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Clinical Review: Emergency management of acute poisoning

Gestion des urgences de l'intoxication aiguë

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KEYWORDS

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Abstract Acutely poisoned patients are commonly encountered in Emergency Centres. Acute poisoning (accidental or intentional) requires accurate assessment and prompt therapy.
The necessity to prevent cross contamination during the initial evaluation should be emphasized. Early identification of the involved toxin/s is crucial and the majority will be identified by a thorough history and physical examination. An ABC-approach should be followed ensuring a protected airway, adequate ventilation and hemodynamic stability. Supportive and symptomatic care remains the cornerstone of treatment. A stepwise approach may be followed to decrease the bioavailability of toxins. Indications, contra-indications, risks and dosage regimens are describe for decontamination procedures including both termination of topical exposures and decreasing exposure to ingested toxins. Furthermore, procedures to increase the elimination of toxins and a short section covering specific toxins and their antidotes are also included.
The aim of this commissioned review was to establish concise guidelines for the initial management of the acutely poisoned patient in the Emergency Centre. The American Academy of Clinical Toxicology and the European Association of Poisons Centres and Clinical Toxicologists are the

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international leaders in the field of toxicology and the guidelines in their position papers were generally followed. Most of the dosage regimes are according to the South African Medicines Formulary.

Abstract Patients empoisonnés de façon aiguë sont couramment rencontrées dans les centres d'urgence. L'intoxication aiguë (accidentelle ou intentionnelle) nécessite une évaluation précise et un traitement rapide.

La nécessité de prévenir la contamination croisée lors de l'évaluation initiale doit être souligné. L'identification précoce de la toxine en cause/s est cruciale et la majorité seront identifiés par une histoire et un examen physique. Un ABC approche devrait être suivie assurer des voies aériennes protégées, une ventilation adéquate et de la stabilité hémodynamique. Les soins de soutien et symptomatique reste la pierre angulaire du traitement. Une approche progressive peut être suivie pour réduire la biodisponibilité des toxines. Indications, contre-indications, les risques et les régimes posologiques pour décrire les procédures de décontamination, y compris à la fois la cessation de l'exposition d'actualité et l'exposition à des toxines ingérées diminue. En outre, les procédures d'augmenter l'élimination des toxines et une courte section portant sur les toxines spécifiques et leurs antidotes sont également inclus.

Le but de cette étude commandée était d'établir des lignes directrices concises pour la gestion initiale du patient empoisonnés de façon aiguë dans le Centre d'urgence. L'American Academy of Clinical Toxicology et l'Association européenne des centres antipoison et de toxicologie clinique sont les chefs de file internationaux dans le domaine de la toxicologie et les lignes directrices dans leurs prises de position étaient généralement suivies. La plupart des régimes posologiques selon le Formulaire des médicaments en Afrique du Sud.

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Introduction

Acutely poisoned patients are very commonly encountered in South Africa. The exposure may be accidental (e.g. medication error, unsafe storage) or intentional (e.g. para-suicide, substance abuse). Virtually all known chemicals have the potential to cause injury or death, if the exposure is large enough. The critical factor is not whether a substance is poisonous or not, but whether exposure to it poses a risk. The practical value of knowing that a product is relatively non-toxic prevents unnecessary healthcare visits, overtreatment and an inappropriate response that may cause panic.

There are limited statistics available in South Africa regarding poisoning as the cause of death. In 2007, the 9th Annual Report of the National Injury Mortality Surveillance System (NIMSS) attributed 4% of deaths to poisoning, with a peak in the 30–34 year age group.[1]

Poison Information Centre (PIC) statistics in the United States of America show that, during 2008, more than 2.4 million human exposures were logged by 61 PICs, of which 1315 were fatalities i.e. 8.2 exposures per thousand population with a fatality rate of 0.05%.[2] Since 1999 the National Poison Information Service (NPIS) in the United Kingdom has provided both a national telephone service as well as free access to an internet database service (TOXBASE) for all professionals registered with the National Health Service. The implementation of the TOXBASE system almost halved the call load to the National Poison Information Service.[3] South Africa does not have a single national poison information service or a facility for access to an online database.

