

Suspected poisoning of puppies by the mushroom *Amanita pantherina*

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ABSTRACT

Amanita pantherina poisoning is suspected as the cause of a severe, transient neurological disorder in three 5-week-old German shepherd puppies. There was very strong circumstantial evidence that this mushroom had been eaten, and the signs encountered were similar to those described in confirmed field cases of intoxication in dogs. It was also in many respects consistent with the syndrome seen in humans. A veterinary perspective on the hallucinogenic *Amanita* spp. is given and the veterinary literature on mushroom intoxication is briefly reviewed as, in contrast to humans, it is not often reported in animals. This is the 1st report of suspected mushroom intoxication of animals in South Africa.

Key words: *Amanita pantherina*, canine, coma, hallucinogenic mushroom, ibotenic acid, intoxication, muscimol.

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INTRODUCTION

Mushrooms are divided into those that are edible, those that are not edible and those that are definitely toxic. The last group are popularly referred to as toadstools, a name anglicised from the German 'todesstuhl' (death chair), apparently derived from the belief that toads sitting on these fungi render them toxic (McKenney and Stutz cited by Ridgway²⁷). From a clinical and diagnostic perspective in humans, toxic mushrooms are grouped by Lampe¹⁸ according to the 6 main syndromes encountered:

1. A diverse group responsible for usually transient gastrointestinal irritation ascribed to a variety of toxins (probably the most commonly seen syndrome).
2. The *Amanita phalloides*-group, containing cyclic polypeptides responsible for the most serious and often fatal intoxication: delayed, irreversible cytotoxicity affecting predominantly the liver and kidney.
3. The *Gyrometra*-group, containing

methylhydrazine and causing similar but less severe cytotoxicity.

4. A group causing muscarine-related symptoms such as salivation and increased gastrointestinal motility.
5. An interesting group that causes sensitivity to alcohol by inhibition of acetaldehyde dehydrogenase (thus not of veterinary importance).
6. The hallucinogenic mushrooms. This category can be subdivided into the *Psilocybe*-group, causing an hallucinogenic syndrome not associated with sleep or coma, and a group causing delirium associated with sleep or coma. Both *Amanita pantherina* and *A. muscaria* fall into the latter group.

Ramaria flavo-brunneescens poisoning of ruminants constitutes an exceptional additional clinical veterinary category, in which the toxin interferes with keratinisation¹⁷.

Mushroom poisoning in humans is relatively common and is well recorded. The most important factors contributing to intoxication are, firstly, confusion of toxic mushrooms with edible species, as considerable experience is required to differentiate between them, and secondly the recently emerging but widespread practice of deliberate use of hallucinogenic mushrooms for 'recreational purposes'²³.

Except for *Ramaria*¹⁷, animals apparently do not eat mushrooms by choice to any extent. Consequently, intoxication is incidental, apparently not frequently

encountered, and therefore seldom recorded. A serious complicating factor in the diagnosis of mushroom intoxication in animals is their inability to communicate that mushrooms have been ingested. Furthermore, the soft texture of the fungi results in rapid fragmentation, so that they may not be readily recognised in vomitus, lavage fluid or at necropsy. In addition, there are the problems of syndrome divergence (*vide supra*) and the lack of knowledge required to identify the causative mushroom and connect it with the signs of intoxication observed. Nevertheless, a number of mushroom intoxications have been reported in the veterinary literature that correspond more or less with the above syndrome classification for humans¹⁸.

This report describes the 1st recorded case of suspected *Amanita pantherina* poisoning in dogs in South Africa, with a brief review of the veterinary literature on mushroom poisoning.

MUSHROOM POISONING IN ANIMALS

A review of the veterinary literature according to species revealed the following:

Mushroom intoxication is rare in ruminants. *Ramaria flavo-brunneescens* is the only mushroom known to regularly cause intoxication in cattle and sheep in Brazil and Paraguay. The syndrome was recently reproduced again in cattle and the pathology described in detail. It appears that the toxin results in no or incomplete and irregular keratinisation of keratinocytes, leading to loss of hooves, horns, tail hair and smoothing of the dorsum of the tongue¹⁷. This unique intoxication syndrome is, to our knowledge, the only mushroom poisoning that has not been recorded in humans. This mushroom is associated with *Eucalyptus* trees and the poisoning is, therefore, commonly known as *Eucalyptus* disease. Neither the syndrome nor the species has been recorded in this region, although a related species, *R. formosa*, occurs in the Western Cape Province in association with bluegum (*Eucalyptus*) trees and ingestion is reported to result in severe diarrhoea in humans³⁰. In

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sheep in northern Europe, *Cortinarius speciosissimus* proved to be the cause of fatal kidney damage²⁴. The genus occurs locally in South Africa¹⁹ but is apparently not common. Only 2 further speculative case reports could be traced, describing gastroenteritis in cattle that might have resulted from ingestion of mushrooms^{7,25}.

