MANAGEMENT OF COMMON POISONING: CHANGING TRENDS & CHALLENGES

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ABSTRACT

Knowledge of poisons, their clinical manifestations, remedial measures, etc. and the causing of death by poisoning, was prevalent in India since time immemorial. The ability to diagnose and manage a case of poisoning (clinical toxicology), identification of the poison on analysis of the body fluids and the samples (analytical toxicology) and understanding the legal aspects of acute and chronic poisoning may have changed with the passage of time. The recent past has witnessed important advances that aid both in the diagnosis and the treatment of poisoned patient, however, the general principles of management including what to do, in what order to do and why to institute a certain procedure, remain unchanged. This paper examines and discusses epidemiology, actions, diagnosis and management of common poisoning cases reporting to the health care systems particularly in the Northern India.

INTRODUCTION

The last few decades have seen tremendous advances in the fields of agriculture, industrial technologies and medical pharmacology. These advances have been paralleled with remarkable changes in the trends of acute poisoning in developing countries, including India. In a study from Chandigarh conducted during 1970-1979[1], a total of 312 cases of poisoning were reported, wherein it was observed that 30.12% fatalities were due to barbiturates poisoning, 19.23% due to agrochemicals and 17.95% due to metallic irritants and corrosives. Over the next decade i.e. 1980-1989, another 555 cases of poisoning were reported from the same region and 31.35% fatalities were attributed to aluminium-phosphide, 27.03% to organophosphates and carbamates, 8.83% to barbiturates and 9.36% to metallic irritants and corrosives[2]. A total of 1035 cases of acute poisoning were studied during 1983 to 1996 at All India Institute of Medical Sciences, New Delhi and the trends showed the increasing use of agro-chemicals[3]. Yet another study from Rohtak in 1993 –1994 analyzed 559 cases of poisoning[4] and Aluminium Phosphide was found to be the most common poison. The present scenario is not different from these reports and agrochemicals continue to be the most common agents responsible for suicidal and/or accidental poisoning.[5]

Mortality due to acute poisoning is a worldwide phenomenon and has enormous medical, legal and social significance. In the management of poisoning cases, a quick and correct decision indeed is always desirable. The familiar adage “TREAT THE PATIENT, NOT THE POISON” is appropriately stated, as it is of no use to make vigorous attempts at removing the ingested poison from patient’s stomach in case there is no breathing or the blood pressure is not recordable. The first step, therefore, is to assess the patient’s condition and to use whatever method is necessary to stabilize his condition. Attempts to identify the toxic agent and to assess its quantity and time of exposure should follow. Decision, whether the substance consumed is toxic or non-toxic, is very essential for planning the treatment.

A toxic substance is one that causes tissue damage (defined as irreversible or slowly reversible structural or functional changes in one or more organ systems that result directly from the poison or its metabolites in the body and not indirectly due to respiratory depression or hypo-tension) during intoxication. Methyl alcohol, Aspirin, Acetaminophen, Theophyllin and heavy metals, when ingested in large amounts, are known to cause tissue damage in patients even if supportive care is provided. Barbiturates and

other sedative / hypnotic drugs, on the other hand, do not cause tissue damage in the presence of proper supportive care.

General approach to manage common poisoning can however be considered under the following headings: Maintain Airways, Breathing and Circulation (ABC); Remove unabsorbed poison by gastric lavage simultaneously taking steps to retard its absorption; Neutralize absorbed poison by using specific antidotes (if available); Remove absorbed poison by diuresis or catharsis and provide supportive care. But treatment plans differ depending upon so many factors like the nature, dose, and form etc of poison as well as route of their administration. Commonly encountered poisons include agrochemicals like aluminium phosphide, organophosphorus compounds and organochlorines and hypnotics and sedative drugs like barbiturates. Bronchodilators have also been reported as common poisons to cause drug-induced deaths.

Aluminium Phosphide (ALP), a widely used solid fumigant, was declared as an ideal fumigant pesticide in 1973 for its effectiveness, easy to use and low cost properties. Available as tablets of 'Celphos', 'Alphos' or 'Quickphos', each tablet weighing 3.0 g liberates 1.0 g of phosphine gas (PH_3). PH_3 being gaseous in nature diffuses uniformly throughout the stored grains, leaving non-toxic residues in the form of phosphite and hypophosphite of aluminium without affecting the food value of grains.

