

Research in Pharmacy and Health Sciences

Review Article

Specific Poisons in Pakistan: A Mini Review

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Abstract:

Poisoning either results from intentional or unintentional exposures, recreational drug use, and therapeutic use of drugs or other agents. Although mortality and serious morbidity in poisonings are uncommon, patients requiring hospitalization often require intensive care. The appropriate management of poisoned patients should improve outcome and decrease complications. Some specific poisons in Pakistan are pesticides, hydrocarbons, iron, insecticides, benzodiazepines, organophosphates etc.

Received: 11-05- 2016

Revised: 26-05-2016

Accepted: 03-06-2016

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Funding: Nil

Competing Interests: Nil

Keywords: *Poison, Symptoms, Treatment, Antidote*

INTRODUCTION:

Poisoning may result from intentional or unintentional exposures, recreational drug use, and therapeutic use of drugs or other agents. Although mortality and serious morbidity in poisonings are uncommon, patients requiring hospitalization often require intensive care. The appropriate management of poisoned patients should improve outcome and decrease complications. Evidence-based information is minimal in toxicology because the variety of drugs and doses that patients are exposed to limits the ability to conduct clinical trials of specific interventions. Current recommendations for severely poisoned patients are based on limited data from animal and human studies, case reports, pharmacokinetic information, known pathophysiology, and often consensus opinion. Studies in animal models and human volunteers do not replicate clinical situations commonly encountered in patients. Therapeutic guidelines can be suggested but may not be supported by definitive evidence.[1]

Poisoning and toxic exposures are major health problems worldwide. In fact, every individual is exposed to toxic chemicals in sub-toxic doses. The expansion in pharmaceutical and chemical industry during the last century has led to an increased accidental and intentional exposure to these chemicals. The Chemical Abstract Service (CAS) registry, a division of the American chemical society has listed 83 million chemical substances. The overall toxicity data of these chemicals is limited and commonly called "data gaps". The toxicity data on high production volume chemicals is limited to only 14 to 25% of products. The epidemiological data on poisoning is very limited in Pakistan, as there is a scarcity of poison surveillance. The studies done in Pakistan are generally case series, based on experiences in a single medical center or intensive care unit (ICU) (9-14). In a national health survey of Pakistan, poisoning was the second commonest

cause of unintentional injuries after fall in people aged five years and above. A hospital-based case series in Karachi, Pakistan reviewed 1900 ICU records and found 40% of them were related to poisoning. The overall mortality was 5.6% and organophosphates were found to be the leading cause of death [2].

In this article we aimed to discuss some common poisons in Pakistan their symptoms and treatment.

SPECIFIC POISONS

1. ENDRIN (insecticide)

From July through Sept 1984, acute convulsions caused by endrin poisoning occurred in the subdistrict of Talagang, Attock District, Punjab province. In Pakistan, endrin use is restricted to application on early cotton and sugar cane crops and access to this pesticide is limited by registration of users.

Symptoms

Patients were consistently reported in good health, with no prior history of seizures. Within two hours after a meal, they collapsed suddenly with vomiting followed by a generalized tonic-clonic seizure. The seizures began with twitching of the orbital and facial muscles, followed by eyes rolling back and bilateral jerking of the upper extremities, then by tonic-clonic jerks in all four extremities, frothing, and occasionally tongue biting. Pupillary size was normal or slightly dilated and weakly responsive to light. The youngest patients had no prodromal symptoms. Older patients (16 years or older) reported experiencing headache, chest tightness, nausea, and/or minor muscular spasms just before the seizures. If patients were left untreated, seizure activity continued for

as many as 15 minutes. Some patients had muscle fasciculations and rhythmic, clonic jerks of the extremities during the postictal period. A low-grade axillary temperature of 37.7°C was recorded for some patients after the seizures. Recovered patients were amnesic to seizure events.

Treatment

Diazepam and phenobarbital in large doses (3 to 5 mg/kg of diazepam and 19 to 20 mg/kg of phenobarbital) and atropine (to control secretions) were an effective therapeutic regimen for most patients.[3]

2: ORGANOPHOSPHORUS (OP)

Organophosphorus insecticides are widely used in agriculture, usually as pesticides. They are common suicidal agents in Pakistan, India, Sri Lanka, and other Asian and South Asian countries.

