More about
Acute poisoning

MUSHROOM POISONING

Only 100 of the thousands of mushroom species are poisonous. Among the potentially poisonous mushrooms, a small number may cause serious intoxication and even fatalities. Although South African mushrooms are well described, information with regard to the poisonous ones and their toxins is limited.

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From a medical point of view, it is more practical to divide poisonous mushrooms into those that cause symptoms and signs of poisoning early (within 6 hours) and late (6 - 48 hours after ingestion). Although poisonings presenting early may be quite serious, they are usually self-limiting, clear up within hours, and have better prognoses than those presenting late. Symptoms and signs of mushroom poisoning vary according to the mushroom ingested, amount eaten, method of preparation and whether alcohol was consumed. Many species of poisonous mushrooms cause gastrointestinal symptoms only, and spontaneous recovery usually occurs within a few hours. However, cyclopeptide (*Amanita phalloides*) poisoning can be fatal in 50% of cases and accounts for 90% of all lethal mushroom poisonings. *A. phalloides* (death cap, duiwelsbrood, slangkos) is found throughout southern Africa. After summer and autumn rains the fruit bodies develop under oaks, pines and poplars. The species is readily recognisable by the olivaceous yellow-green colours of the cap surface, the white lamellae (gills) and stem, the membranous ring, and the white, sac-like volva which envelopes the basal bulb below ground level.

Fig. 1. Amanita muscaria *(top right),* Amanita pantherina *(middle right) and* Amanita phalloides *(middle bottom), all from the Tokai forest, Cape Town. White gills are often associated with poisonous mushrooms.*

Fig. 1. shows some South African mushroom species.

Poisonous mushrooms and clinical syndromes are described in Table I.

General management

One should attempt to ascertain how many types of mushrooms have been eaten, time lapse since ingestion and how many people were involved. Identification of the mushrooms should be attempted and all pieces of mushrooms saved may be helpful in this regard. Where possible, a specimen of a similar mushroom should be obtained. As an initial procedure, activated charcoal is recommended if it can be administered within 4 hours of ingestion. The time interval between ingestion and the onset of symptoms is important for differential diagnostic purposes (described above and in Table I). If the ingested mushroom is likely to be one of the *Amanita* species, the patient should be admitted to hospital, and hepatic and renal function monitored. For the management of mushroom poisoning, it is recommended that you contact your local poison information centre for guidelines.

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Specific management in symptomatic patients

- Monitor liver and kidney functions.
- Manage dehydration and electrolyte/acid-base disturbances where indicated.
- For patients with a disulfiram effect, propranolol may be of benefit. Dopamine may be indicated in the management of hypotension.
- In muscarine poisoning, atropine may be considered, but it may aggravate other types of mushroom poisoning.

