“COMPARATIVE STUDY OF CURRENT DOSING PRACTICE OF ATROPINE IN ORGANOPHOSPHORUS POISONING AT BLDE UNIVERSITY SHRI B M PATIL MEDICAL COLLEGE AND HOSPITAL WITH PROTOCOL OF ADMINISTRATION PRACTICED AT SOUTH ASIAN CLINICAL TOXICOLOGY RESEARCH COLLABORATION"

By

DR.SUPRIYA SINGH

Dissertation submitted to BLDE University, Vijayapur

In partial fulfillment of the requirements for the award of the degree

of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

Under the guidance of

DR. R. C. BIDRI

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BLDE UNIVERSITY’S, SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR, KARNATAKA.

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DR.SUPRIYA SINGH

LIST OF ABBREVIATIONS USED

ACh	-	Acetylcholine
AChE	-	Acetyl cholinesterase
APACHE II	-	Acute Physiology and Chronic Health Evaluation II
CNS	-	Central nervous system
CI	-	% confidence intervals
CPK	-	Creatine phosphokinase
DEF	-	Tribufos
DNA	-	Deoxyribonucleic acid
e.g.	-	For example
ECG	-	Electrocardiogram
GCS	-	Glasgow Coma Scale
GI	-	Gastrointestinal
HETP	-	Hexaethyl tetraphosphate
hrs	-	Hours
i.e.	-	That is
ICU	-	Intensive care unit
IM	-	Intramuscular
IMS	-	Intermediate syndrome
ISRCTN	-	International Standard Randomised Controlled Trial Number
LD	-	Lethal dose
LDH	-	Lactate dehydrogenase
mEq/L	-	Milli equivalents per liter
mg/dL	-	Milligrams per deciliter
mg/kg	-	Milligrams per kilogram
ml	-	Milliliter
mm	-	Millimeter
mRNA	-	Messenger ribonucleic acid
n	-	Total number
NPIC	-	National Poison Information Center
NTE	-	Neuropathy target esterase
OP	-	Organophosphorus
OPC	-	Organophosphate compound

p	-	Probability
P=O	-	Phosphorous oxygen bond
P2AM	-	Pralidoxime
PAM	-	Pralidoxime
POP	-	Peradenya Organophosphorus Poisoning
RCT	-	randomised controlled trials
RBCs	-	Red blood cells
RD	-	Risk difference
SChE	-	Serum cholinesterase
SGOT	-	Serum glutamic oxaloacetic transaminase
SGPT	-	Serum glutamic pyruvic transaminase
SACTRC	-	South Asian Clinical Toxicology Research Collaboration
SD	-	Standard deviation
TEPP	-	Tetraethyl pyrophosphate
U/L	-	Units per liter
WHO	-	World Health Organization
WBC	-	White blood cells

ABSTRACT

The management of Organophosporus compound poisoning includes continuous monitoring and timely intervention, ideally in an intensive care unit and is labour intensive. In our setting where cost is a major factor and a large number of patients who are unable to afford care in an intensive care unit are treated even while they require mechanical ventilation in the general wards/emergency room, a treatment regimen which is simple, easy to follow and includes fixed guidelines may be more easier, efficient and improve care in situations where ICU care is not possible immediately.

This study attempted to formulate, based on the current practice and evaluate an algorithmic protocol of atropine in the management of moderate to severe acute organophosphate poisoning. The current study shows that use of a guideline may result in faster atropinisation and rapid stabilization of acutely sick patients who are treated initially in the emergency room.

