General Management of Poisoned Patients

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INTRODUCTION

* Poisoning is a worldwide problem
* U.S. poison control centers documented 2.38 million human exposures in 2010, with 1146 associated deaths
* Prevention is the key to reducing unintentional poisoning deaths :

- Pharmacists , parents , Teachers and healthcare providers ??

INTRODUCTION

* Exposures occur:

-ingestion

-injection

-inhalation

-cutaneous and mucous membrane exposure

-…

INTRODUCTION

Criteria's for **nontoxic** exposure are:

(1) an unintentional exposure to a clearly identified single substance,

(2) where an estimate of dose is known, and

(3) a recognized information source (e.g., a poison control center) confirms the substance as nontoxic.

Asymptomatic patients with nontoxic exposures may be discharged after a short period of observation, providing they have access to further consultation and a safe discharge destination.

RESUSCITATION

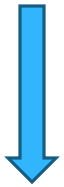
- **Resuscitation** (first priority)

- a structured **risk assessment** (benefit from an antidote, decontamination, or enhanced elimination techniques)

✓Treatment of cardiac arrest :

Advanced Cardiac Life Support **+** interventions

Patients : Young +minimal preexisting organ dysfunction



Prolonged resuscitation

* Stabilization of airway, breathing, and circulation represents initial priorities
* IV crystalloid bolus (**10 to 20** mL/kg) is first-line treatment of hypotension
* Persisting hypotension may respond to a specific antidote.
* Otherwise, cautious administration of an inotropic agent is indicated. (knowledge of the toxin's toxicodynamic properties)

ANTIDOTES

* Stabilization of airway, breathing, and circulation allows further assessment of blood glucose concentration, temperature, and conscious state.
* Although the proper use of antidotes is important, only a few are indicated before cardiopulmonary stabilization :

- naloxone for opiate toxicity,
- cyanide antidotes for cyanide toxicity
- atropine for organophosphate poisoning

HYPOGLYCEMIA

* Treat hypoglycemia with IV dextrose (glucose).
* Altered mental status when hypoglycemia cannot be excluded is an indication for IV dextrose.
* Supplemental ***oxygen, thiamine, glucose, and naloxone*** are often administered empirically as a cocktail in cases of altered mental status. (not cost-effective)
* The decision to administer an antidote should be made after a rapid collateral history is obtained and targeted examination completed.

CARDIAC ARRHYTHMIAS

* Antiarrhythmic drugs are not first-line treatment for toxin induced arrhythmias.
* Most toxin-induced arrhythmias respond to correction of hypoxia, metabolic/acid–base abnormalities, and administration of an antidote (e.g., digoxin Fab).
* Sodium bicarbonate is administered for sodium-channel blocker toxicity with cardiovascular complications, such as wide QRS complex tachyarrhythmia's.
  Ventricular tachyarrhythmia may respond to overdrive pacing

SEIZURES

* Drug-induced seizures are treated with IV benzodiazepines
- isoniazid-induced seizures require pyridoxine
- hypoglycemia and hyponatremia, should be rapidly excluded.
* Barbiturates are second-line agents for benzodiazepine-resistant seizures
  * **There is no role for phenytoin in the treatment of toxin-induced seizures; it has neither theoretical nor proven efficacy, and may worsen toxicity**

AGITATION

* treated with titrated doses of benzodiazepines.
* antipsychotic agents are often used as second-line agents for toxin-induced agitation (disadvantages, including anticholinergic and extrapyramidal effects)
* Droperidol has been associated (rarely) with QT interval prolongation and cardiac arrhythmias

HYPERTHERMIA AND HYPOTHERMIA

* temperatures of >39°C (>102.2°F) require aggressive active cooling measures to prevent complications such as rhabdomyolysis, organ failure, and DIC
* Sedation, neuromuscular paralysis, and intubation are required if active measures are ineffective.
* specific pharmaceutical agents:
- sympathomimetic (benzodiazepines)
- serotonin (cyproheptadine)
- neuromuscular malignant syndrome (bromocriptine).

* Drug-induced coma with subsequent immobility and environmental exposure or inherent drug toxicity (opioids, phenothiazines, ethanol) may produce hypothermia.

