DM SEMINAR
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Approach to the critically ill poisoned patient

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CLINICAL SCENARIO

- A 48 year old unconscious woman is brought to the hospital. No history available. She is convulsing. She is incontinent for urine and stool. On exam her VS: T99, HR50, RR24, BP90/60 . Skin is diaphoretic. She is drooling. Pupils are constricted. Lungs diffuse wheezing.

INTRODUCTION

- **A** high index of suspicion for intoxication is warranted in the practice of critical care medicine.
- The protean manifestations of intoxication challenge even the most astute clinicians, particularly when patients present with altered mental status, are critically ill or when there is no history of intoxication.

EPIDEMIOLOGY

- 2000 annual report, 63 poison centers reported a total of 2,168,248 human toxic exposure cases. Adults accounted for approximately one third of exposures.
- Most exposures were unintentional (71% of cases) and involved a single toxic substance (92%). Oral ingestion was the commonest route of exposure.
- Most exposures occurred at the patient's own residence, and most patients (75%) were managed on-site with assistance from a poison information center and did not require an emergency department visit.
- Only 3% of patients required critical care. The mortality rate was higher in intentional rather than unintentional exposures (79% vs 10.5%, respectively).

Litovitz TL et al. 2000 annual report of the American Association of Poison Control Centers toxic exposure surveillance system. Am J Emerg Med 2001;19:337–395

PGI emergency data .
15 year data 1990-2004
Unpublished thesis data..

- Data reflective only of number requiring admissions. EMOPD excluded.
- Bias toward organophosphate poisoning likely. Also might overestimate mortality data.
- Total number of poisonings admitted: 1420
- Total number of organophosphate poisonings admitted: 557
- Admission as percentage of total: 39.2%
- Outcome Data available for 483 cases.
- Number alive/discharged: 397(82.2%)
- Mortality: 86(17.8%)

POISONING IN INDIA

Table I. Aetiology of poisoning in 84 patients admitted with respiratory failure to the respiratory intensive care unit.

Diagnosis	Number of patients (%) (n=84)	Mortality number (%) (n=17)
Organophosphates	52 (61.9)	8 (15.4)
Barbiturates	8 (9.5)	0
Paraquat	5 (5.9)	3 (60)
Benzodiazepines	4 (4.8)	0
Carbon monoxide	3 (3.6)	0
Others		
Unknown	3 (3.6)	1 (33.3)
Methanol	1 (1.2)	1 (100)
Ethanol	1 (1.2)	0
Lithium	1 (1.2)	0
Corrosive	1 (1.2)	1 (100)
Printer dye	1 (1.2)	1 (100)
Carbamate	1 (1.2)	0
Organochlorates	1 (1.2)	1 (100)
Mercury fumes	1 (1.2)	1 (100)
Opiates	1 (1.2)	0

