance from local health authorities and mycologic societies about wild mushroom picking. In addition, clinicians should be aware of the possibility that eating a wild mushroom may be fatal, especially if symptoms begin after a six-hour latent period. Furthermore, because several types of mushrooms may grow near each other or be consumed together, the presence of early symptoms should not rule out the possibility of more serious poisoning. Finally, if amatoxin poisoning is suspected, prompt and aggressive treatment should be instituted, with arrangements for early consultation to a liver transplantation unit. Our study showed that in California, "serious" mushroom poisoning has a generally favorable prognosis if the patient reaches a hospital. It is up to public health officials, mycologic societies, and enthusiasts to provide education and expert knowledge to reduce the number of "accidental" ingestions of toxic mushrooms.

REFERENCES

1. Pinson CW, Daya MR, Benner KG, et al: Liver transplantation for severe Amanita phalloides mushroom poisoning. Am J Surg 1990; 159:493–499

2. Barbato MP: Poisoning from accidental ingestion of mushrooms. Med J Aust 1993; 158:842-848

3. Perez-Moreno J, Ferrara-Cerrato R: A review of mushroom poisoning in Mexico. Food Addit Contam 1995; 12:355–360

4. Lehmann PF, Khazan U: Mushroom poisoning by Chlorophyllum molybdites in the Midwest United States. Mycopathologia 1992; 118:3-13

5. Office of Statewide Health Planning and Development Discharge Data Tapes. Sacramento, Calif, 1990–1994

 Commission on Professional and Hospital Activities: International Classification of Diseases, 9th Revised—Clinical Modification. Ann Arbor, Mich, World Health Organization, 1978

7. Death Certificate Statistic Tapes. Sacramento, Calif, Dept of Health Services, 1985-1994

8. 1990 Census of Population and Housing—Summary Population and Housing Characteristics, California. Washington, DC, US Dept of Commerce, 1991

9. STATA Statistical Software: Release 4, 6th edition. College Station, Texas, StataCorp, 1995

 Arora D: Mushrooms Demystified. Berkeley, Calif, Ten Speed Press, 1986
Trestrail JH: Mushroom poisoning in the United States: An analysis of 1989 United States Poison Center data. Clin Toxicol 1991; 29:459–465

12. Cole FM: Amanita phalloides in Victoria. Med J Aust 1993; 158:849–850

13. Becker CE, Tong TG, Boerner U, et al: Diagnosis and treatment of Amanita phalloides-type mushroom poisoning—Use of thioctic acid. West J Med 1976; 125:100–109

14. Seeger R, Stijve T: Occurrence of toxic amanita species, In Faulstich H, Kommerell B, Wieland T (Eds): Amanita Toxins and Poisoning. Baden-Baden, Germany, Verlag Gerhard Witzstrock, 1978, pp 3–17

15. Olesen LL: Amatoxin intoxication. Scand J Urol Nephrol 1990; $24{:}231{-}234$

16. Paydas S, Kocak R, Erturk E, Zaksu HS, Gurcay A: Poisoning due to amatoxin-containing Lepiota species. Br J Clin Pract 1990; 44:450–453

17. Jaeger A, Jehl F, Flesch F, Sauder P, Kopferschmitt J: Kinetics of amatoxins in human poisoning: Therapeutic implications. J Toxicol Clin Toxicol 1993; 31:63-80