Table I. Mushroom poisoning syndromes

Clinical syndromes	Mushrooms	Symptoms and signs
• Clinical syndromes presenting early (within 6 hours after ingestion)		
Gastrointestinal group Many toxins involved, mostly unidentified	*Agaricus placomyces, Agaricus semotus, Agaricus xanthodermus* (yellow stainer). *Chlorophyllum molybdites* (false parasol or green-spored parasol). *Hebeloma* species. *Ramaria formosa*. *Scleroderma citrinum* (common earth ball)	Nausea, vomiting, abdominal cramps, diarrhoea, headaches, sweating. Self-limiting. Some may produce CNS, musculoskeletal toxicity. Some people may be affected, others not. Onset within 15 min - 2 h. Subsides within 3 - 4 h. Recovery complete within 1 - 2 days
Muscimol/ibotenic group Ibotenic acid is converted to muscimol which structurally resembles GABA. Other unidentified toxins probably also involved.	*Amanita muscaria, Amanita pantherina*	Patient appears intoxicated. Confusion, ataxia, euphoria, disturbed vision, hyperkinetic activity, spasms and delirium. Onset within 30 - 90 min, peaking at 2 - 3 h. Lasts for 4 - 8 h
Muscarine/histamine group	*Clitocybe toxica, Clitocybe olearia*, and other *Clitocybe* species. *Inocybe eutheles, I. hirtella, I. obscura*	Cholinergic syndrome: profuse salivation, lacrimation, perspiration. Miosis, blurred vision, bradycardia, hypotension, bronchoconstriction. Abdominal cramps and diarrhoea may occur. Onset within 15 min - 2 h. Short-lived, subsiding within 6 - 24 h
Hallucinogenic indole group: Psilocybin, psilocin. Related to LSD	*Psilocybe coprophila*, and other *Psilocybe* species. *Panaeolus papillionaceus. Conocybe* species. *Stropharia aurantiaca, S. semiglobata*	Hallucinations prominent, dilated pupils, confusion, vertigo, ataxia, weakness, drowsiness. Onset usually within 10 - 30 min. Average duration 4 - 5 h
Coprine group Coprine has a disulfiram effect (Antabuse)	*Coprinus atramentarius* (common ink cap)	Flushing, paraesthesias, palpitations, throbbing headache, nausea, vomiting, sweating. Less commonly: chest pain, hypotension, vertigo, blurred vision, confusion, respiratory problems. Poisoning may occur if alcohol is consumed before or within 72 h of ingestion. The disulfiram effect occurs within 30 min - 2 h after ingestion. *Coprinus* mushrooms do not cause toxicity in the absence of alcohol
• Clinical syndromes presenting late (> 6 hours after ingestion)		
Cyclopeptide group ζ-amanitin is the main toxin. It produces severe hepatocellular damage by binding to nuclear RNA polymerase II. Inhibition of mRNA synthesis is the major cause of cell death. Note: the poison is heat stable. Mortality rate of 50%	*Amanita phalloides, A. Phalloides* var. *alba, A. phalloides* var. *umbrina*	After an average period of 10 - 14 h (range 6 - 48 h) there is a sudden onset of nausea, vomiting, colicky abdominal pain, profuse diarrhoea, tachycardia, hypoglycaemia, which may be associated with electrolyte and acid-base disturbances. This is followed by a second phase of temporary recovery, with a relapse on day 2 - 4. During this third stage, fulminant hepatic failure and possibly renal failure become clinically apparent
Monomethylhydrazine group Intoxication resembles isoniazid poisoning	*Gyromitra* species (false morel). Known cases of poisoning mainly from Europe. Poisonous *Gyromitra* species probably not represented in southern Africa	Nausea, vomiting, diarrhoea and abdominal cramps. May develop haemolysis, seizures, fever, inco-ordination, hepatic failure and coma. Onset usually within 6 - 24 h after ingestion
Orellamine group Series of heat-stable nephrotoxic poisons	*Cortinarius* species — probably not found in southern Africa	Anorexia, nausea, vomiting, diarrhoea, headache, joint pain followed by renal failure. Onset after a long latent period, 36 hours - 14 days after ingestion. Symptoms and signs of renal failure may appear even as late as 21 days after ingestion

- In psychoactive mushroom poisonings (hallucinogens, etc.), both cholinergic and anti-cholinergic effects can occur. No specific treatment is necessary. The benzodiazepines may be helpful in agitated patients.
- In suspected *A. phalloides* poisoning, the use of *N*-acetylcysteine has been suggested (refer to paracetamol poisoning regimen, p. 460). Penicillin G, in high doses, may also be of benefit as a receptor-site competitor. Fluid and electrolyte/acid-base imbalance should be treated and hypoglycaemia corrected. Large quantities of carbohydrate appear to protect the liver (5 - 10% dextrose, 4 - 5 l/24 h). Administer vitamin K for bleeding. As soon as fluids can be administered orally, give fruit juices fortified by glucose 120 g/l up to 4 - 5 times daily. Administer silibinin (specific antidote) in a dose of 20 - 50 mg/kg/day intravenously if it is available (see list of antidotes, p. 462). Haemodialysis may be lifesaving in renal failure. For the removal of toxin, haemodialysis or haemoperfusion has not been shown to be of substantial benefit. In patients with hepatic failure, consult a liver specialist for the appropriateness of a liver transplant.[1-6]

References available on request.

POISONOUS PLANTS

For information on traditional medicines of plant origin see p. 481.

In an analysis of 220 consecutive exposures to poisonous plants processed by the Tygerberg Poison Information Centre, most of the symptomatic patients (100/220) ingested plants containing either calcium oxalate (42 cases) or atropine alkaloids (11 cases), or were exposed to plants causing dermatitis (25 cases).

Plant ingestions[1-3]

The most serious toxic effects were encountered with plants containing atropine-like substances (10 out of 11 patients). *Datura stramonium* (stinkblaar) and *Brugmansia* species (moonflower) were most commonly involved. This type of poisoning occurs mostly among teenagers who may be experimenting with drugs.

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The anticholinergic effects include the following: a hot, dry, flushed skin; dry mouth, dilated pupils, urinary retention, tachycardia, fever, disorientation, hallucinations, aggressive behaviour, and delirium, with seizures and coma in severe cases. Rhabdomyolysis is a possible complication for which treatment is primarily symptomatic and supportive. Patients should be closely observed to prevent injuries, nursed in a dark room (photophobia), temperature monitored, and adequate fluid intake should be assured. Benzodiazepines may be given to calm patients and to treat seizures. Urine should be monitored for the presence of myoglobin.

