MUSHROOM POISONING CAUSED BY *AMANITA PANTHERINA*

REPORT OF 4 CASES

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Although mushroom poisoning is well recognized in South Africa, it is not generally appreciated that the toxic effects are extremely variable. This is particularly so with *Amanita pantherina*, which is one of the most common poisonous mushrooms in the Transvaal.[1] There appear to be several reasons for this variable toxicity. Thus, while it is apparent that some individuals tolerate the effect of the toxins better than others, there is good evidence that this is not the only factor and that both the soil in which the particular mushroom grows,[2] and the season during which it is picked, influence the toxicity.[2,3]

The present paper records our recent experience in handling 4 patients who had eaten *Amanita pantherina*. Our findings underline the individual variations in clinical presentation and also highlight some of the therapeutic problems posed by such patients.

CASE HISTORIES

A family of 4 German immigrants, who had been resident in this country for 10 years, picked wild mushrooms found growing in a plantation on the West Rand and ate approximately 2 tablespoonfuls each. About 1½ hours later they arrived at the casualty department of this hospital.

Case 1

J.L., male, aged 62 years. Half-an-hour after the meal he noticed a light-headed 'dizzy' sensation, which he likened to that experienced by a man who had drunk a case of beer. This was followed by tiredness and clouding of vision. However, he retained sufficient presence of mind to collect his family and drive his car to the hospital. On arrival, he became at first excitable and then stuporose. Vomiting started while he was undergoing lavage, and on admission to the ward he was in a semi-comatose state.

Examination. He was a small male, of good physical condition, with no evidence of underlying disease. There was no circulatory collapse or respiratory distress. The blood pressure was 130/90 mm.Hg, and the pulse rate was 120/min. and otherwise unremarkable. Miosis and a moderate degree of salivation was present. The rest of the initial examination was negative. However, within the first hour of admission a noteworthy feature developed. This was a generalized twitching of muscle groups, often spontaneous, but also stimulated by insertion of a needle into a vein, by light touch, or by movement of the bed-clothes. His level of consciousness continued to deteriorate and periods of apnoea were observed. Apart from supportive measures, he was given 1/50 gr. of atropine and a diuresis was induced with 400 ml. of 40% dextrose water. He gradually improved and after 48 hours, during which he was agitated and experiencing visual hallucinations, he became lucid.

Case 2

G.L., female, aged 51 years, the wife of patient 1. She noticed a sensation of 'dizziness' and tiredness immediately after eating the mushrooms, but as the rest of the family seemed unaffected at that time, she attributed her symptoms to the warmth of the evening. However, they gradually became more severe and by the time she reached hospital she was also suffering from nausea. She then induced emesis by pharyngeal irritation. No other noteworthy symptoms were present at this time.

Examination. A middle-aged woman with only very mild distress; there was no evidence of underlying disease. The blood pressure was 100/80 mm.Hg, and the pulse rate was 84/min. and regular. Moderate miosis was present, but no significant salivation, confusion, or twitching was noted. In the ensuing days she complained of non-specific abdominal pain, but there was no diarrhoea or further gastro-intestinal disturbance. Recovery was virtually complete 18 hours after ingesting the toxins.

Case 3

R.L., the 16-year-old son of patients 1 and 2, complained of 'light-headedness' ½ hour after eating the mushrooms. Thereafter he became 'dizzy', felt tired and his vision became clouded. Nausea and vomiting were induced during gastric lavage and on admission to the ward he was in a state of stupor.

Examination. A well-built youth with no respiratory distress or circulatory collapse. The blood pressure was 110/70 mm.Hg, and the pulse rate was 84/min. and regular. Pupil size was not remarkable, but moderate salivation was present. The reflexes were brisk and characteristic twitching similar to that seen in patient 1 was present. Approximately 10 hours after ingestion of the toxins he was sufficiently lucid mentally to answer questions at a ward staff meeting.

Case 4

W.T., a 23-year-old male, a close friend of the family, was sitting in a cinema approximately 1 hour after the meal when he noticed a sensation of 'light-headedness' and found he could not keep his hands still owing to restlessness and tremor. This was so severe that he was unable to light a cigarette. Double vision developed and he left the cinema shortly afterwards. He had to be restrained by his companions as he

staggered and ran down the street, grasping lamp-posts in an inebriated fashion. On the way to hospital he noticed twitching of his limbs and extreme tiredness, and by the time he arrived in the casualty department he was semi-comatose. Gastric lavage induced nausea and vomiting.

