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CASE REPORT

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Hepatotoxicity from ingestion of wild mushrooms of the genus *Amanita* section *Phalloideae* collected in Mexico City: two case reports

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ABSTRACT

We present two cases of acute liver injury resulting from consumption of wild mushrooms. The first case was a male who developed acute hepatitis after ingestion of diverse mushrooms including *Amanita* species. His clinical course was favorable with complete recovery of liver function. The second case was a male who developed acute liver failure (ALF) after ingestion of *Amanita bisporigera.* He required MARS therapy as a bridge to liver transplantation but transplantation was not performed because he succumbed to multiorgan failure. There are few trials demonstrating the efficacy of the different treatments for mushroom poisoning. These cases demonstrate that the consumption of wild mushrooms without proper knowledge of toxic species represents a serious and under recognized health problem.

Key words. Mushroom poisoning. Amanita. Acute liver failure. Treatment. MARS. Amatoxins. Mushroom hepatotoxicity. Fulminant hepatitis.

INTRODUCTION

Ingestion of toxic macroscopic mushrooms (mycetism) occurs infrequently but can be a medical urgency. Patients arrive to the Emergency Department and show diverse symptoms, many of which overlap with gastroenteritis or other benign clinical syndromes. Clinicians must have a high index of suspicion for mushroom toxicity in any patient reporting recent wild mushroom ingestion with a concomitant toxic syndrome. Most lethal exposures (90%) occurring in Europe and North America are attributed to *Amanita phalloides* and *A. virosa;*[1,2] however, in Mexico other species such as *Amanita arocheae*, *A. bisporigera*, *A. verna* and *A.virosa* under the same section *(Phalloideae)* are equally toxic.[3] Fourteen distinct types of mushroom poisoning have been described. *Amanita* species toxicity is characterized by its late onset, occurring between 6 and 24 h or more post-ingestion.[4] We present two cases of hepatotoxicity resulting from ingestion of wild mushrooms gathered in the metropolitan area of Mexico City. Both patients were managed in a tertiary care center yet had dramatically different clinical outcomes.

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CASE REPORT

Case 1

A 62-year-old man was referred from a secondary care hospital with jaundice and a recent history of eating wild mushrooms collected in Tlalpan Park in Mexico City. He presented to the emergency room twelve hours after mushroom ingestion with

crampy, epigastric pain of moderate intensity, nausea and loose stools. The patient was hydrated and received antiemetics and analgesics for the first 24 h. On the third day after ingestion, he developed jaundice and dark urine and was referred to our tertiary care center for abnormal liver biochemistry tests. His past medical history was remarkable only for irritable bowel syndrome treated with tegaserod 6 mg twice a day for three months and alcohol intake less than 30 g every 4 months. At admission his vital signs were normal. His physical exam revealed scleral icterus, jaundice, mild abdominal tenderness, and normal mental status. Laboratory tests were remarkable for elevated ALT > 3,900 U/L, total bilirubin > 20 mg/dL and prolonged PT 40/12 s (Table 1). Serological markers for hepatitis A, B and C were negative. He was given intravenous hydration, ceftazidime, and ursodeoxycholic acid. Within 36 h he showed clinical improvement. He never experienced encephalopathy or serious coagulopathy.

Samples of remnants of the wild mushrooms collected by the patient were evaluated in the Mycology Area of the Department of Comparative Biology of the Faculty of Sciences of Mexico's National Autonomous University (UNAM). The species identify were *Amanita bisporigera, A cf. verna* (section *phalloideae*) *A. flavorubens* (section *Validae*) and *Russula sp*. Some samples were in process of decomposition (Table 2, Figure 1).

Of note, the patient's wife also ate a portion of the wild mushrooms and developed diarrhea with electrolyte abnormalities but no hepatotoxicity. Details of the proportion of each species and quantity of mushrooms consumed by the patient and his wife were not known.

Case 2

A 47-year-old male agricultural worker was referred from a secondary care hospital for acute hepatitis after ingestion of wild mushrooms collected in a

Table 1. Evolution of laboratory test (case 1).