An open-label randomized clinical trial was conducted in , SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, VIJAYPURA(BIJAPUR), Karnataka India on 108 hospitalized individuals with OPC poisoning from January 2016 to September 2017. The aim was to know, 1. Effect of atropinization with different methods and 2. Outcome in terms of duration of hospital stay and patient's recovery .We compared two groups that used a titrated dosing protocol based on a structured monitoring sheet for atropine infusion with another group using an 'ad hoc' regime. The aim was to compare the efficacy and safety of conventional bolus doses with individualized incremental doses of atropine for atropinization followed by continuous atropine infusion for management of OPC

poisoning. Inclusion criteria were patients with a clear history of OPC poisoning with clear clinical signs of toxicity, i.e. features of cholinergic crisis. The patients were observed for up till hospital stay. Immediate outcome and complications were recorded. Out of 108 patients, 54 patients received conventional bolus dose atropine (group A) and 54 patients received rapidly incremental doses of atropine followed by infusion (group B). 36 subjects analyzed in group A and 32 in group B for moderate to severe poisoning. The mortality in group 'A' was 11.1% (4/36) and in group 'B'6.3% (2/32). The mean duration of atropinization in group 'A' was 5.8hrs (348) in minutes compared to time 26.9 minutes for group 'B'.

Conclusion: Administration of atropine using a fixed algorithm is easy and effective in providing the atropine requirement in the management of early phase of acute organophosphorus poisoning. Rapid incremental dose atropinization followed by atropine infusion reduces mortality and morbidity from OPC poisoning and shortens the length of hospital stay and early recovery. Incremental atropine and infusion should become the treatment of choice for OPC poisoning. Given the paucity of existing evidence, further clinical studies should be performed to determine the optimal dosing regimen of atropine that most rapidly and safely achieves atropinization in these patients.

Keywords

Organophosphate compound (OPC). Atropine toxicity; Organ phosphorous poisoning; atropinisation protocols.

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INTRODUCTION

Organophosphorus poisoning (OP) is the most common poisoning in India and is a common emergency health problem worldwide, particularly in developing countries. Organophosphorus poisoning is one of the most common means of attempting suicide in India because of its easy availability[1].

Organophosphorus pesticides are used widely for agriculture, vector control, and domestic purposes. Despite the apparent benefits of these uses acute organophosphorus pesticide poisoning is an increasing worldwide problem, particularly in rural areas. Most cases occur following occupational or deliberate exposure to organophosphorus pesticides. Organophosphorus pesticides are the most important cause of severe toxicity and death from acute poisoning worldwide, with more than 3,00,000 deaths each year in developing countries. Unintentional and intentional OP poisonings continue to be a significant cause of morbidity and mortality in India .In developing countries more than 90% of acute poisoning is due to suicidal attempt[1] Although data are sparse, organophosphates appear to be the most important cause of death from deliberate self-poisoning worldwide[2.]

Organophosphate compounds inhibit both cholinesterase and pseudo-cholinesterase activities. The inhibition of acetylcholinesterase causes accumulation of acetylcholine at synapses, and the resulting overstimulation of neurotransmission at the neuromuscular junction, disturbs transmission at parasympathetic nerve endings, sympathetic ganglia, neuromuscular endplates and certain CNS regions[25]. OP poisoning compounds produce their effects by inhibiting the action of

acetylcholine esterase enzyme, which leads to an increase in acetylcholine, in preganglionic parasympathetic receptors (muscarinic action), sympathetic pregangloinic synapses including adrenal medulla and neuromuscular junctions (nicotinic action). Acetylcholine is also transmitter in central nervous system.

Early diagnosis and appropriate treatment is often lifesaving. The early clinical course of OP poisoning may be quite severe and may need intensive care management. Atropine is the mainstay of treatment of effects mediated by muscarine sensitive receptors.

Basic steps involved in the treatment of poisoning are(1)Prevention of further absorption of the poison(emesis, gastric lavage, chemical absorption, chemical inactivation, purgation) (2)Enhanced elimination of the poison(biotransformation, biliary excretion, urinary excretion, dialysis) and (3)Antagonism and chemical inactivation of the absorbed drug.4)active supportive treatment may be required.

There is great variation in recommendations in textbooks for adequate dose of atropine[18] and atropinisation of an average patient sometimes can take hours and even days to stabilize vital parameters.