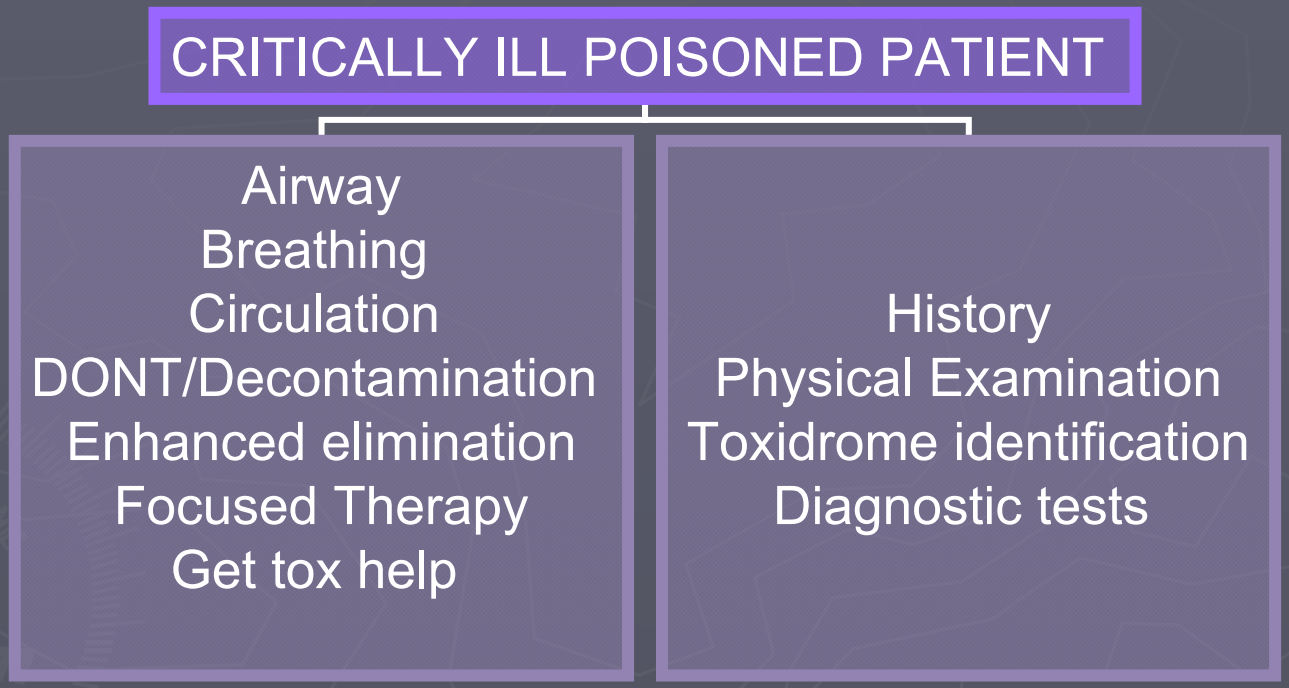
Agarwal R, Srinivas R, Aggarwal A N, Gupta D. Experience with paraquat poisoning in a respiratory intensive care unit in North India. *Singapore Med J 2006; 47(12):1033-1037*

THE CRITICALLY ILL POISONED

- Most critically ill poisoned patients have acutely reversible conditions that will clearly benefit from intensive care intervention.
- Toxicological emergencies have confusing presentations, do not have a well recognized clinical course or predictable complications , nevertheless may be rapidly fatal .
- The therapies, antidotes and complications may be unfamiliar to the **Intensivists.**

Ron A. The therapeutic efficacy of critical care units. Identifying subgroups of patients who benefit. Arch Intern Med 1989;149:338-341

Approach to the Poisoned Patient



Approach to the Poisoned Patient

History

- When to suspect

- Past history of drug overdose or substance abuse
- Suicidal ideation or previous suicide attempt
- History of other psychiatric illness
- Agitation and hallucinations
- Stupor or coma
- Delirium or confusion
- Seizures
- Cardiopulmonary arrest
- Aspiration
- Poly pharmacy

- Time of ingestion
- Medications in the household
- Amount ingested
- Onset of symptoms
- Intentionality
- Underlying medical conditions

Mokhlesi. Toxicology in the critically ill patient. Clin Chest Med 24 (2003) 689–711

Diagnosis

- Physical Exam:
  - Vital signs and general appearance
  - Thorough PE
  - Close attention to neuro exam
    - Pupils
    - Reflexes and posture
    - Mental status
  - Bowel sounds
  - Mucous membranes and skin moisture/appearance
  - Characteristic odors
  - Nosebleeds, needle tracks, “huffer rash”, blistering

Physical Examination
Toxic Vital Signs

Bradycardia (PACED)
Propranolol (beta-blockers), poppies (opiates), physostigmine
Anticholinesterase drugs, antiarrhythmics
Clonidine, calcium channel blockers
Ethanol or other alcohols
Digoxin, digitalis

ERICKSON et al.The Approach to the Patient with an Unknown Overdose.Emerg Med Clin N Am 25 (2007) 249–281

Physical Examination
Toxic Vital Signs

Tachycardia (FAST)
Free base or other forms of cocaine, freon
Anticholinergics, antihistamines, antipsychotics, amphetamines, alcohol withdrawal
Sympathomimetics (cocaine, caffeine, amphetamines, PCP), solvent abuse, strychnine
Theophylline, TCAs, thyroid hormones

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Physical Examination
Toxic Vital Signs

Hypotension **(CRASH)**

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

Hypertension **(CT SCAN)**

Cocaine
Thyroid supplements
Sympathomimetics
CaffeineAnticholinergics,
Amphetamines
Nicotine

ERICKSON et al.The Approach to the Patient with an Unknown Overdose.Emerg Med Clin N Am 25 (2007) 249–281