Examination. A well-built young male in a semi-comatose state. He was not shocked, but some respiratory distress was present. This distress was thought to be due to excessive salivation, to intermittent apnoea and to the aspiration of some stomach contents during vomiting. The blood pressure was 140/80 mm.Hg, and the pulse rate was 70/min. Hyperreflexia and the same twitching of muscle groups as in patients 1 and 3 was present. In this patient, however, minimal stimulation resulted in a major convulsion. The latter feature was progressive. Pupil size was variable in a manner reminiscent of hippus. Large doses of analeptics were required for control of the progressively more severe convulsions. In addition, severe respiratory embarrassment was present: a tracheostomy was therefore performed and the patient breathed artificially for the next 24 hours. The semi-comatose state persisted for approximately 18 hours after ingesting the toxins.

Subsequent Progress of the 4 Patients

Two weeks after the poisoning, all the victims were again ambulatory. A small pneumothorax which had occurred in patient 4 at the time of tracheostomy had resolved, and his neck wound was virtually healed. The only late symptom was an inability to grasp and remember minor details of everyday life. This mild mental deficit was present in all the patients for 6 weeks after the poisoning, but at the time of writing all are fully recovered.

Blood counts, electrolyte estimations, liver-function tests, electrocardiograms and chest X-rays showed no significant abnormality (other than the pneumothorax in patient 4) at any time.

Identification of the Mushrooms

Relatives retrieved the remaining mushrooms which had been picked and Dr. H. J. Swart, of the Department of Botany of the University of the Witwatersrand, identified these as *Amanita pantherina*. He also lent us photographs (Figs. 1 and 2) of some of the more important Amanita species found in

Fig. 1. Amanita pantherina—brown cap and white stem, gills and flesh. (By courtesy of Dr. H. J. Swart, Department of Botany, University of the Witwatersrand.)

Southern Africa. By comparing these with the illustrations in the cookery book used by the patients, we were able to ascertain that they had confused the inedible *Amanita pantherina* with the edible *A. rubescens*. Mistaken identification of these two mushrooms is well documented.[4-6]

After discharge from hospital the patients revisited the site of their mushroom gathering. Since by this time the season had ended they found only some dried specimens of *Amanita pantherina* (Fig. 3). Mr. H. Kundig, of the Department of Pharmacology, extracted any remaining active toxic principles with alcohol, and injected the extract into test rabbits. The only effect noted, however, was a slow contraction and dilatation of the pupils in a similar manner to that described in patient 4.

Fig. 2. Amanita rubescens—lighter coloured cap by comparison with *Amanita pantherina*, with pink stem, gills and flesh. (By courtesy of Dr. H. J. Swart, Department of Botany, University of the Witwatersrand.)

Fig 3. Dried specimens of *Amanita pantherina* obtained from the original site.

DISCUSSION

The arrival of many European immigrants in South Africa in recent years may well increase the incidence of mushroom poisoning, since it is common practice to pick wild mushrooms on the Continent. On general grounds of public health and preventive medicine, it is therefore desirable that the public be made aware of the dangers of mushroom poisoning and of the difficulties in identifying different wild species. In this context it is important to stress that none of the time-honoured methods for identifying non-edible varieties such as 'peeling of the cap', 'blackening of a silver spoon', 'growing near rusty metal objects', and other such 'tests', are reliable.[1,4,5,7]

The features of the clinical presentation of patients suffering from *Amanita pantherina* poisoning were well illustrated by the 4 cases reported in this paper. In parti-

cular, the early onset of symptoms, the predominance of initial neurological manifestations, later occurrence of gastro-intestinal disturbance, and early recovery, serve to distinguish this poisoning from other types.[2,5,8] Thus, although the time of onset of *A. muscaria* poisoning is similar, gastro-intestinal disturbance tends to occur earlier in this form of mycetismus. Similarly, pure muscarine poisoning (e.g. *Inocybe patoullardii*)[6,9,10] has a somewhat later onset. Poisoning due to *A. phalloides* (and other phalloidin-containing mushrooms, e.g. *A. capensis* and *A. verna*) is associated with a late onset of symptoms (6 - 16 hours) and often culminates in anuria, renal and cardiac failure and death after approximately 72 hours in 50% of cases.[2,3,5,9,11-13]

