Chapter

General Approach to Poisoned Patient

Ehab Said Aki and Jalal Alessai

Abstract

Poisoning is a serious worldwide public health problem. Based on the World Health Organization data in 2012, almost 190,000 people died worldwide and the number of deaths due to poisoning in 2008 exceeded the number of deaths due to motor vehicular crashes; also, poisoning death rate nearly tripled worldwide. The number of patients presenting to the emergency departments with overdose had been increased both intentionally and accidentally. All the previous facts make toxicology an important field in emergency medicine. According to the American Association of Poison Control Centers (AAPCC) in the United States, over 2.1 million human exposure calls are reported in 2016. Management of intoxicated patients has a unique approach because of the challenge in diagnosis and treatment of overdose cases. This chapter focuses on general approaches for intoxicated patients and initial management and on how the history and physical examinations could help physicians to have a clue about the drugs that have been abused. Patients are most commonly poisoned via oral ingestion, but other routes could also cause intoxication including inhalation, insufflation, cutaneous and mucous membrane exposure, and injection.

Keywords: initial approach, physical examination, toxidromes, decontamination, toxicology laboratory

1. Introduction

Poisoning is a serious worldwide public health problem. Based on the World Health Organization (WHO) data in 2012, almost 190,000 people died worldwide and the number of deaths due to poisoning in 2008 exceeded the number of deaths due to motor vehicular crashes; also, the death rate due to poisoning nearly tripled worldwide. The number of patients presenting to the emergency departments with overdose had been increased both intentionally and accidentally. All the previous facts make toxicology an important field in emergency medicine [1, 2]. According to the American Association of Poison Control Centers (AAPCC) in the United States, over 2.1 million human exposure calls are reported in 2016.

Management of intoxicated patients has a unique approach because of the challenge in diagnosis and treatment of overdose cases. This chapter focuses on general approaches for intoxicated patients and initial management, explaining how the history and physical examinations could help physicians to have a clue about the drugs that have been abused. Patients are most commonly poisoned via oral

ingestion, but other routes like inhalation, insufflation, cutaneous and mucous membrane exposure, and injection could also cause intoxication.

2. General approach to toxicological cases in emergency medicine

The approach to poisoned patients must be systematic. The range of symptoms and clinical findings in the physical examination are wide in drug poisoning patients; initial management is focused on stabilization of life-threatening conditions. The approach for the poisoned patients in emergency includes: resuscitation, history, physical examination, and management.

Initial screening examination should be done on all patients to find out immediate abnormal measures which need to be stabilized starting with vital signs, conscious level and pupil size, skin temperature, pulse oximetry, and electrocardiogram. Patients who are hemodynamically unstable must be kept in continuous cardiac monitoring. Intravenous access should be done and the blood glucose must be checked especially if the patients have a decreased level of consciousness.

3. Resuscitation

3.1 Airway and ventilation

The initial priorities for a poisoned patient presented to the emergency department are: securing the airway and breathing and stabilizing the circulation. Adequate ventilation and intubation with mechanical ventilation must be done early in the intoxicated patients with depressed mental status, except in cases of easy reversible causes of coma like opioid intoxication or hypoglycemia to prevent complications of intubation like aspiration. Other indications for intubation include severe acid-base disturbances or acute respiratory failure. In intubated patients, development of a respiratory acidosis must be prevented by adequate ventilation; in some cases like high-grade physiologic stimulation, the patient may need sedation and paralysis to prevent complications such as hyperthermia, acidosis, and rhabdomyolysis.

3.2 Hypotension

Drugs cause hypotension by four major mechanisms: decreased peripheral vascular resistance, decreased myocardial contractility, dysrhythmias, and depletion of intravascular volume. First-line treatment of hypotension is IV fluid bolus (10 to 20 mL/kg); if hypotension is not responding to fluid, it may be necessary to add vasopressors such norepinephrine. Norepinephrine is better than dopamine.

3.3 Hypertension

Elevated blood pursues caused by CNS sympathetic overactivity, increased myocardial contractility or increased peripheral vascular resistance, or a combination.