There are currently three PICs in South Africa, answering about 11,000 calls per annum (Personal communication: Dr. DJH Veale; May 2010). Unpublished data from the Tygerberg Poison Information Centre confirm anecdotal reports of poor initial medical management of poisoned patients in South African medical facilities.

Carbon monoxide
Cholinesterase inhibitors
- Organophosphates
- Carbamates

Cyanide
Digoxin
Ethylene glycol
Isoniazid
Metals:
- Arsenic
- Iron
- Lead
- Lithium
- Mercury

Methanol
Methyl bromide
Mushrooms:
- *Amanita phalloides*

Paracetamol
Paraquat
Salicylates
Theophylline

Box 1 Compounds with a high inherent toxicity.

The aim of this commissioned review is to establish concise guidelines for the initial management of the acutely poisoned patient in the Emergency Centre. The American Academy of Clinical Toxicology and the European Association of Poisons Centres and Clinical Toxicologists are the international leaders in the field of toxicology and the guidelines in their position papers were generally followed. Most of the dosage regimes are according to the South African Medicines Formulary (SAMF) as this is the most accessible reference guide for South African healthcare workers.

Approach to the management of the poisoned patient

Acute poisoning requires accurate assessment and prompt therapy. All patients must be thoroughly assessed and stabilized from the start.

History

Attempts should be made to try to identify the specific poison but this however must never delay life-saving supportive care.[4] Therefore, while vital functions are being assessed and stabilised, a proper history should be obtained. Important information to be gained from the history for risk assessment includes the nature of the poisonous substance, the degree of exposure and the time since exposure.[4] Poisons, including medicines, may be divided into two broad categories:[5]

i. Poisons (and/or their metabolites) which directly cause irreversible or slowly reversible structural or functional damage to one or more organ systems.
   These are also considered to be compounds with a high inherent toxicity (Box 1). Included in this category, are substances causing delayed significant symptoms and signs (e.g. paracetamol).
ii. Poisons which do not cause tissue damage directly or those which cause toxic effects that are rapidly and completely reversible.
   Fortunately, most potential poisons fall in this category and appropriate symptomatic and supportive care during the acute phase will usually ensure complete recovery.[4]

When dealing with a suspected toxic exposure or poisoning, one of the major priorities should therefore be to attempt to identify agents with a high inherent toxicity as soon as possible.[5] Early identification will allow for timeous special decontamination and antidotal procedures in order to avoid severe or permanent tissue damage.[5] It should be noted that patients who have ingested poisons with a high inherent tissue-damaging potential often present with severe and persistent gastrointestinal symptoms and signs.[5]

A history obtained from a poisoned patient is often unreliable.[4–7] If possible, relatives and friends should also be questioned as they may have useful information as to what the poison was. A special effort should be made to obtain a sample of the poisonous substance and its relevant container. Pre-hospital personnel should be instructed to collect these from the scene.[4] Examination of the suspected toxic substance or the material ingested is crucial for rapid and positive identification of a poison.

Clinical evaluation

A detailed physical examination must be performed after the initial stabilization of the patient. Critical assessment of signs and symptoms will not only assist in determining the etiology, but may also help to assess the severity of the patient's condition.[8] Serious poisonings are often characterised by severe and persistent gastrointestinal symptoms and signs, hypo- or hypertension, hyperthermia, cardiac dysrythmias, altered mental status, seizures, hypoglycaemia, acid–base and electrolyte disturbances as well as signs of liver and renal impairment.[5] As most deaths due to poisoning are a result of respiratory compromise; special attention should be given to the evaluation of the respiratory system.[5] When dealing with suspected acute poisoning, other life-threatening causes of these presentations must be considered and excluded.[4] Dynamic changes in vital signs may be useful in evaluating the patient's response to supportive or antidotal treatment.[8]

Recognition of a specific toxidrome (a constellation of signs and symptoms associated with a specific poisoning) may sometimes assist in the identification of the class of poisonous substance involved.[8–10] Four common toxidromes are summarised in Table 1. Toxidromes are useful when dealing with unidentified toxic agents, but may be misleading in certain circumstances such as exposure to more than one poisonous substance. The onset of toxic manifestations may also be de-

Table 1 Summary of four common toxidromes.

