

One of the boons of modern civilisation is Stress, physical, mental, emotional or financial. Chinese, though competing with U.S.A in all fields have incurred a tenfold increase in rate of mental illnesses & suicides, equating with Japanese in the latter. Similarly around 2 million poison exposure cases occurred in USA in 1995 with children less than 6 years accounting for 53% of these, although with only 2.8% of fatalities. In adults more often than not the poisoning is intentional (homicidal or suicidal) hence always accompanied by a larger dose and delayed information to attendants with an increased mortality in contrast to children where it is unintentional/accidental hence with reduced mortality.¹ Even in pregnant females there are more than 7000 poisoning exposures yearly. In our Indian set-up also suicidal poisoning a domain of females in the past is equally increasing in young adolescents¹ & past middle-aged males. The means & ways of poison ingestion have also undergone a sea of change from copper sulphate³ (the blue Vitriol), ratti, cannabis, organophosphates & Cellphos⁴ as the common suicidal varieties and marking nut, dhatura and alcohol⁵ mixed with barbiturates as the common homicidal varieties to opiates, benzodiazepines, cyclic antidepressants, Hashish, heroin & calcium channel blockers to insulins.

The word Poison is derived from Fr. Gr. "Potion" that is a substance that produces harmful, toxic effects in one draught only i.e. in minimal amounts, because we know that "in excess every good thing is bad" There is reference of poisoning in B.C. era (300 B.C) of poisoning in Greece. There have been reports of wide spread use of opium smoking in China and near East, causing harm for centuries.

In the United States of America even today the 0.14% annual prevalence of heroin dependence is only 1/3rd the rate of prescription opiate abuse, representing only the tip of an Iceberg. Similarly the 2% role of morphine dependence in South East and South West Asia represents only a partial data and though apparently low as compared to other abused substances their disease burden is substantial, with high rates of morbidity and mortality, disease transmission, increased healthcare, crime and law enforcement cost and less tangible cost of family disturbances and loss of productivity. The fashion today is one of polydrug use involving alcohol, sedatives, opiates, cannabinoids and stimulants, by way of smoking, oral ingestion, I/v ingestion, inhalation or skin patches thereby increasing the chance of transmission of diseases like hepatitis B, AIDS and other communicable diseases.

At the same time these produce complications like meningitis, osteomyelitis and abscesses in various organs, which are dangerous and difficult and costly to treat².

In India usually a patient of poisoning is brought to hospital after all the means of self treatment, ranging from whodwho, Ojha, local quack have been exhausted (from fear of police and society) hence rarely is the victim in original shape of poisoning or conscious enough to narrate the reality, hence more often than not, most of the cases present as one of unknown poisoning putting to test your clinical acumen, general knowledge about the patient and the area he is from and the condition of his attendants. In some of the cases the emanating odour, the physical signs and the history given by the attendants may give a partial lead. Our approach in managing a patient of suspected toxic Ingestion has always been to give an immediate physical assessment and to address the ABC of resuscitation (airway, breathing and respiration) without delay and to concentrate on the History under following "Five Ws": (!) who ---- the patients age, -weight, relationship to others present and gender; (!) what ---- the name and dosage of medication (s) or substances of abuse, coingestants and amount ingested; (!!) when --- the time and date of ingestion; (!v) where --- both the route of poisoning (e.g., ingestion or injection) and the geographic location where the poisoning occurred, and V) why ---- whether intentional or unintentional, and associated details. In addition, a detailed past medical history should be obtained, including previous poisonings, medical conditions and concurrent medications that might affect the patient's response to and metabolism or elimination of ingestants, psychiatric history and history of substance abuse. Particular attention should be devoted to eliciting a history of alcoholism, and renal or hepatic disease. Clinicians should attempt to obtain this information in all cases, even for apparently minor ingestions^{5,6}.

ASSESSMENT AND STABILIZATION

Emergency Assessment

A brief physical assessment should be performed immediately in all patients to determine the effects of toxin (s) and other conditions that might be present. Particular attention should be directed to adequacy of the airway and ventilation, level of mental status and cardiac function. Unstable patients should be placed on a cardiac monitor, with measurement of vital signs every five to 15 minutes until the patient is stabilized to the point that monitoring is no longer necessary.