Mechanism of action

Organophosphorus compounds irreversibly inhibit acetylcholinesterase at neuromuscular junction and in autonomic as well as central nervous system. This results in accumulation of acetylcholine and overstimulation of acetylcholine receptors leading to acute cholinergic crisis,

Symptoms

Bradycardia, bronchorrhea, miosis, sweating, salivation, lacrimation, defecation, urination and hypotension. In addition, skeletal muscle weakness and fasciculations also develop. After severe exposure, slurred speech, convulsions, coma and respiratory depression may also occur. Death occurs acutely due to respiratory failure or cardiovascular collapse and later as a result of peripheral respiratory failure and complications of aspiration and long-term ventilation.

Treatment

Current standard treatment for OP poisoning involves washing of skin and gastric lavage, administration of activated charcoal, atropine/glycopyrrolate, oximes and other newer compounds in addition to ventilatory support which they may require. These are only partly effective, with mortality rate of over 10% or even higher rates.[4]

3: BATRACHOTOXIN

Origin: frog

The famous neurotoxin batrachotoxin 1 is a member of a family of steroidal alkaloids called batrachotoxins.

Mechanism of action

Batrachotoxin intends to affect the nervous system by causing the irreversible opening of Na⁺ channels in the nerves causing a change in ion selectivity of the ion channel by increasing the permeability of the channel toward larger

cations, which results in depolarization of nerves leading to failure of nerve impulse transmission. The persistent lack of nerve transmission to the muscles result in paralysis followed by death of an organism. The interesting feature about this neurotoxin is that it is in fact a cardio-toxin and causes heart arrest by interfering with heart conduction. This toxin has maximum activity at 37°C which is the body temperature of human beings so it is quite difficult to cure a person affected with this noxious and deadly chemical. Much research is required to better understand the mechanism of action of this toxin and its remedies. One aspect of this toxin is that if its action is controlled it can be used as an effective pain killer[5]

4: BENZODIAZEPINE

Benzodiazepine prescription and usage is regulated in most developed countries. However in developing countries, like Pakistan these drugs are easily available without prescription.

Symptoms

The occurrence of the benzodiazepine dependence and withdrawal symptoms is directly dependant on the duration of drug use. Patients using these drugs for four months or more may develop withdrawal symptoms, characterized by anxiety, malaise, dysphasia, depersonalization, and perceptual changes such as hyperacusis and unsteadiness. Although they are considered safe and are generally well-tolerated, there is a considerable risk of misuse and abuse of benzodiazepines. According to several studies, the ingestion of high doses of benzodiazepines is the most common way of self poisoning for suicidal attempts which accounts for 30-40% poisoning cases in the developed countries. In Pakistan more than 80% of self poisoning cases are due to benzodiazepine overdose, a fact which can be generalized to most developing nations as well.

Treatment

Activated charcoal is more likely to produce benefit if it is administered within the first hour after ingestion of benzodiazepine. In our study, patients were treated with activated charcoal, gastric lavage and other symptomatic treatments within one hour of ingestion. Although flumazenil is the only antidote for benzodiazepine poisoning but it was not revealed to be used in the treatment of benzodiazepine poisoning during this study. It may be due to the high cost of the antidote and low socioeconomic status of patients.[6]

5: SNAKE POISON

The common poisonous snakes found in Pakistan is Cobras, Common Indian Krait, Russell's Viper, Saw Scaled Viper, Lavantine Viper, Persian Horned Viper, Malayan Pit Viper and Sea Snakes. The snake venom contains at least 25 enzymes although no single snake venom contains all of these. The composition of snake venom may vary seasonally and locally and depends on the feeding pattern of the snake. The clinical manifestations of envenomation

following snake bite depends on the type and size of the snake, the age and health status of the patient, the location of the bite and its initial management. The distinction of a poisonous snake from a nonpoisonous one is often difficult.