Toxidrome	Common signs	Common causes
Anticholinergic (antimuscarinic)	Delirium Dilated pupils Seizures Raised temperature Dry flushed skin Tachycardia, Dysrythmias Myoclonus Urinary retention Decreased bowel sounds	Antihistamines Antiparkinsonian agents Antipsychotics Antispasmodics Atropine Cyclic antidepressants Mydriatics Plants e.g. *Datura stramonium* (''malpitte'')
Cholinergic (muscarinic and nicotinic receptor stimulation)	Confusion CNS depression Miosis Seizures Muscle weakness (including respiratory muscles) Diaphoresis Salivation Lacrimation Bronchorrhoea Pulmonary oedema Brady-/tachycardia Emesis Gastro-intestinal cramping Urinary/faecal incontinence Muscle fasciculations	Organophosphate and Carbamate pesticides
CNS depressants	Decreased mental alertness Miosis Hyporeflexia Hypothermia Respiratory depression Hypotension Bradycardia Decreased bowel sounds	Amitraz Barbiturates Benzodiazepines Clonidine Ethanol Opioids
Sympathetic nervous system stimulants	Delirium Delusions Paranoia Hyperreflexia Seizures Raised temperature Mydriasis Diaphoresis Piloerection Tachycardia (Brady – if pure α-agonist) Hypertension Dysrythmias	Amphetamines Cocaine Decongestants Ephedrine Methamphetamines Methylphenidate Salbutamol

layed at times e.g. poisonings due to *Amanita phalloides* mushrooms, organophosphates, paracetamol and vitamin K-dependant anticoagulants.

The toxicology laboratory

Although a ''toxicology screen'' may be of value in the identification of poisonous substances, the majority of toxicology-related diagnoses and therapeutic decisions are made from the history or clinical examination.[4,9] The usefulness of a toxicology laboratory is further limited by a prolonged turnaround time and high costs involved to run such a specialised service. Furthermore, the fact that established cut off levels of toxicity have not been determined for many toxins makes the interpretation of test results very difficult.[4] The timing of specimen collection is also important. If collected too early or too late the results may have little clinical correlation.[4] The use of quantitative blood tests should be limited to those drugs and toxins where the specific blood level will either predict toxicity or guide specific therapy.[4] Examples of such drugs are listed in Box 2.

A routine quantitative serum paracetamol level is recommended for ingestions of an unknown drug for two reasons: (i) paracetamol is available in many over-the-counter preparations and (ii) the onset of significant symptoms and signs may be delayed in paracetamol poisoning .[6]

A detailed urine analysis may disclose important clues concerning the diagnosis of an overdosed patient. The presence of calcium oxalate crystals may indicate ethylene glycol poisoning whereas the pH and the colour of the urine may also helpful.[4]

However, the necessity for routine urine drug testing is debatable and possibly of questionable assistance in the emergency setting as supportive treatment remains the cornerstone of the management of the poisoned patient.[9] Furthermore, these test results rarely affect any clinical management decisions.[9]

Without understanding the limitations of toxicology screening tests, physicians may interpret the results incorrectly.[5] If none of the compounds for which the requested tests were designed are identified, the test is reported as negative. Consequently, a negative screening test does not imply that poisoning has not occurred, but only that the compounds tested for are not implicated in the poisoning.[4,5,9] Furthermore, healthcare personnel need to be fully aware of the sensitivity and specificity of the tests they are ordering, as these may differ between different laboratories.[9]

Qualitative urine tests are available for 'drugs of abuse' and the range of drugs tested for depends on the instrumentation available to individual laboratories. Specific reagent kits are purchased for each of the tests and the instrumentation used dictates which kits can be used. Most laboratories (state and private sector) are able to do these tests, depending on their financial resources. Point-of-care qualitative urine screening kits have low sensitivities and should not be used (Personal communication: Dr. DJH Veale, January 2010).

