Approach to the critically ill poisoned patient

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Department of pulmonary medicine
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.
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.
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).

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Unpublished thesis data..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%)
### Table I. Aetiology of poisoning in 84 patients admitted with respiratory failure to the respiratory intensive care unit.

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

CRITICALLY ILL POISONED PATIENT

Airway
Breathing
Circulation
DONT/Decontamination
Enhanced elimination
Focused Therapy
Get tox help

History
Physical Examination
Toxidrome identification
Diagnostic tests
**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

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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

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
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
- Caffeine
  - Anticholinergics,
  - Amphetamines
- Nicotine

Physical Examination

Toxic Vital Signs

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

Physical Examination
Toxic Vital Signs

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

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

Physical Examination

Toxic Vital Signs

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

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), risperidom

**G**: GHB

**I**: Isoniazid, insulin

**C**: Carbon monoxide, cyanide, clonidine

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

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.

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, ecstasy, withdrawal

Alteration of both PNS and SNS

- Serotonin Syndrome
# Common Toxidrome Findings

<table>
<thead>
<tr>
<th>Physical Findings</th>
<th>SYMPATHETIC</th>
<th>ANTI CHOLINERGIC</th>
<th>CHOLINERGIC</th>
<th>SEROTONIN</th>
<th>Sedative-hypnotic</th>
</tr>
</thead>
<tbody>
<tr>
<td>RR</td>
<td>Increased</td>
<td>No change</td>
<td>No change</td>
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<td>Decreased</td>
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<td>HR</td>
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<td>Decreased</td>
<td>Increased</td>
<td>Normal/decreased</td>
</tr>
<tr>
<td>Tem</td>
<td>Increased</td>
<td>Increased</td>
<td>No change</td>
<td>Increased</td>
<td>Normal/decreased</td>
</tr>
<tr>
<td>BP</td>
<td>Increased</td>
<td>No change/increased</td>
<td>No change</td>
<td>Increased</td>
<td>Normal/decreased</td>
</tr>
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<th>SEROTONIN</th>
<th>Sedative-hypnotic</th>
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</thead>
<tbody>
<tr>
<td>Mental status</td>
<td>Alert/agitated</td>
<td>Depressed/Confused/hallucinate</td>
<td>Depressed/Confused/</td>
<td>Agitated</td>
<td>Depressed</td>
</tr>
<tr>
<td>pupils</td>
<td>Dilated</td>
<td>Dilated</td>
<td>Constrict</td>
<td>Dilated</td>
<td>Normal</td>
</tr>
<tr>
<td>Mucus membrane</td>
<td>Wet</td>
<td>Dry</td>
<td>Wet</td>
<td>Wet</td>
<td>Normal</td>
</tr>
<tr>
<td>skin</td>
<td>Diaphoretic</td>
<td>Dry</td>
<td>Diaphoretic</td>
<td>Diaphoretic</td>
<td>Normal</td>
</tr>
</tbody>
</table>
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.

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
  - Methanol or metformin
  - Uremia
  - DKA
  - Paraldehyde or phenformin
  - Iron, INH, Ibuprofen
  - Lactic acidosis
  - Ethylene glycol
  - Salicylates
  - Cyanide
  - Alcohol or acids (valproate)
  - Toluene or Theophylline

Low Anion gap
  -- Lithium
  -- Bromide
Toxins associated with increased OG

- Methanol
- Ethanol
- Ethylene glycol
- Acetone
- Isopropanol

Serum OSM: \( 2[\text{Na}] + [\text{Glc}]/18 + [\text{BUN}]/2.8 \)

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
- hydrogen sulfide
- barbiturates
- benzodiazepines
- cannabinoids
- cocaine
- opioids
- phencyclidine

Management Priorities

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

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

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)
- Antimalarials (quinine), aminophylline (theophylline)
- Barbiturates (phenobarbital)
- Carbamazepine
- Dapsone

Substances not adsorbable by activated charcoal (PHAILS)
- Pesticides, potassium
- Hydrocarbons
- Acids, alkali, alcohols
- Iron, insecticides
- Lithium
- Solvents

## SPECIFIC ANTIDOTES

<table>
<thead>
<tr>
<th>Poison</th>
<th>Antidote</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaminophen</td>
<td>Acetylcysteine</td>
</tr>
<tr>
<td>Acetylcholinesterases, OP’s, physostigmine</td>
<td>Atropine</td>
</tr>
<tr>
<td>Iron salts</td>
<td>Deferoxime</td>
</tr>
<tr>
<td>Methanol, Ethylene glycol</td>
<td>Ethanol</td>
</tr>
<tr>
<td>Mercury, lead</td>
<td>Metal Chelators</td>
</tr>
<tr>
<td>Narcotic drugs</td>
<td>Naloxone</td>
</tr>
<tr>
<td>Anti/muscarinics-cholinergics</td>
<td>Physostigmine</td>
</tr>
</tbody>
</table>

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)
  - Uremia
  - No response to conventional therapy
  - Salicylates
  - Theophylline
  - Alcohols (isopropanol, methanol)
  - Boric acid, barbiturates
  - Lithium
  - Ethylene 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

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 organophosphatate 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)

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 Glasgow Coma Scale score of more than six, and who are not intubated, may not need admission to an intensive care unit.

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 beds-days. Among the patients admitted to the ICU, tricyclic antidepressants, benzodiazepines and alcohol were the most frequently used compounds.


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.

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

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
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.**
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
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.

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

Clinical features of AlP poisoning are severe vomiting, resistant hypotension, and metabolic acidosis.

A characteristic feature of AlP poisoning is myocardial suppression and resistant hypotension.
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.

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.

RECENT ADVANCES

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


► **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.

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.