* A core temperature <32°C (<90°F) is an indication for active rewarming

NALOXONE

* Naloxone is a nontoxic, diagnostic, and therapeutic antidote. (IV, IM, or intranasally)
* Naloxone can be used as a diagnostic (respiratory rate of <12 breaths/min) suggest possible opioid exposure.
* Naloxone is titrated to clinical effect using bolus doses, typically *0.1 to 0.4* milligrams.
* Large initial bolus doses may precipitate *vomiting* and *aspiration*, acute opioid *withdrawal*, or an *uncooperative*, *agitated* patient.
* *Miosis* is an *unreliable* indicator of naloxone's adequate clinical effect

* Doses are titrated to achieve desirable ventilation and conscious state (adequate respiratory rate, normal arterial oxygen saturations on room air, and verbal or motor response to voice).

* Although naloxone may reverse the effects of opioids for 20 to 60 minutes, the effect of many opioids will outlast this time frame with possible return of respiratory depression.

* Patients should be observed for 2 to 3 hours after administration of IV naloxone

RISK ASSESSMENT

* Following initial resuscitation and stabilization, a risk assessment is performed to predict course of clinical toxicity, interventions required, and patient disposition.
* Risk assessment is formulated using history, examination, and ancillary test results.
* Acute poisoning is a dynamic process; therefore, risk assessment may change with time and requires ongoing review

HISTORY

* no clear history due to
- psychiatric illness,
- clinical effects of exposure
- fear of arrest or repercussions from family or friends.

* Information including identity of substances, doses, route of exposure , time ,Emesis ,Reason and Symptoms (from family, friends, previous medical records, and usual healthcare provider)

HISTORY

Prehospital emergency services can provide information regarding empty medication containers or the scene environment (smells, particular materials or substances present).

EXAMINATION

* A systematic physical examination can yield important clues to the nature and potential severity of an exposure
* Examine the skin folds, body cavities if appropriate, and clothing for retained tablets or substances

TABLE 176-3 Examination of the Poisoned Patient

Organ System	Examination	Example of Finding (Possible Significance)
General appearance	General demeanor and dress Signs of injury Odors Mental state Nutritional state Temperature	Unkempt (psychiatric illness) Scalp hematoma (intracranial injury) Malnourished (IV drug use, HIV infection) Smell of bitter almonds (cyanide toxicity)
Central nervous	Conscious state Pupil size and reactivity Eye movements Cerebellar function/gait	Miosis (opioids, organophosphates, phenothiazines, clonidine intoxication) Nystagmus/ataxia (anticonvulsant and ethanol toxicity)

Cardiovascular	Heart rate/blood pressure Cardiac auscultation	Murmur (endocarditis/IV drug abuse)
Respiratory	Oxygen saturation Respiratory rate Chest auscultation	Fever/crepitations/hypoxia (aspiration pneumonia) Bronchorrhea/crepitations/ hypoxia (organophosphate toxicity)
Gastrointestinal	Oropharynx Abdomen Bladder	Urinary retention (anticholinergic toxicity) Oral cavity burns (corrosive ingestion) Hypersalivation (cholinergic toxidrome)
Peripheral nervous	Reflexes Tone Fasciculations Tremor Clonus	Tremor/fasciculations (lithium toxicity) "Lead pipe" rigidity (neuromuscular malignant syndrome) Clonus/hyperreflexia (serotonin toxicity)

BOX 139.1

Agents Affecting Pupil Size

MIOSIS (COPS)

Cholinergics, clonidine, carbamates
Opioids, organophosphates
Phenothiazines (antipsychotics), pilocarpine, pontine hemorrhage
Sedative-hypnotics

MYDRIASIS (SAW)

Sympathomimetics
Anticholinergics
Withdrawal syndromes

Agents Causing Coma or Seizures

COMA (LETHARGIC)

Lead, lithium
Ethanol, ethylene glycol, ethchlorvynol
Tricyclic antidepressants, thallium, toluene
Heroin, hemlock, hepatic encephalopathy, heavy metals, hydrogen sulfide, hypoglycemics
Arsenic, antidepressants, anticonvulsants, antipsychotics, antihistamines
Rohypnol (sedative hypnotics), risperidone
Gamma-hydroxybutyrate (GHB)
Isoniazid, insulin
Carbon monoxide, cyanide, clonidine