Probably because horses are fastidious grazers, only 1 report, describing suspected hallucinogen-containing mushroom intoxication¹³, could be found. Similarly, despite the fact that swine are omnivorous, only a single case of intoxication in a Chinese pot-bellied miniature pig, ascribed to ingestion of *Scleroderma citrinum*, has been recorded¹¹. This common earth ball (or puff ball) is widely distributed in the more humid parts of southern Africa, where it is associated with pine trees³⁰.

Cats also apparently do not eat mushrooms to any extent, as only 1 article describing 2 incidents of suspected mushroom intoxication in this species could be traced²². In 1 case, transient gastrointestinal irritation and near coma occurred in a cat that had been eating an unidentified mushroom. In the other case, neurological signs and neuropathology in addition to liver necrosis of undetermined origin were attributed to possible mushroom intoxication.

The dog is the only species that appears to be affected relatively frequently by toxic mushrooms, with 9 traceable reports of intoxication involving 20 4½–12-week-old pups and 9 adult individuals.

A case of suspected Category 1 mushroom intoxication (gastrointestinal irritation) involved a litter of five 7-week-old boxers that developed black tarry diarrhoea. The faeces contained no blood, and flotation and direct smears of the faeces revealed numerous mushroom spores that unfortunately could not be further identified. Symptomatic treatment resulted in uneventful recovery³¹.

Despite many cases of fatal hepato- and nephrotoxicity (Category 2 poisoning) in humans (also in South Africa) by the highly toxic cyclopeptide-containing *Amanita* spp., most of the literature deals with experimental intoxication in dogs¹⁰. Only 3 reports of field intoxication in dogs were traced: 6 fatal cases occurred in the United States of America, in which the typical histopathological lesions supported the diagnosis. In 3 of these cases, chewed *A. phalloides* was collected on the property and botanically identified, and in the other 3 cases ingestion was suspected¹⁵. In Australia, a 9-week-old spaniel pup that had consumed a small portion of a cap of a botanically identified

specimen of the same species died. No pathological data were, however, recorded⁹. Only 1 case where a 7-week-old pup was seen eating botanically identified *A. verna* a day before death is recorded. The pathology and histopathology in this case, as well as in a 12-week-old pup where the mushroom that was eaten was not available but the owner described something resembling *A. phalloides*, were consistent with this category of intoxication²⁰. In a confirmed case of Category 3 intoxication (less severe hepatotoxicity), a 10-week-old cocker spaniel was observed eating a mushroom, vomited 2–3 h later, became lethargic after 6 h and was presented comatose to the veterinarian. It died 30 min later. Pathological investigation revealed indications of a haemolytic crisis with renal tubular necrosis and periacinar hepatic degeneration, leucocytosis and erythrophagocytosis. Samples collected in the pasture where the dog was found chewing the mushroom proved to be *Gyrometra esculenta* which contains monomethylhydrazine¹. This genus has not been recorded in South Africa (A Eicker, Department of Botany, University of Pretoria, pers. comm., 1997).

Transient vomiting and diarrhoea occurred in a 14-year-old springer spaniel after it had eaten 4 mushroom species, of which the dominant one identified in the vomitus was the known toxic *Inocybe phaeocromis*³². This mushroom belongs to the muscarine-containing Category 4 (*vide supra*) and is represented locally by *I. eutheles*, which occurs in the southern Western Cape Province³⁰.

Concerning hallucinogenic mushroom intoxication in the dog, confirmed cases in both the above Category 6 intoxication syndromes (with and without coma) are recorded:

An adult Labrador showing ataxia, nystagmus, vocalisation and overt aggression without subsequent coma had to be given barbiturate sedation. Subsequently, psilocybin was chemically demonstrated in its blood¹⁶.

In the category of hallucination with concomitant coma, 1 confirmed case in Canada¹² and 1 case of suspected poisoning in the United States of America²¹ are reported. In the former case, *Amanita pantherina* ingestion by a litter of 9 4½-week-old Labradors resulted in severe paresis and opisthotonus in 5 pups, 3 of which died within 3 h of ingestion. Convulsions were encountered for 12 h in the other 2, but they had recovered completely 17 h later¹². Suspected *Amanita muscaria* poisoning in a 4-year-old male collie resulted in apparent confusion,

vestibular signs and convulsions followed by coma. When respiratory arrest occurred 9 h later, artificial respiration was applied for 45 min., but normal breathing only resumed after intravenous corticosteroid therapy. The coma, however, lasted for a further 5 h and was followed by uneventful recovery²¹.