Physical Examination
Toxic Vital Signs

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

ERICKSON et al.The Approach to the Patient with an Unknown Overdose.Emerg Med Clin N Am 25 (2007) 249–281

Physical Examination
Toxic Vital Signs

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

ERICKSON et al.The Approach to the Patient with an Unknown Overdose.Emerg Med Clin N Am 25 (2007) 249–281

Physical Examination
Toxic Vital Signs

Rapid respiration (PANT)
PCP, paraquat, pneumonitis (chemical), phosgene
ASA and other salicylates
Noncardiogenic pulmonary edema, nerve agents
Toxin-induced metabolic acidosis

ERICKSON et al.The Approach to the Patient with an Unknown Overdose.Emerg Med Clin N Am 25 (2007) 249–281

Physical Examination
Toxic Vital Signs

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

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Physical Examination
Toxic Vital Signs

COMA

L: Lead, lithium

E: Ethanol, ethylene glycol, ethchlorvynol

T: Tricyclic antidepressants, thallium, toluene

H: Heroin,heavy metals,hydrogen sulfide, hypoglycemics

A: Arsenic, antidepressants, anticonvulsants, antipsychotics,antihistamines

R: Rohypnol (sedative hypnotics), risperidon

G: GHB

I: Isoniazid, insulin

C: Carbon monoxide, cyanide, clonidine

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Physical Examination
Toxic Vital Signs

Agents that affect pupil size

Miosis (COPS)

Cholinergics, clonidine, carbamates
Opiates, organophosphates
Phenothiazines, pilocarpine, pontine Hemorrhage
Sedative-hypnotics

Mydriasis (SAW)

Sympathomimetics
Anticholinergics
Withdrawal

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Toxidromes

- **Definition**: Constellation of signs and symptoms seen in poisoning, characterized by the type of substance
- Recognizing a toxidrome guides treatment without definitive knowledge of the particular substance

Mokhlesi. Toxicology in the critically ill patient. Clin Chest Med 24 (2003) 689–711

Classification of Toxidromes

Alteration of PNS

- **Diminished**: **Anticholinergic -** Dhatura, Antipsychotics,mushroom,TCA
- **Enhanced**: **Cholinergic -** Pesticide, Sarin, Soman

Alteration of SNS

- **Diminished**: **Opioid/Sedative –** BDZ, Barbiturates
- **Enhanced**: **Sympathomimetic** Amphetamine/Methamphetamine Cocaine , ectasy, withdrawal

Alteration of *both* PNS and SNS

- **Serotonin Syndrome**

Common Toxidrome Findings

Physical Findings	SYMPATHETIC	ANTI CHOLINERGIC	CHOLINERGIC	SEROTONIN	Sedative-hypnotic
RR	Increased	No change	No change	Increased	Decreased
HR	Increased	Increased	Decreased	Increased	Normal/ decreased
Tem	Increased	Increased	No change	Increased	Normal/ decreased
BP	Increased	NoChange/increased	No change	Increased	Normal/ decreased

Common Toxidrome Findings

Physical Findings	SYMPATHETIC	ANTI CHOLINERGIC	CHOLINERGIC	SEROTONIN	Sedative-hypnotic
Mental status	Alert/ agitated	Depressed/ Confused/ hallucinate	Depressed/ Confused/	Agitated	Depressed
pupils	Dilated	Dilated	Constrict	Dilated	Normal
Mucus membrane	Wet	Dry	Wet	Wet	Normal
skin	Diaphoretic	Dry	Diaphoretic	Diaphoretic	Normal

Diagnostic Considerations

- Before proceeding, consider other aspects of the differential diagnosis (CVA, trauma, meningitis, post-ictal state, DKA, behavioral or psych disorders).
- Labs to evaluate glucose, acid-base status and electrolytes, BUN/Cr, carboxyhemoglobin, hepatic enzyme levels, urinalysis, serum osmolality,
- EKG
- Radiography
- Save samples of blood, urine, gastric contents
- In spite of providing direct evidence of intoxication, screening tests alter management in 5% of cases.

