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Amanita phalloides-Type Mushroom Poisoning

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In the fall of 1981 the San Francisco Bay Area Regional Poison Control Center received more than 100 calls regarding wild mushroom ingestion. Ten cases, including three fatalities, had all the features of Amanita phalloides *poisoning. Encephalopathy, coma and renal insufficiency occurred in all three patients who died, but did not occur in those who survived. Two of the three patients who died arrived at the hospital late in the course of their illness, and severe gastroenteritis with accompanying dehydration probably contributed to their deaths. The poison control center promoted public awareness of the mushroom hazard through newspaper and television stories and by notifying local health departments. It also has devised a simple form to improve the quality of data collection and to assist in later verification of suspected* A phalloides *poisoning.*

AS A ROUND-THE-CLOCK toxicology consulting service, the San Francisco Bay Area Regional Poison Control Center (SFBA-PCC) is frequently called on for advice about patient management after wild mushroom ingestion.

In the fall of 1981, a pronounced increase in the number of mushroom-related calls was noted. From October 1 through mid-December, 96 calls were received, a fivefold increase over the same period in 1980. Among these were ten cases of presumed *Amanita phalloides*-type poisoning, of which three were fatal.

This report describes the clinical findings in the ten poisoned patients, who presented initially with acute gastroenteritis and in whom varying degrees of hepatic injury developed. We review the diagnosis and treatment of this type of mushroom poisoning and the identification of *Amanita* species. We also examine possible reasons for the increased number of ingestions in 1981.

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Reports of Cases

CASE 1. A 45-year-old man camping in the Tomales Bay area of Marin County, California, ate two or three wild mushrooms, having eaten wild mushrooms before without problems. No specimens were obtained for identification, but the patient described them as large, having thick stems and being "whitish." After a delay of eight hours, severe nausea followed shortly by abdominal cramping and profuse diarrhea occurred. On arrival in a local emergency room four hours later,

ABBREVIATIONS USED IN TEXT

ALT=alanine amino transferase
AST=aspartate amino transferase

he was alert but was described as appearing dehydrated. His blood pressure was 108/80 mm of mercury and pulse rate was 88 beats a minute. Hematocrit was 53 percent and leukocyte count 14,400 per cu mm. Urinary specific gravity was 1.023 and urine reaction for protein was 2+. Serum electrolyte values were as follows: sodium 135, potassium 4.8, chloride 109 and bicarbonate 16 mEq per liter. Additional laboratory data are given in Table 1.

Intravenous replacement of fluid was started; 36 hours after ingestion, serum aspartate amino transferase (AST, formerly SGOT) and alanine amino transferase (ALT, formerly SGPT) concentrations were greater than 2,500 units per liter. On the fourth day after ingestion, these levels decreased and he appeared to improve. Intravenous administration of thioctic acid, which had just been delivered, was begun. On the fifth day hiccuping developed. Serum AST and ALT concentrations began to rise again, and by the sixth day the patient was in hepatic coma with a blood ammonia concentration of 425 μg per dl. He had a respiratory arrest, was intubated and mechanical ventilation assistance was begun. Generalized motor seizures occurred on the sixth day, for which he was treated with intravenous administration of diazepam (a total of 20 to 30 mg) and phenytoin. Pancuronium bromide was briefly administered intravenously. The patient never regained consciousness, and electroencephalograms on the 7th, 10th and 12th days after ingestion showed progressive diffuse slowing of cerebral activity to a near-isoelectric study. Concurrently, there was progressive loss of brainstem reflexes, and on the 12th day after ingestion, supportive measures were withdrawn and the patient died. Autopsy showed massive hepatic necrosis, renal tubular necrosis and gross cerebral edema.

CASE 2. An 80-year-old man from Marin County who had picked wild mushrooms after each rainfall for years, made a pasta dish containing several mushrooms. He reportedly ate one or two large platefuls. The following day severe nausea, vomiting and pink, watery diarrhea developed. He was admitted to a local hospital 48 hours after the ingestion with severe dehydration and confusion. The hematocrit was 60.2 percent and the leukocyte count was 19,000 per cu mm. Serum electrolyte values were as follows: sodium 154, potassium 4.6, chloride 119 and bicarbonate 11 mEq per liter. Blood urea nitrogen was 52 mg per dl (normal, 8 to 23) and creatinine 4.3 mg per dl (normal, 0.6 to 1.2). Samples of the pasta were obtained and were positive for amatoxins by the Meixner test (see section on identification).