The dose of atropine is usually individualized to the patient's heart rate, pupil size, respiratory secretions and levels of CNS arousal. The guidelines being variable it is difficult for an inexperienced junior doctor to follow[18]. Mortality following OP poisoning remains high despite adequate respiratory support, intensive care, and specific therapy with atropine especially in the early hours. Significant numbers of the subjects needing mechanical ventilation and reaching intensive care units die

within the first 72 h of poisoning. This may be because of failure to stabilize patients early during the course of illness. On the other hand atropine overdose in itself may be responsible for adverse outcome especially in the elderly population.

There is a trend towards using minimum doses of atropine and avoiding Oximes altogether as their efficacy is still controversial* and their use late in the illness may not be useful. The optimum atropine dosage is not yet defined and under or overdosing with atropine is a common occurrence for sick patient presenting to emergency room.

The antidotes of OP poisoning are anticholinergic drugs such as pralidoxime, atropine and glycopyrrolate, atropine being the older of the two medications.

Comparative studies of different protocols have not been done to guide clinicians. South Asian Clinical Toxicology Research Collaboration (SACTRC) works in partnership with local physicians in Sri Lanka to conduct clinical trials in a number of clinical research centers. In this context we have observed considerable variation in the use of atropine within Sri Lanka.

The present study was chosen to compare the current dosing pattern of atropine in OP poisoning at BLDE University Shri B.M. Patil medical college with protocol of administration practiced at South Asian clinical Toxicology research collaboration.

There is no standard regimen, but there are two main approaches to determine the dosing schedule for atropine that were considered. Both rely on the identification of clinical features of atropinization to determine when sufficient atropine has been administered. This is termed ‘individualized treatment’. The first and more conventional approach is bolus dose treatment with atropine initially every 15 min

followed by lower doses at longer intervals until recovery. The second approach consists of more rapid atropinization with incremental boluses followed by an atropine infusion adjusted according to clinical response and tapered until recovery. From our experience, conventional bolus dose treatment is associated with delay in stabilization and ultimately a higher mortality compared to the second approach although this has not been formally investigated.

Thus, a study was done to compare the efficacy and safety of conventional bolus dose treatment with atropine for OPC poisoning with the incremental atropine dosing regimen followed by atropine infusion. The protocol used for each regimen was derived from reviews of the literature [8–9]. The objective of the study was to determine the optimal dosing regimen for atropine therapy in OPC poisoning. The primary outcome measure was mortality. Secondary outcome measures were time to atropinization, total dose of atropine required, incidence of atropine toxicity, incidence of intermediate syndrome (IMS) and duration of hospitalization. It was hoped the study would help to inform development of a future guideline for better management of OPC poisoning to improve outcome.

With the incremental dosing regimen, initial stabilization can be achieved within a very short time, followed by continuous atropine infusion, allowing less frequent follow-up, sustained blood levels of atropine with little chance of fluctuation and ultimately less toxicity and lower mortality[39]

It was felt that the knowledge gained by the study would allow us to decide which method is safer and beneficial to the patients.

AIMS AND OBJECTIVES

1. Effect of atropinization with different methods.

2. Outcome in terms of duration of hospital stay and patients recovery.

REVIEW OF LITERATURE

Acute organophosphorus (OP) poisoning is a common problem in the developing countries [1,2]. It probably kills about 300,000 people every year [3,4].

Most deaths occur in the rural areas of the developing world [2]. In countries like India deliberate self-poisoning is the most common cause of death second to road traffic accidents[5]. Their widespread use as insecticides and easy availability has resulted in serious increase in poisoning. The extent of acute pesticide poisoning in agricultural workers, particularly in less developed countries, has often been based on inadequate information. Epidemiological studies, relying mainly on hospital and poison centre data, have been biased towards the more severe poisonings, whereas field studies indicate that occupational pesticide poisoning is associated with less severe and minor effects. Many reports do not adequately distinguish between intentional, accidental and occupational pesticide poisoning statistics or are dominated by cases of intentional poisoning which, by their nature, result in severe or fatal results.