Brett AS. Implications of discordance between clinical impression and toxicology analysis in drug overdose. Arch Intern Med 1988; 148:437–441

Diagnostic Considerations

- Toxins requiring quantitative levels at a set point:
  - Acetaminophen
  - Carbon monoxide
  - Ethanol, ethylene glycol
  - Heavy metals (24 hour urine)
  - Iron
  - Methanol
  - Methemoglobin
- Toxins requiring quantitative serial levels
  - Aspirin/salicylates, tegretol, digoxin, phenobarbital, phenytoin, VPA, theophylline

Diagnostic Considerations

- **MUDPILES CAT** for high anion gap acidosis
  - **M**ethanol or metformin
  - **U**remia
  - **D**KA
  - **P**araldehyde or phenformin
  - **I**ron, INH, Ibuprofen
  - **L**actic acidosis
  - **E**thylene glycol
  - **S**alicylates
  - **C**yanide
  - **A**lcohol or acids (valproate)
  - **T**oluene or Theophylline

Low Anion gap

-- Lithium

-- Bromide

Toxins associated with increased OG

- Methanol
- Ethanol
- Ethylene glycol
- Acetone
- Isopropanol

Serum OSM: 2[Na] + [Glc]/18 + [BUN]/2.8

OG: Measured OSM-Calculated OSM

Normal OG: -3 to 10 mOSM/kg H2O

Oxygen saturation gap

- An oxygen saturation gap is present when there is more than a 5% difference between the saturation that is calculated from an arterial blood gas analyzer, which uses an assumed standard oxygen-hemoglobin dissociation curve, and the saturation that is measured by co-oximetry. Toxins associated with an increased oxygen saturation gap

carbon monoxide, methemoglobin, cyanide, and hydrogen sulfide. barbiturates, benzodiazepines, cannabinoids, cocaine,opioids, and phencyclidine.

Mokhlesi. Toxicology in the critically ill patient. Clin Chest Med 24 (2003) 689–711

Management Priorities

- ABCs and antidotes.
- DON’T (dextrose + oxygen + naloxone + Thiamine)
- Expose for exam, labs/Enhance elimination
- Friends/Family for history.
- GI decontamination

Management Considerations

- Supportive care is the mainstay of therapy and recovery and may involve decontamination, antidotal therapy, enhanced elimination techniques
- Systemic support for airway security, ventilation, hemodynamic stability, and adequate CNS function.
- Assessment for the need of Intensive unit care for the critically poisoned.
- Activating multi-faceted team approach early and call poison centre for help.

PREVENTION OF ABSORPTION OF POISON

Gastric

Ipecac

- Should *not* be routinely used

Activated Charcoal

- preferred method for gastric decontamination and may be indicated even in the patient with equivocal exposure

Gastric Lavage

Gastro-Intestinal

- Whole Bowel Irrigation

Mokhlesi B et al. Adult toxicology in critical care: part I: general approach to the intoxicated patient. Chest 2003;123(2):577– 92.

"Gastric lavage should not be employed routinely, if ever, in the management of poisoned patients."

J Toxicol Clin Toxicol 2004;42:7:933.

Multiple dose activated charcoal (MDAC)

- MDAC is a potential method of enhanced elimination. can interrupt enterohepatic and enteroenteric recirculation. when the toxins have been absorbed, acting as “gut dialysis.”
- Twenty-five grams every 2 to 4 hours is a reasonable regimen.

Substances adsorbable by activated charcoal (ABCD)

- **A**ntimalarials (quinine), aminophylline (theophylline)
- **B**arbiturates (phenobarbital)
- **C**arbamazepine
- **D**apsone

Substances not adsorbable by activated charcoal (PHAILS)

- **P**esticides, potassium
- **H**ydrocarbons
- **A**cids, alkali, alcohols
- **I**ron, insecticides
- **L**ithium
- **S**olvents

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SPECIFIC ANTIDOTES

Poison	Antidote
Acetaminophen	Acetylcysteine
Acetylcholinesterases, OP's, physostigmine	Atropine
Iron salts	Deferoxime
Methanol, Ethylene glycol	Ethanol
Mercury, lead	Metal Chelators
Narcotic drugs	Naloxone
Anti/muscarinics-cholinergics	Physostigmine

David P .Antidote Use in the Critically Ill Poisoned Patient
Journal of Intensive Care Medicine, 2006: 21;5, 255-277

Extracorporeal elimination of poison

- **Toxins accessible to hemodialysis (UNSTABLE)**
- **U**remia
- **N**o response to conventional therapy
- **S**alicylates
- **T**heophylline
- **A**lcohols (isopropanol, methanol)
- **B**oric acid, barbiturates
- **L**ithium
- **E**thylene glycol

