Consensus Guideline:

Treatment of Poisoning and Self-harm in Afghanistan Hospitals

CG002

General Directorate of Curative Medicine
Ministry of Public Health,
Kabul, Afghanistan

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FOREWORD

Excellence in health practice depends on the quality of care provided to patients and their relatives. The Ministry of Public Health of Afghanistan is committed to advance medical science and to provide the best quality care for those who are using our services. This is a demanding task as the management of each condition, especially in emergency medicine, is unique and varying according to experience and knowledge of health practitioners. As a result, probably, many different approaches are practiced in our emergency rooms. This variation in making clinical decision can sometimes be confusing both to patients and the clinical staff. Clinical guidelines are used to reduce this variation and help patients, their carers and clinicians to make decisions that are clear, based on evidence and most importantly suit individual patients. I am pleased that a dedicated Workgroup under the General Directorate of Curative Medicine managed to compile this guideline for the management of poisoning and self-harm that fulfil the said requirements. This guideline describes good clinical practice and setting standards of care at pre-hospital and hospital levels for treatment of common poisoning and self-harm conditions.

This guideline has drawn the experience of 8 policy experts and 75 senior clinicians, integrated the experience of the staff from Ibnisina Emergency Hospital, and used information from renowned international clinical guidelines. The guideline has gone through extensive public consultation, peer reviewed, discussed at Technical Advisory Board (TAG) and finally approved by the Executive Board of the Ministry of Public Health for practice. This lengthy and consultative approach has made this guideline valid, reliable and useable for our situation.

I recommend that this guideline should be used as a benchmark when providing emergency medical care in poisoning and self-harm conditions in emergency rooms and set standards of practice both at public and private hospitals across the country. It should also be considered as a key reference when managing such patients either in the ambulance or Intensive Care Units.

I would like to acknowledge the excellent contributions of the workgroup and critical reviewers and thank them for their invaluable hard work and dedication for the development of this essential guideline.

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Poisoning, drug overdose and self-harm are medical emergencies that cause major concerns for health service providers worldwide. Self-poisoning with pesticides alone accounted for around a third of all suicides in the world. It is estimated that there are 258,234 (ranging from 233,997 to 325,907) deaths from pesticide self-poisoning alone worldwide each year (Gunnel, et al, 2007). In India about 10 -15 million cases of poisoning are reported annually of which more than 50,000 die (Aggarwal, 2004). Poisons were used in 39% of the 306 reported suicides in Pakistan from 1996-1997 where the organophosphorus pesticides and other agricultural chemicals were the main poisons used. This assertion is supported by findings by Agha Khan University Hospital in Karachi, Pakistan indicating that from 1989–1995 organophosphorus pesticide poisoning accounted for 21% of self-harm admissions. Data for poisoning (deliberate and accidental) in Iran support the view that pesticides are likely to make an important contribution to mortality from suicide in that country (Gunnel, et al, 2007).

The prevalence of poisoning and self-harm in Afghanistan is not known. There is no local data to inform us about the magnitude of this problem in Afghanistan. IbnSina Emergency Hospital in Kabul registered 3,567 out of 67,201 cases of “poisoning” which constituted 5.3% of all patients attended and registered in 18 months period from 21st March 2011 to 21st November 2012. The author of this document observed three cases of poisoning in a short visit: two suicide attempts and one severe alcohol poisoning.

From the medical records of the IbnSina Emergency Hospital it is not possible to know the types of poisons and drugs involved in ‘poisoning’ cases. Anecdotal evidence from key informants and clinicians in this hospital suggest that rodenticides, malathion, paracetamol, carbon monoxide and sedative drugs are the most common causes of accidental or intentional poisoning.

Mortality from poisons, drug overdose and self-harm is also not known in Afghanistan. The medical record of IbnSina Emergency Hospital in Kabul showed that 449 patients died either on arrival to, or in the Intensive Care Unit (ICU) and recovery rooms of the IbnSina Emergency Hospital in the above 18 months. The causes of their death were not known and one can reasonably suggest that at least some of these mortalities could be related to poisoning and self-harm that came to the attention of services. During the compilation of this document in the month of November 2012 alone seven patients died due to “poisoning” in IbnSina Emergency Hospital. The author became aware of two losses due to poisoning in a short visit, in one morning, in November 2012 when visited IbnSina Emergency Hospital: one due to carbon monoxide poisoning and the other was reported intentional drug overdose.

Mortality from poison and self-harm in the community and society at large is not registered. The prevalence of deliberate self-harm is also not certain, as mentioned, in Afghanistan. The evidence, at least from the media especially from Herat province in the recent years, shows that cases of self-harm in the form of self-burning by women are on the rise. Therefore, based on these observation and anecdotal evidence one can hypothesize that cases of poisoning and self-harm are: a) under reported in the country and b) secondly presents one of the major healthcare challenges in Afghanistan.

To tackle the problem of drug overdose and poisoning, the World Health Organisation (WHO) announced a Global Public Health Initiative in 2005. Planned approaches ranged from government regulatory action to the development of new treatments for pesticide poisoning (Bertolote, 2006). This Initiative has not been initiated in Afghanistan yet. Patients with accidental or intentional poisoning are usually assessed and managed in the emergency rooms. Therefore, the emergency services should, ideally, be well equipped to manage such cases. Although some tangible progress has been made in other areas of medicine and health services in Afghanistan, accident and emergency medicine, unfortunately, left behind. For instance, curricula and training schemes have been developed for 21 medical specialties in the Ministry of Public Health, but there is no curriculum, training scheme or a
single accident and emergency specialist exist in the country. Worse than that, there are no permanent members of medical teams based in the emergency departments. Emergency rooms are managed on on-call basis, mostly by junior doctors as part of their training rotation from various branches of medicine, most of the time without direct supervision by senior staff, if not all the time.

On the other hand, Afghanistan is experiencing rapid technological development in all areas of science; including medicine, agriculture and pest control in the last decade. New drugs, household chemicals and pesticides are rapidly introduced in the country. Accidental or intentional poisoning especially in children, farmers, and vulnerable groups due to access to new drugs and chemicals can cause complex and life threatening medical emergencies that require complex bio-psycho-social management.

For these reasons (increasing number of cases of poisoning attending the Emergency Departments, lack of trained accident and emergency specialists in the country, lack of permanent medical staff in the emergency departments, introduction of new drugs, antidotes and treatments in the recent years and the uncertainty about management of poisoning), it is vital to provide doctors, nurses and emergency managers in Afghanistan with clear guidance in the management of poisoning and self-harm in order to provide better care. A survey of guideline practice amongst clinicians was carried out in Kabul Hospitals in the summer of 2012 when and the majority of respondents requested the development of a clinical guideline in emergency medicine, especially in poisoning, as one of their priorities (Rahimi, 2012). This request was indeed compatible with the priorities of MoPH as it outlined in the MoPH strategic plan 2011-2015 (MoPH, 2011).

Following discussions with a number of experts in this field, various stakeholders and policy makers, the General Directorate of Curative Medicine (GDCM) of the Ministry of Public Health (MoPH) decided to develop this guideline. A Guideline Development Group (GDG) was formed to draft this document. A scoping exercise suggested that this guideline should contain some general statements in the management of poisoning and the most prevalent drugs and poisons used in Afghanistan. In the absence of reliable data from within the country to guide clinicians about the most effective management of patients with acute poisoning and deliberate self-harm, this document was developed as a ‘consensus guideline’ by the GDG according to the needs of the country, suggestions made by clinicians on the ground and international literature. The draft version of the guideline was peer reviewed by a group of professionals from Emergency Departments, Kabul Ambulance Services, senior clinicians from various governmental and private hospitals and other stakeholders in a two days workshop. Recommendations of the workshop participants were included in the guideline. Final product was presented before the Technical Advisory Group (TAG) of MoPH and their recommendations were incorporated in the guideline. The guideline was approved by the Executive Board of MoPH in the winter of 2013 for implementation.

This guideline should be read in line with the ‘Administrative Guideline for Emergency Departments, 2012’ (CG003) which was simultaneously developed at the time of the development of this guideline at the GDCM. This document explains the roles, responsibilities, functions, basic infrastructure, emergency processes, staffing, drugs and equipment required for an Emergency Department.
This guideline is intended to cover some general statements in the assessment and management of poisoning and the management of specific drugs and poisons caused accidental or intentional poisoning in Afghanistan. This topic was chosen by the General Directorate of Curative Medicine for reasons outlined in part 1 above, the introduction. This guideline covers basic management of poisoning for all ages until further specific guidelines are developed for children, old age and other vulnerable groups. It is particularly aimed at clinicians working in Emergency Departments and ambulance services who manage such patients in the first instance after poisoning or self-harm. But, it can be used by all health professionals who deal with patients who are either poisoned or have self-harmed.
3. GENERAL CONSIDERATION IN POISONING AND SELF-HARM

Poisons are substances that when introduced into or absorbed by a living organism cause illness or death (Oxford Dictionary). Poisoning occurs when any substance interferes with normal body functions after it is swallowed, inhaled, injected, or absorbed.

The term self-harm is defined as ‘self-poisoning or injury, irrespective of the apparent purpose of the act’. Self-harm is an expression of personal distress, not an illness, and there are many varied reasons for a person to harm himself or herself. People who have self-harmed should be treated with the same care, respect and privacy as any other patient. In addition, healthcare professionals should take full account of the likely distress associated with self-harm (NICE, 2004).