SEIZURES (OTIS CAMPBELL)

Organophosphates, oral hypoglycemics
Tricyclic antidepressants
Isoniazid, insulin
Sympathomimetics, strychnine, salicylates
Camphor, cocaine, carbon monoxide, cyanide, chlorinated hydrocarbons
Amphetamines, anticholinergics
Methylxanthines (theophylline, caffeine), methanol
Phencyclidine (PCP), propranolol
Benzodiazepine withdrawal, botanicals (water hemlock, nicotine), bupropion, GHB
Ethanol withdrawal, ethylene glycol
Lithium, lidocaine
Lead, lindane

Agents With a Characteristic Odor

ODOR	POSSIBLE SOURCE
Bitter almonds	Cyanide
Carrots	Cicutoxin (water hemlock)
Fruity	Diabetic ketoacidosis, isopropanol
Garlic	Organophosphates, arsenic, dimethyl sulfoxide (DMSO), selenium
Gasoline	Petroleum distillates
Mothballs	Naphthalene, camphor
Pears	Chloral hydrate
Pungent aromatic	Ethchlorvynol
Oil of wintergreen	Methylsalicylate
Rotten eggs	Sulfur dioxide, hydrogen sulfide
Freshly mowed hay	Phosgene

Agents Causing Skin Findings

DIAPHORETIC SKIN (SOAP)

Sympathomimetics
Organophosphates
Acetylsalicylic acid or other salicylates
Phencyclidine (PCP)

DRY SKIN

Antihistamines, anticholinergics

BULLOUS LESIONS OR BLISTERS

Barbiturates and other sedative-hypnotics
Mustard gas
Snakes and spiders

FLUSHED OR RED APPEARANCE

Anticholinergics, niacin
Boric acid
Carbon monoxide (in morbid states)
Cyanide (rare)

CYANOSIS

Ergotamine
Nitrates
Nitrites
Aniline dyes
Phenazopyridine
Dapsone
Agent causing hypoxemia, hypotension, or methemoglobinemia

ACNEIFORM RASH

Bromides
Chlorinated aromatic hydrocarbons

TOXIDROMES

* Substances belonging to a particular pharmaceutical/chemical class often produce a cluster of symptoms and signs, or “toxidrome” enabling the identification of potential toxins when a clear history is unavailable

Toxidrome Symptoms

CHOLINERGIC

Muscarinic (DUMBELLS)
Diarrhea, diaphoresis
Urination
Miosis
Bradycardia
Bronchorrhea
Emesis
Lacrimation
Lethargic
Salivation

Nicotinic: Days of Week
Mydriasis
Tachycardia
Weakness
Tremors
Fasciculations
Seizures
Somnolent

ANTICHOLINERGIC

Hyperthermia (HOT as a hare)
Flushed (RED as a beet)
Dry skin (DRY as a bone)
Dilated pupils (BLIND as a bat)
Delirium, hallucinations (MAD as a hatter)
Urinary retention (DRY as a bone)
Tachycardia

OPIOID

Miosis
Hypoventilation
Depressed mental status/coma
Withdrawal
Diarrhea
Mydriasis
Goose flesh
Tachycardia
Lacrimation
Hypertension
Yawning
Cramps
Hallucinations
Seizures (with ethyl alcohol [ETOH] and benzodiazepine withdrawal)

Sympathomimetic
Hyperthermic
Flushed
Diaphoretic
Mydriatic
Agitated
Tachycardic
Seizures

Predicting Toxicity From Vital Signs

BRADYCARDIA (PACED)
Propranolol (β-blockers), poppies (opioids), propoxyphene, physostigmine
Anticholinesterase drugs, antiarrhythmics
Clonidine, calcium channel blockers
Ethanol or other alcohols
Digoxin, digitalis

TACHYCARDIA (FAST)
Free base or other forms of cocaine, Freon
Anticholinergics, antihistamines, antipsychotics amphetamines, alcohol withdrawal
Sympathomimetics (cocaine, caffeine, amphetamines, phencyclidine [PCP]), solvent abuse, strychnine
Theophylline, tricyclic antidepressants (TCAs), thyroid hormones