Parameter	09/03/07 Day 4	09/05/07 Day 6	09/07/07 Day 8	09/10/07 Day 11	10/12/07 Day 42	11/19/08 Day 360
Glucose mg/dL	109	128	95	89	99	-
Creatinine mg/dL	0.9	0.9	0.9	1.0	0.9	-
TB mg/dL	8.6	12.7	**13.83**	**20.58**	1.98	0.71
DB mg/dL	3.9	9.31	9.21	14.11	1.08	0.12
Albumin g/L	3.1	3.4	3.3	3.3	3.8	4.2
ALT U/L	**1,947**	**3,943**	**3587**	688	62	22
AST U/L	**1,290**	626	435	132	40	21
ALP U/L	142	175	170	291	273	94
GGT U/L	-	48	89	396	139	21
LDH U/L	-	-	510	440	304	-
Hb g/dL	15.8	16	16.7	18.1	16.3	-
WBC (10^3/μL)	8,500	8,700	9,100	9,500	8900	-
Platelets (10^3/μL)	106,000	118,000	112,000	95,000	142,000	-
PT (sec)	**40/12**	**33/12**	24.9/12.5	15.5/12.6	12.7/12.4	-

TB: Total bilirubin. DB: Direct bilirubin. ALT: Alanine aminotransferase. AST: Aspartate aminotransferase. PT: Protrombine time. GGT: Gamma glutamyl transferase. LDH: Lactic dehydrogenase. ALP: Alkaline phosphatase. WBC: White blood cells.

Table 2. Identification of wild mushrooms collected (case 1).

Specimen	Species identified
Basidiome*	*Amanita flavorubens*
Stem	*Amanita flavorubens*
Basidiome	*Amanita bisporigera* (subgenus *Lepidella* section *Phalloides*)
Pileus	*Amanita* cf. *verna* (subgenus *Lepidella* section *Phalloides)*
Basidiome	*Russula* sp (impossible to determine species because high degree of decomposition)
Basidiome	*Amanita* cf. *verna*

*Basidiome (fruiting body), is a multicellular structure on which spore-producing structures are borne.