Only one potentially serious complication was encountered with the ingestion of calcium oxalate-containing plants (42 cases). The patient developed laryngeal oedema and stridor. Plants containing calcium oxalate crystals include dumbcane (*Dieffenbachia*), elephant's ear (*Alocasia macrorrhiza* and *Colocasia esculenta*), arum lily (*Zantedeschia* species) and delicious monster (*Monstera deliciosa*). Chewing on parts of the abovementioned plants may produce an immediate, intense pain of the mouth, tongue and lips. In severe cases, especially in children, it may cause laryngeal oedema. These plants may also cause contact dermatitis or keratoconjunctivitis. Management includes clearing the mouth of plant parts and administration of cool liquids or crushed ice. The possibility of laryngeal oedema should be kept in mind.

Ingestion of the ripe berries of the syringa tree (*Melia azedarach*) was the most common plant ingestion. Only 2 of 53 patients presented with mild gastrointestinal symptoms (diarrhoea), which may have been due to other causes. The ripe berry has a very hard kernel and usually passes through the gastrointestinal tract intact. Management is symptomatic and hospitalisation is usually not necessary.

Beans of the castor oil plant.

The number of incidents of exposure to oleander (*Nerium oleander*) and beans of the castor oil plant (*Ricinus communis*) was relatively low (11 and 5 cases, respectively) and only a few patients presented with mild gastrointestinal symptoms (< 12%). Oleander contains cardioactive glycosides and has the potential to cause serious cardiotoxicity. However, the amount ingested by children is usually too small to cause significant poisoning. The seeds of the castor oil plant contain ricin, an

extremely toxic toxalbumin. All parts of the plant are poisonous, but its seeds are especially rich in ricin, which remains in the fibrous residue of the seeds after the castor oil has been extracted from them. Ingestion of the intact, unchewed seed does not result in significant toxicity, because the hard seed coat prevents absorption of toxins. A similar toxalbumin, abrin, is found in the rosary bean or love bead (*Abrus precatorius*). Symptoms and signs of ricin poisoning include gastrointestinal haemorrhage, haemolysis and renal failure. Treatment of exposure to oleander and castor oil plant beans is symptomatic and supportive. Activated charcoal is recommended within the first 2 hours of ingestion.

Four cases of Jerusalem cherry (*Solanum pseudocapsicum*) ingestion were encountered. The brightly coloured red cherries contain varying amounts of solanine and atropine-like alkaloids (the solanine dominates). They are only mildly poisonous and the most common toxic effects are nausea, vomiting, headache and diarrhoea. Other plants which contain solanine include tomato leaves, unripe tomatoes, as well as potato leaves and green tubers. Treatment is symptomatic and supportive.

General management

Most childhood ingestions are non-toxic or relatively benign and require only well-informed explanations and reassurances after the plant has been identified. This is because the amount ingested is generally small. If a known poisonous plant has been ingested, activated charcoal is recommended if given within 2 hours of ingestion. If there are no symptoms and signs of poisoning, regular follow-up is recommended. Symptomatic patients, however, should be admitted to a medical facility. Symptomatic and supportive care is the mainstay of therapy. The same general principles apply even when a plant cannot be identified. Accurate identification of the plant involved can be difficult or impossible. In such cases activated charcoal is recommended, with appropriate follow-up and/or observation. Contact your local poison information centre for help.

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Plant dermatitis (phytodermatitis)[4-9]

The 25 plant dermatitis cases dealt with by the Tygerberg Poison Information Centre were caused mainly by *Euphorbia* plant species, the rainbow leaf plant (*Smodingium argutum*) and the blister bush (*Peucedanum galbanum*).

The following is a simplified classification of plant-induced dermatoses, which is a complex subject (several pseudoplant dermatoses are also recognised, where the primary irritant/trigger is produced by arthropods (mites) and fungi inhabiting the plant, but these are excluded from the classification):

Mechanical injury. These include injuries due to thorns, barbs, etc., with the added potential to cause bacterial or fungal infection.

Direct chemical injury. Examples of plants in this category include the nettles (*Urtica* species), which produce contact urticaria by stinging hairs; *Euphorbia* species, which contain irritant chemicals (diterpenoids) in the milky sap (latex); and the calcium oxalate-producing plants (e.g. *Dieffenbachia*). Several of the local *Euphorbia* species can cause quite severe primary irritant dermatitis. Species include *Euphorbia ingens* (candelabra tree; naboom), *Euphorbia mauritanica* (*gifmelkbos*) and *Euphorbia virosa*. Initial symptoms include erythema and swelling within 2 - 8 hours, followed by vesicle formation within 4 - 12 hours. Skin reactions usually fade within 3 - 4 days. Eye contact may result in corneal ulceration, iritis, conjunctivitis, and even temporary blindness.

