MEDICAL PROBLEMS OF MUSHROOM INGESTION

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Toxic mushroom ingestion is a serious problem in many countries in Eastern and Southern Europe, and many deaths occur each year in several of these countries. In the British Isles, serious poisoning is uncommon and deaths are extremely rare; not more than one or two per annum. For the year 1987, our information service received 337 enquiries (0.68% of total) and recorded no deaths, and no serious illnesses. This is probably because of a much more cautious approach towards the culinary use of mushrooms.

The first step in management of any ingested substance which may be poisonous is to identify the substance and amount taken. With mushrooms, it is necessary to identify the species. The toxicity may vary with the parts of the fungus ingested, the time in its growing season, the amount ingested and individual susceptibility. The evolution (if any) of the illness can be characteristic for different fungal toxins. This has been well described by Pegler & Watling (1982). A common pitfall is the patient who presents with symptoms which were not caused by the mushroom. This is frequently a bacterial exotoxin contaminating the food, but there are many other causes. Table 1 summarizes the main points in diagnosis.

Table 1: DIAGNOSIS OF MUSHROOM POISONING

From the fungus:
- morphology
- habitat and mycorrhizal relationship
- spores
- biochemical tests

From the preparation of the fungus:
- was it dried
- was it eaten cooked or raw; if boiled was the water thrown away
- was it taken with alcohol
- was it taken on one or several occasions

From the patient's symptoms:
- time to initial onset of symptoms
- which symptoms have occurred and in which order
- have all participants been similarly affected

From samples taken from the patient:
- vomit or gastric aspirate for spores
- blood or urine samples for biochemical or immunological techniques of identification

MEDICAL MANAGEMENT

The main options open to the doctor confronted with a case of possible mushroom poisoning are to:
(a) do nothing
(b) empty the stomach by inducing vomiting or by gastric lavage
(c) try to reduce absorption by the body
(d) use specific antidotes

Doing nothing may be sensible if the ingestion is probably non-toxic and especially if it has occurred over 4 h previously, when emptying the stomach will be of little use. The only agent which is nowadays used to induce vomiting is syrup of ipecacuanha, which is the preferred way of emptying the stomach in small children. Salt water should never be used. In adults, gastric lavage ('stomach washout') is very commonly used.

Absorption of a poison by the body can be reduced by giving activated charcoal. It has a large surface area and is capable of combining in a non-specific manner with most poisons, whether they be drugs or chemicals or plant or fungal toxins. It is thus logical to give activated charcoal after emptying the stomach. It has minimal toxicity, though it may cause vomiting. Apart from removing the poison, other managements may also be used, and can be broadly divided into two types: symptomatic and specific.

Symptomatic treatments are aimed at controlling the symptoms produced by the poison, such as giving antiemetics to control vomiting, analgesics for pain, or

a variety of drugs for the management of liver or kidney failure. *Specific treatments* are aimed at pharmacological reversal of the poison.

EARLY-ONSET POISONINGS

Gastrointestinal Irritants. Illnesses of short onset are common with the gastrointestinal irritant group, which comprises a very wide range of fungi. They include *Russula emetica, Lactarius torminosus, Boletus satanas* and many others. Abdominal pain, nausea and vomiting usually commence within 30 min to 3 h of ingestion, and diarrhoea may also occur. Management is symptomatic; there are no specific antidotes for this type of poisoning. Antiemetic drugs (such as prochlorperazine or metoclopramide) can be given if nausea or vomiting are severe, and intravenous fluids will be required if the patient is dehydrated from vomiting or diarrhoea. Activated charcoal may be given in an attempt to bind the toxins.

Hallucinogens. Hallucinogenic mushrooms may be taken accidentally as food but are more commonly taken for their hallucinatory properties. Many contain psilocybin. It takes about 30 fruitbodies of *Psilocybe semilanceata* to produce a hallucinatory experience, which comes on after about 30 min and subsides within 2-3 h.

Other larger fungi which also contain psilocybin are *Psilocybe cubensis, Panaeolus* spp, *Copelandia* spp and *Gymnopilus* spp. The experience may be simply a distortion of colours, shapes and sounds, but sometimes frank hallucinations occur, and occasionally violence and aggression may result. In these cases, the first line of management is to protect the patients from injury, keeping them in a quiet, dark place and to try to establish contact by talking reassuringly ('talking down'). The pupils are dilated and there may be a fast heart rate due to the effects of psilocybin (which is related to lysergic acid, LSD). Drugs for sedation (such as diazepam or chlorpromazine) are rarely needed, and full recovery within six hours is likely. 'Flashbacks' may occur; these consist of repetition of the hallucinatory experience up to six months after ingestion.

