ABSTRACT
Vomiting with abdominal pain is a common presentation in the emergency department (ED). Without a careful history, unusual causes, such as toxic ingestion, may evade diagnosis. We report a case of an Asian couple who presented to the ED with vomiting and epigastric distress. They were discharged with no definite diagnosis, but on a return ED visit the following day were diagnosed with toxic ingestion of *Gyromitra esculenta*, commonly known as the western false morel. The patients were admitted and treated with intravenous hydration and pyridoxine. Both patients developed mild hepatotoxicity but went on to fully recover. This case demonstrates that the western false morel may cause significant toxicity and it highlights the importance of obtaining a complete history in patients who present with non-specific gastrointestinal symptoms.

Key words: *Gyromitra esculenta*, false morel, gyromitrin, monomethylhydrazine, mushroom poisoning

RÉSUMÉ
Le service d’urgence reçoit souvent des patients qui présentent douleurs abdominales et vomissements. Sans anamnèse, des causes inhabituelles, telle que l’ingestion toxique, peuvent échapper au diagnostic. Nous rapportons le cas d’un couple asiatique qui s’est présenté au service d’urgence en détresse épigastrique accompagnée de vomissements. Le couple a reçu son congé sans diagnostic précis, mais s’est présenté à nouveau le lendemain à l’urgence, où on a alors diagnostiqué une ingestion toxique de *Gyromitra esculenta*, communément appelé fausse morille. Les patients ont été admis et traités par hydratation et pyridoxine intraveineuses. Les deux patients ont développé une légère hépatotoxicité pour ensuite se rétablir complètement. Ce cas démontre que la fausse morille peut causer une forte toxicité et qu’il est important de prendre des renseignements complets sur les antécédents des patients qui présentent des symptômes gastrointestinaux non identifiés.

CASE REPORT • OBSERVATIONS DE CAS

Poisoning due to raw *Gyromitra esculenta* (false morels) west of the Rockies

Anne M. Leathem, BSP, MSP; Thomas J. Dorran, MD, MBA

Introduction

Despite the fact that they have a long history of poisoning and are no longer recommended for consumption, *Gyromitra* species, or false morels, have been widely hunted by mushroom lovers. Amateurs sometimes mistake false morels (Fig. 1) for the prized true morels. Both are shades of brown and grow mainly near conifers west of the Rocky Mountains. False morels are irregularly shaped and wrinkled like the surface of the brain, hence their bizarre common names, ‘brain mushrooms’ and ‘beefsteaks.’ *Morchella* species, or true morels (Fig. 2), are conical and the stem is fused to the bottom of the cap. They are hollow from base to apex and have a distinctly pitted head. Western false...
morels have either a hollow or a stuffed stalk.

The North American Mycological Association (NAMA) has collected reports of mushroom poisoning for many years. Cases are voluntarily reported, chiefly by members. In over 30 years, there have been 27 reports of poisoning by *Gyromitra esculenta*. None of these was fatal, but liver damage was reported in 9 cases (33%) and kidney failure in 3 (11%). Unfortunately, these reflect only a small proportion of all cases since NAMA receives only 1% of mushroom poisonings that are handled by US Poison Control Centres. Serious poisonings by *Gyromitra esculenta* are more common in the eastern US and rare west of the Rocky Mountains. It has been suggested that western *Gyromitra* species contain little or no toxin. We present 2 cases of hepatotoxicity resulting from the consumption of raw *Gyromitra esculenta*.

**Case 1**

In springtime in the southern BC city of Kamloops, a Korean couple ate a meal containing wild mushrooms. Two hours later, both people developed gastrointestinal upset and began to vomit. They went to the emergency department (ED) later that night because of continuing symptoms and were treated symptomatically and released. Owing to their inability to speak English, the history of mushroom exposure was not discovered at that time. They returned to the ED the following day with their English-speaking son. A thorough history revealed that the patients had eaten uncooked wild mushrooms. The 49-year-old woman complained of persistent intense abdominal pain, vomiting, and hot and cold chills. She had no diarrhea. She was alert, oriented and distressed; in obvious pain. Vital signs included a pulse of 88 beats/min, a respiratory rate of 20 breaths/min, a temperature of 36.6°C and blood pressure of 100/60 mm Hg. Physical examination revealed lower abdominal tenderness, reduced bowel sounds, dry mucous membranes and a clear chest. Liver function tests revealed an elevated lactate dehydrogenase (LD) at 243 U/L, (normal = 60–190), elevated total bilirubin at 63 µmol/L (normal = 9–26) and increased international normalized ratio at 1.3 (normal = 0.8–1.2).