Epidemiology: Isolated cases of exposure to PH_3 have been reported in the world literature[6], but in India, a series of case reports and studies have appeared from different northern states[7]. The poisoning has been steadily increasing and has achieved alarming epidemic proportion[8]. The poisoning involving primarily the youth is mostly suicidal, occasionally accidental and rarely homicidal[9]. Recently, cases of poisoning with exposed compounds and its combined toxicity with ethyl alcohol have been reported[10, 11].

Mechanism of action: Initial studies carried out on different animals reported non-competitive binding of cytochrome oxidase by phosphine leading to valency change in the haeme component of haemoglobin[12, 13], but later studies, reported significant inhibition of catalase leading to accumulation of hydrogen peroxide[14]. More recent studies reported extra-mitochondrial release of hydrogen peroxide and liberation of oxygen free radicals[15] causing lipid peroxidation and protein denaturation of cell membrane leading to hypoxic cell damage. However, the exact mechanism of action of ALP is still unclear[16].

Diagnosis of ALP poisoning is based on: (a) history of ingestion of the poison, (b) clinical manifestations, (c) Foul or decaying fish like breath odour and (d) cardiac arrhythmias and metabolic acidosis. The confirmation of diagnosis is done by qualitative silver nitrate impregnated paper test for treatment purposes and by chemical analysis for medicolegal purposes.

Management: The main aim of management is to sustain life with appropriate resuscitation measures till PH_3 is excreted from the body. Hence early recognition and institution of therapy are mandatory. The steps to reduce mortality during first 24 hours include:

(a). Preventing absorption of PH_3 through GI tract that can be achieved by:

- Meticulous gastric lavage with potassium permenganate ($KMNO_4$) 1:1000 solution, to be repeated twice or thrice so as to remove / oxidise unabsorbed poison.
- Slurry of activated charcoal (30 to 100 g) may be given to adsorb PH_3.
- Judicious use of antacid orally and H_2 blocker intravenously for symptomatic relief of gastrointestinal manifestations of PH_3.
- Medicated liquid paraffin or magnesium sulphate may be given to accelerate its excretion through gut.

(b). Reducing organ toxicity:

- In the absence of specific antidote and high affinity of PH_3 for enzyme systems, organ toxicity develops rapidly. Most of the organ toxicity is hypoxic due to oxidant injury produced by PH_3 and heart is the most vulnerable organ. Hence, use of magnesium sulphate as an antioxidant has shown significant reduction in the mortality[17]. Magnesium sulphate in addition to having antioxidant effect, is also useful as an anti-arrhythmic and anti-hypoxic agent in ALP poisoning and as such acts as a double edged weapon for protection of the cells in the presence of hypoxia.

(c). Enhancing PH_3 excretion:

- PH_3 is stable and partially water-soluble. It is excreted through breath and urine, therefore, adequate hydration and renal

perfusion by low dose dopamine, 4 – 6 mg / kg / minute must be maintained. Though contraindicated in shock, diuretics may be tried in patients with stable blood pressure of around 80 mm Hg.

(d). Supportive measures:

- Hypoxia is managed by oxygen inhalation, airway patency by endotracheal intubation or assisted ventilation if necessary. Blood gas analysis should be regularly monitored.
- Shock is managed by intravenous fluids, given during first 3 to 6 hours, guided by CVP, PCWP and monitoring of electrolytes. Blood pressure should be maintained above 70 mm of Hg. Low dose dopamine (4 – 6 mg/kg/minute) and hydrocortisone (200 to 400 mg I.V. after 4 to 6 hours) have been reported to be effective. Steroids combat shock, check the capillary leakage in the lungs and potentiate the responsiveness of shock to catecholamines.
- Arrhythmias (both tachy- and bradyarrhythmias) may be controlled by magnesium sulfate that has a membrane stabilizing effect. Conventional anti-arrhythmic drugs like digoxin, xylocaine, etc are reported to be ineffective.
- Acute Respiratory Distress Syndrome (ARDS) can be managed by delivering 100% oxygen by face masks fitted with reservoir inspiratory bags at moderate flow rate of 5 to 10 L, to achieve PO_2 of 60 to 70 with lowest inspired fraction of O_2. Mechanical respiratory support and positive end expiratory pressure (PEEP) therapy is given in haemodynamically unstable patients.

Organophosphorus compounds (OPC) are widely used as agricultural, industrial and domestic insecticides. Poisoning with OPC may occur in isolation after exposure or in epidemics after ingestion of contaminated foodstuffs. Most of these compounds are available either as organophosphates (Malathion, Parathion, Methylparathion, Isomalathion, Diazinon, Dichlorovas, etc.) or carbamates (Carbanyl, Matacil, etc). They are used as sprays after dilution with organic solvents or water. The available formulations contain 1 to 95% of an active ingredient and accordingly the toxicity varies widely.