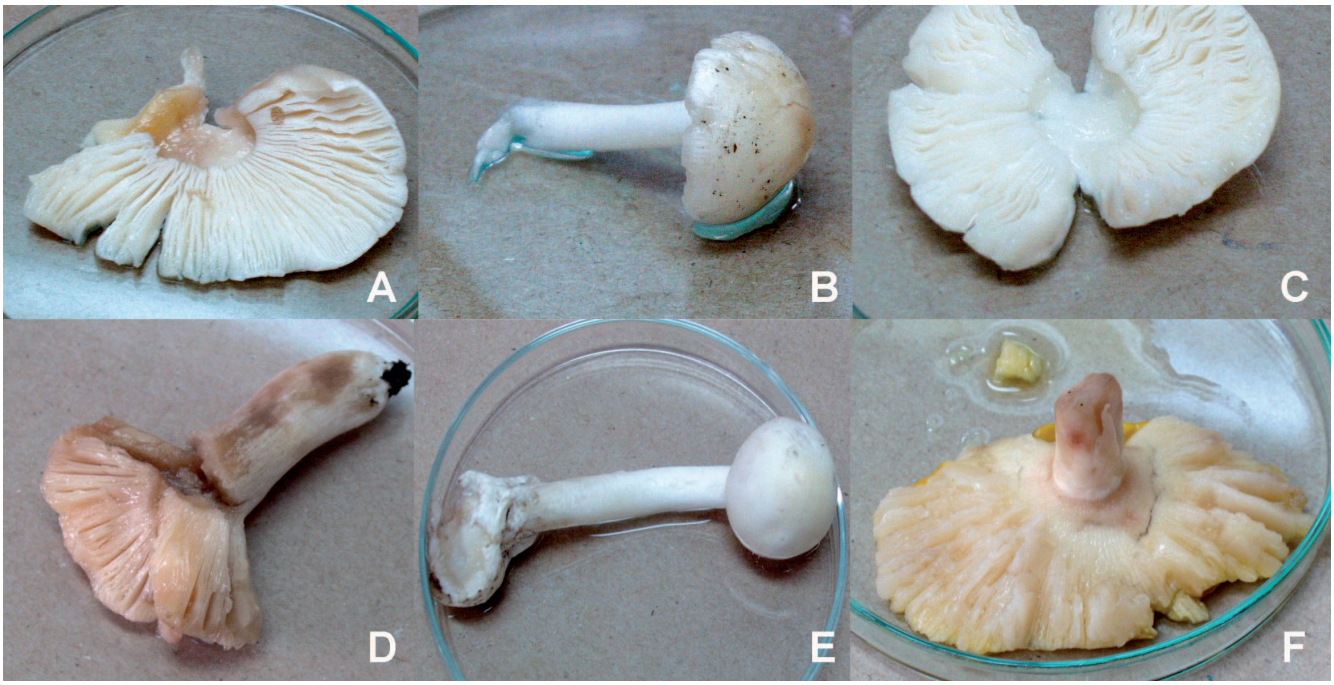

Figure 1. *Macroscopic images of the mushrooms collected in case 1.* ***A.*** *Part of a pileus of the basidiome Amanita cf. verna showing white sheets.* ***B.*** *Incomplete white basidiome of Amanita cf. verna (subgenus Lepidella section Phalloides).* ***C.*** *Bottom of an incomplete white pileus of Amanita verna.* ***D.*** *Basidiome of Russula sp showing the hymenium and stem (the brown coloration is due to the decomposition process).* ***E.*** *Complete white basidiome of Amanita bisporigera.* ***F.*** *Yellow pileus and part of the stem of a reddish-brown basidiome of Amanita flavorubens.*

Table 3. Evolution of laboratory test (case 2).

Parameters	09/15/10 Day 2	09/16/10 Day 3	09/17/10 Day 4	09/18/10 Day 5	09/19/10 Day 6	09/20/10 Day 7	09/21/10 Day 8	09/22/10 Day 9
WBC*	24.8	19.16	16.13	20.36	13.7	19.09	19.5	24.12
Platelets*	365	231	156	92	62	55	75	52
Glucose	105	54	137	65	189	133	176	125
Urea (mg/dL)	137	87	65	33	37	55	91	100
Creatinine	5.3	2.2	1.55	1.22	1.86	3	4.5	4.26
TB (mg/dL)	3.52	7.36	7.19	9.74	12.37	11.2	15	14.7
DB (mg/dL)	3.02	6.52	6.25	7.2	8.18	8.68	11.33	9.42
ALT (U/L)	1,377	2,194	2,675	3,146	1,814	794	511	306
AST (U/L)	1,358	1,663	2,215	2,064	552	114	60	44
GGT (U/L)	-	74	58	69	60	47	53	47
LDH (U/L)	2,080	2,934	3,406	2,239	911	526	649	779
ALP (U/L)	-	104	98	132	139	89	99	101
Albumin	-	3.34	3.04	3.2	3.11	2.3	2.36	2.6
PT (s)	58 /13	76.3/12	75.9/12	58.8	50.3	46.4	60.3	40.9/13

* WBC and platelets reported as ($10^3/\mu L$); glucose and creatinine reported in mg/dL, albumin reported in g/L. **TB:** Total bilirubin. **DB:** Direct bilirubin. **ALT:** Alanine aminotransferase. AST: Aspartate aminotransferase. **PT:** Protrombine time. **GGT:** Gamma glutamyl transferase. **LDH:** Lactic dehydrogenase. **ALP:** Alkaline phosphatase. **WBC:** white blood cell. **Alb:** Albumin.