Enhanced elimination by Charcoal hemoperfusion

- Theophylline
- Barbiturates
- Carbamazepine
- Paraquat
- Glutethimide

Criteria for Admission of the Poisoned Patient to the ICU

- Target organ dysfunction
- Respiratory depression (Paco2 45 mm Hg)
- Emergency intubation
- Seizures
- Cardiac arrhythmia (second- or third-degree atrioventricular block)
- Systolic BP 80 mm Hg
- Glasgow coma scale score 12
- Need for emergency dialysis, hemoperfusion, or ECMO
- Pulmonary edema induced by toxins (including inhalation) or drugs

Mokhlesi B et al. Adult toxicology in critical care: part I: general approach to the intoxicated patient. Chest 2003;123(2):577– 92.

Criteria for Admission of the Poisoned Patient to the ICU

- Hypothermia or hyperthermia including neuroleptic malignant syndrome
- Tricyclic or phenothiazine overdose manifesting anticholinergic signs, neurologic abnormalities, QRS duration 0.12 s, or QT 0.5 s
- Administration of pralidoxime in organophosphate toxicity
- Antivenom administration in Crotalidae, coral snake, or arthropod envenomation
- Need for continuous infusion of naloxone
- Hypokalemia secondary to digitalis overdose (or need for digoxinimmune antibody Fab fragments)

Mokhlesi B et al. Adult toxicology in critical care: part I: general approach to the intoxicated patient. Chest 2003;123(2):577– 92.

POISONING AND ICU – EVIDENCE

- To examine the characteristics of patients admitted to the Medical Intensive Care Unit (MICU) after intentional drug overdose. DESIGN: Retrospective chart review, CONCLUSIONS: Neurologic findings were the best indicators of serious complications after drug overdose. Therefore, patients with a Glascow Coma Scale score of more than six, and who are not intubated, may not need admission to an intensive care unit.

E N Heyman. Intentional drug overdose: predictors of clinical course in the intensive care unit. Heart Lung.1996 ;25 (3):246-52

POISONING AND ICU – EVIDENCE

- **Acute overdose is a common cause of admission to the ICU but has a mortality rate of only 2%. Poisoned patients represented 13.8% of all admissions and 22% of these patients were admitted to the ICU occupying 6% of the available ICU bed-days. Among the patients admitted to the ICU, tricyclic antidepressants, benzodiazepines and alcohol were the most frequently used compounds.**

  Henderson A Experience with 732 acute overdose patients admitted to an intensive care unit over six years. Med J Aust 1993 Jan 4;158(1):28-30.

SAPS II scores calculated within the first 24 hours recognized as good prognostic indicator among patients with acute OPP that required ICU admission. It is concluded that SAPS II score above 11 within the first 24 hours is a predictor of poor outcome in patients with acute OPP requiring ICU admission.

S. Shadnia .A simplified acute physiology score in the prediction of acute organophosphate poisoning outcome in an intensive care unit. Human & Experimental Toxicology (2007) 26, *623—627*

PESTICIDE POISONING

- The number of intoxications with OPs is estimated at some 3,000,000 per year. Fatality rates of 20% are common and the World Health Organization (WHO) has estimated that 200,000 people die each year from pesticide poisoning.
- Most insecticides that are used are organophosphates or carbamates.
- Organophosphate compounds:80% of pesticide-related hospitalization
- Both compounds exert their toxicity through inhibition of acetylcholinesterase and accumulation of acetylcholine at synapses

WHO in collaboration with UNEP. Public Health Impact of Pesticides used in Agriculture. WHO: Geneva; 1990.

PESTICIDE POISONING

- Cause: agricultural use, accidental exposure, suicide.
- Intoxication: onset of symptoms and signs vary with the route and degree of exposure:
- Usually less than 12-24 h,
- Symptoms may persist day to weeks
- Carbamate less toxic and poor CNS penetration than Organophosphate

CLINICAL FEATURES

- Muscarinic overstimulation: -increase parasympathetic tone
  - SLUDGE (salivation, lacrimation, urination, diarrhea, gastrointestinal, emesis)
- Nicotinic effect:
  - Muscle fasciculations, cramping and muscle weakness, tachycardia, hypertension, stimulate adrenal gland
- Cholinergic excess: in CNS
  - Delirium, confusion, coma and seizure
- Cause of death:

  respiratory failure combined with depressed CNS and increase bronchial secretion

Sungur M. Intensive care management of organophosphate insecticide poisoning. Crit Care. 2001 Aug;5(4):211-5.