The initial assessment is especially important in patients with decreased responsiveness and unstable vital signs because, for example, head trauma or penetrating body trauma can masquerade as an overdose. Evidence of head injury, penetrating wounds or chemical burns of the mouth and face mandate specific valuation and management strategies and should not be missed. Focal neurologic signs are suggestive of central nervous system (CNS) vascular events, including cerebrovascular accidents, neurotoxic snake bite poisoning and subdural hematoma. A more detailed physical assessment should be performed after the patient's cardiopulmonary status has been stabilized^{2,6}.

INITIAL STABILIZATION PROCEDURES

Stabilisation of the patient is the first priority in managing toxic ingestions and is performed simultaneously with the initial physical assessment. Treatment should address the "ABCs" (airway, breathing, circulation) without delay. Also, the potential for rapid changes in the patient's condition should be considered in making decisions about airway and ventilatory support. Treatment with naloxone (Narca), dextrose and thiamine should be considered in patients with altered mental status. Naloxone is a competitive antagonist at opiate receptors and can reverse narcotic-induced symptoms when given intravenously, intramuscularly, endotracheally, subcutaneously or intralingually. Successful submental administration of naloxone has also been reported.⁵ Naloxone can safely be given to patients with respiratory and/or CNS depression who have a low likelihood of opioid addiction. Because of concern about withdrawal symptoms and/or unmasking symptoms from coingestants, caution is indicated in cases of suspected opioid addiction and multi-drug poisonings³. Naloxone is administered to adults for treatment of respiratory depression in a dosage of 2.0 mg initially, repeated every two minutes as needed up to a total of 10 mg; in narcotic dependent patients or those with non-life threatening symptoms, the dosage is 0.1 mg initially, doubled every two minutes up to a total of 10 mg; in children older than five years of age or weighing more than 20 kg, the dosage is 2.0 mg initially for patients with respiratory depression and 0.1 to 0.8 mg if respiratory depression is not present, in neonates and young children, the initial dosage is 0.1 mg per kg^{2,6}.

Symptoms of hypoglycemia (e.g, altered mental status, cool, clammy skin, coma) are rapidly reversed with administration of hypertonic dextrose. Patients with altered mental status, absent focal neurologic signs and low or borderline hypoglycemia (blood sugar less than 80 mg per dL on rapid reagent testing) should receive intravenous dextrose (adults: 50 mL of 50% dextrose; children: 4 mL per kg of 25% dextrose; neonates: 5 mL per kg of 10% dextrose) Dextrose may be administered empirically if rapid reagent testing is not available. The safety of hypertonic dextrose in settings of cerebral ischemia has been questioned and, whenever feasible, bedside documentation of hypoglycemia should be obtained before administering dextrose.

In case where intravenous access is difficult, glucagon 1.0 mg intramuscularly, may be given as a temporizing measure. Intravenous thiamine (vitamin B₁) should be given to patients treated with hypertonic dextrose (adults: 100 mg; children: 10 to 25 mg), theoretically before the dextrose is administered, to prevent Wernicke's encephalopathy⁶.

DETERMINATION OF INGESTED SUBSTANCE

Physical findings may suggest the type of toxin (s) ingested but, more often, a detailed history, examination of medication containers or toxicologic analysis reveals the answer. Physical findings, however, often enable the clinician to determine if the toxin is a physiologic stimulant or a depressant, and which common poisons should be considered in the initial management of the patient.

Physical signs following ingestion of stimulants often include mydriasis (dilated pupils), tremor, tachycardia, irritability, diaphoresis, mania, convulsions and tachyarrhythmias. Commonly ingested stimulants include cocaine, amphetamine caffeine, theophylline, tricyclic antidepressants (early symptoms after overdose), antihistaminics and hallucinogens.

Physical findings produced by physiologic depressants include lethargy, decreased responsiveness to verbal and physical stimulation, miosis (constricted pupils), hypothermia and coma. Common sedative-hypnotics include alcohol, benzodiazepines, barbiturates, opiates, muscle relaxants and chloral hydrate.

Cardiovascular agents include antihypotensive agents (angiotensin-converting enzyme inhibitors, beta blockers, calcium channel blockers and centrally acting agents), digitalis, nitrites and antiarrhythmic agents.

LABORATORY EVALUATION

Laboratory evaluation is indicated in most symptomatic patients, when ingested substances are unknown, if the poison has the potential to produce moderate to severe toxicity and if the ingestion was intentional. Routine studies should include a complete blood cell count, determination of serum electrolyte and glucose levels, a chemical screen with hepatic and renal function studies (e.g., calcium, aspartate aminotransferase, alanine aminotransferase, bilirubin, alkaline phosphatase, lactate dehydrogenase, prothrombin time, blood urea nitrogen, creatinine) and urinalysis. Measurement of serum osmolarity may be helpful if poisoning with methanol, ethylene glycol or isopropanol is suspected.