Epidemiology:

The WHO (World health organization) estimates suggest that more than 3 million cases of acute serious pesticide poisoning and 220,000 deaths occur worldwide annually, the majority being caused by organophosphates used for agricultural purposes [1]. In the hospital based poisoning surveys from India 59% of all admissions are due to pesticide poisoning[6].

In a study by Srinivas Rao et al from India there were 8040 admissions in 6

years with an overall case fatality ratio of 22.6%, two thirds of these were less than 30 years old, with 96% rates of intentional poisoining[7] .

In the Shri, B.M. Patil Medical College and Hospital, vijaypura OP poisoning accounts for 12% of all medical intensive care unit admissions and 75% of all poisoning.

History:

Organophosphorus (OP) is the general name for organic derivatives of phosphorus.OP compounds are usually esters, amides, or thiol derivatives of phosphoric,phosphonic, phosphinic, or thiophosphoric acids with two organic and additional side chains such as cyanide, thiocyanate, and phenoxy group . OPs are used as insecticides, nematocides, acaricides, fungicides, erbicides, defoliants, fire retardants, solvents, plasticizers, drugs, and chemical warfare nerve agents. They are the most commonly used insecticides in the world

The first OP compound synthesized by Clermont in 1854 was tetraethyl pyrophosphate (TEPP). It came into use during World War II as an agricultural pesticide substitute for nicotine and for use as a nerve gas in chemical warfare. It is also the most toxic of the OP insecticides. Modern investigations of OP compounds date from the 1932 publication of Lange and Krueger on synthesis of dimethyl and diethyl phosphoflouridates. Schrader described the structural requirements for insecticide activity of these compounds in 1952.(8)

Organophosphorus compounds are diverse group of chemicals used in both domestic Industrial settings .organophosphate compounds are frequently used as festicides in agriculture activities .Examples of organophosphate includes,

insecticides (malathion, parathion, diazinon,fenthion,dichlorvos, Chlorpyrifos, ethion, Nerve gases(soman,sirin,tabun,VX), ophthalmic agents chothiophate, isoflurophate), and antihelmintics (trichlorfon). Herbicides (tribufos [DEF],merphos) are tricresyl-phosphate containing industrial Chemicals [9-10].

Pathophysiology of organophosphorus poisoning:

The primary mechanism of action of OP pesticides is inhibition of acetylcholinesterase (AChE), which is an enzyme found in the nervous system. Its normal action is to break down acetylcholine (ACh).