Some mushrooms were retrieved from the patients’ home and were identified in the ED as *Gyromitra esculenta* by comparing the mushroom sample with photos in a mushroom guide. We contacted the BC Drug and Poison Information Centre and, based on symptoms, time of onset and physical description of the fungi, made a probable diagnosis of poisoning by monomethylhydrazine-containing mushrooms. The patient was rehydrated with saline and treated with meperidine and dimenhydrinate to control pain and nausea. Intravenous pyridoxine 25 mg/kg was given prophylactically. For the next 4 days, profound fatigue, abdominal pain, anorexia, nausea and periodic vomiting continued. The patient received famotidine and antacids as needed for esophagitis caused by vomiting. Jaundice was noted 2 days post-ingestion, and the liver enzymes peaked on day 5, with an LD level of 693 U/L, an aspartate aminotransferase (AST) of 431 U/L (normal = 10–47) and an alanine aminotransferase of 472 U/L (normal = 30–65). The patient’s symptoms gradually diminished, but her abdominal pain persisted 6 days post-ingestion.

**Case 2**

The 56-year-old husband was also admitted the day after ingestion. He was alert and oriented, with a pulse of 76 beats/min, a respiratory rate of 18 breaths/min, blood pres-
Mushroom poisoning should be considered in the differential diagnosis of unexplained gastrointestinal illness. The cases discussed demonstrate that toxin levels in western rivers consume per unit body weight and the frequency of vomiting has occurred.

Poisonings from Gyromitra mushrooms should be relatively easy to differentiate from poisoning by hepatotoxic amanitas because Gyromitra mushrooms grow mainly in the spring and early summer and have a strange ‘brain-like’ appearance, whereas hepatotoxic amanitas grow in the autumn and are typically-shaped (umbrella-like) gilled mushrooms. Various resources, including mushroom guides and web sites are available to help ED staff in the identification of mushrooms. The BC Drug and Poison Information Centre’s Poison Management Manual has a table to help with the identification of mushroom toxin species. Toxin concentration may also vary due to metabolic factors, such as whether a person is a fast or slow acetylator.

The primary toxin, gyromitrin (acetaldehyde-formylmethylhydrazone), is hydrolyzed easily by cooking and by stomach acid into methylformylhydrazine and then into monomethylhydrazine (MMH), a component of rocket fuel, vapourizes at 87.5°C and, while most is boiled off during food preparation, some may remain in the cooking water with the potential for ingestion unless the water is discarded. Because MMH toxin is volatile, the chef may inhale toxin during preparation and become sicker than the dinner guests. Drying the mushrooms for several days can reduce the toxin concentration.

Generally, hydrazines are cytotoxic, and hepatic breakdown products of MMH produce an irreversible blockade of cytochrome P450, aminooxidases and glutathione. Free methyl radicals may cause hepatic necrosis and MMH also inactivates pyridoxine-dependent coenzymes in the brain, which may reduce brain gamma-aminobutyric acid (GABA) levels and predispose one to seizures. MMH may cause gastrointestinal irritation, hemolysis and methemoglobin formation. It is also a low-grade carcinogen. If renal damage occurs, it may be secondary to hemolysis and dehydration. The lethal dose of gyromitrin is estimated to be 20–50 mg/kg in adults and 10–30 mg/kg in children. The lethal dose of monomethylhydrazine is 4.8–8.0 mg/kg in adults and 1.6–4.8 mg/kg in children.

Gyromitra esculenta is also a low-grade carcinogen. If renal damage occurs, it may be secondary to hemolysis and dehydration. The lethal dose of gyromitrin is estimated to be 20–50 mg/kg in adults and 10–30 mg/kg in children. The lethal dose of monomethylhydrazine is 4.8–8.0 mg/kg in adults and 1.6–4.8 mg/kg in children.

Gyromitra esculenta has also been shown to be carcinogenic in animals.

In most cases of poisoning by MMH-containing mushrooms, gastrointestinal symptoms occur 5–8 hours post-ingestion, but they sometimes occur as early as 2 hours post-ingestion. Symptoms are often limited to gastrointestinal discomfort and the patient typically recovers in 2–6 days. In more severe cases, hepatotoxicity and neurologic symptoms (vertigo, ataxia, fatigue, tremor, seizures) may occur. In the 2 cases presented here, the initial gastrointestinal upset was followed by signs and symptoms of increasing hepatic dysfunction. Liver enzymes were increased on the first day post-ingestion and peaked on the fourth post-ingestion day. The only neurologic symptoms noted were generalized fatigue in both patients and a short-term headache in the man. We do not know whether the prophylactic dose of pyridoxine prevented more serious neurologic symptoms.

Treatment for poisoning by MMH-containing mushrooms is symptomatic and supportive, but prophylactic pyridoxine may prevent or arrest seizures. Gastrointestinal decontamination is generally not required if frequent vomiting has occurred.

The patient received saline for rehydration and meperidine for pain and nausea. He was also given an intravenous dose of prophylactic pyridoxine 25 mg/kg. On day 2 post-ingestion, his LD peaked at 236 U/L and jaundice was noted. On day 4, his AST peaked at 116 U/L and on day 5 the patient complained of a transient headache. All symptoms resolved before his discharge on day 6.