Drug screening has limited value because care in most cases of toxic ingestion is supportive and not affected by identification of the ingestant. However, drug screening is a common practice in many medical settings where poisonings are managed and can provide useful information. Qualitative toxicology screening is most useful when the ingested toxin is unknown, in cases of multiple ingestion and when symptoms and physical findings are not compatible with the history. Quantitative

354 toxicology screening is useful when knowledge of drug serum levels may affect patient management; examples include ingestions of acetaminophen, salicylates, ethanol, ethylene glycol, isopropyl alcohol, digoxin, iron, lithium, theophylline, anticonvulsants and methanol.

ANCILLARY TESTS

An electrocardiogram should be performed in patients with arrhythmias and/or suspected ingestion of cardiotoxic drugs. Chest radiographs should be obtained in patients with suspected aspiration, coma or ingestion of medications (salicylates, narcotics, paraquat, sedative-hypnotics) than can produce noncardiogenic pulmonary edema. Abdominal radiographs may detect abnormal densities in patients who have ingested drug packets, salicylates, calcium salts, heavy metals (e.g., iron tablets) or radiopaque foreign bodies. Ingested hydrocarbons may be visualized as a “layer” between gastric fluid and the gastric air bubble.

Ancillary tests such as electrocardiograms, chest radiographs and plain abdominal films need not be routinely ordered but, when appropriate, can provide the clinician with additional useful information.

DECONTAMINATION

Following evaluation and stabilization of the poisoned patient, attention is directed toward decontamination, i.e., decreasing absorption of the ingested poison from the gastrointestinal tract. This can be accomplished by emptying the stomach via gastric lavage, administration of activated charcoal within the gut lumen and use of methods for increasing transit of the toxic substances through the gastrointestinal tract.

Gastric emptying should not routinely be used in all oral poisoning cases because it is ineffective when used at a late stage, may delay more effective interventions and can cause needless complications, such as aspiration. However, it is often beneficial when used early in the treatment of potentially severe poisonings. Gastric emptying is most effective when used within one hour of the ingestion and cannot be justified beyond four hours following the ingestion except in patients with concretions, massive ingestions or ingestions of substances that markedly decrease gastric motility. The stomach may be emptied by gastric lavage or by inducing emesis with syrup of ipecac.

GASTRIC LAVAGE

In most situations, gastric lavage is preferable to administration of ipecac, particularly in emergency departments where prolonged ipecac induced vomiting may delay treatment with activated charcoal. Indications for lavage include ingestion of highly toxic substances (large ingestions or substances associated with high morbidity and mortality); substances not well adsorbed by activated charcoal (i.e., lithium, iron, lead, and methanol) and in patients with the potential for a jeopardized airway (e.g., altered alertness).

Contraindications to gastric lavage include ingestion of corrosives and most hydrocarbons (gastric emptying

is indicated after ingestion of hydrocarbon products containing benzene, toluene, camphor, halogenated hydrocarbon pesticides or heavy metals, or if the ingestion was greater than 4 to 5 mL per kg); patients with depressed gag reflexes who are not intubated and clinically insignificant ingestions. Complications include aspiration, and perforation of the esophagus or bronchus. A 28- to 36-in French tube is suitable for use in children; a 36- to 40-in French tube is suitable for use in adults.

Gastric lavage is accomplished in children using normal saline or tap water in 15 mL per kg aliquots until clear. Lavage in adults uses 300 ml aliquots until clear, up to 10 to 20 L, if necessary. Administration of activated charcoal through the lavage tube before and after lavage may be beneficial in patients with potential fatal ingestions.

IPECAC

Ipecac continues to be useful in management of alert patients unable to travel to a health care facility within one hour of ingestion. It has been shown that ipecac used at home by experienced hospital staff treating paediatric poisonings following ingestions identified as not being “high-risk” decreases paediatric emergency department visits without jeopardizing safety.

Syrup of ipecac is administered in the following dosages: in infants six months to one year of age, 10 mL; in children one to 12 years age, 15 mL; in adolescents over 12 years of age, 30 mL. Water is given immediately after ipecac to enhance the efficacy of gastric emptying with emesis; adults should receive 8 to 16 oz; children should receive 4 to 8 oz; children less than one year of age should receive 5 to 15 mL per /kg body weight.

