Mushrooms

Hans Persson

Abstract

The incidence of fungal poisoning varies considerably globally and is related to local habits, economic factors and lifestyle. Mushroom poisoning is mostly an accidental result of a mix-up between edible and toxic fungi. However, intentional ingestion of psychotropic ('magic') mushrooms has become a problem. Among thousands of mushroom species worldwide, fewer than a hundred are severely toxic. Most fungal toxins cause mild or moderate poisoning, often only gastroenteritis; the ingestion of a few species of extremely poisonous fungi defines the medical dimension of the problem. The most dreaded poisonings are those caused by cytotoxic fungi, for example amatoxins in death cap (*Amanita phalloides*) and destroying angel (*Amanita virosa*); both cause severe gastroenteritis and liver damage. Orellanine, occurring in certain *Cortinarius* spp., can induce severe and persistent kidney damage. Dramatic, but rarely lethal, effects are caused by fungi containing neurotoxins such as muscarine (*Clitocybe* and *Inocybe* spp.), psilocybin (*Psilocybe* and *Panaeolus* spp. – 'magic' mushrooms), isoxazoles (fly agaric, panther cap) and gyromitrin (false morels). Treatment is focused on general symptomatic and supportive care, although antidotes exist for fungi containing muscarine (atropine), gyromitrin (pyridoxine) and amatoxins (silibinin [silibinin is approved e.g. in Sweden and Germany (Legalon SIL D)], penicillin); the benefit of the latter has not yet been fully established.

Keywords amatoxin; antidotes; gyromitrin; isoxazoles; muscarine; mushroom poisoning; orellanine; psilocybin

Introduction

The incidence of mushroom poisoning varies geographically depending on climate, occurrence of toxic fungi, lifestyle and local traditions. Toxic mushroom ingestion can be related to:

- confusion between edible and poisonous species
- ignorance of the risks (careless harvesting)
- intentional ingestion of psychotropic mushrooms
- accidental ingestion by children
- suicidal ingestion (rare).

Diagnosis involves a careful history, assessment of the clinical features and, sometimes, examination of mushroom specimens by a mycologist. If available, chemical analysis can be useful in the acute phase for detecting and measuring the concentrations of, for example, amatoxin.

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Cytotoxic mushrooms containing amatoxins, orellanine and gyromitrin

Amatoxins

These occur in *Amanita phalloides* (Figure 1), *Amanita virosa*, *Amanita verna*, *Galerina marginata* and in some *Lepiota* spp.

Mechanisms of toxicity and toxicokinetics: amatoxins are highly toxic, and large doses can result in death in spite of treatment. Amatoxins are cyclic octapeptides that inhibit transcription from DNA to mRNA because of the blockade of nuclear RNA polymerase II. This will result in impaired protein synthesis and cell death.[1] Other suggested mechanisms are induction of apoptosis, formation of oxygen free radicals and depletion of hepatic glutathione.

Amatoxins are rapidly absorbed and distributed, and uptake is particularly high in the parenchymal cells of the liver, the kidneys and the intestinal mucosa. Excretion is mainly renal, but significant amounts are also excreted in bile and faeces.[2]

Clinical features:[1–3] intense gastrointestinal symptoms, with severe watery diarrhoea, start 8–24 hours (mean 12 hours) after ingestion and persist for 24 hours or longer. Patients become dehydrated, exhausted, hypovolaemic, hypotensive and sometimes hypoglycaemic. Signs of liver damage appear during the second day and hepatic failure can ensue. Impaired kidney function is often seen initially because of dehydration and shock, but its later occurrence is due to amatoxin-induced renal damage, which is a poor prognostic sign. Taken together, these features may suggest a probable fatal outcome.

Management:[1–3] all patients who have eaten a mushroom meal containing amatoxin fungi should be given fluids to replace the heavy loss of body fluids and electrolytes. Acid–base correction should be performed. Hepatic and renal function are supported conventionally.