wooden area next to his home in southern Mexico City. Ten hours after ingestion, he developed severe, crampy mesogastric pain that radiated to the lower back, accompanied by nausea, frequent vomiting, and more than 10 watery bowel movements without mucus, blood or fever. He was initially given intravenous hydration, antiemetics, analgesics, loperamide and trimethoprim-sulfamethoxazole. After 24 h

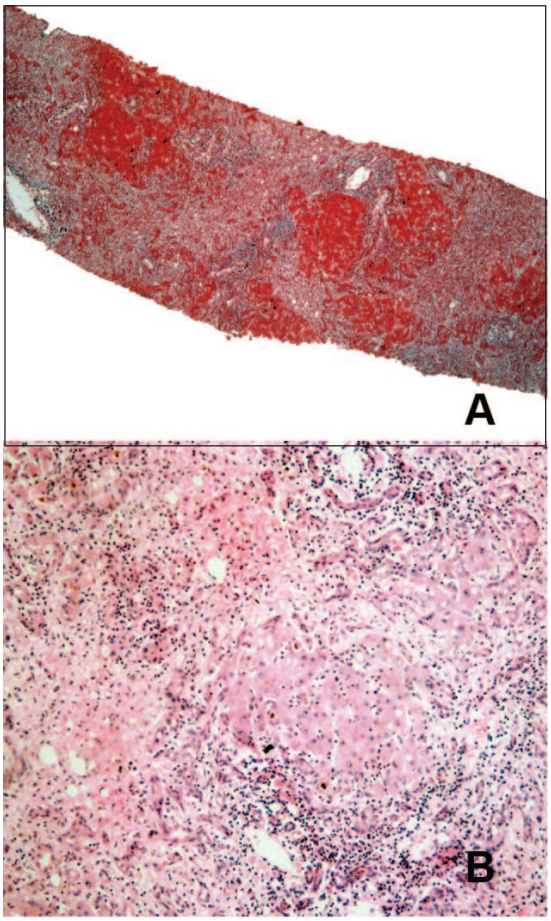

Figure 2. *Post-mortem liver biopsy. Case 2.* ***A.*** *Panoramic 10 x. Masson stain. Massive multiacinar hemorrhagic necrosis, bridge necrosis.* ***B.*** *Hematoxylin & eosin (H&E) 40 x. Multiacinar necrosis with macrovesicular steatosis, canalicular cholestasis with neoformation of bile ducts.*

the patient developed kidney and liver dysfunction and was referred to our tertiary care hospital. His medical history was remarkable only for occasional use of diclofenac for knee joint pain. At admission his blood pressure was 100/60, heart rate 90 beats/min, respiratory rate 20 breaths/min, and he was afebrile. He had scleral icterus, right upper quadrant pain, and a normal neurologic exam without hepatic encephalopathy. Blood work demonstrated acute renal failure, metabolic acidosis, hypoglycemia, prolonged PT and significant abnormalities in the liver biochemistries. Abdominal ultrasound revealed no significant changes in liver or biliary tract. He was admitted to the Intensive Care Unit with an Apache II score of 10 points (Table 3).

Silymarin 420 mg (Legalon®, Nycomed SA, Mexico City) therapy was administered every 8 h. On the fifth day following the ingestion, the patient developed acute pancreatitis with a serum amylase of 455 U/L and lipase of 1418 U/L. Subsequently, hepatic encephalopathy appeared and rapidly progressed to grade IV requiring intubation, norepinephrine infusion, sedation and lactulose. He was evaluated as a potential transplant recipient for acute liver failure (ALF). On the sixth day post-ingestion, therapy with Molecular Adsorbent Recirculating System (MARS® Treatment Kit, PrisMARS, Gambro Rostock GmbH, Friedrich-Barnewitz ST3, Rostock, Germany) was initiated in two sessions (12 and 21 h) as a bridge to transplant. The patient's condition evolved with Glasgow scale of 6, brain CT showing mild swelling, rapid progression of liver and renal failure, and shock refractory to treatment. He died on the tenth day post-ingestion. Liver biopsy was performed post-mortem and showed multiacinar necrosis, bridging necrosis, canalicular cholestasis with neoformation of bile ducts and macrovesicular steatosis (Figure 2).