CASE REPORT

History

The incident occurred in January 1991 on a small agricultural holding just east of Pretoria where the owner had been living for 18 years. A number of well-established trees occur on the property, including a number of *Cedrus deodora* (deodar cedars). A German shepherd bitch with six 5½-week-old pups was kept in a well-constructed, lock-up kennel with a cement floor. During the day the bitch roamed freely and the pups had access to a kikuyu lawn extension in front of the kennel that was temporarily enclosed with loose partitions of coarse mesh wire fencing. This fence had contained the pups successfully up to that stage, but as they became older and stronger they started to crawl through the loose fencing and roam nearby.

At 06:00 on the morning of the incident the owner found a male pup apparently stuck in the fence. When it was released she observed that its 'neck flopped back'. It appeared weak, disorientated and unable to walk. Neck injury was suspected and the pup was admitted to a private practice. By 09:00 a 2nd pup (also male) was found paralysed. Its head was also thrown back, the jaws were clamped, the eyes rolled and it could not rise. This pup also was taken to the veterinarian, who referred both cases to the Veterinary Academic Hospital of the Faculty of Veterinary Science at Onderstepoort (OVAH). At 20:00 on the same day, the owner reported that a 3rd pup developed similar (but less pronounced) signs. According to the owner, it was very lethargic and dull, and although it slept in an apparent coma, it was 'jerky' to tactile and auditory stimuli. This pup appeared normal the following morning, and was not examined by a veterinarian. It passed soft slimy faeces during the night that were collected for laboratory examination.

Clinical and laboratory evaluation and treatment

One male pup was presented in lateral recumbency and semi-comatose, with opisthotonus, right lateral strabismus, paddling and exhibited chewing movements. The other dog was quadriparetic

and depressed, with left lateral strabismus. Both dogs had severely miotic pupils, congested mucous membranes, raised rectal temperatures (39.9 and 39.5 °C), pulse rates >150/min and were panting. A preliminary diagnosis of possible intoxication was made, and the dogs were sedated with diazepam (Valium, Roche) at 0.5 mg/kg intravenously. One of the pups subsequently developed cardio-respiratory arrest. Intubation, positive pressure ventilation, and cardiac massage were successful in resuscitating the pup. Gastric lavage was performed on this pup during the immediate post-resuscitation period while the endotracheal tube was still in place. Consciousness returned after 5 min. Blood samples were collected in EDTA, heparin, sodium fluoride and plain test tubes for haematology, serum chemistry and toxicological investigation. The gastric content was submitted for strychnine determination.

Polyionic fluid (Plasmalyte B, Sabax) was administered to both puppies via jugular catheters, and atropine (1 mg/kg) administered, half intravenously and half subcutaneously. Within 4 h the 2 dogs were ambulatory and appeared clinically normal.

An inflammatory leukogram was present in 1 dog and both were mildly hyperglycaemic (12.8 and 6.9 mmol glucose/l). Normal blood acetylcholinesterase activity ruled out organophosphor toxicity, and the stomach content was negative for strychnine. Faecal smears of the 2 pups revealed small, darkly stained objects suggestive of fungal spores. However, this could not be confirmed.

By the time it was realised that this was most probably a case of mushroom intoxication, the chemical toxicological test on the gastric contents of the sick pup had unfortunately already been completed and both the specimen and container discarded. Consequently, no specimen was available for spore examination. Spores of *A. pantherina* were not found by flotation in the small amount of faeces of the 3rd pup.

Identification of the probable cause of intoxication

The owner was questioned about possible exposure to any known poisonous substances or medicines but was adamant that this could not have occurred. She was, however, requested to reinvestigate this possibility at home. The day following the intoxication she submitted a partially chewed mushroom that had been found next to the pups' run, as well as whole mushrooms collected from under a nearby cedar tree on the property.



Fig. 1: *Amanita pantherina* (photograph: A Eicker).

The mushrooms were recognised immediately as *A. pantherina*, and the identity subsequently confirmed by the Botany Department of the University of Pretoria. An *in loco* inspection the same evening revealed that a young deodar cedar, with the lowest branches almost touching the ground, was growing adjacent to the temporary fence around the pups' kikuyu-lawn run. The low branches and mass of fine needles under the tree created an undisturbed area where several of these mushrooms' fruiting bodies were found. One particular specimen with the cap missing was noticed. As a test of their reaction, the pups were allowed to roam at will and an inquisitive individual crawled in under the branches right up to the stem of the tree.