The treatment of hypertension and agitated patients starts with sedatives such as benzodiazepines; if not responding for initial treatment and there is evidence of end-organ dysfunction, calcium-channel blocker is preferred treatment. The use of beta-blockers is not recommended in the case of sympathetic hyperactivity because it may cause unopposed alpha-adrenergic stimulation and intensified vasoconstriction.

Ventricular tachycardia occurs because of tricyclic antidepressant toxicity. Sodium bicarbonate is first line therapy. Types IA (e.g., procainamide), IC, and III

antiarrhythmic agents may worsen cardiac conduction; hence, they are not recommended; also, using these agents could be potentially dangerous.

Magnesium sulfate can also be used in the case of drug-induced torsade de pointes and prolonged QT intervals on ECG.

Digoxin toxicity with life-threatening tachyarrhythmias or bradyarrhythmias should be treated with specific Fab fragments (Digibind).

3.4 Bradyarrhythmias

Treatment of bradyarrhythmias with hypotension starts with atropine and/or temporary pacing. Calcium, glucagon, or high-dose insulin are used in the case of calcium channel blocker or beta blocker intoxication.

3.5 Seizures

The best treatment of intoxicated patients with seizures is benzodiazepines; we may add barbiturates if necessary. Phenytoin is not recommended to control seizures in poisoned patients.

3.6 Severe hyperthermia

Elevated temperature (hyperthermia) due to drug toxicity (e.g., sympathomimetic overdose, serotonin syndrome, or neuroleptic malignant syndrome) must be treated aggressively to prevent complications like rhabdomyolysis, organ failure, and disseminated intravascular coagulation. Treatment of hyperthermia includes active cooling like ice water immersion; if active cooling is ineffective, the patient may need sedation, neuromuscular paralysis, and intubation.

Patients presenting with signs of opioid overdose (low Glasgow coma scale-GCS respiratory depression, meiosis) must be given naloxone (0.1–2.0 mg I.V) as soon as possible [3].

4. History

History of the present illness is very important and can be obtained from the patients if they are alert and conscious; although the history following intentional ingestion is often unreliable, which makes history taking very challenging especially if the patients are comatose or cannot give their history, in such situations, history can be taken from collateral information from family, friends, ambulance crew, or medical records looking for past psychiatry illness, previous history of suicide or drug abuse, chronic medication, etc.

History must include time, route of entry, quantity, intentional or accidental exposure, availability of drugs at home, and if any member of the family has chronic diseases (hypertension, diabetic, etc.) and missing tablets or any empty pill bottles or other material was found around him [4]. It is very important to ask specifically about the use of traditional or herbal remedies and dietary supplements.

5. Physical examination

Physical examination of poisoned patients may give clues regarding the substance which has been abused and toxidromes. Physical examination includes: general appearance,

- Mental status (agitated or confused)

Some drugs or substances affect the central nervous system either causing agitation or depression.

Central nervous system depression may be caused by the following:

Anticholinergics, antidepressants, antipsychotics, lithium, cholinergic beta blockers, clonidine, and sedative-hypnotics.

Central nervous system agitation

Sympathomimetics, anticholinergics, salicylates, central hallucinogens, drug withdrawal states, carbon monoxide, hypoglycemic agents, and heavy metals.

- Skin (cyanosis, flashing, and physical signs of intravenous drug abuse (track marks)

Red and flushed skin occurs in the case of overdose of anticholinergic agents, antihistamines, TCAs, atropine, scopolamine, and phenothiazines.

Pale and diaphoretic skin occurs in the case of sympathomimetics (cocaine), cholinergic agents (organophosphates), central hallucinogens (lysergic acid diethylamide (LSD) and phencyclidine) and salicylate toxicities.

Cyanotic skin occurs in the case of methemoglobinemia and sulfhemoglobinemia.