Allergic contact dermatitis. This category can be classified as follows:

- Immediate allergic type (hypersensitivity type I, IgE mediated). Allergic reactions are often seen in handlers of apples, tomatoes, potatoes and celery. Symptoms and signs include erythema, oedema, urticaria, and symptoms of itching, burning or tingling at the site of contact within 1 - 2

Dieffenbachia.

hours post exposure. Another example in this category is anaphylactoid reactions after ingestion of nuts.

- Delayed allergic contact dermatitis (type IV, cell-mediated, hypersensitivity contact dermatitis). This reaction develops typically > 6 hours post exposure. Poison ivy-triggered allergic contact dermatitis is a typical example (*Toxicodendron radicans*). *S. argutum* (rainbow leaf, family *Anacardiaceae*) is the plant often responsible for causing allergic contact dermatitis in South Africa. The *Compositae* family is also a common cause of allergic contact dermatitis. This family contains many garden plants, such as chrysanthenums, and dahlias, as well as vegetables, such as lettuce, artichokes, etc. Even a small number of orchids, as well as primrose, have been implicated. The clinical picture of allergic contact dermatitis ranges from transient redness to severe swelling, vesiculation, pruritus and even bullous skin lesions. The dermatitis is usually limited to the site of contact, but may spread later. If the causative agent is removed, the lesions may heal within days to weeks. There may be scaling, cracking and some thickening of the skin. With continued exposure, complications such as infection may occur.
- **Phytophotodermatitis.** This is a light-induced plant dermatitis, which can be a phototoxic, or photoallergic reaction. The furocoumarins are the major photoactive chemicals, of which the psoralens are best known. These chemicals occur in two plant families, the Apiaceae (formerly Umbelliferae) and the Rutaceae. The psoralens strongly crosslink DNA and with exposure to UVA can result in cell damage and cell death. A common local entity is a photodermatitis caused by the blister bush (*Peucedanum galbanum*, family Apiaceae). After accidental skin contact with the sap of the plant, together with direct sunlight, dermatitis develops 24 hours or more after exposure. The skin reaction is characterised by a burning erythema with vesiculation or bullous skin lesions in the areas of contact. The lesions typically go through a hyperpigmentation phase, lasting for weeks to months.

Management of phytodermatoses includes identification and removal of the primary cause, stabilisation of inflammation with either antihistamines and/or corticosteroids, and reparation of the barrier function of the skin with urea (Eulactol), lactic acid (Lacticare) or the ceramides (SBR lipocream).

References available on request.

FOOD-BORNE DISEASE (FOOD POISONING)

Serious acute poisonings are often associated with prominent gastrointestinal symptoms and signs, a feature often overlooked in standard toxicology textbooks. Examples include iron, theophylline, lithium, arsenic, paraquat, digoxin, and mercury poisoning.

Food-borne diseases may present with severe gastrointestinal symptoms and can result from ingestion of a wide variety of foods contaminated with pathogenic micro-organisms, microbial toxins, or chemicals. For differential diagnostic purposes, the food-borne diseases are therefore discussed briefly.

The diagnosis of food-borne disease should be considered when an acute illness with prominent gastrointestinal symptoms (often together with neurological and/or cardiovascular manifestations) affects two or more persons who have shared a meal during the previous 72 hours.[1]

Food-borne poisoning presenting 1 - 6 hours post ingestion

Microbial agents and their toxins

***Staphylococcus aureus* food poisoning** is characterised by vomiting (76% of cases) and diarrhoea (68%). Fever is less common (23%). The illness is caused by a preformed enterotoxin and outbreaks are associated with foods such as ham, poultry, potato, egg salads and pastries.

***Bacillus cereus* food poisoning** is characterised by nausea and vomiting (100%), abdominal cramps (100%) and diarrhoea (33%) and is caused by a heat-stable toxin. Outbreaks are often associated with fried rice that has been kept warm for long periods.

Management includes oral rehydration and other supportive measures.[1]

Non-microbial toxins

Heavy metal poisoning may be due to storage of foods in containers lined with cadmium, copper, zinc, or antimony (enamelled cookware). Ingestion causes nausea, vomiting and abdominal cramps within 5 - 15 minutes. Symptoms usually resolve within 2 - 3 hours after exposure to the offending agent has been terminated.[1]

Scrombroid poisoning — refer to the article on poisonous and venomous marine animals (p. 471).[2,3]

The 'Chinese restaurant syndrome' is an entity that develops within 15 - 30 minutes after dining in certain Chinese restaurants. The symptoms usually last for two hours and resolve without residual effects. Symptoms and signs include numbness or burning at the back of the head, radiating to both the arms and the back, general weakness, palpitations, headache, flushing, sweating, nausea, abdominal cramps and

thirst. Although excessive amounts of MSG (monosodium L-glutamate) used in food have been implicated, several studies did not show a causal link between the two. Other as yet unidentified substances may also play a role.[4] The treatment is symptomatic.