Patients who have features of poisoning especially children and young adults should generally be admitted to hospital. Patients who have taken poisons with delayed action should also be admitted even if they appear well. Delayed action poisons include aspirin, iron, paracetamol, tricyclic antidepressants and modified release preparations. In emergency cases generally and in poisoning and self-harm in particular, clarity of information and clear communication are the key important issues. Information from clinicians, the patient’s notes or files, including what treatment has been given should accompany the patient when transferred between hospitals and wards at all time. Detailed hand over should take place when patients are transferred from ward to ward or hospital to hospital by transferring agent to receiving end.
4. INITIAL ASSESSMENT BY AMBULANCE SERVICES

Ambulance crews are the first who may come to contact with patients who have ingested poison or self-harmed. Therefore, they have a crucial role in the assessment of patients in their environment and early management. For detailed assessment and management of medical emergencies by ambulance staffs please refer to separate guideline on ambulance services. Generally, assessment should cover the following areas in an atmosphere of respect and understanding:

a) The method of self-harm and collecting the evidence for poisoning, e.g. all substances or medications found at the scene of event and pass these to staff upon arrival at the emergency department.

b) If urgent transfer of patient to emergency department is not required, ambulance staff should record relevant information about the patient’s home environment, social circumstances, and history leading to poisoning or self-harm. This information should be passed on to the emergency department staff before or soon after arrival.

c) Ambulance staff should be able to carry out a quick assessment of patient’s physical and mental state at the scene and document them for hand over to emergency staff.

d) Ambulance staff should be able to perform cardiopulmonary resuscitation (CPR) if needed at the scene of event and make sure that the airway is open during transportation of patient to the emergency department.

e) Ambulance staff should offer Activated Charcoal to patients in the following circumstances:
   i) when transporting the patient to the emergency department within one hour of overdose,
   ii) if the patient is considered at risk of serious harm,
   iii) is fully conscious,
   iv) is able to protect his/her own airway and
   v) when expecting a long journey either due to busy traffic or transferring patient from remote areas to the nearest district or provincial hospital to get to the emergency department.

f) Activated charcoal should be offered only when the substances ingested are likely to be absorbed by activated charcoal. A list of these substances is stated in annex 2.

g) In cases where the patient does not require emergency treatment in the emergency department, ambulance staff should consider taking the patient to an alternative appropriate service, such as a mental health hospital or clinic. After arrival at the emergency department ambulance crew should hand over the patient to the triage nurse and get the handover book signed by the head nurse or the manager of emergency department.
5. MANAGEMENT OF POISONING AND SELF-HARM AT THE EMERGENCY DEPARTMENTS

When patients with an episode of poisoning or self-harm arrive at the emergency department they should be assessed for risks of physical deterioration and mental health problems in an environment of respect and understanding. The care pathway of the case should be clearly defined after first assessment.

5.1 Triage

Triage is a formal process of immediate assessment of all patients who present to Emergency Departments especially when many patients present simultaneously. The process is used to ensure that patients receive emergency care according to their clinical needs rather than order of arrival, social status, power or wealth. Categorization of patients all into different levels of priority according to their presenting is beyond the scope of this guideline. A separate guideline needs to be developed for this purpose. It is worth emphasizing that according to our observation of the emergency departments in Kabul, developing a guideline for triage education and practice should be one of the priorities of the emergency departments and the Ministry of Public Health.

Triage assessment is usually carried out by an emergency triage nurse. Patients with poisoning and self-harm are classified into groups according to the degree of their physical and mental health conditions and the type and time of ingestion. Triage staff must make sure that patients with life threatening conditions or risk of physical health and mental health deterioration receive immediate intervention without delay.

5.1.1 In the first instance the patient should be assessed by the triage nurse or doctor without delay for the severity of poisoning, risk of physical health deterioration and their mental state.

5.1.2 If decided that the patient needs immediate medical attention, the medical team should be alerted and the patient directed to the medical emergency assessment and treatment room. The medical assessment and treatment should start without delay.

5.1.3 If the patient was physically stable on arrival, or when the patient becomes physically stable after treatment and the presence of mental health difficulties is suspected, a psychiatric examination should be arranged prior to discharge. Emergency rooms should have access to a psychiatrist 24 hours a day and seven days a week. The psychiatrist should be available for assessment before the patient is discharged during working hours and be available for consultation during the night. Triage staff themselves (whether a doctor or a nurse) should be trained in brief psychiatric assessment and be aware of the appropriate referral system to mental health units or hospital.

5.1.4 If surgical intervention is required due to self-injury the emergency surgery team should be alerted and the patient is directed to the surgical emergency assessment and treatment unit.
6. **Medical Management Of Poisoning And Self-Harm**

The main objective of medical treatment in poisoning and drug overdose should be:

- Prevention of absorption
- Active elimination
- Reducing biological effects of the poison

Superficial and uncomplicated self-injuries may need minor intervention in the emergency surgical unit whilst complicated injuries may need complex surgical intervention. Rapid action, an organized, systematic approach and team work in the assessment and treatment of poisoned patients are key aspects of clinical management. However, when dealing with poisoning or overdose, remember to treat the patient not the poison. Key principles are illustrated in Fig. 1. This algorithm is only a guide and by no means replaces a clinician’s judgment when treating patients.

6.1 Due to lack of toxicology laboratories in Emergency Rooms in Afghanistan at the moment, it is impossible to establish the identity of the poison and the size of overdose. The emergency medical team usually has to rely on the history from the patient and their relatives, ambulance staff; the evidence from the scene if available and their own assessment of patient’s physical and mental states. Even in developed countries it is often impossible to establish with certainty the exact identity and the dose of poison. This is not usually important because only a few poisons such as opioids, paracetamol, and iron have specific antidotes. In the majority of cases treatment of poisoning and self-harm is symptomatic. However, knowledge of the type and the time of poisoning can help physicians in anticipating the course of illness. Therefore, detailed history taking and examinations are vital in the management of poisoning. The information gathered should be interpreted with great caution because it may not be complete or entirely reliable. Patients may present with other underlying illness (es) that confuse the picture and should be assessed with great care.
Fig. 1. Management of poisoned patients. Adapted from Goldfrank, 2007

1. Difficulty breathing?
   - Yes
   - Clear the airway, lift the jaw and start
     - Yes
     - Treat the syndrome
   - No
   - Monitor oxygen level by pulse oximetry
     - Yes
     - Are life threatening abnormalities present?
       - Yes
       - Consider administration of:
         - Hypertonic dextrose
         - Thiamine
         - Naloxone
       - No
       - Consider emergency management of seizure, psychomotor agitation, cardiac arrhythmias and metabolic abnormality
     - No
     - Take detailed and corroborative history, perform thorough physical examination and blood investigation

2. Identified a specific toxic
   - Treat the syndrome

3. Take relevant history and perform physical examination
   - Consider gastric
   - Consider prevention of absorption of poison:
     - Activated charcoal
     - Whole bowel irrigation

4. Assess for enhanced elimination:
   - Multi-dose Activated Charcoal

5. Assess for:
   - ICU admission or to continue treatment in the emergency department
   - Psychiatric assessment or referral
   - Risk of repeated overdose or poisoning before discharge
7. GENERAL CARE IN POISONING AND OVERTDOSE

7.1 Respiration
Respiration is often impaired in unconscious patients. An obstructed airway requires immediate attention. Emergency staff should be trained in first aid, cardiopulmonary resuscitation (CPR) techniques and the ABC of life supports at the start of their jobs and then receive refresher courses on a regular basis, preferably every 6 months. If there is no neck injury the airway should be opened with chin lift and positioning the patient. Intubation and ventilation should be considered in patients whose airway cannot be protected or who have respiratory acidosis because of inadequate ventilation. Such patients should be cared for in the Intensive Care Unit (ICU). Most poisons that impair consciousness also depress respiration. Assisted ventilation (e.g. mouth to mouth) may be needed. Oxygen is not a substitute for ventilation, although it should be given in the highest concentration possible in carbon monoxide and irritant gas poisoning.

7.2 Blood pressure
Severe poisoning can cause central nervous system depression which commonly causes hypotension. A systolic blood pressure less than 70 mmHg may lead to irreversible brain damage or renal tubular necrosis. Hypotension should be corrected initially by raising the foot of the bed and/or administration of an infusion of either sodium chloride or colloid solution. Vasoconstrictor sympathomimetics are rarely required.

7.3 Heart
Cardiac conduction defects and arrhythmias can occur in acute poisoning, particularly with tricyclic antidepressants, some antipsychotics and some antihistamines. Arrhythmias often respond to correction of underlying hypoxia, acidosis, or other biochemical abnormalities. But ventricular arrhythmias that cause serious hypotension require treatment with anti-arrhythmic drugs. If there are ECG changes (QT prolongation) special advice should be sought from a cardiologist. Use of some anti-arrhythmic or major tranquilizer drugs may be inappropriate in such conditions.

7.4 Body temperature
Both hypothermia and hyperthermia require urgent hospital admission for assessment and supportive treatment in the cases of poisoning. Hypothermia may develop after overdose with barbiturates or phenothiazines. It may be missed unless core temperature is measured using a low reading rectal thermometer. Hypothermia should be managed by prevention of further heat loss and appropriately re-warming.

7.5 Convulsion
Single short-lived convulsions do not require treatment. If convulsions recur frequently, lorazepam 4mg or diazepam emulsion 10mg should be given by slow IV injection, at a rate of 1mL (5mg) per minute, into a large vein, repeated once after 10 minutes if necessary. When administering benzodiazepines, facilities for reversing respiratory depression with mechanical ventilation must be immediately available. Benzodiazepines should not be given by IM route for convulsions, but can be used only when oral or IV routes are not available. If the IV route is not readily available diazepam can be administered as a rectal solution, 10-20 mg, repeated once after 10-15 minutes if necessary.

7.6 Unconscious patients
Assessment of conscious level is an essential component of the neurological examination of poisoned patients. Terms such as ‘stuporose’ and ‘semi-consciousness’ are ill defined and a clear description of the patient’s level of arousal and response to stimuli is more helpful. Systematic assessment of unconscious patient by the application of Glasgow Coma Scale (GCS) provides a grading of coma by using a numerical scale which allows serial comparison (Davidson’s, 1999). The scale assesses eye
opening, verbal response and motor responses to motor stimulation, with scores ranging from 3 to 15. GCS of 8 or less is considered sever brain injury. Scores between 9 – 12 is considered moderate brain injury and GCS 13 or greater shows mild brain injury (Grant and Adams, 2009).

Defining patient’s level of consciousness helps to make decision about the level of care needed for the management of poisoned patients at every step of the patient flow. We recommend the use of GCS which is simple, quick and easy as a tool to all ICU emergency department staff. (See GCS in Box 1).