The owner informed us that, depending on rain, this particular mushroom had been encountered periodically for years on the property but that it had never presented any problem.

Description of *Amanita pantherina* (DC.: Fr.) Gonnerm. & Rabenh., Fig. 1

This species (commonly known as the panther, panther mushroom or wanted agaric) has all the characteristics of an ordinary mushroom: at the button-stage the cap (or pileus) is dome-shaped, then becomes convex and finally flattened with a slightly upturned, distinctly striated margin. The colour varies from rather pale creamy-brown or grey-brown to ochre-brown, sometimes with an olive tint. The pileus is covered with persistent, irregularly dispersed, white volval warts.

It has a diameter of 6–10 cm and the gills (lamellae) are white. The white cylindrical to clavate stem (or stipe) of up to 10 cm high and 1.0–1.5 cm wide often thickens to a bulbous base of 2.5–3.0 cm and is surmounted by a free-standing cup, rim or collar of volval tissue just below soil surface. A clearly-defined persistent ring (the annulus) with a double margin occurs around the stem just below the cap^{19,26}. Like most *Amanita* spp., the mycelium of this mushroom interacts symbiotically with the hair roots of a particular host tree in a mycorrhizal association. It is, therefore, chiefly found in pine plantations and in association with exotic trees such as conifers, oaks and *Eucalyptus* spp. This introduced mushroom has a cosmopolitan distribution and in South Africa is prevalent in the Eastern and Western Cape Provinces, KwaZulu-Natal, Gauteng and Mpumalanga²⁶.

The closely related and morphologically similar *A. muscaria* (fly agaric), which causes the same intoxication, has a bright red cap with white warts. It is commonly encountered in the Western and Eastern Cape Provinces, Free State, Gauteng and Mpumalanga during the rainy season^{19,26}.

DISCUSSION

Although the diagnosis could not be confirmed irrefutably in this particular incident, all indications are that it had indeed been a case of *A. pantherina* intoxication. A chewed mushroom was found in the proximity of the puppies' pen and it was evident that the litter had access to the mushrooms growing under the

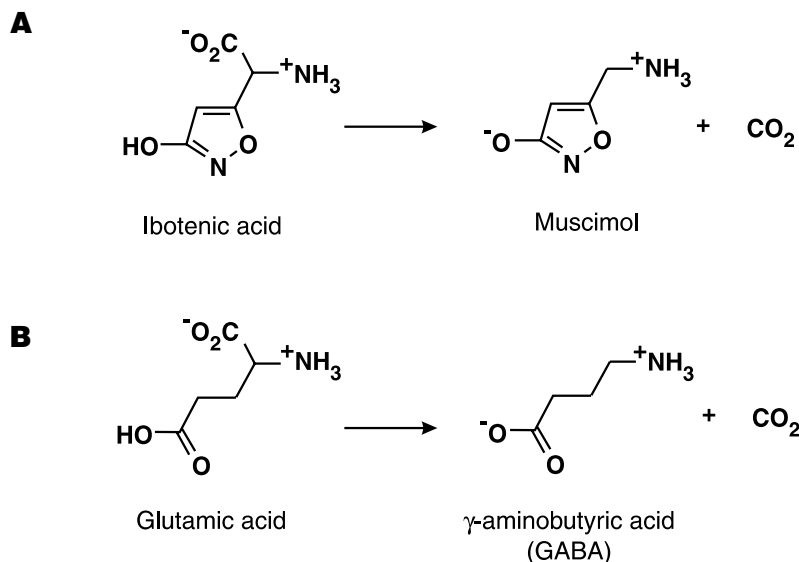


Fig. 2: Structural relationship between false neurotransmitters (A) from *Amanita pantherina* and naturally occurring neurotransmitters (B) (adapted from Lampe¹⁸).

deodar cedar next to their run. It is well-known that pups will chew (and sometimes even swallow) peculiar material, *inter alia* poisonous metallic substances like lead⁶ and certain toxic plants such as *Dieffenbachia* and even cycads⁴.

Failure to find mushroom spores in a small quantity of faeces from the last of the poisoned pups might be ascribed to digestion. Examination of gastric contents in this regard would probably have confirmed the intoxication and should be a standard procedure in all cases of suspected mushroom intoxication. In our case, however, all the gastric contents collected from the 1 pup had been used in the test for strychnine and by the time the diagnosis was made, even the container had been discarded.