The patient was treated with intravenous administration of fluid and dextrose, and the metabolic acidosis and electrolyte imbalance were corrected. However, he remained confused and became progressively more somnolent. Serum AST, ALT and ammonia concentrations and prothrombin time all increased progressively (see Table 1). On the seventh day after ingestion he was deeply comatose. Septicemia developed and he remained hypotensive despite fluid replacement and vasopressor therapy. He died on the ninth day after ingestion. At autopsy his liver was small with a micronodular pattern but no fatty changes. The myocardium showed fatty infiltration but had no evidence of a recent infarct.

A friend who had eaten two tablespoons of the pasta had nausea, vomiting and diarrhea the day after ingestion. She was admitted briefly but had normal liver function values and recovered fully by the third day after ingestion.

CASE 3. A 39-year-old man found a large wild mushroom in the Santa Cruz, California, area. Although advised by a relative that it "looked dangerous," he cooked and ate the entire mushroom. After 12 hours severe vomiting and diarrhea developed. Two days after the ingestion, he visited a physician who, because of the patient's severe gastroenteritis and apparent dehydration, advised admission to hospital. The patient refused. On the third day after ingestion, the diarrhea worsened and hematemesis occurred; 12 hours later he was brought to the emergency room of a local hospital unconscious. His blood pressure was unobtainable, and his hematocrit was 31.9 percent. He had a cardiopulmonary arrest in the emergency room, was resuscitated, but had another arrest four hours later. He died six days after the ingestion without ever regaining consciousness.

During the hospital course his blood glucose level fell to 22 mg per dl and his creatine phosphokinase level rose to 22,000 units per liter with positive MB bands. (Additional laboratory data are presented in Table 1.) The electrocardio-

TABLE 1.—*Pertinent Laboratory Data in 10 Cases of Amatoxin-Containing Mushroom Poisoning*

Case No.	Age/Sex	Symptoms*	Onset (hrs after ingestion)	Day After Ingestion	Laboratory Data: AST U/liter	ALT U/liter	Total Bilirubin mg/dl	Prothrombin Time (% of normal)	Blood Glucose mg/dl	Creatinine mg/dl	Treatment/Course in Hospital
Fatal Cases											
1	45/♂	N,V,C,WD	8	1	51	57	..	100	261	1.4	Rehydrated; fresh-frozen
				2	2,500	2,500	3.0	..	..	..	plasma; vitamin K; thioctic
				4	1,020	2,500	4.2	13	132	1.5	acid given from day 4/
				6	4,220	7,272	11.8	29	130	1.6	Encephalopathy; respira-
				8	79	1,465	11.7	28	273	2.2	tory arrest; seizures;
				11	35	260	12.3	41	272	3.8	coma; oliguria.
											Died on day 12.
2	80/♂	N,V,WD	12	2	1,200	..	1.7	..	..	4.3	Rehydrated/
				4	2,410	2,500	11.0	37	140	1.8	Encephalopathy; coma;
				5	1,410	2,475	21.7	48	174	..	intractable hypotension;
				6	..	..	..	38	..	..	supraventricular tachy-
				8	87	610	26.0	59	..	1.8	cardia; oliguria.
											Died on day 9.
3	39/♂	N,V,D	12	4	4,860	..	..	13	65	..	Transfusions of fresh-
				5	2,820	3,220	6.7	22	22	5.8	frozen plasma/
											Massive gastrointestinal bleeding; coagulopathy; cardiac arrest on admission.
											Died on day 6.
Survivors											
4	18/♂	N,V,C,WD	8	1	42	21	..	100	..	..	Supportive; vitamin K;
				2	1,550	992	..	42	163	..	dexamethasone given
				3	5,280	5,100	..	35	..	..	intravenously/
				4	1,900	3,900	..	..	..	..	Intermittent bradycardia
				5	710	2,750	3.6	..	129	0.5	and hypotension, required
				8	153	1,360	3.5	..	133	..	temporary pacemaker.
				9	84	810	3.0	80	..	..	No encephalopathy.
5	21/♂	N,V,C,WD	12	2	718	648	1.2	93	104	..	Supportive care/
				4	..	241	..	100	..	1.2	No encephalopathy.
6	24/♂	N,V,C	12	2	880	913	..	85	..	..	Supportive care/
				3	940	880	..	..	..	1.0	No encephalopathy.
				4	195	472	1.5	94	..	..	
7	29/♀	N,V,C,WD headache	12	1	66	..	0.6	..	139	..	Supportive care; activated
				3	617	..	1.2	..	..	0.9	charcoal given once/
				4	210	..	1.1	100	..	..	No encephalopathy.
8	37/♂	N,WD	12	2	982	..	1.8	93	..	..	Supportive care/
				3	..	1,590	..	..	..	..	No encephalopathy.
				4	..	650	..	100	..	1.2	
9	28/♀	N,V,D	8	1	37	..	1.2	..	145	..	Supportive; activated
				2	592	765	..	..	..	1.3	charcoal given very 4 hrs/
				3	1,229	2,360	1.4	78	..	1.0	No encephalopathy.
				4	650	1,560	1.7	..	..	..	
10	21/♀	N,V,D	24	2	1,052	..	0.5	81	..	..	Rehydrated; activated
				3	2,565	3,735	..	49	..	..	charcoal given every 12
				4	..	5,870	0.8	60	124	0.7	hrs/Sleepy but no signs
				7	..	1,550	..	..	..	..	of encephalopathy.