Box 1. Glasgow Coma Scale (GCS: Teasdale & Jennett. 1974)

<table>
<thead>
<tr>
<th>Glasgow Coma Skill (GCS)</th>
<th>Response:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td></td>
</tr>
<tr>
<td>Open eyes spontaneously</td>
<td>4</td>
</tr>
<tr>
<td>Opens eyes in response to speech</td>
<td>3</td>
</tr>
<tr>
<td>Open eyes in response to painful stimulation</td>
<td>2</td>
</tr>
<tr>
<td>Does not open eyes in response to any stimulation</td>
<td>1</td>
</tr>
<tr>
<td><strong>Motor response</strong></td>
<td></td>
</tr>
<tr>
<td>Follows commands</td>
<td>6</td>
</tr>
<tr>
<td>Makes localized movement in response to painful stimulation</td>
<td>5</td>
</tr>
<tr>
<td>Makes no purposeful movement in response to noxious stimulation</td>
<td>4</td>
</tr>
<tr>
<td>Flexes upper extremities/extends lower in response to pain</td>
<td>3</td>
</tr>
<tr>
<td>Extends all extremities in response to pain</td>
<td>2</td>
</tr>
<tr>
<td>Makes no response to noxious stimuli</td>
<td>1</td>
</tr>
<tr>
<td><strong>Verbal response</strong></td>
<td></td>
</tr>
<tr>
<td>Is oriented to place, person and time</td>
<td>5</td>
</tr>
<tr>
<td>Converses, may be confused</td>
<td>4</td>
</tr>
<tr>
<td>Replies with inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Makes incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>Makes no response</td>
<td>1</td>
</tr>
</tbody>
</table>

Mild 13-15, Moderate 9-12, Severe 3-8

7.6.1 Emergency staff should also be familiar with and receive regular training in basic and advanced life support skills both for children and adults. For further details and guidance please refer to life support guideline currently under development at the Department of Curative Medicine of the MoPH. For quick reference guide the adult basic and advanced life support
flowcharts introduced in Fig. 2 and Fig. 3: **Adult Basic Life Support**

- **Unresponsive?**
  - **Shout for help**
  - **Open airway**
  - **Not breathing normally?**
    - **Call for Crash Team**
    - **30 chest compression**
    - **2 rescue breath
      30 chest compression**
Advanced Life Support

Unresponsive? Not breathing or only occasional gasps

Call Resuscitation Team

CPR 30:2 Attach defibrillator/ monitor Minimise interruptions

Assess rhythm

Shockable (VF/Pulseless VT)

1 Shock

Immediately resume: CPR for 2 min Minimise interruptions

Non-shockable (PEA/Asystole)

Return of Spontaneous Circulation

Immediately resume: CPR for 2 min Minimise interruptions

IMMEDIATE POST CARDIAC ARREST TREATMENT
- Use ABCDE approach
- Controlled oxygenation and ventilation
- 12-lead ECG
- Treat precipitating cause
- Temperature control/ Therapeutic hypothermia

During CPR
- Ensure high-quality CPR: rate, depth, recoil
- Plan actions before interrupting CPR
- Give oxygen
- Consider advanced airway and caprography
- Continuous chest compressions when advanced airway in place
- Give adrenaline every 3-5 min
- Connect reversible causes

Reversible Causes
- Hypoxia
- Hypovolaemia
- Hypo-/ hyperkalaemia/ metabolic
- Hypothermia
- Thrombosis - coronary or pulmonary
- Tamponade - cardiac
- Toxins
- Tension pneumothorax
8. PREVENTION OF ABSORPTION OF POISON

8.1 Decontamination
Decontamination can be gastrointestinal, topical or respiratory depending on the route of poisoning and the content of the poison. The methods should be carried out promptly to limit the effect of the poison.

8.1.1 Gastrointestinal decontamination is used only for ingested poisons by trained members of staff at the Emergency Department. The gastrointestinal decontamination techniques are used are: dilution, emesis, gastric lavage, catharsis, intestinal lavage, whole bowel irrigation and gastrostomy (a surgical opening into the stomach).

When gastrointestinal decontamination is considered the following criteria should be taken into account:

- a) When the patient present early, within 1-2 hours, post-overdose
- b) When a patient is fully conscious
- c) The airway is fully open and protected
- d) When the patient is at risk of significant harm due to overdose.
- e) If all of the above criteria are met, activated charcoal should be offered if the poison or drugs ingested are likely to be absorbed by activated charcoal (see annex 2).

8.2. Gastric Lavage
Gastric lavage attempts to directly remove stomach contents by means of an oro gastric tube.

8.2.1 Indication:
- h) Ingestion of a substance with high toxic potential within 1 hour of ingestion
- i) Ingested substance is not bound by activated charcoal or has no effective antidote
- j) Potential benefits outweigh risks

8.2.2 Contraindication
- k) Substance does not meet above indications
- l) Spontaneous emesis
- m) Diminished level of consciousness
- n) Loss of or likeliness of airway protective reflexes (intubate first, once intubated, gastric lavage can be performed if otherwise is not contraindicated.)
- o) Ingestion of hydrocarbons (high risk of aspiration) or caustic agents
- p) Foreign body ingestion
- q) Patient is at high risk of oesophageal or gastric injury (gastrointestinal hemorrhage, recent surgery etc.).

8.2.3 Technique
- h) Recommended tube size is 36-40 French for adults, 22-28 French for children
- i) Secure airway via intubation, if necessary
- j) Position patient in left-lateral decubitus position, with head lowered below level of feet
- k) Confirm tube placement following insertion
- l) Aspirate any available stomach contents
- o) m) Lavage with 250mL (10-15mL/Kg in children) of warm water or saline
- p) Continue until fluid is clear and a minimum of 2L has been used.
q) Instill activated charcoal through same tube.

8.2.4 Complications
The primary risk is vomiting, aspiration, and oesophageal injury or perforation.

8.3 Activated charcoal
For the majority of drugs taken in overdose, taking activated charcoal as early as possible, preferably within 1 hour of ingestion, can prevent or reduce absorption of the drug. Activated charcoal should be immediately available for rapid and appropriate use in the emergency departments at all times. The current practice in the Emergency Departments is that the patients’ relative is given a prescription for activated charcoal and several other items of drugs and equipments for the treatment of their patients to buy from local chemists. It will take some time until the relatives return while the patient is waiting for treatment. This practice wastes the golden time for the treatment of patients in emergency situation. Although it implies some cost, this practice produces unnecessary risk and should be stopped and emergency departments should stock needed medications and equipment.

8.3.1 The usual dose of activated charcoal in ADULT and CHILD over 12 years of age: 50g initially then 50g every 4 hours. Vomiting should be reduced with antiemetic drugs because it reduces the efficacy of charcoal treatment. If not tolerated the dose may be reduced and the frequency increased. For example: 25 mg every 2 hours or 12.5 mg every hour, though this may reduce efficacy. CHILD under 12 years of age activated charcoal is given in a dose of 1 g/kg body weight (maximum 50 mg) every 4 hours. The dose may be reduced if not tolerated.

8.3.2 All healthcare professionals who are able to offer activated charcoal to people who have been poisoned should ensure that they know how and when this should be administered. This should include:

f) knowing for which poisons activated charcoal should and should not be used,
g) the potential dangers and contraindications of giving activated charcoal,
h) The skill to encourage and support patients when offering activated charcoal.
i) Explaining the reason for treatment with activated charcoal to the patient.

8.3.3 The evidence for the following interventions in treating poisoning and self-poisoning is weak and can cause serious side effects. Avoid them unless it is absolutely necessary:

q) Multiple use of activated charcoal
r) Emetics, including ipecac (ipecacuanha)
s) Cathartics
t) Gastric lavage unless it is recommended by a senior consultant
u) Whole bowel irrigation

8.4 Collecting samples and interpreting the results
Collecting appropriate sample for laboratory test should be ordered as early as possible. This could be a blood, urine, vomit or gastric contents. When the emergency staff is unsure about the interpretation of results, they should contact the senior medical staff for advice before acting upon the result.
9. **Specific Drugs**

9.1 Paracetamol (acetaminophen) overdose

Paracetamol overdose can result in liver damage which may be fatal. It is rapidly absorbed from the small intestine. In tablet and capsule forms peak serum concentration occur within 1-2 hours and 30 minutes in liquid preparation. Massive paracetamol overdoses causing high serum levels that may cause decreased consciousness, hypoglycemia and hepatic failure. For more information on patients at high risk of liver damage see Box 2.

Patients at high risk of liver damage include those
- Taking liver enzyme inducing drugs such as carbamazepine, phenobarbital, phynetoin, primidone, rifampicin, alcohol, etc.
- Who are malnourished e.g. anorexia nervosa, cystic fibrosis, hepatitis C, HIV positive, etc.
- Who have not eaten for a few days.

These patients can develop toxicity at lower plasma-paracetamol concentration and should be treated if their plasma level is on or above the treatment line.

**Box 2: Risk factors for liver damage.**

**Presentation**

Apart from mild nausea, vomiting, and loss of appetite, patients presenting within 24 h of ingestion are asymptomatic.

Hepatic necrosis becomes apparent in 24-36h with right subchondral pain/tenderness, jaundice (and acute liver failure), vomiting and confusion.

Encephalopathy may worsen over the next 72h.

Oliguria and renal failure.

Lactic acidosis: either <12h (very rare) or late (10% of patients with acute liver failure).

**Complications**

- Acute liver failure with hypoglycemia, cerebral oedema, and gastrointestinal bleeding.
- Severe metabolic (lactic) acidosis.
- Pancreatitis (alone or with liver failure).
- Some 10% of patients develop acute renal failure from acute tubular necrosis which may be seen in the absence of liver failure.
- Very rarely patients with G6PD deficiency develop methaemoglobinemia and hemolysis.

(Ramrakha, 2013)

Although the measurement of paracetamol serum level is currently not routinely available in Afghanistan, however it is important to have some knowledge about the paracetamol serum level and the time of ingestion which is illustrated by a nomogram in table 1. This nomogram with a single treatment line was recently replaced the older version, in order to simplify treatment decisions regardless of the risk of hepatotoxicity, minimize allergic reactions and reduce prescribing error (MHRA, 2012).
Table 1. New serum paracetamol level nomogram introduced following and evidence base review by the Commission on Human Medicines (CHM), UK, 2012.

![Graph](image)

**Time (hours)**

Patients whose plasma-paracetamol concentrations are on or above the treatment line should be treated with acetylcysteine by intravenous infusion.

The prognostic accuracy after 8 hours is uncertain, but a plasma-paracetamol concentration on or above the treatment line should be regarded as carrying a serious risk of liver damage.

Graph reproduced courtesy of Medicines and Healthcare products Regulatory Agency

9.1.1 Management of paracetamol overdose

9.1.1.1 Administration of activated charcoal should be considered if paracetamol in excess of 12g or 150mg/kg, whichever is the smaller, or in excess of 75mg/kg for those who considered at high risk is thought to be ingested in the past 1–2 hours. Other criteria listed above should also be taken into consideration before administering activated charcoal. However, activated charcoal alone is not a life-saving treatment in the paracetamol overdose.

9.1.1.2 Acetylcysteine is the antidote of choice to treat paracetamol overdose. If administered intravenously, within 8 hours, it is virtually 100% effective in preventing the liver damage (MHRA, UK, 2012). Beyond 8 – 10 hours after ingestion, efficacy decreases with increasing delay to treatment.
(Daly, 2008). Acetylcysteine is an effective antidote and protects liver if infused up to and possibly beyond 24 hours of paracetamol ingestion.

In Afghanistan considering that the plasma-paracetamol level cannot be measured, for the time being, acetylcysteine should be administered to all patients judged to be at risk of developing hepatotoxicity (see box above) after paracetamol overdose.

9.1.1.3 When plasma-paracetamol level can be measured, patients whose plasma paracetamol concentrations are above the normal treatment line should be treated with acetylcysteine by IV infusion.