- Eye examination: (pupil size reactivity lacrimation and nystagmus)

Common drugs causing miosis

- Opioids (morphine, hydromorphone, and oxycodone)
- Sedative-hypnotics (barbiturates and benzodiazepines)
- Cholinergic (nerve agents and organophosphate insecticides)
- Sympatholytic (clonidine and oxymetazoline)
- Common drugs causing mydriasis
- Sympathomimetics (cocaine and caffeine)
- Anticholinergics (atropine, scopolamine, and TCAs)
- Hallucinogens (LSD, mescaline, and psilocybin)
- Serotonin syndrome

Common drugs causing nystagmus
Barbiturates, carbamazepine, phencyclidine, phenytoin, and lithium

- Odor (garlic, bitter almonds, glue, alcohol, etc. (**Table 1**).
- Oropharynx hyper salivation or dryness;
- Chest: breath sound, bronchorrhea, wheezing, heart rate, and rhythm regularity;

Substance	Odor
Ethanol, isopropyl alcohol, chloroform, salicylates	Acetone
Cyanide	Bitter almonds
Organophosphates, phosphorus	Garlic
Phosgene	Freshly mown hay
Hydrogen sulfide	Rotten eggs

Table 1.
Substances causing specific odor.

- Abdomen examination (bowel sound, tenderness, and rigidity);
- Limbs (tremors and fasciculation), patient's clothing (looking for any medications and illegal drugs) [3].

5.1 Toxidromes

The term toxidrome was coined in 1970 by Mofenson and Greensher. Toxidromes are a group of abnormal physical examinations and abnormal vital signs known to be present with a specific group of medications or substances. The most common toxidromes are cholinergics, anticholinergics, sympathomimetics, opioids, and serotonin syndrome [4, 5].

5.2 Cholinergic toxidrome

Patients with cholinergic toxidrome present with wet manifestation. SLUDGE+3 Killer B′s and DUMBELLS are simple mnemonics for the common clinical symptoms. Also, patients present with bradycardia, hypertension or hypotension, tachypnoea, or bradypnea.

SLUDGE: salivation, lacrimation, urination, defecation, GI cramping, Emesis + Killer B′s: bronchorrhea, bradycardia, and bronchospasm.

DUMBELLS: diarrhea, urination, miosis (small pupils), bradycardia, emesis, lacrimation, lethargy, and salivation.

Most common causes: organophosphate pesticides, carbamates, some types of mushrooms, and sarin (warfare agent) [4].

5.3 Anticholinergic toxidrome

Patients present with anticholinergic toxidrome with dry manifestation, delirium, tachycardia, dry flushed skin, dilated pupils, hypertension, tachypnoea clonus, elevated temperature, decreased bowl sounds, and urinary retention. Simple mnemonics: "Hot as a Hare, Mad as a Hatter, Red as a Beet, Dry as a Bone, Blind as a Bat."

Most common causes: antihistamines, antiparkinsonians, atropine, scopolamine, amantadine, antipsychotics, antidepressants, muscle relaxants, and plants (jimsonweed) [4].

5.4 Sympathomimetic toxidrome

Patients present with CNS stimulation and psychomotor agitation, elevated blood pressure, tachycardia, dilated pupils, hyperthermia, widened pulse pressure, tachypnoea, hyperpnea diaphoresis, and seizure in severe cases.

Most common causes: cocaine and amphetamine.

5.5 Opioid toxidrome

The most common clinical presentation of opioid toxidrome are: coma, respiratory depression and meiosis, hypotension, hypothermia, bradycardia, and seizure that may occur in propoxyphene overdose, but small pupils not always present may present with normal size pupils such in meperidine and, propoxyphene toxicities [4].

5.6 Serotonin syndrome

Patients present with altered mental status, hypertensive, and tachycardia, myoclonus, hyperreflexia, hyperthermia, and increase in muscle rigidity. Most common causes: SSRI interaction or overdose of SSRIs.

MAOIs, tricyclic antidepressants, amphetamines, and fentanyl [4].

5.7 Neuromuscular malignant

Patients present with severe muscle rigidity, hyperpyrexia, altered mental status, autonomic instability, diaphoresis, mutism, incontinence. Most common causes: antipsychotic medication.

5.8 Sedative/hypnotic

Patients present with central nervous system depression, ataxia, dysarthria, bradycardia, respiratory depression, hypothermia, hypotension, and bradypnea. Most common causes are benzodiazepines and barbiturates.

5.9 Hallucinogenics

Patients present with hallucinations, perceptual distortions, depersonalization, synaesthesia, and agitation.