Abbreviations: AST = aspartate amino transferase (normal, 35 U/liter); ALT = alanine amino transferase (normal, 45 U/liter).

*Symptoms: N = nausea; V = vomiting; C = abdominal cramps; WD = watery diarrhea; D = diarrhea

gram showed nonspecific ST changes without evidence of infarction. Autopsy showed acute massive hepatic necrosis, mucosal necrosis in the small and large bowel, cerebral edema and cerebellar tonsillar herniation. The heart showed fatty infiltration of the subendocardial region and some areas of hemorrhage. The patient's relative who was with him at the time he picked the mushroom brought in a similar specimen, identified by a local mycologist as *A phalloides*.

CASE 4. An 18-year-old man ate approximately ten wild mushrooms that he found while camping in the Santa Cruz area. Eight to ten hours later, nausea, vomiting, abdominal cramps and watery diarrhea began abruptly. He brought a similar mushroom with him to the emergency room of a local hospital and it was identified by a mycologist as *A phalloides*. On admission he appeared mildly dehydrated but was alert. His blood pressure was 110/70 mm of mercury and pulse rate 96 beats per minute. Hematocrit was 48.6 percent and leukocyte count 8,400 per cu mm. He was given fluids intravenously as well as dextrose and dexamethasone. The gastrointestinal symptoms abated, but serum AST and ALT concentrations continued to rise, with a peak AST of 5,280 units per liter on the third day after ingestion (Table 1). He remained alert and awake. On the fourth day after ingestion, he had intermittent severe bradycardia, with a heart rate of 28 to 30 per minute and hypotension necessitating placement of a temporary transvenous pacemaker. Liver enzyme values remained elevated for more than ten days, but he had no symptoms or signs of worsening hepatic failure or of encephalopathy. He recovered without further complications.

CASES 5 through 10. A group of seven Laotian refugees gathered and prepared wild mushrooms from a park in the Santa Rosa, California, area. They and other refugees had frequently gathered and eaten similar mushrooms in the past in this area without ill effects. They stated that in their home country foraging for wild mushrooms was common and that they tested for poisonous species by boiling the mushrooms with rice. Mushrooms that turned rice water red were regarded as poisonous and were not eaten; this batch did not color the water and was consumed. One member of the group ate less than a tablespoon of the dish and remained asymptomatic. In the six others, each of whom ate a sizable portion, nausea, vomiting, abdominal cramps and profuse diarrhea began abruptly 8 to 12 hours after ingestion. All were admitted to a local hospital.

In one patient the ALT reached a peak concentration of 5,870 units per liter (see Table 1); although she was mildly lethargic, there was no other clinical evidence of hepatic encephalopathy. All six patients recovered with supportive care. A sample of the mushroom stew was analyzed by a local biologist who identified all but one specimen as species of the genus *Russula,* which do not contain amatoxins. However, one specimen was a "button" (an immature specimen) that had features of an *Amanita* species.