In most cases of poisoning, standard special investigations (e.g. serum electrolytes, glucose, ECG, etc) are often more rewarding for diagnostic purposes than a toxicology screen.[5]

Anticonvulsants
Carbon monoxide
Digoxin
Iron
Lead
Lithium
Methaemoglobin
Paracetamol
Salicylates
Theophylline
Toxic alcohols

Box 2 Toxic substances where a quantitative result of a blood or urine test may alter clinical management.

Bloemfontein Poison Information Centre (7h00-21h00)	- 082 491 0160
Red Cross War Memorial Children's Hospital Poison Information Service (24hr)	- 021 689 5227
Tygerberg Poison Information Centre (24hr)	- 021 931 6129

Box 3 Poison Information Centres operating in South Africa.

Sources of information

Additional information and guidance regarding the identification and management of the poisoning can be obtained by consulting a local PIC. The contact details and operational times of the South African PICs are available in Box 3. The South African Medicines Formulary (SAMF) is also a good reference to use.[11]

Management of the poisoned patient

Staff should be aware of the necessity of taking universal measures to prevent cross contamination during the initial evaluation, depending on the nature of the poison (e.g. organophosphates, cyanide).[12] An ABC-approach should be followed ensuring a protected airway, adequate ventilation and hemodynamic stability.[12] Supportive and symptomatic care remains the cornerstone of treatment.[4] The poisoned patient should be kept under close observation with frequent re-evaluation of vital signs and level of consciousness.[4]

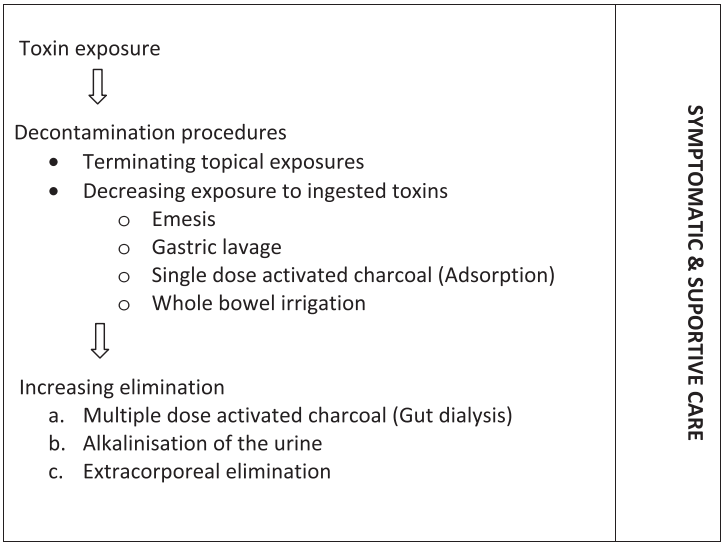
A stepwise approach to decrease the bioavailability of a toxin is illustrated in Box 4.

Decontamination procedures

Terminating topical exposures

Patients exposed to any form of cutaneous contact should have their clothes removed if contaminated. The affected areas should be well rinsed with tap water and then washed with soap or shampoo if available.[13] In the event of serious skin damage (e.g. chemical burns) the affected areas should be rinsed with water alone.

A chemical burn of the eye is one of only a few ophthalmological emergencies.[14] One dose of a local anaesthetic eye drop may be instilled into the affected eye to aid in examination and irrigation, the lids everted and any solid particles swept out of the fornices with a cotton bud.[12] Copious irrigation with normal saline or tap water is of utmost importance and should be initiated as soon as possible.[14] The eye must be irrigated for at least 20 min.[14] Neutralization of an acid or alkali should never be attempted as it will result in an exothermic reaction. All



Box 4 A stepwise approach to decrease the bioavailability of a toxin.

chemical burns involving the eyes need to be evaluated by an ophthalmologist as an emergent case.[15]