Ipecac should not be given children who are less than six months of age, patients who are already vomiting, patients with altered mental state or impaired gag reflexes, and patients who have ingested medications that cause seizures or decreased responsiveness. Use of ipecac should be avoided following ingestion of corrosive (acids or alkalis), sharp objects, most hydrocarbons (similar to gastric lavage) or when treatment with activated charcoal is anticipated within 60 to 90 minutes.

ACTIVATED CHARCOAL

Activated charcoal forms the mainstay of gastric decontamination and is effective for most oral poisonings when given alone or following gastric emptying. Exceptions include ingestions of caustic acids and alkalis, alcohols, lithium and heavy metals (e.g., iron, arsenic). Activated charcoal is inert and remains within the gastrointestinal tract, offering a large surface area for adsorption of ingested toxins. In addition activated charcoal may decrease the absorption of drugs that undergo enterohepatic or enterohepatic circulation.

The usual dosage is 1 to 2 g per kg for children and adults, usually given as a single dose combined with a cathartic. The charcoal is mixed with water in a ratio of 1:4 to 1:8 (1 part charcoal to 4 or 8 parts water) to form a slurry; small quantities of fruit juice or chocolate powder can improve

the taste. Multiple dosing (1 g per kg every two to six hours) had been shown to be effective for poisonings with phenobarbital, phenytoin, carbamazepine. Salicylates, digitalis, theophylline and dapsone. When multiple dosing is used, a cathartic can be given with the first dose but should not be administered with subsequent doses to prevent serious fluid and electrolyte imbalances. Use of activated charcoal is contraindicated in mechanical obstruction and ileus.^{6,7}

WHOLE BOWEL IRRIGATION

Since most often the patient is brought in an unconscious state and in most of the poisonings the laboratory assessment may not come to our rescue hence again it is only the clinical knowledge which will be beneficial in light of the signs and symptoms of the patients. The involvement of various systems will be a pointer to the poison involved e.g. unconsciousness, stupor or convulsions in case of opiate poisoning are cannabis poisonings and tachycardia, hypertension, arrhythmias and pulmonary edema in cases of stimulants like amphetamine etc. Hence a brief description of signs and symptoms along with management is rendered as below.

METHANOL POISONING

Also known as wood alcohol poisoning, moonshine and Hooch-poisoning is a very common type of tragedy usually occurring in slums, rural areas and in places of illicit distillation. The poisoning is almost entirely to its ingestion as a substitute for ethanol or to the drinking of denatured ethyl alcohol. Methanol is oxidized in the body to formaldehyde and formic acid and the enzyme alcohol dehydrogenase appears to be responsible for first step in oxidation. The accumulation of toxic metabolites of methanol damage the optic nerve and retina. Accumulation of formic acid as also the inhibitory effect of formate on oxidation of carbohydrates, results in severe acidosis, manifestations of poisoning, usually occurring 12-24 hrs after ingestion usually include nausea, vomiting, severe abdominal pain, vasomotor disturbances, CNS depression, respiratory failure and visual disturbances, ranging from mild blurring of vision to total blindness, due to toxic optic neuropathy, which may progress to optic atrophy. Serious poisoning also causes severe acidosis with a wide anion gap. For efficient management of acute poisoning gastric lavage is useful only if performed within first hour or two. The specific treatment involve alcohol dehydrogenases, institution of a saline or osmotic diuresis, thiamine and pyridoxine supplements, fomepizole or ethanol and hemodialysis. The intravenous administration of alcohol dehydrogenase inhibitor fomepizole (15mg/kg as a loading dose) or ethanol intravenously to achieve a level of 22mmol /L (100mg/dL serves to lessen the toxicity by competing with methanol for metabolism by alcohol dehydrogenase. I/V methyl prednisolone and retrobulbar triamcinolone have shown recovery in visual salvage. Fomepizole though costly is a better agent as it does not produce excessive obtundation caused by ethanol.^{5,9,10}

OPIATE POISONINGS

Commonly used both in peace and war as euphoric, analgesics and somniferent since ages. The hero of Odyssey also used Nopenthe (free from sorrow) during war. In U.S.A alone around 9,000 adolescents become opiate abusers everyday and not through drug dealers but through the family members the most common presenting poisonings symptoms include shallow and slow respiration, pupillary miosis, bradycardia, hypothermia and stupor and coma. Usually the patients is a chronic addict belonging to all strata of society and usually a history is elicited which is helpful in managing the patient. The specific treatment in addition to ABC (vide-supra) involves.