Paralytic shellfish poisoning — refer to the article on poisonous and venomous marine animals (p. 471).

The 'Chinese restaurant syndrome' is an entity that develops within 15 - 30 minutes after dining in certain Chinese restaurants.

Miscellaneous mushroom poisonings.[5] Various poisonous mushroom species may cause severe gastrointestinal or nervous system toxic effects within 30 minutes - 2 hours post ingestion. Patients with mushroom poisoning presenting within 6 hours of ingestion usually have a more favourable outcome than those presenting late (6 - 48 hours post ingestion). (See detailed discussion in article on mushroom poisoning on p. 475.)

Puffer fish poisoning is caused by the ingestion of the flesh, viscera or skin of fish containing the neurotoxin tetrodotoxin. Headache, a floating sensation, sweating, oral and peripheral paraesthesias occur within 10 minutes - 3 hours. Typical gastrointestinal symptoms include epigastric pain, nausea, vomiting, and salivation. Hypotension, bradycardia, and fixed, dilated pupils indicate severe poisoning. An ascending paralysis may lead to respiratory arrest within 4 - 24 hours. Puffer fish poisoning is uncommon in southern Africa.[6]

Sodium nitrite poisoning (nitrate is metabolised to nitrite). Sodium nitrite is used in small quantities as a curing agent in processed meat (200 - 500 ppm). Sodium nitrite has been sold on the street as 'table salt', leading to multiple cases of severe methaemoglobinaemia and, in some instances, fatalities. Less than 1 g is potentially lethal in children. Sodium nitrite oxidises haemoglobin ($Hb\text{-}Fe^{++}$) to methaemoglobin ($metHb\text{-}Fe^{+++}$), which has no oxygen-carrying capacity. Cyanosis occurs when 15% of Hb has been converted to metHb.[7]

- **Clinical features.** If substantial amounts (> 1 g) are ingested, symptoms and signs of poisoning may occur within 1 hour. These include: gastric irritation, nausea and vomiting, a throbbing headache and dizziness. Chocolate cyanosis of the skin, lips and nails develops when the metHb concentration reaches 30 - 40%. Levels above 60% usually result in stupor, seizures and respiratory failure.
- **Management** includes immediate life-support measures, such as 100% oxygen. Activated charcoal is useful if administered within 1 hour after ingestion. Methylene blue (1 - 2 mg/kg in a 1% solution) should be administered if the metHb concentration rises above 20%; additional doses of methylene blue may be required and total doses of 7 mg/kg may be required for adequate treatment although the higher doses carry a risk of causing haemolysis. Oxygen therapy should be continued for at least 2 hours after methylene blue has been given. Exchange transfusion should be considered in severe cases.

Food-borne poisoning presenting more than 6 hours post ingestion

Microbial agents and their toxins[1]

Abdominal cramps and diarrhoea within 8 - 16 hours. *Clostridium perfringens* food poisoning is caused by an enterotoxin-produced *in vivo*. Vomiting and fever are uncommon. An enterotoxin producing *B. cereus* causes a similar clinical picture. Symptoms and signs of both entities usually resolve spontaneously within 24 hours. Meat and vegetable dishes are usually implicated in outbreaks.

Fever, abdominal cramps, and diarrhoea within 16 - 48 hours. Pathogens responsible include salmonellae (from beef, poultry, eggs and dairy products), shigellae (egg salads and lettuce), *Campylobacter jejuni* (poultry and raw milk), *Vibrio parahaemolyticus* (shellfish), and invasive *Escherichia coli* (undercooked ground beef and raw milk). These food-borne diseases are caused by tissue-invasive pathogens. Vomiting may also be a feature in these cases.

Clostridium perfringens food poisoning is caused by an enterotoxin produced in vivo.

Abdominal cramps and watery diarrhoea within 16 - 72 hours. The major pathogens responsible include enterotoxin-producing strains of *E. coli* (beef and raw milk), *V. parahaemolyticus* (seafood), and *Vibrio cholerae* (ingestion of contaminated water, seafoods and other foods). *C. jejuni*, salmonellae, and shigellae may also be responsible. Severe cholera is characterised by

profuse watery diarrhoea, vomiting, muscular cramps, dehydration, oliguria, and cardiovascular collapse.