Diagnosis and Treatment of Amatoxin-Containing Mushroom Poisoning

Many species of *Amanita* mushrooms contain amatoxins (α-amanitine and β-amanitine), cyclopeptides that attack the intestines, liver and kidneys. The characteristic course of amatoxin poisoning has three stages: (1) The first phase is delayed in onset, rarely beginning before six hours and sometimes not until 24 hours after ingestion. Severe abdominal cramping, vomiting and profuse watery diarrhea occur and may cause profound dehydration and circulatory collapse. (2) During the next 24 to 36 hours, there is often a remission in symptoms and patients appear to be recovering. During this so-called latent phase, elevations of liver enzymes persist and may even increase. (3) In the third phase, hepatic damage becomes apparent clinically and jaundice, encephalopathy, hypoglycemia, seizures and coma may occur. Death is due to hepatic and renal failure and to secondary effects on the cardiovascular and central nervous systems.[1] Mortality has been reported as anywhere from 10 percent to 90 percent.[1-3] The range reflects the wide variation both in amounts ingested and in individual susceptibility.

Treatment is primarily supportive, with careful attention to restoration of fluid losses, replacement of clotting factors, prevention of hypoglycemia and minimization of hepatic encephalopathy. More experimental or invasive treatments such as hemodialysis or hemoperfusion, repeated doses of activated charcoal given orally and administration of steroids, penicillin, cytochrome *c* and thioctic acid have been proposed, but to date no controlled clinical studies have been done to confirm their effectiveness.[3] Uncontrolled human trials are unreliable, given the great variability in amount of toxin ingested. Animal studies, when

carefully carried out, show no apparent benefit from thioctic acid.[3,4] Previously our impression was that amatoxin poisoning carried with it a mortality of up to 90 percent. Initial results with the use of thioctic acid thus appeared very favorable. Since then we have concluded that the true mortality is probably closer to 30 percent to 40 percent. Our current opinion is that thioctic acid is not of value in the treatment of amatoxin poisoning.

With other "antidotes," treatment was successful only when given to the animals along with the amatoxin or within the subsequent eight hours[5]; this is probably because most amatoxin is excreted within six hours.[3] It is difficult to extrapolate from these results to accidental poisonings because patients often seek treatment much later.

Discussion of Cases

In this series of ten patients the mortality was 30 percent. In all patients who died, encephalopathy and coma developed and evidence of renal insufficiency was present.

The death rate might have been lower had the patients in cases 2 and 3 sought medical attention sooner. The patient in case 3 arrived at the hospital moribund with severe blood loss and coagulopathy; both might have been corrected or prevented had he agreed to earlier admission to the hospital. At autopsy, the patient in case 2 had a small liver with micronodular changes consistent with cirrhosis. His advanced age and probable lack of hepatic and cardiovascular reserve made recovery from the severe gastroenteritis phase less likely.

The patient in case 1 died despite early and vigorous supportive care. No conclusion may be drawn about the effectiveness of thioctic acid in this case because it was not started until about eight hours after mushroom ingestion. It is unlikely that the diazepam, used to control seizures on day 6, significantly affected the electroencephalographic findings or outcome because the dose was small. Although pancuronium may cause prolonged apnea in patients with oliguria and hepatic insufficiency, it would not be likely to cause the electroencephalographic changes seen in this patient.

Other mushroom species may produce toxic symptoms; these have been reviewed previously.[1,6] Hepatotoxicity has been described following ingestion of the "false morel" or *Gyromitra esculenta*. This and similar species contain gyromitrin, which is hydrolyzed to monomethylhydrazine. The onset and character of symptoms are similar to amatoxin poisoning. *Gyromitra* species are very distinctive in appearance, with great brain-like convoluted surfaces. They are principally found in the spring or early summer. Only 20 cases of poisoning due to gyromitrin have been reported in the United States since 1900.[6] It is very unlikely that gyromitrin was responsible for any of the cases reported here; all of these poisonings occurred in the late fall, and in no case did the victim describe finding or ingesting such distinctive mushrooms.

Many other species of mushrooms may cause gastrointestinal upset. However, symptoms occur shortly after consumption and do not result in hepatotoxicity.

Although confirmation by direct examination of specimens or assay for amatoxins was not possible in each case, it is highly likely that amatoxin poisoning was responsible for these illnesses. The onset and character of symptoms, the delayed hepatotoxicity, the description of the mushrooms and the time of year all implicate amatoxin-containing *Amanita* as the cause of poisoning.