Muscimol and Ibotenic Acid. Some hallucinogenic mushrooms contain toxins such as muscimol, and ibotenic acid and pantherin. *Amanita muscaria* and *A. pantherina* are the best known in this group and are abused for their hallucinogenic properties. They may cause elation, excitement and delirium, though they often produce unwanted symptoms such as vomiting, twitching cramps and drowsiness.

Convulsions are rare. Management is usually symptomatic, Atropine should not be given; physostigmine may be tried in severe cases since the toxic effects are mainly not muscarinic but anticholinergic.

Muscarinic Poisoning. Muscarine is present in *Inocybe fastigiata* and *Clitocybe dealbata.* These and similar species may produce rapid onset of sweating, salivation and running nose and eyes, features typical of muscarinic toxicity. More severe poisoning may be associated with abdominal cramps, blurred vision and wheezing. The pupils are constricted and the pulse rate is slowed. These signs of parasympathetic overactivity can be reversed by atropine.

Coprine. Coprinus atramentarius and *Coprinus comatus* are well-known edible mushrooms, but must not be taken with alcohol, since they contain coprine, which is metabolised to 1-aminocyclopropanol. This inhibits aldehyde dehydrogenase, resulting in an accumulation of acetaldehyde in the body. This reaction is the same as the disulfiram ('Antabuse') reaction. Flushing, nausea, vomiting, headache and possibly collapse may occur shortly after alcohol is drunk for up to 48 h or 72 h after eating the mushroom. Severe cases may need intravenous fluids, drugs such as dopamine to raise blood pressure, and drugs to treat cardiac arrhythmias.

LATE-ONSET POISONINGS

Amatoxins. Fungi which cause symptoms more than six h after ingestion are much more serious as a group, and the most important among these are the

species which contain amatoxins. This is a collective name for a number of toxic cyclic octapeptides found in such well-known species as *Amanita phalloides, A. verna* and *A. virosa* and some *Galerina* and *Lepiota* species. The time to onset of symptoms is particularly important; it is never less than six h, and if it is in the region of 7-8 h a fatal outcome is much more likely than if symptoms start at 10 h or more after ingestion. The symptoms of nausea, severe vomiting, colicky pain and watery diarrhoea last for about 24 h and are followed by a 'silent interval' of over 24 h till symptoms of hepatic (liver) failure occur 48-96 h after ingestion. Death is from liver failure; the mortality is now generally given as 20%. Treatment is controversial. Continual duodenal suction and oral activated charcoal are recommended, to interrupt the enterohepatic circulation of amatoxin. Other treatments which may be effective are intravenous penicillin (in very high doses) and silibinin, and alcohol taken with the fungus has a protective effect. This topic has recently been reviewed thoroughly (Floersheim, 1987).

Orellanin. Orellanin, also a cyclopeptide, is found in some *Cortinarius* species (particularly *C. speciosissimus* and *C. orellanus*), and is stable to drying and cooking. The patient has no symptoms for 3-17 days after ingestion; the first symptoms are usually thirst, polyuria (frequent urination) and abdominal pain, and acute renal failure (kidney shut down) may follow (Holmdahl et al, 1984). It is possible that this type of poisoning may go unrecognised as the cause of renal failure because of the long interval between ingestion and symptoms. The medical management is to replace the kidney function with peritoneal dialysis or haemodialysis.

Gyromitrin. Gyromitra species are sought after as edible mushrooms. They contain a compound (loosely called gyromitrin) which in many people is converted to monomethylhydrazine, a competitive inhibitor of the coenzyme pyridoxal phosphate. However, the precusor is volatile and also water-soluble, so that mushrooms do not cause toxicity if dried or if boiled with an excess of water which is then discarded. Cooks and cannery workers have become poisoned by inhalation. Symptoms come on suddenly 6-12 h after ingestion and consist mainly of fatigue, headache, nausea, vomiting and diarrhoea. Convulsions, coma and liver damage, however, may occur, and this poisoning has a 15-35% mortality. In Eastern Europe, it causes up to one-third of mushroom fatalities. Management is to give pyridoxine in large doses intravenously; after this symptoms should subside.

CONCLUSION

This brief outline of the medical aspects of mushroom poisoning should provide an insight into medical management for mycologists, and a guide to symptoms and management for first aiders and medical practitioners. Further details may be found in larger texts on toxicology, or in books on toxic mushrooms, such as Lincoff/Mitchel (1977).

It should be stressed of course that prevention is better than cure. Mycologists can make a valuable contribution in the safe use of mushrooms by encouraging and educating their local communities and also by making themselves available to the medical profession for the emergency identification of specimens.

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