Management of the latter two diarrhoeal illnesses includes replacement of fluid losses. Antimicrobial agents are usually indicated in the treatment of shigellosis (a fluoroquinolone, trimethoprim-sulphamethoxazole, amoxicillin), cholera (tetracyclines, a fluoroquinolone, trimethoprim-sulphamethoxazole), and invasive salmonellosis and typhoid (ceftriaxone, a fluoroquinolone, amoxicillin, chloramphenicol, trimethoprim-sulphamethoxazole).

The abrupt onset of acute gastrointestinal symptoms, together with a descending weakness or paralysis, strongly suggests food-borne botulism.

Bloody diarrhoea (haemorrhagic colitis) without fever within 72 - 120 hours. This is caused by cytotoxin-producing strains of *E. coli* (Shiga toxin). The toxins damage endothelial cells in target organs such as the gut and kidney. Haemorrhagic colitis is accompanied by an acute onset of severe abdominal cramps and watery diarrhoea that typically becomes grossly bloody within 24 hours. Development of fever may indicate complications. Approximately 50% of the cases are complicated by a haemolytic-uraemic syndrome. Treatment is supportive, with fluids and a bland diet.

Nausea, vomiting, diarrhoea, and paralysis within 18 - 36 hours. The abrupt onset of acute gastrointestinal symptoms, together with a descending weakness or paralysis, strongly suggests food-borne botulism. In humans the disease is caused by one of three heat-labile neurotoxins, produced by the anaerobic growth of *Clostridium botulinum*. Growth frequently occurs in underprocessed, non-acid canned foods (pH > 4.6). The exotoxins inhibit the release of acetylcholine at peripheral nerve endings. Home-canned foods are the most common sources and vegetables, fish, fruits, and condiments are the most common vehicles, but beef, milk products, pork, and poultry have been involved. Non-canned foods, e.g. foil-wrapped baked potatoes and chopped garlic in oil, have also caused outbreaks.[8]

- **Clinical features.** The onset of food-borne botulism is abrupt, usually within 18 - 36 hours post ingestion, although the incubation period may vary from 4 hours to 8 days. Nausea, vomiting, abdominal cramps, and diarrhoea frequently precede neurological symptoms. Neurological symptoms and signs are characteristically bilateral and symmetrical, beginning at the cranial nerves and followed by descending weakness or paralysis. Constipation is common after neurological impairment. There are no sensory disturbances and fever is absent. Major complications include respiratory failure and pulmonary infections.[8]
- **Management.** Gastric lavage and activated charcoal, if instituted early, may be helpful in cases where consumption of contaminated food is suspected. Respiratory impairment requires management in an ICU. The antidote, trivalent antitoxin (A,B,E), should be administered as soon as possible. The antitoxin does not inactivate toxin that is already bound at the neuromuscular junction; pre-existing impairment cannot be reversed rapidly. The antitoxin may, however, slow or halt further progression.[8]

Non-microbial toxins

Abdominal cramps and diarrhoea within 6 - 24 hours, followed by hepatorenal failure. Mushrooms containing cyclic oligopeptides (cyclopeptides), particularly alpha-amanitin, are responsible for this syndrome. Mushrooms containing these oligopeptides include *Amanita phalloides, A. phalloides* var. *alba* and *A. phalloides* var. *umbrina*. They occur throughout southern Africa, under broad-leafed and pine trees. The illness is typically triphasic. The early stage is characterised by abdominal pain, nausea and vomiting, and cholera-like diarrhoea. This is followed by an asymptomatic stage of 1 - 2 days' duration. During the third stage, fulminant hepatic failure, and possibly acute renal failure, sets in (see article on mushroom poisoning, p. 475).

Of the serious non-food-borne acute poisonings presenting with prominent gastrointestinal symptoms and signs most will usually present clinically within 6 hours post ingestion. However, digoxin and sustained-release theophylline and lithium may often present later (6 - 12 hours post ingestion).[5]

References available on request.

TRADITIONAL MEDICINES AND ACUTE POISONING

Traditional healers (e.g. *inyanga, ngaka, sangoma*) form an integral part of the life of most indigenous peoples of southern Africa. Health professionals need to be aware that 60 - 80% of all South Africans consult traditional healers and use the medicines they prescribe. They provide a relatively inexpensive alternative form of medical care that is readily accessible, even in remote parts of the country.

Most traditional medicines contain substances of plant origin. These medicines are generally not associated with prominent or significant adverse/toxic effects. This is probably because the concentration of pharmacologically active substances in the medicines is either too low, or no active ingredients are present at all. Some traditional medicines, however, do contain potent pharmacoactive chemicals which may, in certain circumstances, cause toxic side-effects or poisoning.

Health professionals need to be aware that 60 - 80% of all South Africans consult traditional healers and use the medicines they prescribe.