Mydriasis, hyperthermia, tachycardia, hypertension, tachypnoea, and nystagmus. Most common causes:

phencyclidine, LSD, mescaline, psilocybin, and MDMA [“Ecstasy”].

5.10 Ethanolic

Patients present with central nervous system depression, ataxia, dysarthria, and odor of ethanol.

5.11 Extrapyramidal

Patients present with dystonia, torticollis, muscle rigidity, choreoathetosis, hyperreflexia, and sometimes seizures. Most common causes: risperidone, haloperidol, and phenothiazines.

5.12 Salicylate

Patients with salicylate toxidrome present with altered mental status, mix respiratory alkalosis, metabolic acidosis, tinnitus, tachypnoea, tachycardia, diaphoresis, nausea, vomiting, and hyperpyrexia.

Most common toxin: aspirin and oil of wintergreen (methyl salicylate).

6. Management

6.1 Electrocardiogram (ECG)

ECG should be done on all patients who are symptomatic or who have been exposed to cardiotoxic agents looking for the rate and conduction; ECG abnormalities may help in diagnosis or may help as prognostic information. Specific attention should be paid to QRS interval and QT interval; in the case of prolongation of QT or QRS sodium bicarbonate infusion should be strongly considered.

6.2 Radiographic studies

Imaging examinations are not necessary in every poisoned patient but may be useful in some situations where the toxins are radiopaque [6]. The toxins which are radiopaque can be summarized by the mnemonic "CHIPES" (**Table 2**); also, "body packers" may be seen on plain films (**Figure 1**). Chest x-ray is useful in the case of noncardiogenic pulmonary edema and the acute respiratory distress syndrome due to exposure to certain toxins.

6.3 Abdominal ultrasound

Ultrasound abdomen is not helpful in poisoned patient and the use of ultrasound is very limited and does not appear to be a reliable method of detecting ingested toxins [7].

6.4 Laboratory test

Blood test must be done with all intoxicated patients; especially in the case of intentional overdose, the laboratory test should include basic lab (full cell count and kidney function liver function and electrolytes). Acetaminophen screening is very important in every patient presenting with altered mental status or intentional overdose [8].

For the patients with an acid-base abnormality, serum osmolarity needs to be checked, looking for increasing osmolar gap, which rolls out toxic alcohol ingestion.

In the case of presence of anion gap, metabolic acidosis may help and give to physician a clue of ingestion of certain toxins like (salicylates, ethylene glycol, and methanol or other drugs which may cause high anion gap metabolic acidosis; also serum creatinine, glucose, ketones, and lactate should be tested to detect other causes of the anion gap acidosis.

C	**Chlorinated hydrocarbons, calcium salts, and crack vials**
H	Heavy metals (iron, arsenic, mercury, thallium, and lead)
I	Iodinated compounds (e.g., thyroxine)
P	Packets of drugs ("body packers"), Play-Doh potassium salts, psychotropics (e.g., phenothiazines, lithium, and cyclic antidepressants
E	Enteric-coated tablets (aspirin)
S	Salicylates, sodium salts, and sustained-release drugs
	Sustained-release preparations

Table 2.
Radiopaque toxins.