Symptoms

The most common symptom following snake bite is fright. The patient may appear semi-conscious with cold clammy skin, feeble pulse and rapid shallow breathing. Local effects of snake bite include erythema, oedema and necrosis. The systemic effects in case of Elapidae and Hydrophidae bites are mainly due to neurotoxins and cardiotoxins. Early signs are ptosis and glossopharyngeal paralysis followed by dryness of throat, paralysis of tongue, respiratory distress and myasthenia, leading to shock. Death can ensue due to respiratory paralysis. In case of viperine bites the main clinical features are hemorrhages from different sites of the body, hematuria, epistaxis, melaena and haemoptysis, which may lead to hypovolaemic shock and death. It is not always possible to identify the type of snake because usually the snake is not brought for identification in the health facilities. Sometimes the snakes are not trapped and killed and at times not even seen. This is compounded by the fact that most snake bites occur after sunset, when it is generally very dark in rural areas.

Treatment

In the absence of reliable snake identification clinicians are obliged to use polyvalent anti-venom. Secondary and even primary health care facilities are able to make an accurate diagnosis of envenomation by history and physical examination and are able to administer polyvalent anti-snake venom safely.[7]

6. Hydrocarbons

Hydrocarbons that contain carbon and hydrogen are miscellaneous group of organic substances. The respiratory and central nervous system are effected by toxic exposure to hydrocarbons. Hydrocarbons are found in substances such as furniture polishes, lamp oil, lighter fluid, glues, nail polishes, gasoline, kerosene, paints, paint removers, pine oil. Hydrocarbons are often mixed with agents that have systemic toxicity, such as camphor, aniline dyes, heavy metals, and pesticides. Hydrocarbon toxicity can be classified according to potential for toxicity:

Low toxicity: asphalt, tars, motor oil, and axle grease, mineral oil and petroleum jelly,

Systemic toxicity: Systemic toxicity occurs after ingestion of halogenated and aromatic hydrocarbons or after ingestion of compounds that combine aliphatic hydrocarbons with toxic additives (e.g. organophosphates, heavy metals, camphor). The acute systemic toxicity is determined by their degree of absorption and volatility from the GI tract or respiratory system.

Mechanism of Toxicity:

Majorly it affects the central nervous system and respiratory system. Direct injury effect the respiratory system. Low viscosity, low surface tension and solvent properties of aspirated hydrocarbons together determine a compound's ability to cause chemical pneumonitis. Severe necrotizing pneumonia is caused by hydrocarbons. It also effects the airway epithelium, alveolar septae and pulmonary capillaries as well as solubilization of the lipid surfactant layer. The inflammatory response from chemical irritation causes temperature elevation, usually within hours of exposure. Volatile hydrocarbons enter the circulation through the lungs and rapidly diffuse throughout the body and into the CNS because they are highly lipid soluble. Neurons, which have a high lipid content, are particularly susceptible to severe pulmonary injury and hypoxia.

Management

Patients in respiratory distress should receive oxygen, serial chest radiographs and beta-2 agonist. If their respiratory status continues to decline, they require endotracheal intubation. Patients with seizures should receive IV benzodiazepines. Stabilization depends on the degree of symptoms including respiratory distress and altered mental status. Gastric lavage, ipecac administration and activated charcoal is NOT recommended because of increased risk of vomiting and additional pulmonary aspiration. Remove all contaminated clothing, clean affected hair and skin to reduce risk of additional irritation and inhalation. Dermal and ocular exposure can be treated with copious water irrigation.[8]

7. Cocaine

Recently Cocaine poisoning is increased. Because of its sudden onset and rapidly fatal course survival is rare.

Symptoms

The symptoms of cocaine poisoning are severe respiratory and metabolic acidosis acute agitation, diaphoresis, and tachycardia, and was complicated by grand mal seizures, apnea, and accelerated idioventricular rhythm.

Management

The patient made a quick recovery after control of the seizures with diazepam and treatment of the acidosis with ventilation and bicarbonate, the ventricular dysrhythmia abated. Seizures are a major determinant of lethality in cocaine poisoning. Treatment of the seizures is of prime importance, and correction of the acidosis can normalize cardiac rhythm and function in these critically ill patients.[9]

8. Iron

In childhood the accidental ingestion of iron-containing preparations is relatively common, in adults intentional over dosage with iron is seen. Iron ingestion can result in profound mental retardation or death. Deferoxaminemesylate, a specific and tenacious chelator of

iron. The necessities for its early administration demand that the physician be aware of a rational approach to the therapy for iron poisoning.