9.1.1.4 Dose and administration: Infusion of acetylcysteine is carried out in 3 stages, ADULT and CHILD over 12 years:

- **Initial infusion**: An initial dose of 150mg/kg (max 16.5g) of acetylcysteine diluted in 200mL of 5% glucose and infused over 15 to 60 minute.
- **Second infusion**: Initial infusion is followed by a continuous infusion of 50mg/kg (max 5.5g) of acetylcysteine in 500 mL of 5% glucose over the next 4 hours
- **Third infusion**: Second infusion is followed by a continuous infusion of 100mg/kg (max 11g) of acetylcysteine in 1000 mL of 5% glucose over the next 16 hours.

9.1.1.5 CHILD under 12 years if body weight is over 20kg, initially 100mL, given over 15 minutes, then 250mL over 4 hours, then 500mL over 16 hours. CHILD with body weight under 20kg initially 3mL/kg given over 15 minutes, then 7mL/kg over 4 hours, then 14mL/kg over 16 hours.

- **9.1.1.6** 5% Glucose is preferred infusion fluid; 0.9% sodium chloride is an alternative if 5% glucose is unsuitable.

- **9.1.1.7 Side effects of acetylcysteine**: Acetylcysteine can cause hypersensitivity-like (anaphylactoid) reactions that are manifested by rash, wheeze or mild hypotension and occurs in 10%–50% of patients during the first two acetylcysteine infusions (Daly et al, 2004).

- **9.1.1.8 Management of side effects** is supportive, with temporary halting or slowing of the infusion, and administration of antihistamines (Prescott, et al. 1977). Severe life-threatening reactions are very rare, but may occur in predisposed individuals, such as patients with asthma.

9.1.2 **Methionine**

In remote areas if acetylcysteine is not available or cannot be given immediately, then methionine by mouth is an alternative provided that the overdose has been taken within 10-12 hours and the patient is not vomiting. Once the patient arrives in hospital the need to continue treatment with the antidote will be decided by emergency senior medical team.

9.1.2.1 **Dose**: ADULT and CHILD over 6 years initially 2.5 g, followed by 3 further doses of 2.5 g every 4 hours. CHILD under 6 years initially 1g, followed by 3 further doses of 1g every 4 hours.

9.1.2.2 **If the time of ingestion is known**, The Australian and New Zealand guideline for the management of paracetamol poisoning produced an easy to follow and step by step pathway in the management of paracetamol overdoses that is reproduced here in Fig. 4.
ALT = alanine aminotransferase. NAC=N-acetylcysteine. OD=overdose.
This flow chart is applicable only if the treating clinician is confident of an accurate time of ingestion.

*Co-operative adults who have potentially ingested greater than 10g or 200mg/kg, whichever is less
9.1.3 Investigations

Recommended investigations according to time from paracetamol ingestion to acetylcysteine treatment is summarized in Box 3.

Box 3. Investigations in paracetamol overdose

<table>
<thead>
<tr>
<th>Test</th>
<th>Time after paracetamol ingestion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1-8 hours</td>
</tr>
<tr>
<td>Serum paracetamol</td>
<td>At 4 hours or as soon thereafter as possible</td>
</tr>
<tr>
<td>Transaminases (ALT/AST)</td>
<td>On admission and end of acetylcysteine infusion</td>
</tr>
<tr>
<td>INR/prothrombin time</td>
<td>On admission</td>
</tr>
<tr>
<td>Creatinine and urea</td>
<td>On admission</td>
</tr>
<tr>
<td>Glucose</td>
<td>On admission</td>
</tr>
<tr>
<td>Arterial blood pressure</td>
<td>On admission</td>
</tr>
</tbody>
</table>

Adapted from ‘Frank F S Daly et al: Guidelines for the management of paracetamol poisoning in Australia and New Zealand, 2008’

9.2 OPIOID OVERDOSE

Opioids (narcotic analgesics) cause coma, respiratory depression, and pinpoint pupils. The specific antidote naloxone is indicated if there is coma or bradypnoea.

9.2.1 Naloxone Hydrochloride

Naloxone is an opioid receptor antagonist used in the treatment of opioid overdose when there is impaired consciousness and/or respiratory depression (see Glasgow Coma Scale in Box 1).

Since naloxone has a shorter duration of action than many opioids close monitoring and repeated injections are necessary according to the respiratory rate and depth of coma. When repeated administration of naloxone is required, it can be given by continuous IV infusion and the rate of infusion is adjusted according to the patient’s vital sign. The effect of some opioids such as buprenorphine, are only partially reversed by naloxone. Methadone has very long duration of action; patients may need to be monitored for long periods following large overdose.

The minimum effective dose of naloxone should be used to reverse respiratory depression. Too much naloxone itself causes side effects such as:

- nausea and vomiting
- anxiety, fear, agitation
- hypotension, hypertension, vascular tachycardia and fibrillation, cardiac arrest
- hyperventilation, dyspnoea, pulmonary oedema
- headache, pupillary dilatation, dizziness
Naloxone precipitates opioid withdrawal symptoms, such as agitation, aggression and violence, in opioid dependent patients. Therefore, naloxone should be given slowly and preparation should be made to deal with possible withdrawal symptoms.

When reversing the effects of long acting opioids such as methadone, consider the use of intravenous infusion of naloxone.

When using naloxone monitoring of vital signs, including oxygen saturation, should be taken routinely until the patient remains conscious with adequate spontaneous respiration for a long period.

9.2.1.1 Dose:  *By IV injection:*
ADULT: 0.4 - 2mg; if no response, repeat at interval of 2-3 minutes to a max. of 10mg (then review diagnosis); further doses may be required if respiratory function deteriorates.
CHILD: 10micrograms/kg; if no response give subsequent dose 100micrograms/kg (then review diagnosis); further doses may be required if respiratory function deteriorate.
*By continuous IV infusion:* using an infusion pump

**ADULT** and **CHILD**: rate adjusted according to response. Initially, rate may be set at 60% of the initial resuscitative IV injection dose per hour. (The initial resuscitative IV injection dose is that which maintained satisfactory ventilation for at least 15 minutes).
Naloxone infusion table: Continuous in 5% glucose or 0.9% sodium chloride diluted to a concentration of up to 200 micrograms/mL and administered via a an infusion pump.
9.3 BENZODIAZEPINE OVERDOSE

Benzodiazepines taken alone can cause drowsiness, ataxia, dysarthria, nystagmus, respiratory depression and coma. Benzodiazepines potentiate the effects of other central nervous system depressants taken at the same time.

9.3.1 Activated charcoal
Activated Charcoal can be given within 1 hour of ingesting a significant quantity of benzodiazepine, provided the patient is awake and the airway is protected.

9.3.2 Admission to ICU
The possibility of admission to ICU should be considered at the earliest opportunity if suspected mixed overdose of benzodiazepines with other drugs especially when patient’s clinical progress is not satisfactory.

9.3.3 Flumazenil
Flumazenil a benzodiazepine antagonist that reverses the central sedative effect of benzodiazepines. Flumazenil has a shorter half life and duration of action than diazepam and midazolam so patients may be re-sedated once the effect of flumazenil is over. Flumazenil should be prescribed and administered only when a positive diagnosis of poisoning with benzodiazepines is made. Flumazenil given cautiously can reduce the need for admission to ICU, endotracheal intubation and artificial ventilation. ‘Caution’ means that patients should receive comprehensive physical examination including the status of respiration and the ability to protect their own airway before considering flumazenil. Extra caution should be taken when using flumazenil in patients with history of epilepsy or receiving long term benzodiazepine treatment for epilepsy (risk of convulsion), benzodiazepine dependency (may precipitate withdrawal symptoms), or taken epileptogenic drugs such as antipsychotics and/or antidepressants. Avoid rapid injection in high risk or anxious patients, following major surgery, head injury, elderly and children.

Flumazenil should be used at smaller (minimum effective) doses in benzodiazepine poisoning, compared to other conditions, and administered slowly to avoid the risk of more serious side effects of flumazenil.

Flumazenil should be prescribed only as far as it is clinically necessary and only by those who have been trained in the use of flumazenil and preparations are made to manage agitation if emerged and preferably resuscitation equipment is immediately available.

9.3.4 Dose

9.3.4.1 ADULT:

The doses of flumazenil mentioned here are recommended by BNF for the use in Intensive Care only. Clinician’s own clinical judgment and above recommendations should guide them in the appropriate use of this drug in Afghanistan.

By IV injection: 300 micrograms over 15 seconds, then 100 micrograms at 60 second intervals if required. Max. total dose 2 mg. If drowsiness recurs 300 microgram by IV injection or 100-400 micrograms/hour by IV infusion, adjusted according to response.

9.3.4.2 Neonate and CHILD:
Flumazenil is not licensed for use in children (BNF for children, 2012). However, in severe cases for reversal of sedative effects of benzodiazepines use cautiously as IV injection over 15 seconds.
**By IV injection:**

**Neonate:** 10 micrograms/kg repeated at 1 minute interval if required.

**Child 1 month - 12 years:** 10 micrograms/kg (max. single dose 200 micrograms), repeated at 1-minute interval if required; max. total dose of 40 micrograms/kg (1mg) or 2mg in ICU).

**Child 12-18 years:** 200 micrograms, repeated at 1-minute intervals if required; max. total dose 1 mg (2 mg in ICU).

**By IV infusion if drowsiness recurs after injection:**

**Neonate:** 2-10 micrograms/kg/hour, adjusted according to response.

**Child 1-month – 18 years:** 2-10 micrograms/kg/hour, adjusted according to response; max. 400 micrograms/hour.

**Administration:** for continuous intravenous infusion, dilute with 5% glucose or 0.9% sodium chloride.
10. Other Poisons

10.1 Cyanides
Cyanide is a mitochondrial toxin that is among the most rapidly lethal poisons known to man. Used in both ancient and modern times as a method of execution, cyanide causes death within minutes to hours of exposure. Though significant cyanide poisoning is uncommon, it must be recognized rapidly to ensure prompt administration of life-saving treatment (HSE, UK).

Causes: The most common cause of cyanide poisoning is domestic fires. Cyanide can be liberated during the combustion of products containing both carbon and nitrogen. These products include wool, silk, polyurethane (insulation/upholstery), plastics, melamine resins (household goods), and synthetic rubber. Vehicular fires can also expose victims to cyanide.

10.1.1 Clinical features
If it is inhaled it can cause nausea, vomiting and weakness.
When ingested it can cause dizziness, rapid respiration, vomiting, flushing, headache, drowsiness, fall in blood pressure, rapid pulse, unconsciousness, convulsion and death.
Contact with cyanide can cause skin burns or epidermal necrosis.

10.1.2 Management of cyanide toxicity
Remove contaminated clothing and rinse contaminated skin thoroughly. Remember ABC of life support.

10.1.3 Oxygen
Oxygen should be administered to patients with cyanide poisoning. The choice of antidote depends on the severity of poisoning, certainty of diagnosis, and the cause.