Mechanism of action: Organophosphorus compounds are potent inhibitors of true acetylcholinestrase (AchE) present in central nervous system (CNS) and the red blood cells (RBC) and pseudo- cholinesterase (Pseudo-ChE) present in liver, plasma and serum. The inhibition of these enzymes is due to irreversible binding of phosphate radicals of organophosphates to active sites of enzymes. In case of carbamates this binding is reversible. The pharmacological and toxicological effects are due to accumulation of acetylcholine at synapses resulting in initial stimulation followed by paralysis of neurotransmission at cholinergic synapsis[18] present in CNS, somatic nerves, autonomic ganglion, para-sympathetic nerve endings and some sympathetic nerve endings like in sweat glands[19].

Diagnosis is based on: (1) history and circumstances leading to exposure, (2) clinical manifestations like broncho-constriction, fasciculations, pinpoint pupils etc., (3) clinical and therapeutic response to atropine and oximes. Confirmation of diagnosis is by measurement of anticholinestrase enzyme in RBCs or plasma pseudocholinestrase enzyme for treatment purpose and chemical analysis of body fluids (blood, urine, gastric lavage) for medicolegal purpose.

Management: All cases of OPC poisoning should be sent to hospital as quickly as possible. Although symptoms may develop rapidly, delay in onset or steady increase in severity may be seen up to 24 hours after ingestion. The therapy may be graded according to severity of intoxication[20].

- Latent poisoning (Serum ChE activity 50 to 90% of normal value) having no clinical manifestations has excellent prognosis without any treatment. However, observation for few hours is necessary.
- Mild poisoning (Serum ChE activity 20 - 50% of normal value) demands atropine 1 – 2 mg I.V and pralidoxime (PAM) 1 gm I.V stat to be repeated if necessary. Prognosis is good.
- Moderate poisoning (Serum ChE activity 10 to 20% of normal value) with widespread muscarinic and nicotinic effects without pulmonary oedema or respiratory paralysis demands atropine 1 – 2 mg I.V every 20 to 30 minutes till signs and symptoms of poisoning disappear or the signs of atropinization like clearing of rales and drying of pulmonary secretions appear. Pralidoxime 1 gm I.V stat is repeated if necessary. Treatment with atropine, with same dose at increasing intervals may need to be continued for 2 – 3 days.
- Severe poisoning (Serum ChE activity less than 10% of normal value) may demand following modalities:

(a). Evacuation of stomach

- Meticulous gastric lavage with lukewarm water or sodium chloride to remove the unabsorbed poison. However, the success

of this procedure depends upon the time since ingestion and speed of absorption of poison.
- Slurry of activated charcoal (30 to 100 g) may be given to adsorb the poison.
- Catharsis with sodium or magnesium sulphate may be given to accelerate its excretion in faeces.

(b). Prevention of absorption from other sites

- Remove the contaminated clothing.
- Meticulous washing of skin with alkaline soap or sodium bicarbonate solution.
- Extensive eye irrigation with water or saline.
- In the event of inhalation remove the patient to fresh air.

(c). Supportive measures

- Maintain airway by oropharyngeal suction or endotracheal tube.
- Maintain respiration with Ambu bag or mechanical means.
- Monitor blood gas analysis, respiratory rate, pulse, blood pressure and urine output.
- Administer intravenous fluids.
- If convulsions are not controlled with atropine and PAM, diphenylhydantoin may be administered.

(d). Administration of specific antidote

- Intermittent atropine therapy is given in the dose of 2 – 5 mg I.V in adults and 0.05 mg/kg I.V slowly in children, every 5 to 10 minutes till parasympathetic manifestations are controlled or early signs of atropinization appear. Tachycardia and papillary dilation are not reliable indicators of atropinization. The maintenance dose is given after the initial bolus at a continuous infusion at the rate of 0.02 – 0.08 mg/kg/hr. continued for 3 – 5 days and slowly withdrawn on 6^{th} or 7^{th} day. Sudden withdrawal may produce relapse or exacerbation of signs and symptoms.
- Some studies have reported that high dose continuous administration of atropine (150 mg in 5% Dextrose) drip over a period of 6 hours is equally effective as intermittent therapy.
- Oximes (cholinesterase enzyme reactivators) as for example praldoxime is given in dose of 1 – 2 g I.V over 5 – 10 minutes or mixed in 250 ml of normal saline and infused over 30 minutes. The dose may be repeated after one hour and subsequently every 6 – 12 hours if muscle or diaphragmatic weakness or coma are not relieved. In severe poisoning praldoxime can be administered by continuous infusion (Adults: 500 mg/hr to a maximum of 12 g/24 hr and Children: 9 – 19 mg/kg/hr after an initial bolus). The titration should be based on clinical response and the oxime therapy should be monitored by measuring cholinesterase activity.