Discussion

Toxic reactions to Gyromitra mushrooms and related species vary widely in severity, perhaps because the amount of toxin varies greatly between, and even within, species. Toxin concentration may also vary according to growing conditions, differences in maturity and geographical area. Gyromitra esculenta generally contain the highest toxin concentration, but individual reactions to Gyromitra poisoning also vary. Gyromitra toxins are unstable and volatile, so the method of their preparation affects the amount of toxin ingested. In addition, the amount of mushroom consumed per unit body weight and the frequency of consumption may determine whether toxic effects occur. Individual reactions may also be affected by metabolic factors, such as whether a person is a fast or slow acetylator.

The primary toxin, gyromitrin (acetaldehyde-formylmethylhydrazone), is hydrolyzed easily by cooking and by stomach acid into methylformylhydrazine and then into monomethylhydrazine (MMH), a component of rocket fuel, vapourizes at 87.5°C and, while most is boiled off during food preparation, some may remain in the cooking water with the potential for ingestion unless the water is discarded. Because MMH toxin is volatile, the chef may inhale toxin during preparation and become sicker than the dinner guests. Drying the mushrooms for several days can reduce the toxin concentration.

Generally, hydrazines are cytotoxic, and hepatic breakdown products of MMH produce an irreversible blockade of cytochrome P450, aminooxidases and glutathione. Free methyl radicals may cause hepatic necrosis and MMH also inactivates pyridoxine-dependent coenzymes in the brain, which may reduce brain gamma-aminobutyric acid (GABA) levels and predispose one to seizures. MMH may cause gastrointestinal irritation, hemolysis and methemoglobin formation. It is also a low-grade carcinogen. If renal damage occurs, it may be secondary to hemolysis and dehydration. The lethal dose of gyromitrin is estimated to be 20–50 mg/kg in adults and 10–30 mg/kg in children. The lethal dose of monomethylhydrazine is 4.8–8.0 mg/kg in adults and 1.6–4.8 mg/kg in children.

Gyromitra esculenta is also a low-grade carcinogen. If renal damage occurs, it may be secondary to hemolysis and dehydration. The lethal dose of gyromitrin is estimated to be 20–50 mg/kg in adults and 10–30 mg/kg in children. The lethal dose of monomethylhydrazine is 4.8–8.0 mg/kg in adults and 1.6–4.8 mg/kg in children.

Gyromitra esculenta has also been shown to be carcinogenic in animals.

In most cases of poisoning by MMH-containing mushrooms, gastrointestinal symptoms occur 5–8 hours post-ingestion, but they sometimes occur as early as 2 hours post-ingestion. Symptoms are often limited to gastrointestinal discomfort and the patient typically recovers in 2–6 days. In more severe cases, hepatotoxicity and neurologic symptoms (vertigo, ataxia, fatigue, tremor, seizures) may occur. In the 2 cases presented here, the initial gastrointestinal upset was followed by signs and symptoms of increasing hepatic dysfunction. Liver enzymes were increased on the first day post-ingestion and peaked on the fourth post-ingestion day. The only neurologic symptoms noted were generalized fatigue in both patients and a short-term headache in the man. We do not know whether the prophylactic dose of pyridoxine prevented more serious neurologic symptoms.

Treatment for poisoning by MMH-containing mushrooms is symptomatic and supportive, but prophylactic pyridoxine may prevent or arrest seizures. Gastrointestinal decontamination is generally not required if frequent vomiting has occurred.

Poisonings from Gyromitra mushrooms should be relatively easy to differentiate from poisoning by hepatotoxic amanitas because Gyromitra mushrooms grow mainly in the spring and early summer and have a strange ‘brain-like’ appearance, whereas hepatotoxic amanitas grow in the autumn and are typically-shaped (umbrella-like) gilled mushrooms. Various resources, including mushroom guides and web sites are available to help ED staff in the identification of mushrooms. The BC Drug and Poison Information Centre’s Poison Management Manual has a table to help with the identification of mushroom toxin group, based on symptoms and time of onset post-ingestion. Regional poison centres may also be able to help identify a mushroom or toxin, or to refer care providers to local mycologists. The ED should be prepared to provide information about the mushroom, its habitat and the number of different species ingested by the patient.
Gyromitra species may be sufficient to cause poisoning, especially if eaten raw. The variability of toxin content, which is dependent on many factors including food preparation, emphasizes the need to extract a complete and accurate history from patients and underscores the need to have relevant poison information resources readily available in the ED.

Competing interests: None declared.

References


4. Beug MW, Shaw M, Cochran KW. Thirty years plus of mushroom poisoning: summary of the approximately 2,000 reports in the NAMA case registry. McIlvainea [Forthcoming].


Correspondence to: Anne M. Leathem, anne@leathem.ca