Laboratory finding

- Routine lab finding: nonspecific: nonketotic hyperglycemia, hypokalemia, leukocytosis, pulmonary edema.
- Definite diagnosis of OP intoxication:
  - Decreased cholinesterase activity in the blood
- RBC cholinesterase is more accurate but less available, serum cholinesterase is more sensitive but less specific
- Mild case: deceased cholinesterase level to 20-50%, severe case decreased less than 10%
- Some chronic, lowgrade intoxication may show normal level of cholinesterase
- Carbamate poisoning is less useful due to cholinesterase level may return to normal in 4-8 hr

Management

- Establishment of airway and supportive therapy
  - Initial objective treatment: establishment of airway and adequate ventilation
  - In agricultural exposures it is extremely important to remove all contaminated clothing and cleanse the hair and skin thoroughly to decrease absorption.

Atropine use

- Atropine:
  - Atropine is competitively blocking the action of Ach at muscarinic receptor (not nicotinic receptor), decrease parasympathetic stimulation
  - Adult dose: 2 mg IV (6 mg IV for life-threatening cases) followed by 2 mg every 15 minutes. If after 3–5 min a consistent improvement has not occurred, then double the dose, and continue to double each time that there is no response until adequate atropinization has occurred
  - **Target end-points for atropine therapy**
- 1. Clear chest on auscultation with no wheeze
- 2. Heart rate >80 beats/min
- 3. Pupils no longer pinpoint
- 4. Dry axillae
- 5. Systolic blood pressure >80 mmHg
- **IMPROVEMENT IN ALL 5 PARAMETERS NEEDED.**

ATROPINE INFUSION

- Ensure that the two IV drips have been set up (one for fluid and drugs, the other for atropine). Give 500–1000 ml (10–20 ml/kg) of normal saline over 10–20 min.
- In the infusion, try giving 10–20% of the total amount of atropine that was required to load the patient every hour. Larger doses may be required if oximes are not available. It is rare that an infusion rate greater than 3–5 mg/ hour is necessary.

- **Markers used to assess atropine toxicity**
- 1. Confusion
- 2. Pyrexia
- 3. Absent bowel sounds (Urinary retention)

Reduce agitation with diazepam(preferred over haloperidol) 10 mg given by slow IV push,repeated as necessary in an adult, up to 30–40 mg per 24 hours.

Michael Eddleston.*Early management after self-poisoning with an organophosphorus or carbamate pesticide – a treatment protocol for junior doctors Critical Care* 2004, **8:**R391-R397

Oximes : Current status

- The clinical benefit of oximes for OP pesticide poisoning is not clear, being limited by the type of OP, poison load, time to start of therapy, and dose of oxime.
- Oximes are not recommended for carbamate poisoning.

- Current World Health Organisation guidelines recommend giving a 30 mg/kg loading dose of pralidoxime over 10–20 min, followed by a continuous infusion of 8–10 mg/kg per hour until clinical recovery(12–24 hours after atropine is no longer required or the patient is extubated) or 7 days, whichever is later.

- Where obidoxime is available, a loading dose of 250 mg is followed by an infusion giving 750 mg every 24 hours. Too rapid administration will result in vomiting, tachycardia and hypertension (especially diastolic hypertension).

Michael Eddleston.*Early management after self-poisoning with an organophosphorus or carbamate pesticide – a treatment protocol for junior doctors Critical Care* 2004, **8:**R391-R397

EXPERIENCE AT PGI

- Continuous 2-PAM infusion(7.5 mg/ kg body weight/h) along with aggressive atropinisation (2.5 mg q 10 min) after initial decontamination improved the outcome but not the duration of MV in severely intoxicated patients with organophosphate compounds who required assisted ventilation

Singh S, Chaudhry D, Behera D, Gupta D, Jindal SK. Aggressive atropinisation and continuous pralidoxime (2-PAM) infusion in patients with severe organophosphate poisoning: experience of a northwest Indian hospital. Human Exp Toxicol2001; 20:15–18.