Decreasing exposure to ingested toxins
Patients who have ingested any irritant or corrosive substance may drink small sips of water. This procedure may assist in establishing if the patient is able to swallow or not. The total volume of water should not exceed 125 ml, and this volume should be reduced in children.[16] No attempts should be made to neutralize ingested acids or alkalis with other agents. In addition to causing a potentially harmful exothermic reaction, excessive carbon dioxide (CO_2) will be released which may result in gastric distension and perforation. [16]

Gastro-intestinal decontamination
The American Academy of Clinical Toxicology and the European Association of Poisons Centres and Clinical Toxicologists do not recommend the routine use of gastric decontamination, but advise that it may be considered in selected cases.[17–21] Although controversial, healthcare practitioners must always determine whether the benefits outweigh the associated risks.[4]

Emesis. The use of syrup of ipecacuahna to induce vomiting is no longer recommended for several reasons: it possesses inherent toxicity if dosed incorrectly and can cause delayed and protracted vomiting, which delays other decontamination measures and oral treatment.[17]

However, emesis itself is a non-invasive physiological mechanism.[17] In our experience, inducing emesis using the gag reflex may be useful but is not always successful. Giving a glass of water before pharyngeal stimulation may prove effective. In the alert paediatric patient, emesis induced in this way is easier and less traumatic to perform than gastric lavage, as well as being more effective for removing large tablets and objects such as moth balls. It must only be considered in patients presenting within 1 h post ingestion. The patient should be given no more than 250 ml of water to drink, followed by mechanical stimulation of the pharynx.

Induction of emesis is contra-indicated when corrosive substances, volatile hydrocarbons (e.g. paraffin, petrol) or central nervous system stimulants have been ingested as well as in any patient with a decreased level of consciousness.[17]

Gastric lavage. Gastric lavage should not be performed during the routine management of poisoned patients.[18] The serious risks of this procedure usually outweigh the possible benefits.[18] It is unethical to use gastric lavage as a punitive measure.[4]

Gastric lavage is only indicated, if ever, in patients with a suspected lethal overdose who present within one hour after ingestion.[18] It is contra-indicated in patients with an unprotected airway, after ingestion of corrosive substances or substances with a high aspiration potential (such as hydrocarbons), and those with pre-morbid conditions where a risk of gastro-intestinal tract bleeding is present.[18] Serious risks of the procedure include hypoxia, pharyngeal perforation, laryngospasm, aspiration pneumonitis, gastro-intestinal tract perforation, fluid and electrolyte disturbances and dysrythmias.[18]

In order to perform a gastric lavage effectively, the correct bore orogastric tube needs to be used. A large bore 36–40 French gauge tube (external diameter 12–13.3 mm) should be used for adults and 24–28 French gauge tube (diameter 7.8–9.3 mm) in children.[18]

Single dose activated charcoal. Activated charcoal (due to its large surface area) adsorbs a significant amount of poisonous

Acids
Alcohols
Alkalis
Cyanide
Heavy metals (e.g. arsenic, lead, mercury)
Iron salts
Lithium
Petroleum distillates (volatile hydrocarbons)

Box 5 High risk factors for further self harm.

substance in the gastrointestinal tract.[19] Currently there are limited studies to guide the use of single dose activated charcoal and there is no evidence that it improves clinical outcomes.[19]

As with other decontamination measures, the administration of activated charcoal should not be routinely used in the management of poisoned patients.[4] It may be considered in any patient who ingested a potentially toxic amount of a poison, which is known to adsorb to charcoal, up to one hour post ingestion.[19] Administration after 1 h of ingestion remains controversial.[19]

Box 5 lists the toxins that are not adsorbed by activated charcoal.[19,21] Administration of activated charcoal is contra-indicated in patients with an unprotected airway, patients with a risk of gastrointestinal obstruction and where its use will increase the possibility of aspiration.[19]