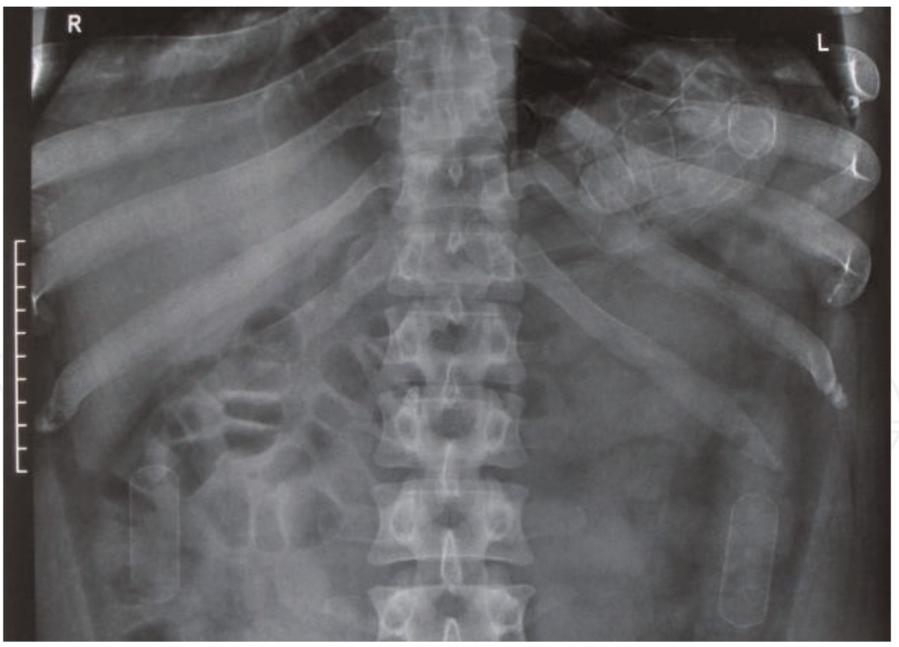

Figure 1.
Body packers.

When serum creatinine is elevated with a normal BUN, this finding is seen in the case of isopropyl alcohol toxicity (or with diabetic ketoacidosis). Co-oximetry can be used for rapid diagnosis of carbon monoxide toxicity and methemoglobinemia.

6.5 Toxicology screening

Toxicology screening is not necessary in case of nonintentional ingestion are asymptomatic patient or have clinical findings that are match with the medical history.

Drugs of abuse to opioids, benzodiazepines, cocaine metabolites, barbiturates, tricyclic antidepressants, tetrahydrocannabinol, and phencyclidine can be detected by using immunoassay screens in urine.

Positive and negative screens for drugs do not necessarily confirm diagnosis of acute poisoning but require further investigations.

6.6 Limitations of toxicologic drug screening assays

- Nonspecific—because most tests can detect only typical drugs within a class: opioids, amphetamines, benzodiazepines, cannabinoids, cocaine, barbiturates. For example, opioid screens do not detect meperidine and amphetamine screens do not detect methylenedioxymethamphetamine

- Drugs may be detected days to weeks after exposure. A positive test may not mean acute poisoning

- Cross reactivity in the case of carbamazepine, cyproheptadine, and chlorpromazine; the test can be positive for tricyclic antidepressants

- Test can be negative if tested urine was diluted.

7. Decontaminations

Decontamination of poisoned patient means removing the patient from the toxin and removing the toxin from the patient, either outside the patient's body by gross washing or inside the body by gastrointestinal decontamination or enhanced elimination.

7.1 Gross decontamination

Patient must be fully undressed and washed thoroughly with copious amount of water twice regardless of how much time has elapsed since the exposure. All the clothing must be removed and placed in plastic bags, and then the bags must be sealed; no need to neutralize an acid with a base or a base with an acid because that may lead to more tissue damage because the heat could be generated by this reaction. Using any greases or creams must be avoided because they will only keep the xenobiotic in close contact with the skin and ultimately make its removal more difficult.

Decontamination must be done in an isolated specific area. Gross decontamination is used in chemical, biological, and radiation exposure. Healthcare providers must wear universal precautions (gown, gloves, and eye protection) and sometimes may need personal protective equipment.

7.2 Ocular decontamination

In the case of eye exposures to chemical substance, initially, application of a local anesthetic agent (e.g., 0.5% tetracaine) may be needed, then copious irrigation with crystalloid solution. Lid retraction facilitates the irrigation. Alkalis cause more injury than acids because of deep tissue penetration via liquefaction so may need prolonged irrigation (1 to 2 hours). pH of conjunctival sac should be tested and irrigation should be continued until pH is <7.4.

7.3 Gastrointestinal decontamination

There are multiple methods used for gastrointestinal decontamination including:

- Emesis

Induced vomiting by ipecac syrup can decrease absorption and was used in the past but now is rarely indicated because there is no evidence supporting its effectiveness in reducing toxin absorption. It may also increase the risk of complications. Syrup ipecac may be considered in conscious, alert patients with ingestion of a potential number of toxic drugs and present in a very short time after ingestion (<1 hour).