Most traditional medicines are mixtures of a variety of plants and are of very variable composition and concentration. Seasonal variation in chemical concentration found in some plants may also contribute towards inconsistent concentrations of extracted chemicals, e.g. the bufadinaloids (cardiac glycosides) in *slangkop* (*Urginea sanguinea*).[1-3]

Table I lists some of the more common poisonous plants used in traditional medicines. Target organs and main toxic effects are also listed.[3-5]

Clinical features

Gastrointestinal symptoms and signs predominate in poisoning caused by traditional medicines. Many traditional medicines contain potent gastrointestinal irritants and purgatives, included for their 'purifying' actions. Enthusiastic use of enemas may be contributory. Symptoms and signs include upper abdominal discomfort, nausea, vomiting and diarrhoea of varying degrees and duration. In severe cases, haematemesis and melaena are seen. Complications include dehydration, disturbances in electrolyte and acid-base balance, haemolysis and a reduced haemoglobin, which may compromise kidney function. Perforation of the lower gastrointestinal tract should be considered in patients presenting with an acute abdomen.

McVann *et al.*[6] advise that in cases of poisoning where gastrointestinal symptoms are prominent, cardiac glycosides are the most likely cause — e.g. the medicine *sekanama* (Tswana), obtained from the bulb of *Urginea* plant species (see table).

Other organs targeted by potentially poisonous traditional medicines include the kidneys, liver and CNS. Symptoms and signs of urogenital damage include dysuria, haematuria and oliguria. Renal damage/failure is often secondary to hepatocellular damage. Patients with hepatic damage may present with raised liver enzymes, an increase in prothrombin time (or INR), metabolic acidosis and hypoglycaemia. Frank jaundice, which may occur occasionally, carries a poor prognosis.

Seeds of Datura stramonium *(stinkblaar).*

Some traditional medicines may directly cause CNS toxic effects such as confusion, delirium, hallucinations, respiratory failure and convulsions. However, in many cases symptoms and signs of CNS toxicity are secondary to metabolic disturbances, such as acidosis, hypoglycaemia, electrolyte disturbances, uraemia or hepatic failure.

Poisoning due to the ingestion of *stinkblaar* seeds (*Datura stramonium*) is relatively common. The plant is known as *mokhura* to Tswana traditional healers and is used as an aphrodisiac. The dry seeds are sometimes smoked with dagga. Acute poisoning is caused by the muscarinic receptor-blocking action of atropine and related alkaloids. The toxic effects include a hot, dry and flushed skin, dry mouth, dilated pupils, urinary retention, tachycardia, fever, disorientation, hallucinations, aggressive behaviour, delirium, and seizures and coma in severe cases. Rhabdomyolysis is an occasional complication.

Sometimes traditional medicines may contain potentially poisonous substances of animal origin, e.g. cantharidine, and/or of non-biological origin, such as dichromate, arsenic or lead. Even conventional (allopathic) medicines may be incorporated. Hepatorenal syndrome with severe coagulopathy and intravascular haemolysis has been described in poisonings due to dichromate-containing medicines.

Cantharidine, a powerful lipid-soluble toxin/vesicant, derived from many indigenous blister beetle species (family Meloidea), is the active ingredient in a medicine known as *seletsa*. It is used as an abortifacient and aphrodisiac. Over-enthusiastic oral use of this medicine may cause severe necrosis of the oesophageal and gastric mucosa, congestion and irritation of the urinary tract with dysuria, haematuria and renal failure. Liver function may also be compromised. Ventricular dysrhythmias, coagulopathy and shock may develop in severe cases. As little as 10 mg may be fatal.

Table I. **Plants used in traditional medicines reported to be associated with acute poisonings**

