Mushroom and Toadstool Toxicology

There is no difference between mushrooms and toadstools, although the term toadstools has gradually become applied to toxic types. They are all agarics (ie fungi with gills beneath the cap).[1]

Mushrooms found in gardens or on lawns are highly unlikely to be seriously toxic but may cause gastrointestinal upsets or be hallucinogenic.

Epidemiology

- Poisoning with potentially lethal mushrooms in the UK is extremely rare.
- Highly toxic types are not likely to be found in towns or cities.
- *Amanita phalloides* (death cap) is the most toxic UK species and is found in all areas with the possible exception of Sutherland and Western Ross. The equally toxic *Amanita virosa* is found in the birch woods of the north.
- *Cortinarius* spp. are becoming more frequent but only three cases of poisoning have been reported in Britain. Symptoms may not start until several days after ingestion.

Presentation

- Mushrooms which cause features within six hours of ingestion are unlikely to be seriously toxic.[1]
- A long latency period (>6 hours), seen in a small number of cases (estimated at about 2% in one series), can be observed with amatoxins, orellanus and gyromitrin syndromes.[2] This generally signifies a serious ingestion and should be considered potentially life-threatening.
- Gastrointestinal disturbance nearly always occurs - eg, nausea, vomiting, abdominal pain.
- Other symptoms include blurred vision, headache, loss of consciousness and confusion.
- In severe cases there may be features of organ failure - eg, jaundice, haemorrhage.[2]

Differential diagnosis

- Food poisoning.
- Other intoxications, including illicit or prescription drugs.
- Other causes of acute kidney injury.
- Other causes of liver failure.
- Psychosomatic: some patients have been reported to develop anxiety-related symptoms after learning that they have eaten wild mushrooms.

Investigations

- Further investigations will be dependent on the clinical features and well-being of the patient and identification of the ingested mushroom.
- Urinary amanitin analysis is a valuable diagnostic tool.[3]

Management

See also separate Acute Poisoning - General Measures article.

- Pre-hospital: make every attempt to identify the ingested mushroom. In the UK, Toxbase® has descriptions and photographs to help with identification.[1] Care is otherwise supportive.
- Early onset of gastrointestinal symptoms predicts a benign course so nothing more than symptom control may be warranted.
- Consider activated charcoal in those presenting with gastrointestinal symptoms six hours after ingestion or if a patient presents before symptom development following known ingestion of hepatotoxic or nephrotoxic species.
- There is evidence to suggest that multi-dose activated charcoal may be more beneficial in amanita poisonings, as some of the toxins undergo enterohepatic circulation.[4]
- In the UK, contact the National Poisons Information Centre.[5]
- Observe for six hours to exclude central nervous system (CNS) and gastrointestinal toxicity. Treat symptomatically.
- Atropine for cholinergic symptoms (eg, as with inocybe mushroom toxicity) and pyridoxine for gyromitra.
- The very rare hepatotoxic mushrooms can cause gastrointestinal symptoms from six hours onwards.
- N-acetylcysteine is sometimes used as an antidote for hepatotoxicity associated with mushroom poisoning but the evidence for its effectiveness is limited.[6, 7]
- For the UK, consult specific entries in Toxbase® if the mushroom has been identified.

Specific mushroom toxicities
**Amanita phalloides**

- The most poisonous mushroom toxins are produced by *A. phalloides*.
- The main toxins responsible are the amatoxins. The amatoxins cause necrosis of the liver and, to a lesser degree, the kidney. [8]
- Poisoning is characterised by a delay of between 6 and 24 hours in the onset of symptoms following ingestion. Violent vomiting, diarrhoea and abdominal pain are the first symptoms. Patients then make an apparent recovery but this is followed a few days later by hepatic failure and acute kidney injury. [8, 9]
- Therapy includes:
  - Supportive care, including correction of hypoglycaemia and electrolyte imbalance.
  - Gastric lavage and activated charcoal.
  - Anecdotal and animal studies suggest a potential benefit of high-dose penicillin, silibinin (a constituent of the extract silymarin derived from the milk thistle, *Silybum marianum*), cimetidine, aucubin (an iridoid glycoside of *Aucuba japonica*) and kutkin.
  - Fulminant hepatic failure may require a liver transplant. [10]
- Factors associated with a poor prognosis are low blood pressure, encephalopathy, mucosal haemorrhage, oliguria, hypoglycaemia, thrombocytopenia, hyponatraemia, raised urea, hepatic failure with raised international normalised ratio, and raised activated partial thromboplastin time values. [11]

**Cortinarius spp.**

- Orellanine is a nephrotoxin found in some *Cortinarius* spp.
- Early symptoms are often vague, with gastrointestinal dysfunction 1-2 days after ingestion. Therefore, poisoning may initially be overlooked.
- Patients often first present with acute renal failure which may follow at any time from 36 hours to 2 weeks after ingestion. Supportive care is the only therapeutic option. [12]
- No specific treatment is available and therapy is directed toward the renal failure, including dialysis and possible transplantation. [13]

**Inocybe spp.** [1]

- Common names: common white fibre head, peaked inocybe, red-staining inocybe, straw-coloured fibre head, turnip foot inocybe, woolly inocybe.
- There are over eighty recognised *Inocybe* species found in the UK and Ireland. Differentiation between different fungi within the genus is very difficult.
- Some inocybe mushrooms contain muscarine.
- Features develop between 15 minutes and 2 hours of ingestion and include perspiration, salivation, lacrimation, vomiting, abdominal pain, diarrhoea, urgency of micturition, flushing, bradycardia, hypotension, constricted pupils and blurred vision.
- Deaths are uncommon.
Magic mushrooms