HYPOTHERMIA (COOLS)
Carbon monoxide
Opioids
Oral hypoglycemics, insulin
Liquor (alcohols)
Sedative-hypnotics

HYPERTHERMIA (NASA)
Neuroleptic malignant syndrome (NMS), nicotine
Antihistamines, alcohol withdrawal
Salicylates, sympathomimetics, serotonin syndrome
Anticholinergics, antidepressants, antipsychotics

HYPOTENSION (CRASH)
Clonidine, calcium channel blockers
Rodenticides (containing arsenic, cyanide)
Antidepressants, aminophylline, antihypertensives
Sedative-hypnotics
Heroin or other opioids

HYPERTENSION (CT SCAN)
Cocaine
Thyroid supplements
Sympathomimetics
Caffeine
Anticholinergics, amphetamines
Nicotine

RAPID RESPIRATION (PANT)
PCP, paraquat, pneumonitis, phosgene
Acetylsalicylic acid (ASA) and other salicylates
Noncardiogenic pulmonary edema, nerve agents
Toxin-induced metabolic acidosis

SLOW RESPIRATION (SLOW)
Sedative-hypnotics (barbiturates, benzodiazepines)
Liquor (alcohols)
Opioids
Weed (marijuana)

SEGMENT/INTERVAL	APPEARANCE	AGENT(S)
QT/QTc	Prolonged	Antipsychotics (typical and atypical), citalopram, hydrofluoric acid, methadone, ethylene glycol (oxalate byproduct)
T wave	Peaked	Hydrofluoric acid (hyperkalemia)
	Flattened	Lithium
U wave		Barium, beta-agonists, lithium, methylxanthines (caffeine, theophylline), toluene

DIAGNOSTIC TESTING

* A serum acetaminophen concentration is a routine screening test in poisoned patients.
* An electrocardiogram is a useful test to detect cardiac conduction abnormalities
* Toxicologic screening tests seldom directly influence patient management, and toxicology screening has limitations
* Toxicologic screening may be appropriate for medicolegal reasons, especially in pediatric cases
* A positive urine drug screen for an illicit substance is an indication to involve local child protection services

Toxicologic Electrocardiogram Manifestations

SEGMENT/INTERVAL	APPEARANCE	AGENT(S)
P wave	Absent	Digoxin Cholinergics Hyperkalemia
	Notched	Quinidine
PR interval	Prolonged	Beta-antagonists, calcium-channel antagonists, magnesium
QRS interval	Prolonged	Type 1 antidysrhythmics, cocaine, diphenhydramine, tricyclic antidepressants
ST segment	Scooped	Digoxin ("Salvador Dali's moustache")

DECONTAMINATION

* Decontamination is required for toxic exposures affecting large dermal areas.
* Healthcare providers wearing personal protective equipment
(if indicated) or observing universal precautions (gown, gloves, eye protection) should assist with undressing and washing the patient using copious amounts of water.
* Contaminated clothing is collected, bagged, and properly disposed.
* Decontamination ideally occurs in a separate area adjacent to the ED, minimizing cross-contamination

OCULAR DECONTAMINATION

* Eye exposures may require local anesthetic (e.g., 0.5% tetracaine) instillation and lid retractors to facilitate copious irrigation with crystalloid solution.
* Alkalis produce greater injury than acids due to deep tissue
  penetration via liquefaction so that prolonged irrigation (1 to 2 hours) may be required.
* Ten minutes after irrigation (allowing equilibration of crystalloid and conjunctival sac pHs), conjunctival sac pH is tested.
  Irrigation continues until pH is <7.4.
* Ophthalmologic consultation is indicated for all ocular alkali injuries.

GASTROINTESTINAL DECONTAMINATION

* Gastric decontamination is not a routine part of poisoned-patient management
* Gastric decontamination may be considered in individual

patients after a three-question risk-benefit analysis:

(1) Is this exposure likely to cause significant toxicity?

(2) Is gastrointestinal decontamination likely to change clinical outcome?

(3) Is it possible that gastrointestinal decontamination will cause more harm than good

Emesis

* Traditionally, ipecac syrup was administered to induce vomiting, theoretically emptying the stomach of poisons.
* ipecac may be used in rare circumstances in remote locations, but this recommendation has been questioned.