Management

The simultaneous oral and continuous intravenous (IV) administration of deferoxamine offers the most rational specific therapy for this condition. [10]

9. Heavy metals

Heavy metals are metallic elements having relatively high density compared to water . Heavy metals include metalloids, such as arsenic, which induces toxicity at low level of exposure. Environmental contamination is caused by these metals. Also, human exposure has risen dramatically as a result of an exponential increase of their use in technological applications, several industrial, agricultural and domestic Reported sources of heavy metals in the environment include geogenic, industrial, agricultural, pharmaceutical, domestic effluents, and atmospheric sources .

Arsenic:

People are exposed to arsenic chronically throughout the world. Exposure to arsenic occurs via the oral route (ingestion), inhalation, dermal contact, and the parenteral route to some extent, toxicity of arsenic depends on the exposure dose, frequency and duration, the biological species, age, and gender, as well as on individual susceptibilities, genetic and nutritional factors. Arsenic exerts its toxic effect is through impairment of cellular respiration by the inhibition of various mitochondrial enzymes, and the uncoupling of oxidative phosphorylation and arsenic trioxide induces DNA damage in human.

Cadmium:

Cadmium is a heavy metal. The highest level of cadmium compounds in the environment is accumulated in sedimentary rocks, and marine phosphates. The main routes of exposure to cadmium are via inhalation or cigarette smoke, and ingestion of food. Human exposure to cadmium is possible through a number of several sources including employment in primary metal industries, eating contaminated food, smoking cigarettes, and working in cadmium-contaminated work places. Exposure to cadmium is commonly determined by measuring cadmium levels in blood or urine. Cadmium is a severe pulmonary and gastrointestinal irritant, which can be fatal if inhaled or ingested. After acute ingestion, symptoms such as abdominal pain, burning sensation, nausea, vomiting, salivation, muscle cramps, vertigo, shock, loss of consciousness and convulsions usually appear within 15 to 30 min. Cadmium causes damage to cells primarily through the generation of ROS , which causes single-strand DNA damage and disrupts the synthesis of nucleic acids and proteins.[11]

10. Pesticides

Pesticides are a group of chemicals used predominantly in agriculture and against vectors in vector-borne diseases such as malaria. there are certain undesirable and unwanted effects of using pesticide.[12]

| | |
|---|---|
| Suicides and mass poisoning Pesticide formulators, mixers. Applicators and pickers. | Single and Short-term, Very high-level and Exposure |
| Pesticide manufacturers, Mixers, applicators and pickers | Long-term high-level, Exposure |
| All population groups | Long-term low-level exposure |

Usage of pesticides in Pakistan has controlled the pests, but it has started causing environmental problems in the area. In some areas of Punjab and Sindh groundwater has been found contaminated and is constantly being under the process of contamination due to pesticide use. Farmers have overused and misused pesticides especially in cotton-growing areas. Farmers are at higher risk for acute and chronic health effects associated with pesticides due to occupational exposure. The intensive use of pesticides (higher sprays more than the recommended dose) in cotton areas involves a special risk for the field workers, pickers, and of an unacceptable residue concentration in cottonseed oil and cakes.[13]

11. Amantadine

Amantadine (Symmetrel) is an antiviral agent that is also effective in the treatment of Parkinson’s disease and for prophylaxis against the parkinsonian side effects of neuroleptic agents.

Overdose symptoms:

Although there is limited information about its effects in acute overdose, it has been associated with seizures, arrhythmias, neuroleptic malignant syndrome, and death.

Mechanism of toxicity:

Amantadine is thought to both enhance the release of dopamine and prevent dopamine reuptake in the peripheral and central nervous systems. In addition, it has anticholinergic properties, especially in overdose.

Toxic dose:

The toxic dose has not been determined. Because the elimination of amantadine depends entirely on kidney function, elderly patients with renal insufficiency may develop intoxication with therapeutic doses.

Clinical aspects:

Amantadine intoxication causes agitation, visual hallucinations, nightmares, disorientation, delirium, slurred speech, ataxia, myoclonus, tremor, and sometimes seizures. Anticholinergic manifestations include dry mouth, urinary retention, and mydriasis. Rarely, ventricular arrhythmias including torsade de pointes and multifocal premature ventricular contractions may occur. Amantadine has also been reported to cause heart failure.