10.1.4 Perform gastric lavage with 400mL 5% sodium thiosulphate solution. Treat metabolic acidosis if it occurs. Administer activated charcoal. Provide symptomatic and supportive treatment

10.1.4 Dicobalt edetate
Dicobalt edetate is the antidote of choice when there is a strong clinical suspicion of cyanide poisoning. Dicobalt edetate itself is toxic, associated with anaphylactoid reactions, and is potentially fatal if administered in the absence of cyanide poisoning. Due to its toxicity edetate dicobalt must only be used when the diagnosis of poisoning with cyanide is definite and the patient is starting to lose conscious or is unconscious. It must NOT be used as precautionary measure.

Dose: By IV injection, ADULT 300mg over 1 minute (5 minutes if condition is less serious) followed immediately by 50ml of glucose IV infusion 50%. If response is inadequate a second dose of both may be given, but there is a risk of cobalt toxicity.

10.1.5 Hydroxocobalamine
Hydroxocobalamine is also indicated for cyanide poisoning.

Dose: By IV infusion, ADULT 5 g over 15 minutes. A second dose of 5 g can be given over 15 minutes to 2 hours depending on severity of poisoning and patient stability. CHILD under 18 years with body weight of 5 kg and over 70mg/kg (max. 5 g) over 15 minutes. A second dose of 70mg/kg (max. 5 g) can be given over 15 minutes to 2 hours depending on severity of poisoning and patient stability.
11. **Carbon Monoxide Poisoning**

Carbon monoxide poisoning is usually due to inhalation of smoke, car exhaust, or fumes caused by blocked flues or incomplete combustion of fuel gases in confined spaces.

11.1 Management: Immediate treatment of carbon monoxide poisoning is essential. The person should be removed to fresh air, the airway cleared and high flow of 100% oxygen administered through a tight fitting mask with an inflated face seal.

11.2 Artificial respiration should be given as necessary and continued until adequate spontaneous breathing starts, or stopped only after persistent and efficient treatment of cardiac arrest is failed.

11.3 The patient should be admitted to hospital because complications may arise after a delay of hours or days. Cerebral oedema may occur in severe poisoning and is treated with IV infusion of mannitol.

11.4 If patient is pregnant, has been unconscious and develop systemic complications hyperbaric oxygen treatment should be considered in the hospital. These cases should be consulted with specialists in the relevant fields.
12. Rodenticides

Rodenticides, known in Afghanistan as mouse killers (مرگ موش) are a heterogeneous group of compounds that exhibit markedly different toxicities to humans and rodents. They are among the most toxic substances regularly found in homes. Mouse killers are taken accidentally by children or as a means of self-harm and suicide attempt in Afghanistan.

Rodenticides are broadly classified as first generation and second generation rodenticides. The first generation rodenticides used before the mid-20th century were quick acting acute poisons such as heavy metals (arsenic, thallium), zinc phosphate and barium carbonate. Second generation rodenticides include slow acting multiple doses poisons such as rodafarin, rotafin and bromadiolone anticoagulant rodenticides that came to market since the mid-20th century. Warfarin was the initial ingredient in these products, but rodents quickly developed resistance to its effects. Longer-acting, more potent anticoagulants were developed in the 1970s and the term “superwarfarins” was coined to describe them. Currently, most mouse and rat baits available to the public worldwide contain these long-acting anticoagulant rodenticides (LAARs) but in Afghanistan the 1st generation rodenticide are still widely available and sold on the streets in large quantities with no control. The majority of cases with rodenticide poisoning attending the Avicenna Emergency Hospital in the last few years were reported as zinc sulphate poisoning.

12.1 First generation rodenticides

12.2 Long Acting Anticoagulant Rodeaticides (LAARs)

These can be absorbed through the digestive tract, through the lungs, or through skin contact. The substance is a vitamin K antagonist. The lack of vitamin K in the circulatory system reduces blood clotting and will cause death in large dosages due to internal hemorrhaging.

12.3 Clinical presentation

Bleeding is the major risk following ingestion of LAAR compounds. The clinical manifestations after ingestion of LAARs range from being asymptomatic to active bleeding manifested as hematuria, epistaxis (nose bleed), menorrhagia (bleeding from uterine), soft tissue bruising, hemarthrosis (bleeding into joint spaces), anemia, hemoptysis, hematemesis, retroperitoneal or intra-abdominal and intracranial hemorrhage. Patients can require weeks to months of supplemental vitamin K therapy to correct the coagulation defect. Spontaneous intra-abdominal hemorrhage.

12.4 The PT or INR

The PT or INR is the best screening test when performed 48–72 hours after exposure. Coagulation studies are expected to be normal in acute exposures to anticoagulants. These patients may require an International Normalized Ratio (INR) check on a daily basis for a couple days to rule out or confirm toxicity.

12.5 Management

Give all patients with rodentine overdose activated charcoal as soon as possible to prevent further absorption of ingested toxins.

If a coagulopathy is documented, institution of vitamin K therapy is suggested. However, in the absence of documented coagulopathy, prophylactic vitamin K therapy is absolutely contraindicated. This would potentially mask the onset and severity of ingestion and would obscure the time required for clinical and/or laboratory monitoring.
Intentional exposure to an anticoagulant rodenticide for suicidal may require substantial treatment with vitamin K for a protracted period of time, particularly in the face of exposure to one of the superwarfarins.

12.6 Vitamin K\textsuperscript{1} dosage

v) Vitamin K\textsubscript{1} dosage: The optimal dose for vitamin K\textsubscript{1} is unclear. Recommended dosages vary from 0.25-2.5 mg/kg in warfarin (coumarin) exposure, to 2.5-5 mg/kg in the case of long-acting rodenticide intoxication (diphacinone, brodifacoum, bromadiolone). Reported cases of LAAR poisoning have required as much as 50-250 mg of vitamin K\textsubscript{1} daily for weeks to months. Whereas, a single dose of 1-10 mg is usually sufficient in patients who are overanticoagulated with warfarin. A reasonable starting dose for a patient who has intentionally overdosed on either warfarin or a LARR is 25-50 mg of vitamin K\textsubscript{1}, orally 3-4 times daily (due to short-lived duration of action) for 1-2 days.

w) The INR should be monitored and the vitamin K\textsubscript{1} dose adjusted accordingly. Once the INR is <2, a downward titration in the dose of vitamin K\textsubscript{1} can be made on the basis laboratory analysis.

x) The onset of the effect of vitamin K\textsubscript{1} is not immediate, regardless of the route of administration. However, IV administration of vitamin K\textsubscript{1} should be reserved for life-threatening bleeding and serious bleeding at any elevation of INR. Under these circumstances patients may also be supplemented with prothrombin complex concentrate, fresh-frozen plasma (9 mL/kg) or whole blood (20mL/kg) intravenously to replace clotting factors and RBC if bleeding is severe. The oral form of Vit. K\textsubscript{1} may be used daily after the first day, commonly at the same level as the loading dose.

y) To minimize the risk of an anaphylactoid reaction, the preparation should be diluted with preservative free 5% dextrose, 0.9% sodium chloride solution, or 5% dextrose in 0.9% sodium chloride solution and administer slowly.

z) One week of vitamin K\textsubscript{1} treatment is usually sufficient for first-generation anticoagulants. For intermediate and second-generation anticoagulants or if anticoagulant type is unknown, treatment should continue for 2-4 weeks to control long term effects.

12.7 Final Recommendations

aa) Patients with exposure due to accidental or suspected self-harm, or potentially malicious administration of LAARs should be referred to an emergency department immediately.

bb) Regardless of the doses reported transportation to an emergency department should not be delayed for administration of activated charcoal.

cc) Patients with symptoms of LAAR poisoning (e.g., bleeding, bruising) should be referred immediately to an emergency department for evaluation regardless of the doses reported.

dd) The administration of vitamin K is not recommended prior to evaluation for coagulopathy.

e) Physicians’ offices or outpatient clinics must be able to obtain coagulation study results in a timely manner, preferably in less than 24 hours, for patients who require outpatient monitoring.
**13. MALATHION**

Malathion is an organophosphate pesticide. It is a yellow to deep brown liquid with garlic-like odor which does not provide adequate warning of hazardous concentration. It is widely used to kill insects on agricultural crops, on stored products and in home gardens. It also used to kill mosquitoes and fruit flies in large outdoor areas. In addition, it is used to kill fleas on pets and to treat head lice on humans and by farmers as a pesticide on fruits, vegetables, nuts, and grains. Commercial pesticides often dissolved in a hydrocarbon solvent, which is flammable and can cause illness.

### 13.1 Routes of Exposure

#### 13.1.1 Inhalation
As malathion has low vapor pressure, significant inhalation is unlikely at ordinary temperature. However toxic effect can occur after inhalation of malathion sprays or dusts. The hydrocarbon solvents (most commonly toluene and xylene) used to dissolve malathion are more volatile than malathion itself, and toxicity can result from inhalation of solvent vapor as well.

#### 13.1.2 Skin
Malathion is absorbed through skin during malathion application spray onto the crafts or in houses in Afghanistan. It is readily absorbed through intact skin causing systemic toxicity especially in children. Persons whose skin or clothing is contaminated with liquid or powdered malathion can cause secondary contamination by direct contact.

#### 13.1.3 Ingestion
Malathion is rapidly absorbed by ingestion and in large amounts can cause severe and acute toxic effect including rapid fatal systemic poisoning.

### 13.2 Clinical features
Systematic malathion toxicity result from excess cholinergic stimulation from all routes of exposure. Symptoms include:
- abdominal cramps, vomiting, diarrhoea
- pinpoint pupils and blurred vision
- excessive sweating, salivation and lacrimation
- wheezing, excessive tracheobronchial secretions
- agitation, seizures, bradycardia or tachycardia
- muscle twitching and weakness
- urinary and fecal incontinence

Seizures are much more common in children than in adults. Death results from loss of consciousness, coma, excessive bronchial secretions, respiratory depression and cardiac irregularity. Toxicity of malathion depends on metabolic activation; thus, symptoms may appear from a few minutes to a few hours after exposure.

### 13.3 Laboratory Investigation
The diagnosis of malathion toxicity is mainly clinical. However, WBC, glucose and electrolytes should be requested for general information. A chest x-ray is useful to examine for hydrocarbon aspiration and non-cardiogenic pulmonary oedema.

### 13.4 Management

#### 13.4.1 Management by ambulance staff
Malathion is highly contaminating and systemic effects can occur from all routes. When malathion exposure is suspected the ambulance crew should take care of their own safety first by dressing appropriately to protect themselves from exposure. Victims whose skin or clothing is contaminated with liquid or powdered malathion can secondarily contaminate others by direct contact or evaporation of solvent vapor. Clothing and leather goods (e.g., belts or shoes) cannot be reliably decontaminated; they should be incinerated. Commercial malathion products often contain hydrocarbon solvents, such as xylene or toluene, which themselves can cause toxicity. Treatment for breathing the solvent is fresh air. Remove the patient to fresh air.