ALUMINIUM PHOSPHIDE(ALP)

- AlP is a highly toxic, low cost rodenticide. Upon exposure to moisture, it liberates phosphine gas, which is absorbed rapidly by inhalation, dermally, or gastrointestinally.
- In a study of 559 cases of acute poisoning in India, 68% were due to AlP exposure, with 60% mortality.

- Toxicity of phosphine is related to oxidant free radicals and associated inhibition of enzymes of metabolism, such as cytochrome c oxidase

Singh D, Jit I, Tyagi S. Changing trends in acute poisoning in Chandigarh zone: a 25-year autopsy experience from a tertiary care hospital in northern India. *Am J Forensic Med Pathol* 1999;**20**:203-10

CLINICAL FEATURES

- Clinical features of AlP poisoning are severe vomiting, resistant hypotension, and metabolic acidosis.
- Characteristic garlic smell of phosphine gas
- A characteristic feature of AlP poisoning is myocardial suppression and resistant hypotension.

Goel A .Pesticide poisoning.
Natl Med J India 2007 Jul-Aug; 20(4):182-91.

MANAGEMENT

- Current management is supportive; however, survival is unlikely if more than 1.5 g is ingested
- Novel therapies such as *N*-acetylcysteine, replenishing cellular glutathione, and magnesium, which has been reported to have antioxidant properties.
- Other agents include trimetazidine, which switches myocyte metabolism to glucose from fatty acids, thus reducing oxygen consumption, and may have a potential role.

Duenas A, Perez-Castrillon JL, Cobos MA, *et al.* Treatment of the cardiovascular manifestations of phosphine poisoning with trimetazidine, a new anti-ischemic drug. *Am J Emerg Med* 1999;**17**:219â€“20

Paraquat poisoning

- Paraquat, a widely-used herbicide, remains a major cause of death in developing countries.
- Paraquat poisoning can be classified into three categories:
- Mild poisoning (20 mg per kg) minor gastrointestinal symptoms but usually fully recover;
- Severe poisoning (20-40 mg per kg)

  acute renal failure,acute lung injury and progressive pulmonary fibrosis with death occurring in two to three weeks from respiratory failure;
- Fulminant poisoning (40 mg per kg)

  multiple organ failure and death within hours to a few days after ingestion.

MANAGEMENT

- Management of paraquat poisoning has remained mostly supportive and the results of treatment for paraquat poisoning, including absorbents, pharmacological approaches, radiotherapy, haemodialysis and haemoperfusion were disappointing.
- Paraquat poisoning is characterised by severe pulmonary inflammation, and is also the primary cause of death. One major step towards attenuation of lung inflammation has been the use of immunosuppressive drugs including glucocorticoids and cyclophosphamide.

Agarwal R, Srinivas R, Aggarwal A N, Gupta D.Experience with paraquat poisoning in a respiratory intensive care unit in North India Singapore Med J 2006; 47(12) : 1034

RECENT ADVANCES

- **OP pesticides poisoning** : blood alkalinization with sodium bicarbonate and also magnesium sulfate as adjunctive therapies are promising.

  Balali-Mood M .Neurotoxic disorders of organophosphorus compounds and their management. Arch Iran Med. 2008 Jan;11(1):65-89.
- **Acetaminophen toxicity:** A 21-hour intravenous infusion protocol with the total administered NAC dose of 300 mg/kg has recently been approved by the US FDA.
- The latest toxicology antidotes include fomepizole for ethylene glycol and methanol poisoning and high-dose insulin for calcium channel antagonist poisoning.
- **Carbon monoxide–poisoning**. The current Cochrane Database concludes that existing randomized trials do not establish whether the administration of HBO to patients who have carbon monoxide poisoning reduces the incidence of adverse neurologic outcomes

ERICKSON et al. The Approach to the Patient with an Unknown Overdose. Emerg Med Clin N Am 25 (2007) 249–281

TAKE HOME MESSAGE

- **The management of the critically ill poisoned patient who has an unknown exposure can be diagnostically and therapeutically challenging.**
- **The history and physical examination, along with a small dose of detective work, can often provide the clues to the appropriate diagnosis.**
- **Careful resuscitation with appropriate use of antidotes, followed by good supportive care and observation in a rapid and timely manner is required to manage this subset of poisoned patients .**

- **Careful monitoring, appropriate management, early recognition of need for intensive care may decrease the mortality rates among these patients.**