Amantadine withdrawal:

Either after standard therapeutic use or in the days following an acute overdose, may result in hyperthermia and rigidity

Diagnosis:

It is based on a history of acute ingestion or is made by noting the above-mentioned constellation of symptoms and signs in a patient taking amantadine.

Treatment:

Emergency and supportive measures

1. Maintain an open airway and assist ventilation if necessary
2. Treat coma, seizures, arrhythmias, and hyperthermia if occur.
3. Monitor the asymptomatic patient for at least 8–12 hours after acute ingestion.

Specific drugs and antidotes:

There is no known antidote. Although some of the manifestations of toxicity are caused by the anticholinergic effects of amantadine, physostigmine should not be used.

1. Treat tachyarrhythmias with beta blockers such as propranolol or esmolol.
2. Hyperthermia requires urgent cooling measures and may respond to specific pharmacologic therapy with dantrolene. When hyperthermia occurs in the setting of amantadine withdrawal, some have advocated using amantadine as therapy

Decontamination:

1. Prehospital- Administer activated charcoal, if available.
2. Hospital- Administer activated charcoal. Gastric emptying is not necessary if activated charcoal can be given promptly.

Enhanced elimination:

Amantadine is not effectively removed by dialysis, because the volume of distribution is very large (5 L/kg). The serum elimination half-life ranges from 12 hours to 34 days, depending on renal function. In a patient with no renal function, dialysis or hemoperfusion may be necessary. [14]

12. Over-the-counter (OTC) cough and cold medications:

These medications are marketed widely for relief of common cold symptoms, and yet studies have failed to demonstrate a benefit of these medications for young children. In addition, OTC medications can be associated with significant morbidity and even mortality in both acute overdoses and when administered in correct doses for chronic periods of time.

Toxicity symptoms:

The potential toxicities of cough and cold medicines vary with their composition. Many products contain multiple substances including a decongestant, cough suppressant, antihistamine, and/or antipyretic/analgesic. Pseudoephedrine and PPA are sympathomimetics that reduce nasal congestion by stimulating the α -adrenergic receptors on vascular smooth muscles. Clinical toxicity presents with central nervous system (CNS) stimulation, hypertension, and tachycardia with ephedrine or pseudoephedrine ingestion, and bradycardia with PPA ingestion. CNS stimulation can manifest as extreme agitation, restlessness, insomnia, psychosis, and seizures. Serious complications after decongestant ingestions and/or overdoses include hypertension, tachycardia, bradycardia, seizures, stroke, and cerebral hemorrhage. Dysrhythmias, myocardial infarction, and ischemic bowel infarction have also been reported.

Clinical toxicity of antihistamines:

These are characterized by a spectrum of anticholinergic symptoms and CNS depression. Tachycardia, blurred vision, agitation, hyperactivity, toxic psychoses, and seizures may be evident. Cardiac dysrhythmias including torsades de pointes have also been reported.

Clinical toxicity of Dextromethorphan:

It is an antitussive, has also been associated with toxic side effects such as lethargy, stupor, hyperexcitability, ataxia, abnormal limb movements, and coma.

Treatment:

Its treatment depend on type of otc medications used.[15]

| OTC agents | Antidote |
|------------------|--|
| Antihistamines | There is no specific antidote for antihistamine overdose. As for anticholinergic poisoning, physostigmine has been used for treatment of severe delirium or tachycardia. However, because antihistamine overdoses carry a greater risk for seizures, physostigmine is not routinely recommended. Sodium bicarbonate, 1–2 mEq/kg IV, may be useful for myocardial depression and QRS interval prolongation after a massive diphenhydramine overdose. |
| Caffeine | Beta blockers effectively reverse cardiotoxic effects mediated by excessive beta-adrenergic stimulation. Treat tachyarrhythmias or hypotension with intravenous propranolol, 0.01–0.02 mg/kg or esmolol, 0.025–0.1 mg/kg/min (p 443), beginning with low doses and titrating to effect. Because of its short half-life and cardioselectivity, esmolol is preferred. |
| Dextromethorphan | Naloxone has been reported effective in doses of 0.06–0.4 mg, other cases have failed to respond to doses up to 2.4 mg. |

13. Quinine and chloroquine:

These are antimalarial agents used in treatment of malaria. Quinine, the oldest antimalarial drug, has been used for over 300 years. Quinoline derivatives, particularly quinine and chloroquine, are highly toxic in overdose.