Organochlorines - commonly used, as pesticides have become an indispensable part of the agricultural world. The term 'pesticide' has been defined by the US Federal Environmental Pesticide Control Act as (1) any substance or mixture of substances intended for preventing, destroying, repelling or mitigating any pest (insect, rodent, nematode, fungus, weed, other forms of terrestrial or aquatic plant or animal life, virus bacteria or any microorganism which the administrator declares to be pest. (2) Any substance or mixture of substances intended for use as a plant regulator, defoliant or desiccant.[21] Based on the combination of biological activity and chemical structure, the pesticides have been classified as: (i) Chlorinated ethane derivatives such as DDT and its analogs, methoxychlor, etc., (ii) Hexachlorocyclohexanes such as Lindane, (iii) Cyclodiene compounds such as chlordane, aldrin, dieldrin, endrine, endosulfan, etc.

DDT (dichloro-diphenyl trichloro ethane) was synthesized by Zeidler in 1874 but its insecticidal properties were discovered by Muller in 1939. In 1950s and 1960s, it was used in India, on a large scale in National Malaria Eradication Program but due to the development of resistant strains and its metabolites being found to accumulate in natural food chains, its use became markedly restricted.

Management includes emesis with syrup of Ipecac, gastric lavage with 2 to 4 liters of tap water, followed by activated charcoal and cathartic, ventilatory support, if there is respiratory failure and seizure control with diazepam. Solvents used to dissolve DDT are known to complicate the situation by causing CNS depression leading to less seizures but more severe respiratory depression.

Barbiturates Introduced in 1903, barbiturate overdose became a leading cause of drug-induced death by the early 1970s.

Recognition of the overdose and abuse potential led to their replacement by the safer and more efficacious benzodiazepines as sedatives. However, they continue to be a common anticonvulsant, particularly in the pediatric and refractory adult population. They are also widely used to treat anxiety, pain and sleep disorders. The basic structure of all barbiturates is barbituric acid, which has no intrinsic central nervous system depressant properties. It is the addition of an alkyl or aryl group that confers them sedative properties. Based on their elimination half-lives, they are divided into four categories:

- Ultra-short acting – thiopental, methohexital, thiamylal, etc.
- Short acting – pentobarbital, secobarbital, hexobarbital etc.
- Intermediate acting – amobarbital, aprobarbital, butabarbital, etc.
- Long acting – mephobarbital, Phenobarbital, metharbital, etc.

Barbiturates act via inhibitory g-aminobutyric acid (GABA) synapses in the brain. They may also depress noradrenergic activity selectively. They abolish central respiratory drive leading to respiratory depression (the commonest cause of death in barbiturate poisoning). High doses of barbiturates suppress skeletal, smooth and cardiac muscle leading to depressed myocardial contractility, vasodilatation and hypotension. Reduced gastrointestinal mobility results in ileus. Biochemical effects include binding to cytochrome P450 and induction of hepatic microsomal enzymes.

The onset of symptoms depends on the type of barbiturate ingested. Victims of short-acting barbiturate overdoses develop symptoms within 15 to 30 minutes and these effects peak in 2 to 4 hours. Toxic effects of long acting barbiturates begin at 1 to 2 hours and peak at 6 to 18 hours. Early fatalities result from cardio-respiratory arrest. Later causes of death include circulatory failure, aspiration pneumonia, and pulmonary and cerebral edema.

Management: Emesis is contraindicated. Because of decreased gastrointestinal mobility and delayed gastric emptying, gastric lavage may be performed with a large bore tube up to 6 to 8 hours post-ingestion, taking adequate measures for airway protection. Barbiturates are well adsorbed by charcoal. The suggested dose of charcoal is ten times the ingested dose of barbiturate or 1g/kg in adults. Repeat doses of activated charcoal have been reported to reduce the average serum half-life of intravenous and oral Phenobarbital whereas serial activated charcoal has been reported to decrease the Phenobarbital elimination half-life in the overdose setting; however, the exact role of serial charcoal has not been confirmed.