The optimal dose of activated charcoal is unknown, but a recommended dose is 1–1.5 g/kg of powder.[19] This dose must be adapted for the paediatric patient and 0.5–1 g/kg is recommended.[19] As a guide, a tablespoon of powder equals about 3.5–6 g. Tablets and capsules containing activated charcoal are unsuitable. The slurry of activated charcoal is ideally prepared by mixing the required dose with water in the proportion of 8ml of water to 1g of charcoal, e.g. 50 g of powder in 400 ml water.[16,22] The slurry must be swallowed within a 30 min time frame to be most effective.[19] It should be administered via a naso-gastric tube in patients who refuse to drink the slurry.[19] The appearance of the slurry can be disguised by pouring it into an empty cool drink can to make it more attractive for children to drink. Adding cola may also help to disguise the taste.[23]

Whole bowel irrigation. Whole bowel irrigation (WBI) involves the oral administration of a large volume of an iso-osmotic polyethylene glycol electrolyte solution in order to reduce drug absorption by rapid mechanical cleansing of the entire gastrointestinal tract.[20] It does not usually affect the fluid or electrolyte balance.[20] There is no conclusive evidence that the outcome of poisoned patients is improved using this method.[20] It may be considered after ingestion of the following substances:

Iron or heavy metals
Sustained-released or enteric-coated drug formulations
Packets of illicit drugs (''body-packers'')[20]

Contra-indications include bowel obstruction, perforation or ileus; haemodynamically unstable patients and patients with unprotected airways.[20]

The iso-osmotic polyethylene glycol electrolyte solution is best administered via a naso-gastric tube. The recommended dosage is:

1500–2000 ml/h (adults and adolescents)
1000 ml/h (6–12years)
500 ml/h (9 months–6 yrs)

Administration of this solution should be continued until the rectal effluent is clear.[20]

Increasing elimination

Increasing the elimination of the toxin can be accomplished in three different ways.

Multi-dose activated charcoal (Gut dialysis)
The theoretical benefit of multi-dose activated charcoal (MDAC) is to interrupt the entero-enteric and entero-hepatic circulation of drugs.[21] The role of MDAC in improving morbidity and mortality in the poisoned patient is still unclear.[21] The optimal dosage regimen has not yet been clearly established. Expert opinion suggests that MDAC should only be considered in patients presenting with a potentially lethal dose of carbamazapine, phenobarbitone, theophylline, dapsone or quinine.*21* It should not be used in patients who are unable to protect their airways or in patients with gastrointestinal obstruction.[21]

After an initial loading dose of 50 g activated charcoal in 400 ml water, repeat dosages (a slurry of 25 g every two hours or 50 g every 4 h) is administered, preferably via naso-gastric tube.[16] The dosage should be reduced to 0.5 g/kg every two hours or 1 g/kg every four hours in children (1–12 yrs). This dosage regime should be continued until the patient's clinical condition improves and laboratory parameters or blood levels (if available) decrease.[21] The concurrent administration of cathartics (e.g. sorbitol) is not recommended and could result in fluid and electrolyte imbalances, particularly in children.[21]

The use of MDAC is relatively free from serious adverse effects. However, aspiration, constipation, impaction and obstruction may occur and the patient should therefore be evaluated frequently for ability to protect the airway and evidence of decreased peristalsis or obstruction. [16,21]

Urine alkalinisation
Intravenous sodium bicarbonate is administered to raise the pH of urine. The rise in urinary pH increases the ionization of drugs which are weak acids (e.g. salicylates). This effect reduces re-absorption in the renal tubules (ion-trapping).[24]

Urine alkalinisation is the first line treatment for patients with acute salicylate poisoning unless signs of neurotoxicity are present (in which case haemodialysis becomes the treatment of choice).[24] Other possible indications include phenobarbitone, fluoride or methotrexate poisoning.[24] An initial bolus of intravenous sodium bicarbonate (1 m Eq/kg) is administered and additional boluses can be given there is no clinical improvement.[24] Although many toxicology books advise to maintain urine pH between 7.5 and 8.5, we found it to be extremely difficult to achieve in practice; furthermore, there is the potential of overdosing the patient with sodium bicarbonate.