13.4.2 Management at the Emergency Department
Treatment for malathion poisoning consists of thorough decontamination, cardiopulmonary resuscitation and support, and administration of the antidotes atropine and pralidoxime. In cases of severe poisoning, diazepam should also be administered.

13.5 ANTIDOTES
Antidotes should be administered as prevention even if the diagnosis is in doubt.

13.5.1 Atropine
Atropine will reverse the muscarinic effects of acetylcholine and is given by intravenous injection in a dose of 2mg (20 micrograms/kg in child, max. 2mg) as atropine sulphate every 5-10 minutes (according to the severity of poisoning) until the skin becomes flushed and dry, the pupils dilate, and bradycardia is abolished.

13.5.2 Pralidoxime Chloride
Pralidoxime Chloride is a cholinesterase reactivator, used in adjunct to atropine in moderate to severe poisoning. It improves muscle tone within 30 minutes of administration. Pralidoxime chloride is continued until the patient has not required atropine for 12 hours.

13.5.3 Dose
By IV infusion, ADULT and CHILD, initially 30 mg/kg over 20 minutes, followed by 8 mg/kg/hour, usual max. dose is 12 g in 24 hours. If pulmonary oedema is present or IV infusion is not practical, the loading dose may be administered by IV injection (diluted to a concentration of 50 mg/mL with water for injection) over at least 5 minutes.
14. Some Other Substances Commonly Caused Accidental Poisoning or Used as Means of Selanistan

14.1 Iron salts
Iron poisoning in children is usually accidental.

14.1.1 Symptoms
Symptoms are nausea, vomiting, abdominal pain, diarrhoea, haematemesis, and rectal bleeding. Hypotension and hepatocellular necrosis can occur later. Coma, shock and metabolic acidosis indicate severe poisoning. There are five stages described for iron toxicity, though one should not solely rely on the temporal relationship between symptoms and ingestion time as patients do not follow the same temporal course through these stages.

14.1.2 The five stages of iron toxicity
ff) **Phase I (0.5 – 2 hrs):** Gastrointestinal phase characterized by nausea, vomiting, diarrhoea, abdominal pain, GI bleeding. If there is no vomiting within 6 hours there is no toxicity.

gg) **Phase II:** Latent phase or second phase refers to 6-24 hours post ingestion. GI complaints resolve but there is ongoing cellular toxicity until overt systemic toxicity develops. Patients with mild toxicity will not progress beyond this stage.

hh) **Phase III (2 - 24 hours):** Systemic toxicity. Recurrence of GI symptoms, anaerobic metabolism, multi-organ involvement with cardiac dysfunction, bleeding, renal failure will lead to shock and metabolic acidosis.

ii) **Phase IV:** Sudden and intensive hepatic failure may occur 2-5 days post-ingestion and is characterized by hepatic failure due to cellular oxidative injury.

jj) **Phase V:** Delayed sequelae, occurs rarely, 2-8 weeks post-ingestion from gastrointestinal scarring. Gastric outlet obstruction may occur due to intestinal strictures and scarring from the initial gastrointestinal injury.

14.1.3 Diagnosis
kk) History of overdose or accidental poisoning and presence of gastrointestinal symptoms indicate toxicity with iron.

ll) Presence of symptoms of hepatic toxicity and metabolic acidosis especially lactation indicates severe and cellular toxicity.

mm) Laboratory investigation of total iron serum level of more than 500 micrograms/dL at 4-8 hours post-ingestion confirms severe toxicity. Abdominal x-ray shows radio-opaque tablets in the digestive system but a negative result does not rule out iron ingestion or intoxication.

14.1.4 Treatment
Mortality is reduced by intensive and specific therapy with desferrioxamine which chelates iron. The serum iron concentration is measured as an emergency. Remember that activated charcoal does NOT bind to iron. Whole bowel irrigation is ONLY indicated if a large amount of pills seen on x-ray film. IV desferrioxamine given to chelate absorbed iron in excess of the expected iron binding capacity. In severe toxicity IV desferrioxamine should be given immediately without waiting for the result of serum iron measurement.

Desferrioxamine Mesilate: By continuous IV infusion ADULT and CHILD up to 15 mg/kg/hour, reduced after 4-6 hours; max 80 mg/kg in 24 hours. The total duration of therapy has not been established, however, it is recommended to continue desferrioxamine up to 24 hours after the patient’s urine is clear, serum iron falls to <100 microgram/dL, or patient is asymptomatic.

Treat coagulopathy, coma, seizures and metabolic acidosis if they occur.

14.2 TRICYCLIC ANTIDEPRESSANTS

14.2.1 Clinical features
- dry mouth, dilated pupil
- coma of varying degrees
- hypotension
- hypothermia
- hyperreflexia, extensor plantar responses
- convulsions
- respiratory failure
- cardiac conduction defects and arrhythmias
- urinary retention

Metabolic acidosis may complicate severe poisoning; delirium with confusion, agitation, and visual and auditory hallucinations are common during recovery.

14.2.2 Management
Assessment in hospital is strongly advised in case of poisoning by tricyclic and related antidepressants but symptomatic treatment can be given by attending doctor or ambulance staff before transfer. Supportive measures to ensure a clear airway and adequate ventilation during transfer are mandatory. IV lorazepam or IV diazepam (preferably in emulsion form) may be required to treat convulsions. Activated charcoal is given within 1 hour of overdose reduces absorption of the drug. Although arrhythmias are worrying, some will respond to correction of hypoxia and acidosis. The use of antiarrhythmic drugs is best avoided, but IV infusion of sodium bicarbonate can arrest arrhythmias or prevent them in those with an extended QRS duration. Diazepam given by mouth is usually adequate to sedate delirious patients but large doses may be required.

14.3 Household products: acid and acid related corrosives and caustics
Including all poisonous household products is beyond the scope of this guideline. We discuss corrosives that are found in most households in Afghanistan in recent years, especially in major cities, for the purpose of disinfecting and de-scaling bathrooms. Sulphoric acids are sold in large quantities freely in shops for this purpose. Deliberate or accidental poisoning with acid is reported in Afghanistan and the management is reported to be unclear amongst clinicians.

14.3.1 Toxicity
Ingestion of 1 mL of corrosive acid may cause death. Acids destroy tissues by direct chemical action. The tissue protein is converted to acid proteinate.

14.3.2 Clinical features
Inhalation
Acids readily dissolve fluids of mucous membrane and lung tissue to produce inflammation leading to:

a) acute chemical pneumonitis, pulmonary oedema manifested by cough, chest pain, cyanosis, dyspnoea, haemoptysis; blood pressure may be high or low.
b) chronic cough with bronchopneumonia.

**Ingestion:**
Corrosive burns of the oropharynx, oesophagus and stomach causing burning pain, vomiting with or without blood, fever, and rigid abdomen. Can also stricture of pylorus and oesophagus. Oxalate ingestion may also produce convulsions, respiratory collapse and renal stones with or without anuria.

**Contact:**
Skin: severe pain and brownish or yellowish stains
Eyes: conjunctival oedema and corneal destruction, pain and tearing.

14.3.2.1 Laboratory Tests
FBC, electrolytes, glucose, arterial blood gases, chest X-ray, abdominal X-ray

14.3.2.2 Antidote
Do not use chemical antidote eg. bicarbonates

14.3.2.3 Management of Toxicity

**Inhalation:**
- maintain respiration
- treat shock - maintain BP by infusion fluids
- treat pulmonary oedema
  - decrease respiratory rate with IM morphine 10 mg
  - give oxygen
  - IV infusion, aminophylline 250-500 mg for bronchoconstriction
  - reduce oedema with oral or IV frusemide 20 - 80 mg
- treat pneumonia with antibiotics

**Ingestion:**
- Give water or milk (120 mL to 240 mL in adults; 60 mL to 120 mL in children). For oxalic acid give milk, calcium lactate or calcium carbonate to precipitate oxalate.
- Avoid emesis and gastric lavage
- If perforation is suspected, keep nil by mouth until endoscopic examination
- Treat asphyxia
- Treat shock - maintain BP by infusion fluids
- Reduce pain with morphine 5 - 10 mg 2-4 hourly prn
- In oxalic acid poisoning, give fluids up to 4 L daily to prevent precipitation of oxalate stones in renal tubules

**Contact**
- eye contact
  - flood eye with running water for 15 minutes
  - relieve pain with analgesic, bandage and refer to ophthalmologist
- skin contact
  - flood area with running water for 15 minutes
  - relieve pain with analgesic and treat burns
15. ANIMAL STINGS

15.1 Snake bites
Snake bites occur when a snake bites the skin. They are medical emergencies if the snake is poisonous. Snake bites can be deadly if not treated quickly. Children are at higher risk for death or serious complications due to snake bites because of their smaller body size. Snakes found in and near water are often mistaken as being poisonous. Most species of snakes are harmless and many bites are not life-threatening, but unless you are absolutely sure that you know the species, treat it seriously.

15.1.1 WHO classified venomous snake species according to the risk or threat to public health in various countries and territories around the world into two categories:

**Category 1:** Highly venomous snakes which are common or widespread and cause numerous snakebites, resulting in high levels of morbidity, disability or mortality. Species listed in this category within a country, territory or area should be considered as being of highest priority for anti-venom production on the basis that available knowledge implicates them as being responsible for the greater burden in that particular setting.

**Category 2:** Highly venomous snakes capable of causing morbidity, disability or death, for which exact epidemiological or clinical data may be lacking; and/or which are less frequently implicated (due to their activity cycles, behaviour, habitat preferences or occurrence in areas remote to large human populations).

There are numerous other venomous species that rank as lesser threats in countries, territories and other areas that is not included in the above categories.

WHO listed the categories of venomous snakes for Afghanistan as follows:

**Category 1:** Elapidae: Naja oxiana; Viperidae: Echis carinatus; Macroviper a lebetina

**Category 2:** Elapidae: Bungarus caeruleus (east), Bungarus sindanus (east), Naja naja (reported in south-east); Viperidae: Eristicophis macmahonii (south-west); Gloydis halys.

15.1.2 Snake venoms are complex in their composition depending on their species, age and the season. Snake venoms were classified as cytotoxins, nephrotoxins, myotoxins, hemorrhagins, coagulants/anticoagulants, neurotoxins and etc. in the past and each one were ascribed to different families of snakes. This characterization is misleading as the snake venom should be considered as an aggregate of different effects and symptoms they produce in human beings. Therefore, the right antivenom can save a person's life. Getting to an emergency room as quickly as possible is very important. If properly treated, many snake bites will not have serious effects. The World Health Organisation and the International Programme on Chemical Safety convened a Working Group on Natural Toxins (WGONT) in 1989 which developed general guidelines for the management of all venomous bites and stings. This document forms the basis of guidelines on both first aid and medical treatment recommendations.