An immature specimen of the wild mushroom collected was also evaluated in the Mycology Area, UNAM. Because the specimen had not yet developed all its morphological characteristics, it could identified only as *Amanita aff. bisporigera*. In this case, the patient's wife also ate a small portion of the mushroom and experienced only gastrointestinal upset without liver or kidney injury.

DISCUSSION

We present two cases of mushroom-induced hepatotoxicity with distinct clinical courses. The first was an acute toxic hepatitis caused by consumption of *Amanita* species, with decreased albumin and prolongation of PT but without complications such as hemorrhagic diathesis or encephalopathy. The second case died of multi-organ failure due to ALF despite MARS therapy initiated as a bridge to liver transplantation. Although the albumin dialysis therapy appeared to result in an initial improvement, its utility was likely limited because of the delay in initiation.

In the first case of mycetism, the only edible species were *Rusulla sp;* the other species such as *Amanita flavorubens* (section *Validae*) may result in abdominal pain and toxicity when eaten raw.[5] Unfortunately, the distinction between edible and toxic mushrooms is based on detailed knowledge of their morphological characteristics. There is no practical, simple method that could allow most individuals to make this distinction. For example, while different species of edible mushrooms are white like the typi-

cal white button mushroom (*Agaricus spp*, *Amanita tuza*, etc.) or yellow *(Amanita caesarea, A. yema, A. tullossi*, or A. *flavorubens),* most toxic species in North America are also white.

Nearly 74,000 species of mushrooms in the world have been identified,[6] of which three main families contain lethal amatoxins:

- ***Amanitaceae.*** Within genus *Amanita*, including most of the 44 species described for the section *phalloideae.*
- ***Cortinariaceae.*** Of the genus *Galerina* including species such as *G. autumnalis, G. badipes, G. marginata, G. sulciceps, G unicolor, G.venenata*.
- ***Agaricaceae.*** Including *Chlorophyllum molybdites* and species of the genus *Lepiota,* among the most notable *L. Helveola, L. brunneoincarnata, L. sunincarnata*.[5,7]

Toxins of the genus *Amanita* are classified into three groups: amatoxins, phallotoxins and virotoxins. The virotoxins and phallotoxins act quickly, typically in 1-2 h. The phallotoxins are poorly absorbed and are responsible for the gastrointestinal symptoms. Amatoxins (cyclo-octapeptides) have a slower onset, typically 10 to 15 h post-ingestion, but are 10 to 20 times more toxic. Amatoxins are resistant to heat and freezing, and cannot be denatured by cooking or digestive enzymes.[8-10]

The lethal dose of amatoxins is < 0.1 mg/kg of body weight and a mature mushroom can contain a fatal dose of 8-12 mg. The amatoxins are mainly present in the pileus, ring and stem of the basidiome (or fruiting body, on which spore-producing structures are borne). The severity of poisoning depends on the amount of mushroom ingested.[11-13] The amatoxins inhibit protein synthesis in enterocytes, hepatocytes and renal proximal tubular cells. Hepatocellular damage is due to the recapture of amatoxins, mediated by the organic anion polypeptide transporter located in the cytoplasmatic membrane. This polypeptide binds to the subunit of RNA polymerase II transcription, interfering with DNA, suppressing the production of RNA, blocking protein synthesis, and causing cell death.[1,9,14,15]

Amanita poisoning presents with different clinical stages:

- The *incubation stage*, which is an asymptomatic period between 6 and 12 h after ingestion.
- The *gastrointestinal stage* characterized by abdominal pain, nausea, vomiting and diarrhea for up to 24 h, which can lead to dehydration and shock.
- The *cytotoxic stage* which typically consists of an apparent clinical improvement after 24 to 48 h followed by a progressive deterioration in renal or liver function.[4,14,16]