Compromised renal function is a contraindication for urine alkalinisation, and extreme care should also be taken in patients with significant pre-existing heart disease.[24] The administration of bicarbonate may lead to alkalaemia and hypokalaemia (easily corrected with potassium supplementation).[24]

Clinical improvement and a decrease in blood drug levels are indications for the discontinuation of alkalinisation of the urine.[24]

Extracorporeal elimination
The removal of toxins by extracorporeal techniques such as haemodialysis, haemofiltration and haemoperfusion is indicated for only a small select group of poisons.[25] Agents with low protein binding, a low volume of distribution and a low molecular weight are ideally eliminated using extracorporeal techniques.[25] Haemodialysis may be indicated in poisoning

Table 2 Antidotes and their public sector availability

Indication	Antidote	Availability in SA
Anticholinergics	Physostigmine	Not available
Arsenic, Lead, Mercury	BAL (Dimercaprol)	Not available
	DMSA (Succimer®)	Not available
	D-Penicillamine	Limited availability[a]
Benzodiazepines	Flumazenil	Available
B-blockers	Glucagon	Available
	Insulin and Glucose	Available
Ca^{+} channel blockers	Calcium	Available
	Glucagon	Available
	Insulin and Glucose	Available
Cyanide	Nitrite/sodium thiosulphate regimen (Tripac-Cyano®)	Available
	Dicobalt edetate	Not available
	Hydroxocobalamin	Not available
Digoxin and cardiac glycosides	Digoxin-specific Fab fragments	Not available[b]
Ethylene glycol	Fomepizole	Not available
	Pyridoxine	IV not available
	Thiamine	Available
Hydrofluoric acid	Calcium gluconate	Available
Iron	Desferrioximine	Available
Isoniazid	Pyridoxine	IV not available
Methanol	Folic acid	Available
	Leucovorin	Available
	Ethanol	Available
Oxidants (Methaemoglobin-forming agents)	Methylene blue	Short supply
Opioids	Naloxone	Available
Organophosphates and carbamates	Atropine	Available
	Obidoxime	Not available[b]
Paracetamol	N-acetylcysteine (IV and oral)2	Limited availability[a]

[a] Restricted stocks available, approval required from the Medicines Control Council.
[b] Still awaiting approval from the Medicines Control Council.

by salicylates, lithium, toxic alcohols (methanol, ethanol and ethylene glycol) and theophylline.[25] The decision to use extracorporeal techniques must be made in consultation with a nephrologist.[25]

Antidotes

Despite popular misconceptions, the administration of an antidote is indicated in only the minority of poisoning cases.[4] Basic supportive and symptomatic measures might be all that is needed with the possible addition of gastric decontamination or methods to increase elimination.[10]

The administration of an antidote must only be considered if the identity of the toxin has been confirmed.[4] The clinician should be aware of the specific indications and contra-indications associated with the administration of the antidote.[10] Table 2 lists the most common antidotes, possible indications and current availability in South Africa (Personal communication: Dr. DJH Veale; December 2010). The availability of certain antidotes remains a problem in the South African public healthcare sector and treating physicians should focus on providing optimal supportive care.[26]

Pearls and pitfalls of managing poisonings and respective antidotes

Paracetamol. Paracetamol is the most common medicinal overdose reported to PICs.[27] The Rumack–Matthew nomogram can be used for predicting the potential for hepatotoxicity and serves as a guide to whether antidotal therapy is necessary.[28] Interpretation of the nomogram is meaningless in chronic ingestions or for blood levels drawn less than 4 h post ingestion or 24 h post ingestion. Patients that are malnourished or have induced cytochrome p-450 enzymes (e.g. alcoholics or those taking enzyme-inducing drugs concurrently) are regarded at high risk for development of hepatotoxicity and the lower ''high risk'' treatment line should be used as the guide to the necessity for antidote administration.[27] N-acetylcysteine remains the cornerstone of therapy and can be administered intravenously or orally.[29] Superiority of N-acetylcysteine to methionine is unclear, but N-acetylcysteine seems superior to other antidotes (dimercaprol, carbocisteine, cysteamine).[30] Prolonged treatment with N-acetylcysteine for 2–3 days may be of benefit in patients who are seriously poisoned and where antidotal therapy was started late.[11]