Identification of *Amanita* Mushroom Species

Several factors may account for the increase in ingestions of amatoxin-containing mushrooms. First, mycologists are finding *A phalloides* in larger quantities over greater areas of California. Second, there are growing numbers of amateurs interested in foraging for fungi to use for food or for hallucinogenic effects. Some of these collectors may naively sample unidentified species, and others may mistake a toxic for an edible member of the *Amanita* genus (for example, *Amanita calyptroderma*). Third, early and heavy rains followed by warm weather in the fall of 1981 were responsible for an increase in the total number of mushrooms growing in northern California.

The amatoxin-containing amanitas that are commonly found in the western states are *A phalloides* (California, Oregon), *Amanita ocreata* (California) and *Amanita verna* (Washington, questionably California). There are other species of *Amanita* with different toxicologic properties that do not contain amatoxin, for example, *Amanita pantherina* and *Amanita muscaria*; their toxicology is not reviewed here. However, all of these mushrooms have common morphologic characteristics that may be used to classify them

in the *Amanita* genus. The description offered here cannot be used to differentiate among the amanitas or to exclude the possibility of amatoxin ingestion because other non-*Amanita* sp may contain amatoxin (for example, several species of *Galerina, Cortinarius, Conocybe* and *Lepiota*).[6] However, mushrooms that fit the description should be suspect, and the ingestion of any *Amanita* mushroom is ill-advised.

The *Amanita* mushrooms undergo development from a membrane-covered egg stage to a full-sized specimen with stem and open cap (Figure 1). At its early stage, the *Amanita* may easily be mistaken for an edible puffball unless it is cut open to reveal the developing stem, cap and gills inside. As the stem elongates, the membrane breaks and may sometimes be seen on the cap as a patch or warts, depending on the species. This feature is not commonly seen in *A phalloides*. As the mushroom matures, the cap opens outward and tends to flatten with age. As it does so, a second membrane pulls away from the undersurface of the cap to expose the gills. This membrane usually persists as a ring around the midportion of the stem. The gills of *Amanita* sp are white and do not attach to the stem. This can be seen by twisting the stem off the cap to leave the gills intact with no part remaining on the stem itself. An extremely important distinguishing feature of the amanitas is the volva at the bottom of the stem. This is a cuplike membrane around the bulbous base of the mushroom. Unfortunately, it is frequently left behind because the inexperienced collector breaks the specimen off at the stem.

Color is not a very reliable characteristic for identification because it may vary with weather, soil conditions, age and other factors. The cap of *A phalloides* is usually olive yellow or olive brown, whereas that of *A ocreata* is initally white and then turns orange-buff and that of *A verna* remains white.

Although there are many excellent mushroom guidebooks,[7,8] a clinician should confirm the identity of a suspicious mushroom through a local expert. Often a poison control center, a university biology department or a mycological society can assist (in the San Francisco Bay Area, call Dr. Harry Thiers, San Francisco State University, telephone [415] 469-2439).

If possible, it is most helpful to obtain a specimen of the ingested mushroom. An experienced mycologist also may be able to make the identification from spores seen in the vomitus, gastric aspirate or leftovers of the meal itself. Few laboratories are set up to do chromatographic analysis of the mushroom.

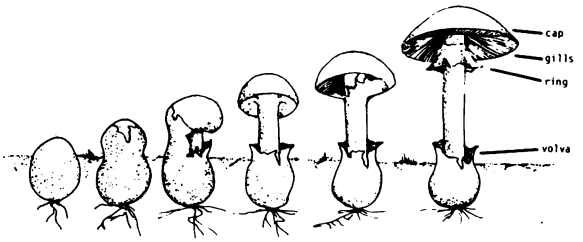

Figure 1.—Development of an *Amanita* showing "button" (far left), often mistaken for edible puffball, and mature specimen (far right). (Reproduced with permission from Lincoff and Mitchel.[6])

A recently evaluated field test for amatoxins described by Meixner consists of rubbing a piece of the mushrooom cap onto newsprint, which is then allowed to dry. A drop of concentrated hydrochloric acid is applied to the paper and yields a bluish-green color if amatoxins are present. The test is highly sensitive, but a negative result should not be used to indicate edibility of a mushroom.[9]

It is apparent from the cases reported here that amateurs should be extremely careful about which mushrooms they choose. So-called rules of thumb, such as the Laotians' rice water test, are unreliable. Unfortunately, untrained persons continue to believe that a poisonous mushroom will tarnish a silver cooking spoon, that a mushroom with nibble marks left by an animal must be safe or that cooking or processing *A phalloides* will destroy its toxins.