Table I was compiled in an attempt to correlate the presentation of *Amanita pantherina* poisoning with the toxin responsible for each feature. At least two groups of signs and symptoms occur, and possibly a third group, although the latter may be due to a combination of groups 1 and 2. Group 1 consists of salivation, bradycardia, miosis, sweating, dyspnoea and gastro-intestinal stimulation, and is thought to be due to muscarine and choline.[2,5,6,9,10,14] Group 2 is attributed to the action of 'pilzatropine', mycoatropine, or L-hyoscyamine,[2,5,6,9,12] the features being mental irritation, confusion, delirium, convulsions, dry mouth and skin, and mydriasis. The third group, which is thought to be due to bufotenine, manifests as light-headedness, purple flushing, air hunger, visual hallucinations, mydriasis and nausea.[2,6,9,15]

TABLE I. TENTATIVE CLASSIFICATION OF THE *A. pantherina* TOXINS

Toxin	*Clinical features*
1. Muscarine and choline	Salivation, sweating, miosis, bradycardia, dyspnoea, gastro-intestinal disturbance
2. Pilzatropine, mycoatropine, or L-hyoscyamine	Mental irritation, confusion, delirium, convulsions, mydriasis, dry mouth and skin
3. Bufotenine or pilztoxin	Light-headedness, nausea, purple flushing, air hunger, mydriasis, visual hallucinations

The conflicting effects of the two major toxins of *Amanita pantherina* pose difficulty in therapy, particularly in regard to the administration of atropine ;[4,6,14,16,17] it would appear that its use is only indicated where the clinical presentation indicates that the 'pilzatropine' effect has failed to cancel the muscarinic effect.

The variability of response to the toxic effects of *Amanita pantherina* in individual patients has evoked much discussion in the literature.[2,6,9,14,16-19] It is usually claimed that different batches of mushrooms contain differing amounts of the various toxins. Thus specimens collected by Lewis[17] at the Cape had a high content of L-hyoscyamine, whereas those harvested in the winter and spring in the USA by Brady and Tyler[20] contained no such alkaloid. These authors also mention the work of Wieland and co-workers,[21] who claimed to have isolated bufotenine from their specimens of *Amanita pantherina*. The former authors were, however, sceptical as to its significance in mycetismus. It is therefore apparent that there is either tremendous variation in the proportions of the toxins in each batch of *A. pantherina*, or that the tests used for analysis are not uniform.

No estimates of the incidence of *A. pantherina* poisoning in the Republic have been published. However, 1,000 of 5,000 cases of mycetismus reported in Germany[5] in 1946 were due to this species. The mortality rate in this country is also not certain, although Silberbauer and Mirvish[22] reported 7 deaths in 7 cases in 1927. Krause[23] also reported a high mortality rate (7 out of 8 cases), but the generally accepted figure is 10 - 20%. The syndrome described by Silberbauer and Mirvish was somewhat different from that generally described, since jaundice, renal failure and death occurred at approximately 72 hours. These features are reminiscent of phalloidin poisoning, and it seems probable that, although *A. pantherina* was positively identified by these authors, an admixture of *A. phalloides* or *A. capensis* was also present. The pathology described by these authors is thus also suspect, since many of their findings were indistinguishable from those due to phalloidin poisoning.[3,5,11-13,24]

The treatment of the present cases of *A. pantherina* poisoning is set out in Table II. Additional measures which have been advocated include the use of charcoal for adsorption of the toxin, high colonic lavage and purgation, exchange blood transfusion, haemodialysis, the oral administration of ground-up rabbit stomach and brain, the administration of antiphalloidin serum (Pasteur Institute, Paris) and therapy for renal, hepatic and cardiac failure. The last form of treatment is usually only necessary in cases caused by the much more lethal *A. phalloides, A. verna* and *A. capensis*, and was fully reviewed by Elliott and co-workers[25] in 1961, and previously by others.[3,5,26]

TABLE II. TREATMENT OF 4 CASES OF *A. pantherina* POISONING

Treatment	*Case 1 (62 yrs)*	*Case 2 (51 yrs)*	*Case 3 (16 yrs)*	*Case 4 (23 yrs)*
Gastric lavage or emesis	+	+	+	+
Atropine	+	+	+	+
400 ml. 40% dextrose water	+	+	+	+
Sedation and analgesia	+	+	+	+
Supportive measures	+	—	—	+
Tracheostomy and respirator	—	—	—	+
Antibiotic	+	+	—	+

SUMMARY AND CONCLUSIONS

1. Four cases of mushroom poisoning due to *A. pantherina* are described.

2. The fallacy of the well-known lay 'tests' for edibility of wild mushrooms is stressed and a plea is made for the re-education of the mushroom-eating public in this regard.

3. The toxicological basis of the 'Pantherina syndrome' is presented.

4. The therapy of the condition is briefly discussed and the dangers of the use of atropine are stressed.

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