15.1.3 Clinical features: A detailed clinical presentations of poisoning by snakebites by all species is beyond the scope of this guideline. Some general points important for the assessment in the emergency departments are covered. Fatal envenoming is rare but the potential for severe envenoming must not underestimated. Envenoming from snake bites may cause local and systemic effects.

- Local effects include severe pain, burning of the skin, fang marks on the skin, swelling, bruising, skin discoloration, bleeding from wound, and tender enlargement of regional lymph nodes and tissue death.
Systemic effects include early anaphylactic symptoms (transient hypotension with syncope, angioedema, urticaria, abdominal colic, diarrhea and vomiting) with later persistent or recurrent hypotension, ECG abnormalities, spontaneous systemic bleeding, coagulopathy, adult respiratory distress syndrome, and acute renal failure. Other symptoms may include: blurred vision, convulsions, diarrhea, dizziness, excessive sweating, fainting, fever, increased thirst, loss of muscle coordination, nausea and vomiting, numbness and tingling, rapid pulse and weakness.

15.1.4 Management
First Aid by Ambulance staff:

- Keep the patient calm, reassuring them that bites can be effectively treated in an emergency room. Restrict movement, and keep the affected area below heart level to reduce the flow of venom.
- If you have a pump suction device, suck the area.
- Remove any rings or constricting items because the affected area may swell. Create a loose splint to help restrict movement of the area.
- If the area of the bite begins to swell and change color, the snake was probably poisonous.
- Monitor the person's vital signs—temperature, pulse, breathing, and blood pressure—if possible. If there are signs of shock (such as paleness, decreased consciousness), lay the person flat, raise the feet about a foot, and cover the person with a blanket.
- Arrange for transfer of the patient to the nearest Emergency Department.
- Bring in the dead snake only if this can be done safely. Do not waste time hunting for the snake, and do not risk another bite if it is not easy to kill the snake. Be careful of the head when transporting it—a snake can actually bite for several hours after it is dead.

15.1.5 Some simple advice on NOT TO DO for anybody who attempts to provide first aid, especially ambulance and emergency department staff, in cases of snake bites is provided in Box 4.
Box 4. NOT TO Dos in case of snake bite

Do NOT allow the person to become over-exerted. Carry the person to safety.
Do NOT apply a tourniquet.
Do NOT apply cold compresses to a snake bite.
Do NOT cut into a snake bite with a knife or razor.
Do NOT try to suck out the venom by mouth.
Do NOT give the person stimulants or pain killers if not sure about the interaction and side effects.
Do NOT give the person anything by mouth.
Do NOT raise the site of the bite above the level of the person’s heart.

15.16 Medical Management in the Emergency Department
If the patient is in anaphylaxis shock secure the airway, restore blood pressure and administer adrenaline as first line treatment.

15.1.7 Adrenaline
The intramuscular (IM) route is the first choice for the administration of adrenaline in patients with anaphylaxis. The best site for IM injection is the anterolateral aspect of the middle third of the thigh. The needle used for injection needs to be sufficiently long to ensure that the adrenaline is injected into muscle length. Dose: The Working Group of the Resuscitation Council (UK), 2008, recommends the following doses of adrenaline in their Emergency Treatment of Anaphylaxis Guideline for Healthcare Providers, for the management of anaphylaxis, which is presented in Box 5.

Box 5. Adrenalin doses according to age

<table>
<thead>
<tr>
<th>Age</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult</td>
<td>500 micrograms IM (0.5 mL)</td>
</tr>
<tr>
<td>Child more than 12 years</td>
<td>500 micrograms IM (0.5 mL)</td>
</tr>
<tr>
<td>Child 6-12 years</td>
<td>300 micrograms IM (0.3 mL)</td>
</tr>
<tr>
<td>Child less than 6 years</td>
<td>150 micrograms IM (0.15 mL)</td>
</tr>
</tbody>
</table>

Adrenaline IV to be given only by experienced specialists
Titrate: Adults 50 micrograms; Children 1 microgram/kg.

15.1.8 Systemic envenoming should be treated with anti-venoms. The symptoms of systemic envenoming include hypotension, ECG abnormalities, vomiting, haemostatic abnormalities, and marked local envenoming (when swelling extends beyond the wrist or ankle within 4 hours of the bite).

15.1.9 European viper venom antiserum, ADULT and CHILD, one vial (10mL) given by IV injection over 10-15 minutes or intravenous infusion over 30 minutes after diluting in sodium chloride 0.9% (use 5mL diluents/kg body weight). The dose can be repeated after 1-2 hours if symptoms of systemic envenoming persist.

15.1.10 In severe envenoming (e.g. shock, ECG abnormalities, or local swelling that advanced from foot to above the knee or from the hand to above the elbow within 2 hours of the bite) an initial dose of 2 vials (20mL) of antiserum is recommended.
15.2 Scorpion Poisoning
Scorpions are nocturnal animals and hide under the logs, rocks, cavities and wall cracks during the day. They usually feed on other insects, spiders, centipedes and even small vertebrates.

There is no scientific information available on the types of different kinds of scorpions found and the prevalence of scorpion bites in Afghanistan at the time of compiling this guideline. However, anecdotal evidence suggests that scorpion bites are not an infrequent medical problem in Afghanistan.

15.2.1 Clinical features
- Local reactions include severe pain and mild oedema.
- General reactions include parasympathomimetic symptoms (also known as ‘autonomic storm’) such as increased or decreased blood pressure, bradycardia, mydriasis, increased salivation, myocardial damage which may result in pulmonary oedema and central nervous system manifestation. Intoxication is usually more severe in children.

15.2.2 Management
Treatment of scorpion poisoning is usually symptomatic to treat symptoms of ‘autonomic storm’ with vasodilators. For general advice please refer to the management of snake poisoning as above. The specific type of antiserum available for scorpions in Afghanistan is going to be added to this guideline later.
15.3 Spider Poisoning

There are about 30,000 species of spiders in the world and all of them are considered venomous, as most of them possess a pair of venom glands. However only 20-30 are potentially dangerous to human beings. The venom glands of primitive spiders, such as tarantulas, are quite small and situated inside the jaws. In contrast, most other spiders have relatively large poison glands that may extend out of the jaws and reach far into their body. Each poison gland consists of a long cylindrical sac and an adjoining duct which opens slightly away from the tip of the fang.

Anecdotal evidence from Afghanistan suggests that the most common and poisonous spider in Afghanistan is called Ghondal (غندل) but no scientific information is available about the type of its venom at the time of compilation of this guideline.

15.3.1 Clinical features

- **Local reactions:** There will be pain at the site of bite, followed by swelling and redness. In severe cases local necrotic symptoms are also seen. Following the painful bite, the skin turns dark and black and then becomes an eschar with dry skin. The lesions sloughs in few days and a deep, granular area surrounded by normal skin will appear. This ulcer may take many weeks for healing.
- **General reactions:** Neurotoxic symptoms are mainly seen after spider bites include headache, nausea, vomiting muscle spasms and tremors. Cardiovascular symptoms might also be present.
- **Systemic reaction** such as intravascular hemolysis and hemoglobinuria has been reported following bite by a ‘brown recluse spider’.

15.3.2 Treatment

General first aid as mentioned in the snakes poisoning should be considered if clinically required. Treatment is mostly symptomatic. Specific antivenom for specific spider in Afghanistan is not known and will be included in the guideline by the time it goes to publication. Oral dapsone (100 mg b.d.) could be given to necrotic tissues. Calcium gluconate (10 ml of 10% solution iv) is used to reduce the muscle spasm.
16. PSYCHO-SOCIAL ASSESSMENT

16.1 Patients who are poisoned, accidentally of deliberately (self-harmed) should receive a comprehensive assessment of risk and needs to avoid further episode of poisoning whether accidental or deliberate.

16.2 Patients who are poisoned, whether accidentally or deliberate are under enormous distress. The assessment process and interactions between the emergency staff and the patient should be reassuring, therapeutic, enhance trust and engage the patient.

16.3 All patients who have self-harmed must receive comprehensive psychiatric assessment in particular for depression, psychosis, suicidal and homicidal ideation and intention.

16.4 The assessment should be written clearly in the patient’s notes with clear recommendation for further action.

16.5 Attempts should be made to use a standardised and validated mental health triage scale to screen for mental disorders and risk of suicide. SAD PERSONS score which has low sensitivity but high specificity can be one of the options and is produced in Box 6.

Box 6. SAD PERSONS Scale

<table>
<thead>
<tr>
<th>Factors</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>S = Sex (Male)</td>
<td>1</td>
</tr>
<tr>
<td>A = Age (&lt;19 or &gt;45 years)</td>
<td>1</td>
</tr>
<tr>
<td>D = Depression</td>
<td>1</td>
</tr>
<tr>
<td>P = Previous suicide attempt</td>
<td>1</td>
</tr>
<tr>
<td>E = Ethanol abuse</td>
<td>1</td>
</tr>
<tr>
<td>R = Rational thinking loss</td>
<td>1</td>
</tr>
<tr>
<td>S = Social support lacking</td>
<td>1</td>
</tr>
<tr>
<td>O = Organized plan</td>
<td>1</td>
</tr>
<tr>
<td>N = No spouse</td>
<td>1</td>
</tr>
<tr>
<td>S = Sickness (chronic debilitating disease)</td>
<td>1</td>
</tr>
</tbody>
</table>

Score less than 2: Discharge with psychiatric outpatient referral or appointment
Score 3 - 6: Consider for hospitalisation or at least very close follow up like psychiatric assessment and follow up in the next day or so.
Score 7 or above: admit for close supervision to avoid further suicide attempt.

Source: Patterson et al, 1983.

16.6 Nursing staff in collaboration with assessing doctors in the emergency departments should be trained in assessing the risk of repeated self-harm and poisoning.

16.7 A risk assessment form to guide the emergency staff in this regard as well as general risk assessment is produced in annex 3. If a standardised risk assessment scale is used to assess risk, this should be used only to aid in the identification of people at high risk of repetition of self-harm or suicide. A risk assessment score is based on the objective and subjective assessment carried out by emergency staff or junior doctor. It cannot replace the objective assessment by an expert emergency staff.
17. POISONING AND SELF-HARM IN SPECIAL AGE GROUPS

17.1 Children and younger people
Children under age 16 – 18 who are presented with poisoning whether accidental or deliberate have special needs given their vulnerability.

17.1.1 Emergency departments should have a trained children’s nurse for the assessment of children and younger people under age 16.