The Poison Center Response

All of the reported cases were originally received through the San Francisco Bay Area Regional Poison Control Center. Consultation was obtained quickly with the backup physician staff of the Clinical Pharmacology Unit at San Francisco General Hospital Medical Center. In addition, the public health division of the California Department of Health Services was notified so that investigation and preventive measures could be initiated. This "epidemic" was also reported to local news agencies, resulting in several television and newspaper reports warning the public against consuming wild mushrooms.

A review of mushroom-related calls received in our poison center over the past three years was attempted. However, retrospective verification of suspected amatoxin cases was difficult because

MUSHROOM-RELATED CALL DATA FORM

Poison Control Center Case #_____ Date _____

☐ ASYMPTOMATIC
☐ child ☐ adult ☐ emesis advised ☐ asymptomatic on follow-up

☐ SYMPTOMATIC—hallucinations or neuropsychiatric symptoms
Onset ____ hours after ingestion Follow-up 1 day: ☐ symptomatic ☐ asymptomatic

☐ SYMPTOMATIC—simple gastroenteritis, self-limited
Onset ____ hours after ingestion Follow-up 1 day: ☐ symptomatic ☐ asymptomatic

☐ SYMPTOMATIC—possible amatoxin-type poisoning
Onset ____ hours after ingestion Admitted ____ hours/days after ingestion

ON ADMISSION:
Symptoms: ☐ abdominal cramps ☐ vomiting ☐ other _____
☐ diarrhea (describe severity: _____
Exam: ☐ alert ☐ lethargic ☐ comatose ☐ dehydrated ☐ seizures
Labs: AST ____ prothrombin time ____ ammonia ____ glucose ____
ALT ____ creatinine ____ BUN ____ CPK ____

3-DAY FOLLOW-UP: (____ days after ingestion)
Exam: ☐ alert ☐ lethargic ☐ comatose ☐ seizures ☐ other ____
_____
Labs: AST ____ prothrombin time ____ ammonia ____ glucose ____
ALT ____ creatinine ____ BUN ____ CPK ____

☐ REQUIRES LONGER FOLLOW-UP

MUSHROOM IDENTIFICATION:
Description: _____
☐ original specimen ☐ similar mushroom specimen ☐ confirmed by mycologist
☐ Meixner test used ☐ positive ☐ negative
(Rub mushroom cap on newsprint, allow to dry; add 2-3 drops of concentrated hydrochloric acid—blue-green color may indicate amatoxins)
OUTCOME: ☐ survived ☐ died
COMMENTS/TREATMENT: _____
_____
_____

Figure 2.—Mushroom-related call data form. Abbreviations: ALT = alanine amino transferase; AST = aspartate amino transferase; BUN = blood urea nitrogen; CPK = creatine phosphokinase; exam = examination; labs = laboratory data.

data collection was frequently incomplete. A special mushroom call data form was devised (Figure 2). It should improve our ability to collect essential information and to verify suspected amatoxin poisonings.

Conclusion

Identification of mushrooms by nonexperts, particularly by telephone, is unreliable. Even though *Amanita* sp have distinctive characteristics, there are several other genera of mushrooms that contain amatoxins, as noted earlier.

Our poison control center recommends that in all cases of wild mushroom ingestion, regardless of the description, emesis be induced with syrup of ipecac as soon as possible. When mushroom description or clinical presentation (or both) strongly suggests an amatoxin-type poisoning, we recommend thorough gastrointestinal evacuation by emesis, administration of cathartics and repeated doses of activated charcoal. Nasogastric tubes are too small to remove mushroom pieces and should not be used. Along with intensive supportive care, specimens of the ingested mushroom should be sought and identification by an expert obtained. At present, available evidence does not support the use of other treatments such as dialysis, penicillin, steroids or thioctic acid.

Our experience shows how a poison control center can serve (1) as a resource for the immediate care of a poisoned patient, (2) as an agency for data collection and review that promotes recognition of a new health hazard and (3) as an early warning system for state health agencies, the professional community and the public.

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