Toxic symptoms:

The toxic effects are related to their quinidine-like actions on the heart and include circulatory arrest, cardiogenic shock, conduction disturbances and ventricular arrhythmias. Additional clinical features are coma, convulsions, respiratory depression. Blindness is a frequent complication in quinine overdose. Hypokalaemia is consistently present, although apparently self-correcting in severe chloroquine poisoning.

Toxicokinetic studies:

Toxicokinetics studies of quinine and chloroquine showed good correlations between dose ingested, serum concentrations and clinical features, and confirmed the inefficacy of haemodialysis, haemoperfusion and peritoneal dialysis for enhancing drug removal.

Management:

General management:

The general management of antimalarial overdose includes gastric lavage and symptomatic treatment.

Chloroquine and quinine overdose management:

- It requires early monitoring of vital signs
- ECG, blood pressure and intensive supportive treatment of cardiovascular disturbances:
- Adrenaline (epinephrine) for circulatory arrest
- Isoprenaline (isoproterenol) for shock and conduction disturbances
- DC countershock for ventricular tachycardia or fibrillation and sometimes pacemaker stimulation for later ventricular arrhythmia.

- Correction of hypokalaemia should be done very cautiously when cardiac depression has been rectified.
- After ingestion of a large dose or when cardiotoxic symptoms are present, diazepam should be given systematically as a loading dose of 1 mg/kg followed by a continuous infusion.
- Dapsone-induced methaemoglobinaemia requires methylene blue administration and repeated oral doses of activated charcoal.
- Haemodialysis may be indicated if methaemoglobinaemia reoccurs.
- Chloroquine overdose remains the most severe and frequent cause of antimalarial drug poisonings. Its prognosis and high mortality should be improved by adequate supportive treatment and by the systematic and early treatment with diazepam. [16]

14. Isoniazid

Isoniazid (INH), a hydrazide derivative of isonicotinic acid, is the bactericidal drug of choice for tuberculosis. INH is well known for its propensity to cause hepatitis with chronic use. Acute isoniazid overdose is a common cause of drug induced seizures and metabolic acidosis.

Acute overdose symptoms:

Isoniazid produces acute toxic effects by reducing brain pyridoxal 5-phosphate, which is the active form of vitamin B6 and an essential cofactor for the enzyme glutamic acid decarboxylase. This result in lower CNS levels of gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter, which leads to uninhibited electrical activity manifested as seizures. INH may also inhibit the hepatic conversion of lactate to pyruvate, exacerbating the lactic acidosis from seizures.

Chronic toxicity symptoms:

Peripheral neuritis with chronic use is thought to be related to competition with pyridoxine.

Management:

Treatment of isoniazid overdoses include intravenous pyridoxine (one gram IV pyridoxine for each gram of ingested isoniazid), intravenous diazepam or phenobarbital for continued seizures, and gastric decontamination with lavage and activated charcoal (1 g/kg). Extraordinary measures such as early haemodialysis and haemoperfusion should be reserved for those patients with persistent coma or refractory seizures. [17]

15. Anesthetics, local:

Local anesthetics are widely used to provide anesthesia via local subcutaneous injection, topical application to skin and mucous membranes, epidural, spinal, and regional nerve blocks.

Mechanism of toxicity:

Local anesthetics bind to sodium channels in nerve fibers, blocking the sodium current responsible for nerve conduction and thereby increasing the threshold for conduction and reversibly slowing or blocking impulse generation. In therapeutic concentrations, this results in local anesthesia. In high concentrations, such actions may result in CNS and cardiovascular toxicity. Some local anesthetics (eg, benzocaine) have been reported to cause methemoglobinemia.

Symptoms:

The severity of cardiovascular and central nervous system (respectively, CVS and CNS) toxicity is directly related to the local anesthetic potency, dose, and rate of administration. Signs of CNS stimulation, ranging from tremors to convulsions and perhaps cardiac dysrhythmias, can be described in terms of a chaos-derived state change in which the local anesthetic appears to act as an initiator. [18]

Treatment

Emergency and supportive measures includes:

1. Maintain an open airway and assist ventilation if necessary
2. Treat coma, seizures, hypotension, arrhythmias, and anaphylaxis if they occur.