Plant species	Vernacular name(s)	Toxic ingredients/target organs and features of poisoning
Asclepias fruticosa	Milkweed, *melkbos, tontelbos, usinga-lwesalukazi* (Zulu), *modimolo* (S Sotho)	Cardiac glycoside. GIT
Boophane disticha	Bushman poison bulb, *gifbol, iBadi* (Zulu), *inCwadi* (Xhosa)	Buphanidrine and other alkaloids. CNS (dizziness, restlessness, hallucinations, respiratory failure)
Bowiea volubilis	*uGibisisila* (Zulu), *Mgaqaqana* (Xhosa)	Cardiac glycosides. GIT, cardiovascular, haemolysis
Chenopodium ambrosioides (mucronatum)	*Setla-botsha*	Active ingredient not known. CNS (psychosis)
Callilepis laureola	Ox-eye daisy, *impila* (Zulu)	Atractyloside (strychnine-like poison). GIT, kidney, liver
Corchorus tridens (? capsularis)	*Ghumbughumbu*	Cardiac glycoside. GIT
Eucomis utumnalis (E. undulata)	*ubuHlungu becanti* (Xhosa), *uMathunga* (Zulu)	Haemolytic saponin: GIT, kidney
Gloriosa superba	*iHlamvu* (Zulu, Xhosa) *uHlamvu* (Zulu)	Colchicine. Mucous membrane irritation, GIT, hypotension, respiratory failure
Jatropha curcas	Purging nut, *purgeerboontjie, Mathlapametse* (Tswana)	Curcin and curnanoleic acid, which is similar to ricinoleic acid (in castor oil). GIT, kidney, potent irritant laxative, severe diarrhoea/dehydration
Ledebouria spp	*Letswitlana*	Active ingredient not known. GIT, kidney, liver, CNS
Rhoicissus tridentate (? cuneifolia)	*Morokolopudi*	Active ingredient not known. GIT, kidney, CNS, respiratory failure
Ricinus communis	Caster oil plant, *kasterolieboom,* Mokhura (N Sotho), *Umhlakuva* (Xhosa, Zulu)	Two toxic ingredients in seed (not in the oil) ricinin and ricin. Ricin is extremely toxic. GIT (haemorrhage), haemolysis, kidney, liver. Ricinoleic acid is a potent GIT irritant, causes diarrhoea. Oil contains ricinoleic acid (as in castor oil)
Scadoxus puniceus	Red paintbrush, *rooikwas, Umphompo* (Zulu)	Patalensine and other alkaloids. CNS (stimulation and convulsions)
Scilla nervosa	*inGcino* (Zulu), *Magaqana* (Xhosa)	Digitalis glycosides. GIT, cardiovascular
Spirostachys africana	African mahogany, tamboti, *umtomboti* (Xhosa, Zulu), *morekuri* (Sotho)	Latex contains diterpenes. GIT (potent irritant)
Urginea sanguinea (? burkei)	*Slangkop, sekanama* (Sotho, Tswana), *Skanama* (Zulu)	Digitalis-like glycosides. GIT, cardiovascular system (cardiac conduction abnormalities)

Management

Management of patients with poisoning due to traditional medicines should follow the same general principles and guidelines as applied to any other poisoning. Basic life support measures should always be a first priority. Serum electrolytes, acid-base balance, state of hydration, blood glucose, renal and liver function, full blood count and coagulation status should be assessed and monitored. Fluid replacement and the correction of acid-base balance and electrolytes (especially serum potassium) should be a priority. In cases of gastrointestinal bleeding, the haematocrit and haemoglobin need to be monitored regularly. Because cardiac glycosides are often the cause of poisoning, ECG monitoring is recommended in cases of suspected cardiac glycoside poisoning. Urine should be screened for the presence of myoglobin to exclude rhabdomyolysis. Antiemetics and antidiarrhoeals may be required for the treatment of severe vomiting and diarrhoea. In patients with renal failure, haemodialysis should be considered.

Some traditional medicines may directly cause CNS toxic effects such as confusion, delirium, hallucinations, respiratory failure and convulsions.

In poisoning due to Indian traditional medicines, dichromate and other heavy metals have been implicated. These should also be considered in patients presenting with acute renal failure, gastrointestinal haemorrhage and hepatic failure.

Management strategies should include peritoneal or haemodialysis.

In cantharidine poisoning, IV fluids should be administered in patients with significant vomiting and/or diarrhoea, haematuria or gastrointestinal bleeding. ECG monitoring is essential. In cases where renal failure occurs, haemodialysis may be necessary. Treatment of *Datura stramonium* poisoning (anticholinergic syndrome) is primarily symptomatic and supportive. Although physostigmine is recommended by most textbooks, its use is generally not advisable. Patients should be closely watched to prevent injuries, they should be nursed in a dark room (patients are photophobic), temperature monitored and adequate fluid intake assured. Benzodiazepines may be given to calm patients and to treat seizures.[1-7]

No routine tests exist for the identification of plant toxins.

References available on request.

SINGLE SUTURE

Asthma and nitrogen dioxide exposure

There is a suggested link between exposure to nitrogen dioxide (NO_2) as an air pollutant and respiratory disease. It is also known that viral infections are a major cause of asthma exacerbations. A recent paper in *Lancet* showed that high levels of NO_2 in the week before the start of a respiratory viral infection, at levels within current air quality standards, were associated with an increase in the severity of a resulting asthma exacerbation.

(Chauhan AJ, *et al. Lancet* 2003; **361:** 1939-1944.)

This is an important consideration in cities such as Cape Town and Johannesburg where levels of air pollutants are far in excess of accepted international norms — Editor.