To achieve elimination enhancement, hemodialysis is most effective for long-acting barbiturates, which display less protein binding and lipid solubility than other barbiturates. Its use, however, should be restricted to those patients of long-acting barbiturate poisoning who fail to respond to supportive care or who have lethal blood barbiturate level. Hemoperfusion is more effective than dialysis for short and intermediate-acting barbiturates. However, rebound may occur from the release of barbiturates from tissue stores that may be accompanied by clinical deterioration.

Urinary alkalinization increases Phenobarbital excretion 5 to 10 times and also enhances metharbital excretion. Forced alkaline diuresis requires a urine flow of 3 – 4 ml / kg / min and a pH over 7.5. Care must be taken not to aggravate cerebral or pulmonary edema with excessive fluid loads. Alkaline diuresis is ineffective for short and intermediate-acting barbiturates. Furthermore the use of alkaline diuresis requires adequate renal and cardiac function to be effective and therefore, is not indicated in patients requiring vasopressor support.

SUPPORTIVE MEASURES:

- Take a careful history estimating the amount of drug ingested and time of ingestion. Evaluate for associated trauma and signs of drug abuse.
- Respiratory arrest is the major cause of early death. Assess the patency of the airways and the adequacy of ventilation first. Appropriate corrective measures include supplemental oxygen, head tilt, chin lift, intubation and assisted ventilation.
- Establish an intravenous line with Ringer's lactate & give a fluid challenge of up to 2 liters for hypotensive patients, at the same time monitoring for complications of alkalosis and fluid overload.
- Patients who have ingested long-acting barbiturates may be comatose for as long as 2 to 3 days. Furthermore, severely intoxicated patients with significant depression of myocardial function (manifested by hypotension unresponsive to fluid challenge) require hemodynamic monitoring. Such patients may require pulmonary artery catheter and / or arterial pressure lines to guide fluid management. Early use of hemodialysis / hemoperfusion needs to be considered in such patients.

- EEG displays a predictable progression of changes depending on the depth of coma and an EEG pattern dominated by frequencies in the alpha range is usually associated with recovery without neurologic sequelae. However, it should be remembered that EEG might occasionally be isoelectric without the presence of irreversible brain damage in severe cases.

Benzodiazepines (BZD) – are by far the safest and most commonly used hypnotics / sedatives. They are the drugs of first choice as antianxiety agents and in addition are used as anticonvulsants, muscle relaxants and general anesthetics. They act on the BZD-GABA receptor complex in the CNS and augment GABA-mediated chloride channel opening like barbiturates but by acting at a different site on the complex.

Benzodiazepines have a flatter dose response curve, a wider therapeutic index and margin of safety. They have excellent oral bioavailability, are highly lipophilic and have good CNS penetrability. The duration of action of various drugs of this class determine their clinical use. For example shorter acting drugs (oxazepam, temazepam, triazolam, etc.) are used as hypnotics / sedatives. These drugs do not have active metabolites and hence there is no prolongation in the duration of drug effects. The longer acting BZDs (diazepam) are used mostly as antianxiety, anticonvulsant and muscle relaxant. Physical dependence is seen with the benzodiazepines particularly with the long acting agents, when used for a prolonged period as in anxiety neurosis.

Benzodiazepines are rarely fatal even in high doses unless other CNS depressants are concomitantly ingested. Ethanol, in addition to synergizing with the CNS depression of benzodiazepines, also enhances their absorption from the gastro-intestinal tract. The effects of an overdose are generally prolonged sleep (arousable) unless there is cardio-respiratory depression. However, the availability of specific antidote – flumazenil with certain supportive measures like maintenance of airway, plasma volume, renal output and cardiac function has rendered the mortality almost negligible.

Bronchodilators – may occasionally be associated with poisoning due to drug overdose. Those commonly used include methylxanthines, beta-agonists and anticholenergics. Amongst all the drugs used for bronchial asthma, theophylline has the greatest potential for serious toxicity. A variety of preparations containing theophylline and aminophylline are currently available. Theophylline has a narrow therapeutic index and can produce toxic effects towards the high end of its therapeutic range (serum concentrations > 30 mg / L).