Opioids. Opioid poisoning may be reversed with naloxone, an opioid receptor antagonist. The potential for precipitating acute withdrawal in addicts is often detrimental and unpredictable; therefore the goal should not be complete arousal, but just enough to alleviate respiratory depression.[31] Administration of naloxone should be via the intravenous route in order to facilitate better dose titration. Patients should be monitored continuously as the half-life of naloxone is very short in comparison to that of the opioid drugs and resedation often occurs.[31]

Socio-economic factors:
- Male
- Elderly
- Recent loss of life partner
- Unemployed

Medical history:
- Depression
- Terminal illness

Planned suicide:
- Evidence of planning of overdose
- Suicide note
- Precautions against discovery

Box 6 High risk factors for further self harm.

Cholinesterase Inhibitors (Organophosphate and Carbamate insecticides). Atropine is used to control the excessive muscarinic receptor stimulation. Different treatment regimes exist,[32] but we suggest boluses of 0.05 mg/kg (max 4 mg) every 15 min until control of excessive pulmonary secretions is evident.[11] Large doses of atropine might be needed. Maintenance therapy of atropine (intermittent or continuous infusions) may be given at a rate of 0.05 mg/kg/h; titrate as needed using lung sounds and oxygenation as endpoints.[11] Tachycardia is not a contraindication for atropine but a urinary catheter should be inserted to prevent urinary retention. As the patient improves, the atropine dose should be tapered off slowly over at least 24 h with close observation as rebound effects of the poisoning may occur.[11]

The addition of glycopyrrolate (which does not cross the blood brain barrier) to the atropine regimen can be considered when atropine-induced CNS toxicity becomes evident but the use of atropine is still indicated.[33] Repeated intravenous doses (1–2 mg in adults and 0.025 mg/kg in children) may be required as needed.[33]

Oximes (acetylcholinesterase reactivators such as pralidoxime and obidoxime) are also used in the treatment of acute organophosphate poisoning, but current evidence is insufficient to indicate whether they are harmful or beneficial.[34] However, these drugs are not currently available in South Africa.[26]

Benzodiazepines. Flumazenil (a benzodiazepine antagonist) is only indicated in patients with marked respiratory depression or deep coma e.g. in small children, the elderly and patients with obstructive airway diseases.[11] Extreme care should be taken in benzodiazepine-dependent patients and in patients with a history of epilepsy. Flumazenil is contraindicated in co-ingestions of pro-convulsant drugs or tricyclic antidepressants.[35] Resedation may occur after reversal, therefore patients should be observed carefully.9

Tricyclic antidepressants. Because of the strong protein binding and large volume of distribution of the tricyclic antidepressants, urinary alkalinisation, dialysis or haemoperfusion are ineffective in the management of poisoning.[11] Alkalinisation with sodium bicarbonate is of benefit in convulsing patients and those with signs of cardiotoxicity (ventricular dysrhythmias, QRS duration greater than 0.10 s) and in adequately hydrated patients with persistent hypotension.[11,36] Benzodiazepines are used to treat seizures.[11]

Disposition

Any patient with a suspected poisoning should be admitted to a medical facility for observation.

Patients with intentional self poisoning should undergo a risk assessment for further self harm before discharge. High risk factors (Box 6) should prompt the involvement of the psychiatric team for a full psychiatric evaluation.[37] These patients should always be discharged in the care of a responsible adult.[37] Social support must also be offered to substance abuse patients. This includes assistance with rehabilitation.

Conclusion

This document proposes concise guidelines for the initial management of the acutely poisoned patient in the Emergency Centre. The history and physical examination are important in the identification of the toxin/s involved. Supportive and symptomatic care remains the mainstay of treatment in poisoning cases. A stepwise approach may be followed to decrease the bioavailability of toxins.

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