Naloxone (an opiate antagonist) 0.4–2 mg IV Or IM, with an expected response within 1 to 2 minutes and if the overdose is due to buprenorphine 10 mg or more naloxone and if this does thus not produce the desired effect then another cause of toxicity, poly drug ingestion should be suspected, like benzodiazepines and an additional dose of flumazenil at 0.2 mg/ minute can be given to a maximum of 3gm/hr, but it may precipitate seizures and increase intracranial pressure. Like naloxone, administration for a prolonged period is usually required since most benzodiazepines remain active for considerably longer than flumazenil. Support of vital functions includes oxygen and positive pressure breathing, IV fluids, pressor agents for hypotension and cardiac monitoring to detect QT prolongation, which might require treatment.

1. For detoxification of opiate withdrawal methadone dose tapering regimens (from 60 to 70 mg daily, usually even upto 150 mg daily) range from 2 to 3 weeks to as long as 180 days. Buprenorphine, a partial agonist of morphine allows a shorter detoxification period compared to methadone and is superior to alpha - 2 -adrenergic agonist clonidine.
2. Medication- free- treatment, these may be carried out in inpatient, residential or outpatient setting but 1 to 5 years outcome are very poor and involve the coordination of physician, a counsellor and detoxification expert.^{7,11}

COCAINE AND OTHER PSYCHO- STIMULANT DRUGS

Derived from the leaves of coca plant (*Erythroxylon coca*) cocaine is a stimulant and a local anaesthetic with potent vasoconstrictor properties and produces physiologic and behavioural effects when administered orally, intranasally, intravenously are via inhalation during smoking (pyrolysis) the reinforcing effects appear to be related to activation of dopaminergic neurons in the mesolimbic system. It is widely abused all the world over and in all strata of society commonly abused as a polydrug. Cocaine produces a brief dose related stimulation & enhancement of mood and an increase in cardiac rate and blood pressure, there is significant rise in body temperature and high doses are associated with lethal pyrexia or hypertension. In addition to

356 generalized seizures neurologic complications may include headache, ischemic or hemorrhagic stroke, or subarachnoid hemorrhage with death from respiratory depression cardiac arrhythmias and convulsions. Hepatic necrosis may also occur along with paranoid ideation and visual and auditory hallucinations that resemble alcoholic hallucinosis. The disorders of cerebral blood flow and perfusion in these patients can be detected with magnetic resonance spectroscopy studies. Coca-ethylene a metabolite of cocaine can be detected in blood and urine. Although people may use it for euphoria and as an aphrodisiac, its long term use is associated with loss of libido, impotence and gynecomastia in males and in females derangement of menstrual cycle, galactorrhea, amenorrhea and infertility. In pregnant women cocaine abuse is instrumental in producing congenital malformations in the foetus and perinatal cardiovascular and cerebrovascular disease in the mother^{9,11}. Treatment of cocaine overdose characterised by an hyperadrenergic state (vide-supra) is an emergency, to be managed in an intensive care unit. For control of seizures I/V diazepam in doses upto 0.5mg/kg administered over an 8 hr period has proven to be effective and for ventricular arrhythmias 0.5 to 1mg of propranolol has shown success. In case the desired response is still not achieved a multydrug toxicity should be suspected, especially heroin and managed accordingly. treatment of chronic cocaine abuse is a long process, requiring the coordination of a physician, a psychiatrist and a psychosocial care taker^{2,13}.

Marijuana and cannabis compounds: In India these are obtained from cannabis indica (Bhang) which in addition to the psycho active substance, delta-9-tetrahydrocannabinol (THC) contains many other toxic compounds. Marijuana is obtained from the concentrate of its leaves and flowering tops. Hashish is prepared from concentrated resin of C.indica and contains a THC concentration of between 8-12mgm% by wt. Smoking is the most common route of use as marijuana and being cheap is most popular among adolescents. Acute intoxication produces a feeling of relaxation and euphoria akin to alcohol intoxication and is accompanied by some impairment in thinking, concentration and perceptual and psychomotor function. Physical effects primarily include conjunctival injection and tachycardia, but angina may be precipitated in patients with a history of coronary insufficiency. Decreased sperm count and motility besides other morphological abnormalities of spermatozoa have been detected in chronic marijuana smokers in addition to chronic bronchial irritation. Therapeutic uses in the form of synthetic oral cannabinal (dronabinol) include as anti-emetic in patients of chemotherapy as an appetiser in AIDS patients for reduction of intraocular pressure in glaucoma and for reduction of spasticity in multiple sclerosis and other neurologic disorders^{13,14}.