Extracorporeal circulatory assistance (eg, balloon pump or partial cardiopulmonary bypass) has been used for short-term support for patients with acute massive overdose with 20% lidocaine solution.

3. Monitor vital signs and ECG for at least 6 hours.

Antidotes:

There is no specific antidote. [19]

References:

1. Zimmerman JL. Poisonings and overdoses in the intensive care unit: general and specific management issues. *Crit Care Med.* 2003;31(12):2794-2801.
2. Khan NU, Mir MU, Khan UR, Khan AR, Ara J; Raja K et al., The Current State of Poison Control Centers in Pakistan and the Need for Capacity Building. *Asia Pac J Med Toxicol.* 2014;3(1):31-35.
3. Rowley DL, Rab MA, Hardjotanojo W, Liddle J, Burse VW, Saleem M, Sokal D et al., Convulsions caused by endrin poisoning in Pakistan. *Pediatrics.* 1987;79(6):928-934.
4. Shaikh JM, Siddiqui FG, Soomro AG. Management of acute organophosphorus insecticide poisoning: An experience at a university hospital. *JLUMHS.* 2008.7(2):97-101.
5. Sultan A, Raza AR, Khan KM. Towards the Synthesis of Batrachotoxin-Formation of Alkynyl Stannanes. *J Chem Soc Pak.* 2013; 35(5): 1371-1385.
6. Saleem U, Mahmood S, Ahmed B. Benzodiazepine Poisoning cases: A Retrospective Study from Faisalabad, Pakistan. *Pak J Pharm.* 2010;23(1):11-13.
7. Suleman MM, Shahab S, Rab M. Snake bite in the Thar Desert. *J Pak Med Asso.* 1998. 48:306-307.
8. Signs V. Hydrocarbon Poisoning. Available at: <https://pedclerk.bsd.uchicago.edu/page/hydrocarbon-poisoning> (Accessed March 10, 2016)
9. Jonsson S, O'Meara M, Young JB. Acute cocaine poisoning: importance of treating seizures and acidosis. *Am J Med.* 1983;75(6):1061-1064.
10. Robotham JL, Lietman PS. Acute iron poisoning: a review. *Am J Dis Child.* 1980;134(9):875-879.
11. Tchounwou PB, Clement G Yedjou, Anita K Patlolla, Dwayne J Sutton. *Heavy Metals Toxicity and the Environment.* Exs. 2012;101:133-64.
12. Jeyaratnam J. Acute pesticide poisoning: a major global health problem. *World Health Stat Q.* 1990; 43(3):139-44.
13. Tariq MI, Afzal S, Hussain I, Sultana N. Pesticides exposure in Pakistan: A review. *Envi Int.* 2007; 33:1107-1122.
14. Olson KR, Anderson IB, Benowitz NL. 2011. *Poisoning & drug overdose.* 6th Ed.

15. Gunn VL, Taha SH, Liebelt EL, Serwint JR. Toxicity of over-the-counter cough and cold medications. *Pediatrics*. 2001;108:e52-e52.
16. Jaeger A, Sauder P, Kopferschmitt J, Flesch F. Clinical features and management of poisoning due to antimalarial drugs. *Med Toxicol Adverse Drug Exp*. 1987;2:242-273.
17. Siefkin A, Albertson T, Corbett M. Isoniazid overdose: pharmacokinetics and effects of oral charcoal in treatment. *Hum Toxicol*. 1987;6:497-501
18. Mather LE, Copeland SE, Ladd LA. Acute toxicity of local anesthetics: underlying pharmacokinetic and pharmacodynamic concepts. *Reg Anesth Pain Med*. 2005;30:553-566.
19. Zink W, Graf BM. The toxicity of local anesthetics: the place of ropivacaine and levobupivacaine. *Curr Opin Anaesthesiol*. 2008;21:645-650.

Cite this article as: Arshad S, Abid F, Aziz HA, Anjum R, Nadeem I. Specific Poisons in Pakistan: A Mini Review. *Res Pharm Healt Sci*. 2016;2(3):179-186.