- This is a nonspecific term for fungi which are hallucinogenic.
- In the UK they are commonly liberty cap mushrooms (*Psilocybe semilanceata*), which are often found in parks, golf courses and football pitches.
- The fatal dose is not known and potency is variable.
- Hallucinogenic effects occur after ingestion of 20-100 mushrooms. Signs of intoxication generally develop within one hour and may persist for up to 15 hours.
- Features include nausea, vomiting, abdominal pain, flushing of the face and neck, tachycardia, dilated pupils, diastolic hypertension and drowsiness.
- Rhabdomyolysis leading to renal failure, arrhythmias and myocardial infarction have been reported.
- Behavioural effects vary from restless overactivity with lack of co-operation, and aggressiveness, to withdrawn, uncommunicative staring.
- Perceptual abnormalities include visual hallucinations, distorted body image, sounds and tactile sensation.
- Ability to judge heights and distances may be grossly impaired.
- Management:
  - All symptomatic patients should be referred to hospital.
  - Activated charcoal (50 g for adults; 1 g/kg for children) only if the patient presents within one hour of ingestion.
  - Observe for at least four hours after ingestion. Monitor pulse, temperature, blood pressure, cardiac rhythm and perform a 12-lead ECG.
  - Check renal function, electrolytes and creatine kinase in symptomatic patients.
  - Sedate if necessary with intravenous lorazepam or diazepam.
  - Frequent or prolonged seizures should be treated with intravenous diazepam or lorazepam.

Other mushroom toxicities

- Gastrointestinal mushroom syndromes present with abdominal discomfort, cramping, nausea, vomiting and/or diarrhoea. Dehydration is the most common complication. Most symptoms resolve by 24 hours and the prognosis is generally good.
- Anticholinergic symptoms can occur: dizziness, lack of co-ordination, ataxia, seizures, hallucinations, muscle spasms, flushing and dilated pupils may be observed.
- Cholinergic effects may result from muscarine ingestion: salivation, bladder cramping, diarrhoea and difficulty with visual accommodation.[14]
- Neuropsychiatric symptoms: include hallucinations or delirium. Muscle weakness, drowsiness, hallucinations, hyperkinesis and mydriasis have been described. Patients may have hallucinations while awake and then have a prolonged sleep that lasts hours. The prognosis is generally good and symptoms resolve within 24 hours.
- The Coprinus syndrome: rapid onset of nausea, vomiting, tachycardia, palpitations, paraesthesiae, diaphoresis and flushing. Hypotension may also occur. This syndrome has been referred to as a disulfiram-like reaction, as it is associated with ethanol use from 30 minutes to five days following a mushroom meal. Symptoms generally last 2-4 hours.
- Gyromitrin toxicity may manifest at 4-6 hours post-ingestion with gastrointestinal dysfunction. Dehydration, hepatic dysfunction, methaemoglobinemia, intravascular haemolysis and CNS effects (eg, malaise, tremor, myoclonus, delirium, seizures, encephalopathy) may occur.

Complications

- Respiratory: aspiration pneumonia, non-cardiogenic pulmonary oedema.
- CNS complications: convulsions are common in gyromitrin poisoning, but they also may be due to hypoxia, acidosis, and metabolic abnormalities. Cerebral oedema may be a complication of hypoxia, acidosis and hepatic failure.
- Hepatic complications: liver failure and hypoglycaemia are complications for amatoxin and gyromitrin poisoning.
- Renal failure is a common complication of orellanine poisoning but also may be due to hypoperfusion and shock and may be part of the hepatorenal syndrome.
- Methaemoglobinemia and haemolysis may complicate gyromitrin poisoning.
- Trauma may complicate hallucinogenic mushroom poisoning.
- Hypovolaemia and electrolyte disturbances may complicate any mushroom poisoning.

Prognosis

- The prognosis in the great majority of cases of mushroom poisoning is excellent.
- Amatoxin poisoning: with good supportive care, the mortality rate for amatoxin poisoning may be reduced from 60% but, owing to progressing fulminant hepatic failure, is still high (5-20%).[11]
- Gyromitrin poisoning: most recover and death is rare.
- Orellanine poisoning: a shorter time course between ingestion and toxicity correlates with a worse prognosis. Mortality is high but mild renal insufficiency may resolve within months of the ingestion.

Prevention

- Don't take any risks with wild mushrooms and keep children well away.
- Education: even experienced mycologists may not be able to distinguish edible from poisonous mushrooms without microscopic examination.
- Identifying mushrooms is an exact science that is very difficult and time-consuming.
- Gourmet cooks who have harvested wild mushrooms have been poisoned. To be safe, avoid all wild mushrooms.

Further reading & references


4. National Poisons Information Service


Disclaimer: This article is for information only and should not be used for the diagnosis or treatment of medical conditions. Patient Platform Limited has used all reasonable care in compiling the information but makes no warranty as to its accuracy. Consult a doctor or other healthcare professional for diagnosis and treatment of medical conditions. For details see our conditions.

View this article online at: patient.info/doctor/mushroom-and-toadstool-toxicology

Discuss Mushroom and Toadstool Toxicology and find more trusted resources at Patient.

Ask your doctor about Patient Access

© Patient Platform Limited - All rights reserved.