Contradictions:

- Unprotected airway
- Corrosive/hydrocarbon ingestion
- Unstable patient status (hypotensive-seizure) [9].
- Gastric lavage

Gastric lavage is an intervention widely used to remove the ingested toxin drugs from the stomach by an orogastric tube. Because of the absence of published evidence that shows that orogastric lavage may change the outcome, now orogastric lavage is rarely indicated. It may be considered in the case of recent (<1 hour) ingestion of life-threatening amount of a toxin for which there is no effective treatment once absorbed.

Contraindications:

- Corrosive/hydrocarbon ingestion
- Supportive care/antidote likely to lead to recovery
- Unprotected airway
- Unstable, requiring further resuscitation (hypotension and seizures).

Complications:

- Aspiration pneumonia
- Water intoxication
- Hypothermia
- Laryngospasm
- Mechanical injury to gastrointestinal tract.
- Activated charcoal:

Activated charcoal is a super-heating carbonaceous material. Activated charcoal works by reducing the absorption of a substance in the gastrointestinal lumen but it is not effective in metal, alcohols, corrosives, and lithium. The most effective action can be achieved when activated charcoal is given within the first hour of ingestion. In the case of intubated patients, activated charcoal may be administered via an orogastric or nasogastric tube.

Dose:

- Children 1 to 12 years of age: 25 to 50 g or 0.5 to 1.0 g/kg (maximum dose 50 g)
- Adults: 25 to 100 g (with 50 g representing the usual adult dose).

Contraindications:
Substances not adsorbed by activated charcoal.

- Unprotected airway
- Corrosive ingestion
- Upper gastrointestinal perforation.

Complications:

- Vomiting
- Aspiration of the activated charcoal
- Reduce absorption of orally administered antidotes [10]
- Whole-bowel irrigation:

Whole-bowel irrigation is a mechanical cleansing of the whole gastrointestinal track reducing toxin absorption. The whole-bowel irrigation can be done by Polyethylene glycol solution. Polyethylene glycol is an osmotically balanced electrolyte solution; polyethylene glycol can be given orally to cooperative, awake patients. Patient positioning (head up 30°) reduces the risk of pulmonary aspiration; during whole-bowel irrigation also bowel sounds must be present. Clear rectal effluent and imaging shows the absence of foreign bodies considered as endpoint of whole-bowel irrigation treatment.

Indication:

- Iron ingestion >60 mg/kg with opacities on abdominal radiograph
- Life-threatening ingestion of diltiazem or verapamil
- Body packers or stuffers
- Slow-release potassium ingestion
- Lead ingestion (including paint flakes containing lead)
- Symptomatic arsenic trioxide ingestion
- Life-threatening ingestions of lithium
- Contraindications
- Unprotected airway.

Gastrointestinal obstruction absent bowel sound or perforation [11].
Recurrent, unstoppable vomiting.
Complications:

- Nausea and vomiting
- Pulmonary aspiration
- Vomiting, bloating, and rectal irritation.

8. Enhanced elimination

Enhanced elimination is a method used to increase the rate of toxic removal from the body so as to reduce the severity and duration of clinical intoxication.

Enhanced elimination methods are not routinely used in poisoned patients. The indications for enhanced elimination include: [4].

- Severe toxicity
- Poor outcome despite supportive care/antidote
- Slow endogenous rate of elimination.

There are different techniques to enhance elimination:

Multiple dose activated charcoal (MDAC).

MDAC is defined as at least two sequential doses of activated charcoal [12]. Multidose activated charcoal can be given via orogastric or nasogastric tube to intubated patients.

Mechanism of action:

- Prevents ongoing absorption of toxin that persists in the GI tract (modified-release preparation)
- Enhances elimination in the post absorptive phase by delayed enterohepatic recirculation or enteroenteric recirculation ("gut dialysis").

Indications:

Ingestion of a life-threatening amount of carbamazepine, dapsone, phenobarbital, quinine, salicylates, or theophylline.
Ingestion of a life-threatening amount of another toxin that undergoes enterohepatic or enteroenteric recirculation and that is adsorbed to activated charcoal.
Ingestion of a significant amount of any slowly released toxin.

Contraindications:

- Unprotected airway
- Bowel obstruction.

Complications:

- Vomiting
- Pulmonary aspiration
- Constipation
- Bowel obstruction or perforation.