The fourth phase may begin abruptly with coagulopathy, hepatic encephalopathy, hypoglycemia, and development of fulminant hepatic failure combined with acute renal failure.[9,17,18] The diagnosis can be confirmed by the detecting the presence of alpha amanitin in urine. Different methods of analysis (RIA-radioimmunoassay, HPLC-High Performance Liquid Chromatographic method, ELISA) are highly sensitive for detecting alpha amanitin in blood or urine if they are performed within 48 h prior to ingestion. However, these tests are not accessible at all sites and are not routinely performed.[19] Mortality from mushroom poisoning has been found to be as high as 20% in adults and 50% in children. Without transplantation, the probability of surviving due to mushroom poisoning ranges between 10 and 30%.[14,16,20] The following risk factors have been found to confer a higher morality risk: age < 10 years, female gender, short interval between ingestion and onset of diarrhea (< 8 h), severe coagulopathy, severe hyperbilirubinemia, elevated creatinine, and a rapid increase in prothrombin time.[21]

There is no specific antidote for mushroom poisoning. An accurate taxonomic identification of the mushroom can be useful in determining prognosis. Treatment should begin with vigorous fluid resuscitation and an attempt to evacuate the GI tract. Ipecac syrup is effective only in the first hour after ingestion. Nasogastric lavage followed by activated charcoal every 2-4 h to reduce absorption is also recommended. Forced diuresis with sodium bicarbonate have been used to eliminate the toxin in the first hours.[9,17] Nasobiliary drainage by endoscopic cholangiography (ERCP) has been used successfully to remove amatoxins from enterohepatic circulation but is not performed routinely.[9]

Other medications that have been used in mushroom poisoning include silymarin (*Silybum marianum*-Milk Thistle), which inhibits the binding of toxins to hepatocytes, and competes for the transmembrane transporter, thereby interrupting enterohepatic circulation of the toxin and reducing oxidative stress.[15,22] Silymarin has been used at doses of 20-50 mg/kg/d orally or through its intravenous form silybin (5 mg/kg bolus and 20 mg/kg/24 h

infusion for three days).[14,23] Silymarin has been used in combination with other agents so its single contribution is unknown. Comparative prospective studies are needed to demonstrate its real benefits. Other drugs that have been used empirically are high-dose penicillin G, ceftazidime, N-acetylcysteine, or cimetidine.[9,10] Randomized control trials demonstrating the efficacy of these different therapeutic modalities are lacking.

Several extracorporeal detoxification methods such as charcoal hemoperfusion, plasmapheresis, hemodialysis and MARS, have been used in many transplant centers. MARS therapy is a albumin dialysis method which may remove primary and secondary toxins and support the excretory function, thereby maintaining hemodynamic stability and preventing organ failure.[20,24] MARS is most useful if started before the onset of gastrointestinal symptoms, and can act as a bridge to liver transplantation.[25] On the other hand, MARS therapy is an invasive and expensive modality, and its utility in *Amanita* intoxication has only been demonstrated in uncontrolled case reports. There are several reports of successful liver transplantation for mushroom poisoning when ALF is identified early with proper consideration of prognostic factors. One year survival of 65% has been reported.[26,27]

CONCLUSION

Although mushroom poisoning occurs infrequently, it must be recognized promptly so as to implement therapies to limit the absorption of lethal toxins within the first critical hours. Patients with evidence of hepatic impairment should be managed aggressively in tertiary hospitals with liver transplant capacity. In Mexico there is a wide variety of mushrooms and an extensive traditional knowledge about edible mushrooms.[28,29] However, these cases show that the consumption of wild mushrooms without a clear distinction of edible *vs.* toxic species represents a serious and under recognized health problem.

ABBREVIATIONS

- **ALF:** Acute liver failure.
- **MARS:** Molecular adsorbent recirculating system.
- **ALT:** Alanine aminotransferase.
- **AST:** Aspartate aminotransferase.
- **TB:** Total bilirubin.
- **PT:** Prothrombin time.

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