Methamphetamine: Famous as Meth, Speed, Chalk, Crank, Ice, Glass or Crystal is second only to cocaine abuse as threat to the society involving young adults (18-25 years). It is consumed in the form of smoking, snorting, I/V injections or oral administration, resulting in euphoria

and decreased fatigue. Adverse effects include, headache, difficulty in concentrating, diminished appetite, abdominal pain, vomiting or diarrhoea, disordered sleep, paranoid or aggressive behaviour and psychosis.

Severe toxicity involves, hypertension, cardiac arrhythmias, or cardiac failure, sub-arachnoid hemorrhage, ischemic stroke, intracerebral hemorrhage, convulsions or coma. The magnetic resonance spectroscopic studies show neuronal damage in the frontal areas and basal ganglia.

The treatment is symptomatic involving use of ammonium chloride for acidification of urine and to enhance clearance of the drug. For hypertension sodium nitroprusside or α -adrenergic antagonists are used. Sedatives may be used for reduction of agitation and other signs of central nervous system hyperactivity. Chronic dependence may be treated similar to those described for cocaine abuse. Ecstasy or MDMA (3,4-methylenedioxyamphetamine) is a derivative of methamphetamine, usually taken orally but may be injected or inhaled. Its effects last for 3 to 6 hrs. and in addition to amphetamine like effects it can induce hyperthermia and vivid hallucinations and other perceptual distortions^{6,13}.

Lysergic Acid Diethylamide (LSD) : A very potent hallucinogen, even in 20 micro gm oral doses induces profound psychological and physiologic effects, within minutes tachycardia, hypertension pupillary dilation, tremor and hyperpyrexia occur. A variety of bizarre and often conflicting perceptual and mood changes, including visual illusions, synesthesias and extreme lability of mood, usually occur within 30 minutes after LSD in take and may last for 12 to 18 hrs. Even though the half life of the drug is only three hours.

The most frequent acute medical emergency caused by LSD use is a panic episode (the "Bad trip" which may persist up to 24 hrs and is best managed by supportive reassurance ("talking down") and if need be by administration of small doses of anxiolytic drugs. Its chronic use makes one prone towards enhanced risk for schizophreniform psychosis and derangements in memory function, problem solving and abstract thinking. All these are amenable to psychiatric treatment.

Phencyclidine (PCP) : A cyclohexylamine derivative, widely used in veterinary medicine to briefly immobilize large animals is also sometimes used as a dissociative anesthetic. It is easily synthesized and primarily young people and polydrug users are its victims. It is used orally, by smoking, by snorting or by IV injection, and also as an adulterant in THC, LSD, amphetamine or cocaine, the commonest street preparation is "angeldust". Low doses (5 mg) produce agitation, excitement, impaired motor coordination, dysarthria and analgesia. However toxicity produces horizontal or vertical nystagmus, flushing, diaphoresis and hyperacusis. The behavioural changes include distortions of body image, disorganization of thinking and feelings of estrangement. Higher doses of PCP (5 to 10 mg) may produce salivation, vomiting,

myoclonus, fever, stupor, or coma. Still higher doses of ten mg or more cause convulsions, opisthotonos, and decerebrate posturing which may be followed by prolonged coma.

Since the initial symptoms of PCP overdose (anxiety, paranoia, delusions, hallucinations) may mimic an acute schizophrenic reaction hence the clinical diagnosis is difficult however it can be confirmed by determination of PCP levels in serum or urine because it remains in urine for 1 to 5 days following high dose intake. The treatment requires life support measures, including treatment of coma convulsions and respiratory depression in an intensive care unit. Although there is no specific antidote for PCP, its excretion from the body can be enhanced by gastric lavage and acidification of urine (vide-supra). Death usually occurs because of aspiration, hyperthermia, respiratory depression, severe hypertension, seizures, hypertensive encephalopathy and intracerebral hemorrhage. Acute psychosis because of PSP is a psychiatric emergency, because the patient may be at a high risk for suicide or violence towards others and is successfully managed by haloperidol (5mg IM) on an hourly basis to induce suppression of psychotic behaviour. Chronic PCP use induces insomnia, anorexia, severe social and behavioural changes and even chronic schizophrenia^{7,13}.

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