Dose: no optimal dose of MDAC has been established. But the acceptable regimen of 50 g is administered every 4 hours, or 25 g every 2 hours. Study on volunteer found no difference in effectiveness of larger doses spread out over time compared to smaller, more frequent dose [13].

- Urinary alkalinization

Urine alkalinization is a treatment regimen which enhances the elimination of toxins by administration of intravenous sodium bicarbonate to produce urine with pH > or = 7.5.

Alkaline urine acts on ionization of acidotic toxins within renal tubules, stopping resorption of the ionized drug back across the renal tubular epithelium and enhancing elimination through the urine [14].

Characteristics of drugs which respond to urinary alkalinization are [15].

- Eliminated predominantly unchanged by the kidney
- Distributed primarily in the extracellular fluid compartment
- Minimal protein-bound
- Weak acids (3.0 to 7.5).

Urinary alkalinization for poisoned patients can be done by the following steps:

- Correct hypokalemia
- Start with 1 to 2 mEq/kg IV sodium bicarbonate bolus
- Infuse 100 mEq of sodium bicarbonate mixed with 1 L of D5W at 250 mL/h
- Potassium chloride (20 mEq) can be added to maintain normokalemia
- Monitoring serum potassium and bicarbonate level every 2 to 4 hours to prevent hypokalaemia
- Urine pH should be checked regularly, keeping urine pH between 7.5 and 8.5.

Indications:

- Moderate to severe salicylate toxicity
- Phenobarbital severe toxicity
- Chlorophenoxy herbicide severe toxicity
- Chlorpropamide severe toxicity.

Contraindications:

- Pre-existing fluid overload
- Renal impairment
- Uncorrected hypokalaemia.

Complications:

- Hypokalaemia

- Volume overload
- Alkalemia.
- Urinary acidification (urine pH below 5.5) with ammonium chloride or ascorbic acid was used in the past to treat toxicity of weak bases such as amphetamines, quinidine, or phencyclidine. However, this practice is not used now because of lack of evidence of efficacy and complications such as iatrogenic toxicity (from severe academia) and rhabdomyolysis may occur.
- Extracorporeal elimination includes hemodialysis, hemoperfusion, and continuous renal replacement therapies, this method has limited indications in intoxicated patients, extracorporeal elimination need critical care setting also this procure are expensive and invasive, are not always available extracorporeal elimination were used in less than 0.1% of cases reported to U.S. poison control centers in 2010 [16]. The toxins need to have a number of criteria to be effectively removed by extracorporeal elimination [17]:
- low volume of distribution (<1.0 L/kg),
- low molecular weight (<500 Da), relatively
- low protein binding
- low endogenous clearance.

Indications:
Life-threatening toxicity of

- Lithium
- Metformin lactic acidosis
- Phenobarbital
- Salicylates
- Valproic acid
- Methanol/ethylene glycol
- Potassium salts
- Theophylline.

Contraindications:

- Hemodynamic instability
- Active hemorrhage
- Severe thrombocytopenia
- Sever coagulopathy

9. Extracorporeal membrane oxygenation (ECMO)

ECMO: extracorporeal technique used when the patients are critically ill and they cannot provide an adequate amount of gas exchange or perfusion to sustain. This technique may be used in the case of severe and massive overdose especially cardiotoxic drugs (beta blockers, calcium channel blocker) [18].

10. Antidotes

Although supportive care is the main treatment of most poisoned patients, there are cases in which administration of a specific antidote is potentially life-saving. Antidote is a substance that can prevent further poisoning from specific substances. **Table 3** shows the most common antidote used in the emergency department (see **Table 1**) [4].