17.1.2 Each emergency department should have a named senior paediatrician allocated to that emergency department for consultation. This paediatrician can be based in the paediatrics ward/department but should have overall responsibility for the treatment and care of children and young people who are presented with poison and self-harm in the local emergency department.

17.2 Older age people
Although the definition of old age is not clearly defined in Afghanistan, but older people are usually considered those who are above 65 years of age.

17.2.1 Any older person who present with poisoning should be considered at high risk of suicide. All acts of self-harm in older people should be regarded as evidence of suicidal intent until proven otherwise because the number of people in this age range who go on to complete suicide is much higher than in younger adults.

17.2.2 This group of patients should be assessed by a mental health specialist during their stay in the emergency department.

17.2.3 Assessment should follow the same principles as for younger adults who self-harm, but should also pay particular attention to the potential presence of depression, cognitive impairment and physical ill health, and should include a full assessment of their social and home situation.

17.2.4 Considering the high risks of completion of suicide amongst older adults who have self-harmed, admission to the psychiatric unit or hospital should be considered for mental health, risk and needs assessment. Longer observation in a psychiatric unit or hospital will also give an opportunity to observe the patient for change in their mental state and also for cognitive impairment.
18. REFERRAL, ADMISSION AND DISCHARGE FOLLOWING POISONING AND SELF-HARM

Following initial assessment and treatment of poisoning at the emergency department a decision should be made whether to discharge the patient, refer to other agencies for further assessment and treatment or admit to the hospital. This decision should be based on comprehensive physical and psychiatric assessment and discussion with senior members of staff or consultant. The decision should be clearly documented in the patient’s notes.

Following any poisoning or act of self-harm temporary overnight admission should be considered especially for people who are severely distressed; returning to unsafe or potentially harmful environment, transferred from one of the distant provinces or need longer term assessment. The patient should be reassessed the next day before discharge by the ward doctor.
19. GUIDELINE REVIEW DATE

This guideline is developed in consultation with policy makers at the MoPH and clinicians at Avicenna Emergency Hospital in Kabul city and adaptation of some international literature like NICE guidelines for Afghanistan. Although this guideline is reviewed by experts in the field before its approval by the Ministry of Public Health for implementation, it is written single handedly in a short period of time which is not an ideal way of developing a guideline. However, there is a huge thirst for policy development and research in the MoPH in recent years that may result in some evidence being produced in Afghanistan in the years to come to help the revision of this guideline. Therefore, we recommend that this guideline should be reviewed as soon as new evidence emerges or the provision of emergency services change in Afghanistan or at least in the next 3-5 years.
Reference

2. All India Institute of Medical Sciences, (2008), Acute Poisoning – Management Guidelines, Delhi, India.
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6. Block, Barbara, First Aid for the Emergency Medicine Boards, [Book], Division of Emergency Medicine, Department of Surgery, University of Colorado Denver School of Medicine, Aurora, Colorado, 2009
11. Daly, F. S. et al, (2010), Guidelines for the management of paracetamol poisoning in Australia and New Zealand,
15. Descotes, Jasques; (1996), Human Toxicology [book], Department of Pharmacology and Medical Toxicology & INSERM U80, Lyon Laennec Faculty of Medicine, Lyon, France Elsevier Publication.

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The following drugs are mentioned in this guideline:

- Activated charcoal
- Adrenaline
- Antihistamines
- Atropine
- Diazepam Emulsion 10 mg
- Dicobalt edetate
- Drugs mentioned in this guideline
- European viper venom antiserum
- Flumazenil
- Hydroxycobalamine
- Lorazepam
- Methionine
- N-acetylcysteine
- Naloxone Hydrochloride
- Oxygen
- Pralidoxime chloride
- Sodium thiosulphate 5% solution
- Vitamin K₁
A) Drugs that Activated Charcoal is Effective in Adsorbing

Activated charcoal has been shown to be effective in adsorbing the following drugs:

- Acetaminophen
- Tricyclic antidepressants
- Antipyrines
- Arsenic
- Aspirin
- Atropine
- Chlorpheniramine and related antihistamines
- Chlorpromazine and related phenothiazines
- Dextro-amphetamine
- Digoxin
- Glutethimide
- Isoniazid
- Meprobamate
- Salicylates

- Morphine
- Paraquat
- Phenobarbital and other barbiturates
- Penicillin
- Phenylpropranolamine
- Phenytoin
- Propoxyphene
- Quinidine
- Quinine
B) Drugs that Activated Charcoal is NOT Effective in Adsorbing

Activated charcoal has not been shown to be effective in adsorbing the following:

- Acids and caustic alkalis
- Aromatic alcohols
- Boric acid
- Ethylene glycol
- Heavy metals
- Iron
- Lithium
- Malathion
- Methylcarbamate
- Methanol
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معالجی
ریاست شفاخانه

نام و تخلص بیمار:

پرستار بخش:

دکتر متخصص:

شماره دوسيه:

پرستار بخش:

شماره دوسيه:

دکتر متخصص:

تاریخ تولد/ سن:

داکتر متخصص:

شماره دوسيه:

1. خطر خود آزاری/ خودکشی (Risk of Self-Harm/ Suicide)

شدت خطر (1= خفیف، 5= شدید) 5

فاکتور های شناخته شده که باعث تشدید این خطر می‌گردد:

- پیشینه خودآزاری و آگاهی
- حملات از شیوه‌های خطرناک (مانند حلقویت)
- افتخار افزایشی
- تومای بررسی‌های
- اضطراب و تاراگری
- نگرفتن دارو
- مشکل
- بیکاری/ بی خانگی

2. خطر استفاده سوء از بیمار (Risk of Exploitation)

شدت خطر (1= خفیف، 5= شدید) 5

فاکتور های شناخته شده که باعث تشدید این خطر می‌گردد:

- سن (کودکی، طفولت و بیری)
- اشکال اجتماعی
- اختلال در گفتار
- اختلال در درک
- اعتیاد به مواد مخدر
- انتظار به دست افراد
- اعمال به دست افراد
- بیکاری و کشف

وزارت صحت عامه
ریاست عملیه طب
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ریاست شفاخانه

نام و تخلص بیمار:

پرستار بخش:

دکتر متخصص:

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- اختلال در گفتار
- اختلال در درک
- اعتیاد به مواد مخدر

وزارت صحت عامه
ریاست عملیه طب
معالجی
ریاست شفاخانه

نام و تخلص بیمار:

پرستار بخش:

دکتر متخصص:

شماره دوسيه:

دکتر متخصص:

شماره دوسيه:

دکتر متخصص:

شماره دوسيه:

1. خطر خود آزاری/ خودکشی (Risk of Self-Harm/ Suicide)

شدت خطر (1= خفیف، 5= شدید) 5

فاکتور های شناخته شده که باعث تشدید این خطر می‌گردد:

- پیشینه خودآزاری و آگاهی
- حملات از شیوه‌های خطرناک (مانند حلقویت)
- افتخار افزایشی
- تومای بررسی‌های
- اضطراب و تاراگری
- نگرفتن دارو
- مشکل
- بیکاری/ بی خانگی

2. خطر استفاده سوء از بیمار (Risk of Exploitation)

شدت خطر (1= خفیف، 5= شدید) 5

فاکتور های شناخته شده که باعث تشدید این خطر می‌گردد:

- سن (کودکی، طفولت و بیری)
- اشکال اجتماعی
- اختلال در گفتار
- اختلال در درک
- اعتیاد به مواد مخدر

وزارت صحت عامه
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نام و تخلص بیمار:

پرستار بخش:

دکتر متخصص:

شماره دوسيه:

دکتر متخصص:

شماره دوسيه:

دکتر متخصص:

شماره دوسيه:
3. خطر به دیگران (Risk to Others):

شدت خطر (1=خفی، 5=شدید)

- فکتورهای شناخته شده که باعث تشکیل این خطر می‌گردند:
  - سابقه تخریب کاری
  - سابقه نگرفتن داروی تجویز شده
  - بی حوصله و تغییر عادات
  - توهات (ارمینی)
  - تنبه بیش از حد
  - اظهار قصد تهدید

4. خطر به اطفال (Risk to Children): (اطفال در اینجا اشخاص زیر سن 18 ساله است)

شدت خطر (1=خفی، 5=شدید)

- فکتورهای شناخته شده که باعث تشکیل این خطر می‌گردند:
  - سابقه جرمی (مانند قتل اطفال)
  - سابقه کودک آزاری و غفلت از کودک
  - سابقه جرمی (مانند قتل اطفال)

5. خطر بازگشت مرض (Risk to Relapse):

شدت خطر (1=خفی، 5=شدید)

- فکتورهای شناخته شده که باعث تشکیل این خطر می‌گردند:
  - سابقه بازگشت مكرر بیماری
  - سابقه بستری شدن مکرر در شفاخانه
  - سابقه بازگشت مکرر بیماری
  - سابقه بستری شدن در شفاخانه

6. خطر عدم همکاری یا فرار از شفاخانه (Risk of non-engagement or absconding):

شدت خطر (1=خفی، 5=شدید)

- فکتورهای شناخته شده که باعث تشکیل این خطر می‌گردند:
  - بیشینه ی فرار از شفاخانه
  - اظهار قصد فرار
  - ناامنی بیشینه
  - اختلال شعور و جهت بی‌پایی
  - استفاده از مواد مخدر
  - بیمارانی که از زندان اخراج شدند
برنامه پیشگیری خطر:
برای هر یک از شش خطی که در این فرم شناسایی شده اند باید برنامه پیشگیری دقیق طرح شود.

برنامه مدیریت خطر

<table>
<thead>
<tr>
<th>نام پرستار کلیدی تطیف گیرنده برنامه</th>
<th>برنامه مدیریت خطر (Risk Management Plan)</th>
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</tbody>
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آیا این برنامه با خود بیمار مشوره گرفته است؟
آیا این برنامه با اقارب بیمار مشوره گرفته است؟

در صورتی که جواب منفی (نه) باشد دلایل را بنویسید:

نام پرستار کلیدی: .................................................................
تاریخ: ..............................................................................
امضاء: ..............................................................................
ANNEX 4

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VISION  All citizens reach their full potential in health contributing to peace, stability and sustainable development in Afghanistan.

VALUES  Equity, Integrity, Right to Health, Accountability, Trust

MISSION STATEMENT
The Mission Statement of the Ministry of Public Health of the Government of the Islamic Republic of Afghanistan is to prevent ill health and achieve significant reductions in mortality in line with the national targets and sustainable development goals and to reduce impoverishment due to catastrophic health expenditure. Also to be responsive to the rights of citizens through improving access and utilization of quality, equitable, affordable health and nutrition services among all communities especially mother and children in rural areas, and through changing attitudes and practices, promoting healthy lifestyles and effectively implementing other public health interventions. All in coordination and collaboration with other stakeholders

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