



Foraged Mushroom Toxicity Presenting to Urgent Care with Acute Kidney Injury

Urgent message: Though it occurs relatively rarely, mushroom toxicity can result in irreversible organ damage and, in certain cases, death if not recognized quickly. Diagnosis can be difficult due to the facts that toxicity may present at different intervals from time of ingestion, depending on the species of mushroom, and initial symptoms are nonspecific and similar to those of benign gastrointestinal illnesses. Timely consultation with a poison control center may be life-saving.

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Introduction

Mushroom foraging is a popular activity in the U.S. Pacific Northwest (PNW). Based on cultural traditions, many Asian and European immigrants commonly forage for mushrooms, as well. A case of mushroom misidentification may occur when a poisonous species in the U.S. is mistaken for an edible species in an individual's country of origin, which occurs most commonly among species from Europe and Asia. There are also poisonous local native species which can be confused with edible species with similar appearances. *Amanita smithiana* is an example of a poisonous native PNW mushroom which is similar in appearance to, and grows in the same densely forested habitat as, an edible species: the pine mushroom (or *matsutake* as it is known in Asia), which is used in many traditional Asian dishes.¹

Amanita smithiana is known to cause delayed renal failure when ingested, due to the nephrotoxic compound allenic norleucine.² Gastrointestinal symptoms generally begin within 6 hours of ingestion, but renal toxicity does not manifest until 1 to 4 days after ingestion; as such, it may not be evident on initial laboratory evaluation. The treatment is supportive and often requires several weeks of dialysis.³



The case presented here concerns suspected *A smithiana* toxicity with subsequent acute renal failure.

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Case Presentation

A generally healthy 52-year-old man of East Asian descent presented to an urgent care center complaining of loose stools the previous night followed by severe vomiting. He was unable to tolerate any oral intake. He spontaneously reported that he had eaten mushrooms foraged from a forest in Western Oregon several hours prior to the onset of his symptoms. He reported that he thought he had eaten three different species and was uncertain of the identity of one of the mushrooms. His wife, who also ate some of the foraged mushrooms, was asymptomatic. The patient denied fever, abdominal pain, fevers, chills, rash, hematochezia, dysuria, or darker urine.

His physical exam, including abdominal exam, and vital signs were normal. He was uncomfortable, but in no distress. A dipstick urinalysis showed 3+ protein, pH 9, and 3+ glucose. The patient had no known history of kidney disease. The patient was sent to the emergency department for further evaluation, given concerns for the possibility of nephrotoxic mushroom ingestion. In the ED, his vitals and exam remained normal. On laboratory evaluation, the patient had mildly elevated transaminases (ALT 179 U/L, AST 49 U/L) and significantly abnormal renal function (creatinine 2.8 mg/dL). Poison control was contacted. Given locality, timing of symptom onset, and presentation of acute kidney injury (AKI), the consulting toxicologist suspected the most likely species of mushroom was the *Amanita smithiana*.

Clinical Course

The patient was admitted to the hospital and received intravenous fluids. The following morning he was anuric and his labs showed worsening renal function but stable transaminases (creatinine of 5.24 mg/dL, ALT 176 U/L, and AST 48 U/L). A viral hepatitis panel was nonreactive. Hemodialysis was initiated via a temporary internal jugular dialysis catheter. A renal biopsy 3 days later revealed acute tubular necrosis (ATN). A tunneled hemodialysis catheter was subsequently placed on day 7; the patient was discharged 2 days later with plans to continue dialysis and follow up with nephrology. Creatinine at the time of discharge was 8.2 mg/dL and he had begun producing urine. His creatinine was 1.59 mg/dL when seen by nephrology on day 20 of his illness and the transaminases at that time had returned to normal. The dialysis catheter was removed 9 days later (29 days after ingestion). The patient experienced some mild weakness after hospitalization, but was able to return to work 1 month after hospital discharge.

Discussion

Acute nausea, vomiting, and diarrhea are extremely common complaints in urgent care medicine, often due to viral gastroenteritis, and typically resolve with supportive care. In rare cases, however, such as after toxic mushroom ingestion, these symptoms can suggest the possibility of imminent organ failure.

In this case, the history of GI symptoms following ingestion of foraged wild mushrooms was critical for expanding the differential diagnosis. The dipstick urinalysis, which demonstrated proteinuria and glucosuria in a patient without known renal disease, raised further concern for mushroom toxicity. ED referral was helpful in allowing for further immediate laboratory assessment, as well as admission for monitoring.

Urgent care providers are constantly faced with decisions regarding possible escalation of care to an ED setting. Often, the indications are obvious; other times, however, they are more subtle.

While *Amanita smithiana* poisoning causes predominantly renal injury, there are many other species of poisonous mushrooms which can affect kidney function or result in toxicity to other target organs. Most notably, *Amanita phalloides*, commonly referred to as the “death cap,” can cause severe liver injury and fulminant hepatic failure, even in minute quantities.⁴ Initial GI upset, however, is a common feature in most cases of foraged mushroom poisoning, with misidentification being the most common cause of accidental ingestion.⁵ Therefore, a history of eating foraged mushrooms is worth exploring in patients presenting with GI distress. Even with mild presenting symptoms, these patients should be discussed with a specialist from the local poison center and, generally, referred to an ED immediately for full renal and liver function testing. Such GI symptoms may be a harbinger for impending organ failure over subsequent days, and determining with certainty that the mushroom species consumed was nontoxic is rarely achievable in urgent care. ■

References

1. Tuloss R, Lindgren J. *Amanita smithiana*: taxonomy, distributions, and poisonings. *Mycotaxon*. 1992;45:373-387.
2. Kirchmair M, Carrilho P, Pfab R, et al. *Amanita* poisonings resulting in acute, reversible renal failure: new cases, new toxic *Amanita* mushrooms. *Nephrol Dial Transplant*. 2012;27(4):1380-1386.
3. West PL, Lindgren J, Horowitz BZ. *Amanita smithiana* mushroom ingestion: a case of delayed renal failure and literature review. *J Med Toxicol*. 2009;5(1):32-38.
4. Santi L, Maggioli C, Mastroberardino M, et al. Acute liver failure caused by *Amanita phalloides* poisoning. *Int J Hepatol*. 2012;2012:487480.
5. Brandenburg WE, Ward KJ. Mushroom poisoning epidemiology in the United States. *Mycologia*. 2018. 110(4):637-641.