Toxin	Antidote
Acetaminophen	N-acetylcysteine 150 mg/kg dextrose IV over 15–60 min, then 50 mg/kg NAC IV over 4 hrs. Then 100 mg /kg NAC IV over 16 hrs.
Cholinergic (organophosphates, carbamates)	Atropine 1-2 mg every 2–3 min, until there is drying of secretions, pralidoxime (2-PAM) 70 mg/kg IV, then infusion at 500 mg/hour
Anticholinesterases	Physostigmine 0.5–1 mg IV as a slow push over 5 min and repeated every 10 min
Benzodiazepines	Flumazenil 0.2 mg repeated; max dose: 2 mg
β-Blockers	Glucagon 3–10 mg
Calcium channel blockers	Calcium gluconate 10% 10–30 mL IV
Cyanide	Amyl nitrite, sodium thiosulfate, sodium nitrite (3% solution), and Vitamin B12
Digoxin	Digoxin Fab 5–10 vials
Isoniazid	Pyridoxine (vitamin B6) 70 mg/kg IV (maximum 5 g)
Methanol, ethylene glycol	Ethanol loading: 8 ml/kg of 10% ethanol then 1–2 ml/kg/hour of 10% ethanol; fomepizole Loading: 15 mg/kg in 100 ml IV over 30 min. Maintenance: 10 mg/kg IV over 30 min every 12 hours for 48 hr.
Narcotics	Naloxone 0.1–0.4 mg, may be repeated
Tricyclic antidepressants	Sodium bicarbonate 1–2 mEq/kg IV bolus followed by 2 mEq/kg per h IV infusion
Iron	Deferoxamine IV infusion dose of 15 mg/kg/hour
Methaemoglobinaemia	Methylene blue 1–2 mg/kg (0.1–0.2 ml/kg of 1% solution) IV slowly over 5 min
Local anesthetics	Intravenous lipid emulsion 1–1.5 ml/kg 20% IV bolus over 1 min, repeat bolus at 3–5 min, then infuse 0.25 ml/kg/min
Wernicke's syndrome, wet beriberi	100 mg IV
Insulin, oral hypoglycemics	Dextrose (glucose) 1 g/kg IV

Table 3.
Antidote.

11. Disposition

If the patient has persistent and toxic effects, the patient will require prolonged care course. Admission is indicated for completing his treatment and observation; in the case of severe toxicity, the patient may need admission to intensive care unit.

In the case of mild toxicity or asymptomatic patient, a 6-hour observation period is sufficient to exclude the development of serious toxicity.

A number of toxins have delayed onset clinical toxicity, for example (but not limited to): modified-release preparations of calcium channel antagonists, selective norepinephrine reuptake inhibitors (tramadol and venlafaxine), and newer antipsychotics (amisulpride); this means that the duration of observation should be longer than usual.

The decision to admit a patient with a toxic exposure to an intensive care setting should be based upon clinical criteria that relate to the stability of the airway, respiratory system, cardiovascular system, and the patient's level of consciousness.

A retrospective study which was done in more than 200 patients with drug overdoses shows that clinical assessment in the emergency department could reliably find out patients who are at high risk for complications and need intensive care unit admission [19].

Based on the following clinical criteria: if the patient has one of any of the following clinical criteria, the patient may need admission to intensive care unit:

- $PaCO_2$ > 45 mmHg
- Intubated patient
- Seizures post-ingestion
- Unresponsiveness to verbal stimuli
- Abnormal cardiac rhythm (nonsinus)
- Atrioventricular block (Second- or third-degree)
- Systolic blood pressure below 80 mmHg
- QRS duration ≥0.12 seconds.

12. Conclusion

The first step in the approach to intoxicated patient should start with stabilization measures including protected airway and adequate ventilation and circulation and control of the convulsion.

History in poisoning cases could be difficult; especially in self-harm poisoning or comatose patients, the physician must use collateral information from friends, family, prehospital personal and medical records.

Always ask, especially about the use of over-the-counter drugs and traditional or herbal preparations.

Physical examination may help to find the toxidrome and complication of toxins; physical examination should include all systems.

Focused laboratory test helps physicians to understand the severity of toxicity and suspected toxin and guides in management, making sure the drug level is sent

at proper time not so early or late to avoid wrong interpretation; urine or blood toxicology screen assays have limited value in the case of acute over dose. Most of poisoned patients only supportive care with decontamination will be sufficient for them, but antidotes same times is the cornerstone of the treatment.

In the end, remember all the time "treat the patient, not the poison."

Author details

Ehab Said Aki* and Jalal Alessai
Emergency Department, Hamad Medical Corporation, Doha, Qatar

*Address all correspondence to: akiehab2004@gmail.com

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