There is no role for the induction of emesis in the ED

Orogastric Lavage

* ***orogastric lavage*** is now rarely indicated. changes outcome ? complications ??
* may be considered in cases of:

- ingestion of a life-threatening amount of poison within the previous hour where institution of supportive care and antidotal therapy would not ensure full recovery.

* When orogastric lavage is performed in a resuscitation area:
  • Ensure a protected airway if consciousness level is reduced.
  • Use a 36 to 40F-gauge orogastric tube (22 to 24F in children).
  • Position the patient on the left side with the head down 20 degrees.
  • Pass lubricated tube down the esophagus a distance equal to that between chin and xiphoid process.
  • Confirm tube position by insufflation of air.
  • Gently lavage with 200 mL (10 mL/kg in children) of warm tap water.
  • Continue until returned fluid is clear.
  • Consider administration of activated charcoal via orogastric tube before removal

Single-Dose Activated Charcoal

* Toxins within the GI lumen are adsorbed onto the activated charcoal.
* Activated charcoal does not effectively adsorb metals, corrosives, and alcohols.
* The decision to give activated charcoal requires individual patient risk assessment and is not considered routine management.
* may be effective when given >60 minutes after ingestion of substances known to slow GI motility (e.g., anticholinergics) or after massive ingestion of a substance associated with bezoar formation (e.g., salicylates).
* There are insufficient published data supporting the routine use of
  a cathartic agent added to activated charcoal

Substances That Do *Not* Bind to Activated Charcoal

PHAILS

Pesticides
Heavy metals
Acids/alkalis
Iron
Lithium
Solvents

Whole-Bowel Irrigation

* Polyethylene glycol can be administered orally to cooperative, awake patients, but consider formal airway control if consciousness is likely to deteriorate.
* Minimize risk of pulmonary aspiration during whole-bowel irrigation by:

patient positioning (head up 30 degrees), ensuring bowel sounds are present during fluid administration, and utilization of cuffed endotracheal tubes.

* Nonsurgical treatment of asymptomatic body drug packers using whole-bowel irrigation is increasingly common
* metoclopramide may be required to control polyethylene glycol–induced gastric distension and vomiting.
* The endpoint is clear rectal effluent and imaging demonstrating absence of foreign bodies

ENHANCED ELIMINATION

* MULTIDOSE ACTIVATED CHARCOAL
* URINARY ALKALINIZATION

MULTIDOSE ACTIVATED CHARCOAL

* ***Indications:***

- Carbamazepine coma (reduces duration of coma)
- Phenobarbital coma (reduces duration of coma)
- Dapsone toxicity with significant methemoglobinemia
- Quinine overdose
- Theophylline overdose if hemodialysis/hemoperfusion unavailable

URINARY ALKALINIZATION

* Alkaline urine favors ionization of acidotic drugs within renal tubules, preventing resorption of the ionized drug (most effective for weak acids)
* Hypokalemia will reduce the effectiveness of urinary alkalinization.
* Indication: moderate to severe salicylate toxicity when criteria for hemodialysis have not been met.
* Urinary alkalinization for adult patients can be instituted as follows:
  - Correct any existing hypokalemia.
  - Administer a 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.

* 20 mEq of potassium chloride may be added to the solution to maintain normokalaemia.
  • Monitor serum potassium and bicarbonate every 2 to 4 hours to detect hypokalemia or excessive serum alkalinization.
  • Check urine pH regularly (every 15 to 30 minutes), aiming for a pH of 7.5 to 8.5
* **GOAL :**

serum pH of approximately 7.5 and a urinary pH of approximately 8.0

DISPOSITION

* should be part of initial risk assessment.
* Admission is indicated if the patient has persistent and/or severe toxic effects
* In most cases, a 6-hour observation period is sufficient to exclude the development of serious toxicity.
* Onset of clinical toxicity can be delayed after :
  - calcium channel antagonists
  - tramadol, venlafaxine
  - newer antipsychotics (amisulpride)

  hence a period of extended observation is indicated.

* Patients who have deliberately self-poisoned require appropriate mental health assessment before disposition

از توجهتان متشکرم