Repeated doses of charcoal can enhance the excretion of amatoxin. In adults, an initial dose of activated charcoal 50 grams should be given and should be followed by repeated doses of activated charcoal 25 grams every 4 hours for 3 days. In children, an initial dose of activated charcoal 25 grams is given and followed by charcoal 10 grams every 4 hours for 3 days. A modestly increased diuresis (around 200 ml/hour in adults) is advised for the first 24–48 hours to support toxin elimination, although the benefit has not been demonstrated.

The value of silibinin or benzylpenicillin in reducing amatoxin-induced hepatic toxicity is not proven conclusively, but silibinin 5 mg/kg intravenously over 1 hour followed by 20 mg/kg/24 hours by continuous infusion should be considered in cases of substantial ingestion. Treatment is usually given for 3 days after ingestion. Benzylpenicillin 300 mg/kg/24 hours by continuous infusion can be used if silibinin is unavailable. Some recent experimental and clinical data suggest that acetylcysteine might also be of value. Liver transplantation occasionally has to be considered in severe, life-threatening poisoning, normally on day 4–8 after ingestion.

Orellanine

Orellanine is a potent nephrotoxin occurring in a number of fungi belonging to the *Cortinarius* genus, in particular *Cortinarius*

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Diversity of species of the genus *Conocybe* (Bolbitiaceae, Agaricales) collected on dung from Punjab, India

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Abstract

A study of diversity of coprophilous species of *Conocybe* was carried out in Punjab state of India during the years 2007 to 2011. This research paper represents 22 collections belonging to 16 *Conocybe* species growing on five diverse dung types. The species include *Conocybe albipes*, *C. apala*, *C. brachypodii*, *C. crispa*, *C. fuscimarginata*, *C. lenticulospora*, *C. leucopus*, C. *magnicapitata*, *C. microrrhiza* var. *coprophila* var. nov., *C. moseri*, *C. rickenii*, *C. subpubescens*, *C. subxerophytica* var. *subxerophytica*, *C. subxerophytica* var. *brunnea*, *C. uralensis* and *C. velutipes*. For all these taxa, dung types on which they were found growing are mentioned and their distinctive characters are described and compared with similar taxa along with a key for their identification. The taxonomy of ten taxa is discussed along with the drawings of morphological and anatomical features. *Conocybe microrrhiza* var. *coprophila* is proposed as a new variety. As many as six taxa, namely *C. albipes*, *C. fuscimarginata*, *C. lenticulospora*, *C. leucopus*, *C. moseri* and *C. subpubescens* are the first time records from India. *Conocybe velutipes* is the first time record from North India.

Key Words – Basidiomycota – dung – hymeniform – lecythiform – taxonomy

Introduction

The genus *Conocybe* belongs to the family Bolbitiaceae and can be separated from the other genera of the family by lecythiform cheilocystidia with a round capitellum and pileal margin not plicate-sulcate. It is characterized by its delicate carpophores which are small to medium sized and are reported to grow on a variety of substrates such as on the ground, in woods, pastures, gardens, etc. and on dung, occasionally attached to plant remains and vegetable refuse, sometimes even on sand dunes as discussed by Singer (1986), Pegler (1977, 1983, 1986).

Kirk et al. (2008) recognized 200 species of *Conocybe* the world over. From India, about 30 species of *Conocybe* are known (Bilgrami et al. 1991, Saini & Atri 1995, Natarajan et al. 2005, Atri et al. 2009, 2012, Kumar et al. 2014). Natarajan & Raaman (2005) reported 03 species, namely *C. ambigua* (Kühner) Sing.: Sing., *C. plumbeitincta* (Atk.) Singer and *C. semiglobata* Kühner: Singer as growing on cow dung from Tamil Nadu state in India. Thomas et al. (2001) reported three species, namely *C. pseudopubescens* K. A. Thomas, Hauskn. & Manimohan, *C. volvata* K. A. Thomas, Hauskn. & Manimohan growing on elephant dung and *C. zeylanica* (Petch) Boedijn on a

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