Management: Acute theophylline toxicity requires emergency management. The patient is often alert and oriented, having tachycardia, metabolic acidosis, hypokalemia, hyperglycemia and leucocytosis. Gastric lavage with a large bore nasogastric tube should be instituted immediately followed by 1 g / kg up to at least 30 g of activated charcoal every hour. The repeat dose of charcoal acts by trapping theophylline in the gastrointestinal tract as it back-diffuses into the lumen of the intestines and is adsorbed to the charcoal. Even if the toxicity occurs due to intravenous administration, activated charcoal should be given in the manner prescribed (gastrointestinal dialysis). Whole bowel irrigation may be useful in the removal of sustained release preparations. Theophylline level should be monitored every hour unless two successive levels show decline. In case the serum theophylline levels continue to rise, or seizures or ventricular arrhythmias occur, charcoal hemoperfusion should be initiated. In laboratory studies, Phenobarbital has been shown to protect against theophylline-induced seizures. O_2 and respiratory support are significant.

Chronic toxicity occurs due to prolonged period of excessive exposure to theophylline. Patient often presents with anorexia, nausea, palpitations or vomiting and occasionally seizures. Management is mainly symptomatic and supportive with gastric evacuation and close monitoring.

Poisoning with locally available plant poisons

It will be appropriate to mention here that acute poisoning is a medical emergency, which poses a major health problem all over the world. However, its type, associated morbidity and mortality vary from place to place and change over a period of time. Knowledge of general pattern of poisoning in a particular region can be of great significance in early diagnosis and treatment of such cases, thus leading to a decrease in mortality and morbidity.

Common problems faced in the management of poisoning

- The patient with alleged history of consumption of some poisonous substance, or his relatives, due to some ulterior motives, may not provide proper history regarding the poisonous substance. They may also try to disguise it as an episode of illness/gastroenteritis, etc. This deliberate uncooperative attitude by the patient/ relatives causes great difficulty to the treating doctor.
- Poisoning patients, on coming to a govt. dispensary/hospital, first come in contact with an EMO / GDMO, who usually has little knowledge of the various manifestations of particular poisons and the means to diagnose and treat them. Even when a proper history is given, the doctor on duty may not know the exact chemical constituents of the alleged poison, whose trade name is told to them.
- Proper history taking should include: a) the alleged poison consumed, b) its approximate quantity, c) date, time and place of consumption, d) whether taken alone or in combination with other drugs/ alcohol, etc., e) all the clinical manifestations observed by the patient/ relatives prior to coming to the hospital, f) history of depression/ recent familial or financial or career loss etc. Exact records regarding the treatment given, progress etc. should be well maintained. Such a history recording is usually not seen in the case sheets.
- Most of the time, almost all govt. hospitals and dispensaries are in acute shortage of specific antidotes and life saving drugs. Infrastructural facilities like emergency care, analytical and diagnostic laboratories etc. are also lacking in most of these hospitals.
- Proper collection, preservation and handling of samples in the casualty and emergency wards, is a must for the same to be allowed as evidence by a Court of Law. This exact knowledge is found wanting in the doctors working in the emergency wards and the casualties, which are the main centers where the samples have to be collected, preserved and sealed for onward submission to the chemical analyzer.
- The absence of a specialized toxicology wing, even in a tertiary care hospital, is very much felt, when due to wrong diagnosis and treatment, as a result of lack of infrastructural facilities, the life of the victim is compromised.

CONCLUSION

During the recent past, mortality as a result of acute poisoning has been mainly due to agrochemicals, which appears to be a byproduct of the “green revolution” in India. Increase in crop yields in the granary of India may be the result of the use of high yield varieties of seeds, which are thought to be more disease-prone than native varieties. This may have necessitated the enhanced use of insecticides & pesticides. Higher production also implies storage problems & further need for using grain fumigants. On the other hand, occasional crop failure, despite the use of high-tech agriculture often resulted in the frustrated farmers committing suicide by using the same pesticides used for better crop yields. Involvement of more & more agricultural workers in acute accidental poisoning also indicates the lack of adequate preventive care & protective gear during agriculture related activities. Furthermore, over the last few decades, an alarming increase in drug overdose has been reported all over the world. This has made the management of poisoning not only complex but also more challenging.

A high index of suspicion coupled with a good history of the drugs that patient had been taking, may help in making an early diagnosis that in turn may be life saving at times. The management of poisoning, in general, involves stabilization of the patient, a quick clinical evaluation, attempts at eliminating the poison (from either the gastrointestinal tract, skin, eyes, etc.) administration of an antidote, followed by supportive therapy, that in turn requires, skilled staff, specially trained for this purpose. Accordingly, we recommend well-equipped toxicological emergency wings, at least, at the secondary and tertiary health care levels throughout the country.

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