The hallucinogenic principles in both *A. pantherina* and *A. muscaria* that result in subsequent coma are ibotenic acid and its decarboxylation product, muscimol (Fig. 2). Both constituents have the same pharmacological effects, although muscimol is 5–10 times more potent than ibotenic acid²⁹. Contrary to earlier belief, these mushrooms contain insignificant amounts of muscarine. They possibly contain as yet unidentified components that may contribute to the intoxication syndrome. However, ibotenic acid and muscimol, together with their metabolites, appear to be responsible for the signs of this poisoning: nausea, hallucinations, delirium, muscular spasms and sleep⁸. Ibotenic acid is a conformationally restricted derivative of glutamic acid, as muscimol is of gamma-aminobutyric acid (GABA)⁸. Ibotenic acid, like glutamic acid, is an excitant of isolated interneurons and

Renshaw cells, whereas muscimol, like GABA, is a powerful inhibitor of firing of central neurons¹⁴. Unlike glutamic acid and GABA, ibotenic acid and muscimol cross the blood/brain barrier, apparently by active transport, and these false neurotransmitters appear to be the main cause of the syndrome (Chilton⁸ citing Balkar and Krogsgaard-Larsen).

The toxin content in this mushroom appears to vary considerably. In a recorded incident of poisoning in humans in South Africa, it was estimated that the adult individuals involved each had only consumed approximately a tablespoonful of cooked mushroom. This resulted in severe intoxication³. In contrast, in the United States, where this species is widely used for the deliberate induction of a hallucinogenic state, Ott²³ states that 'half a cup of sauteed mushrooms is usually enough' to produce the desired effect. Apparently the inconsistent transient nausea prior to hallucination is no deterrent to the habitual user. However, in accidental intoxication, this nausea, followed by severe, unexpected delirium, is clearly a most frightening experience.

It is interesting also that the insecticidal properties of these mushrooms (whence the popular names 'fly agaric' or 'Fliegenpilze' for *A. muscaria*) are also ascribed to the ibotenic acid and muscimol⁵.

Except for the shorter period of convulsions, the signs encountered in the current incident were very similar to those seen in the only other confirmed field incident of this intoxication in the dog¹², as well as to those encountered experimentally with ibotenic acid and muscimol in canines²⁹. The syndrome in dogs also can be equated to that seen in

humans³. It is surmised that, in the dog, the equivalent of the extended stage of delirium seen in humans would manifest as a transient disorientation. Fortunately no mortality occurred in this particular case, as opposed to 3 out of 5 pup deaths in the intoxication recorded by Hunt¹². The quantity consumed in this instance might have been less.

In humans, mortality due to *A. pantherina* is rare¹⁸, in spite of the fact that it is the most common mushroom intoxication encountered in Europe³ and the Pacific North-West²³ and that, in all probability, it is the mushroom most commonly used to obtain a psychotropic effect in the United States of America²³. In contrast, in confirmed intoxications in dogs relatively high mortality has been reported^{12,29}. This could perhaps be ascribed to greater sensitivity of young animals (4½ to 5½ weeks old). Glutamic acid and related excitatory amino-acids like ibotenic acid produce convulsions in immature rats in which the blood/brain barrier is not completely developed (Johnston as cited by Chilton⁸). However, mature animals are protected only from the convulsive properties of glutamic acid and GABA and not from those of muscimol and ibotenic acid, which cross the blood/brain barrier (Balkar and Johnston and Krogsgaard-Larsen and Johnston as cited by Chilton⁸).

In the present case, successful general symptomatic treatment was instituted, as a specific diagnosis had not been made. It consisted of controlling the nervous signs and stabilising the electrolyte balance. Where a case of known poisoning by the *A. pantherina*/*A. muscaria*-group of mushrooms is presented, the serious nervous signs should first receive attention, and then further absorption of toxins should be prevented. Great care must be exercised in the use of sedatives, as the administration of small doses of diazepam or phenobarbitone in muscimol-treated experimental animals induces a flaccid paralysis and an EEG-pattern similar to deep anaesthesia²⁸. It is, therefore, possible that the cardio-respiratory arrest in one of the pups in this incident was induced by the intravenous administration of diazepam. It is suggested that this could have occurred either as a result of potentiation of the muscimol-induced GABA effect (*i.e.* neuro-inhibition), or due to the inherent hypotensive effect of diazepam². Further absorption should be limited by the judicious use of apomorphine (or if this is contra-indicated by the state of consciousness of the patient, gastric lavage), followed by activated charcoal at 2 g/kg.

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