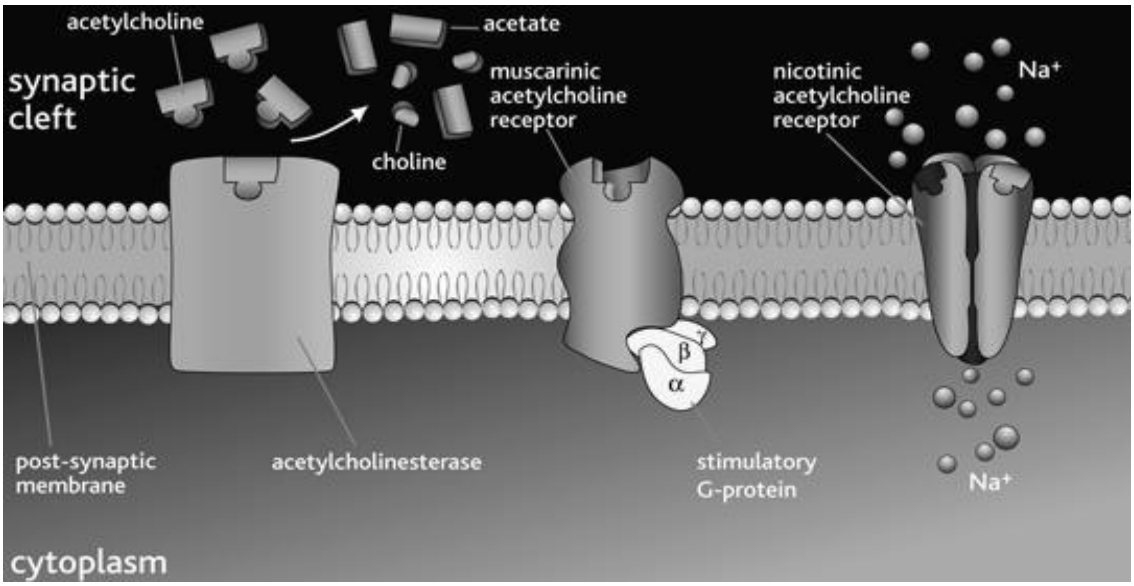


Figure 1: Action of acetylcholinesterase

OPs inactivate AChE by phosphorylating the serine hydroxyl group located at the active site of AChE. The phosphorylation occurs by loss of an OP leaving group and establishment of a covalent bond with AChE. Once AChE has been inactivated, ACh accumulates throughout the autonomic ervous system, the somatic nervous system, and the brain, resulting in overstimulation of the muscarinic and nicotinic receptors.

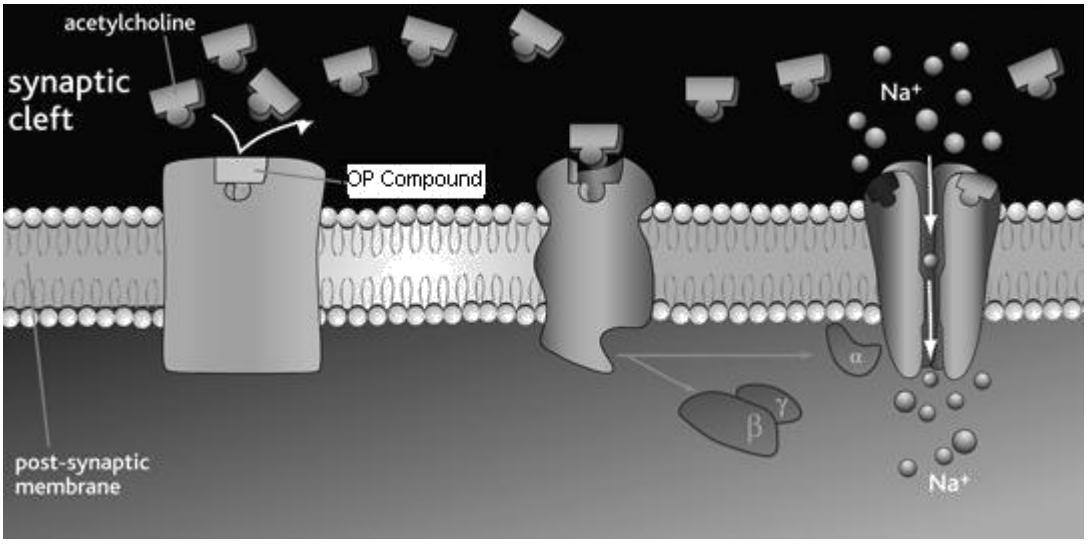


Figure 2: Inactivation of AChE and accumulation of ACh in the synapse.

(Figures r*eproduced from;* Pharmacology, *4th edition. Rang HP, Dale MM and Ritter JM. Edinburgh, UK: Harcourt Publishers Ltd, 2001:110–138.)*

The preganglionic and postganglionic neurons in the parasympathetic nervous system release ACh. Postganglionic ACh acts on muscarinic receptors on the heart, eyes, glands, GI tract, and respiratory system. Somatic motor axons emerge from the spinal cord and directly innervate muscle cells at the neuromuscular junction, releasing ACh on nicotinic receptors. The brain and spinal cord both contain muscarinic and nicotinic receptors. Cholinergic pathways in the brain are associated with various behaviors and functions, including hunger, thirst, thermoregulation, respiration, aggression, and cognition.

Once an OP binds to AChE, the enzyme can undergo 3 processes, including

(1) Endogenous hydrolysis of the phosphorylated enzyme by esterases or paraoxonases,

(2) Reactivation by a strong nucleophile such as pralidoxime (2-PAM), and

(3) Biological changes that render the phosphorylated enzyme inactive (aged).

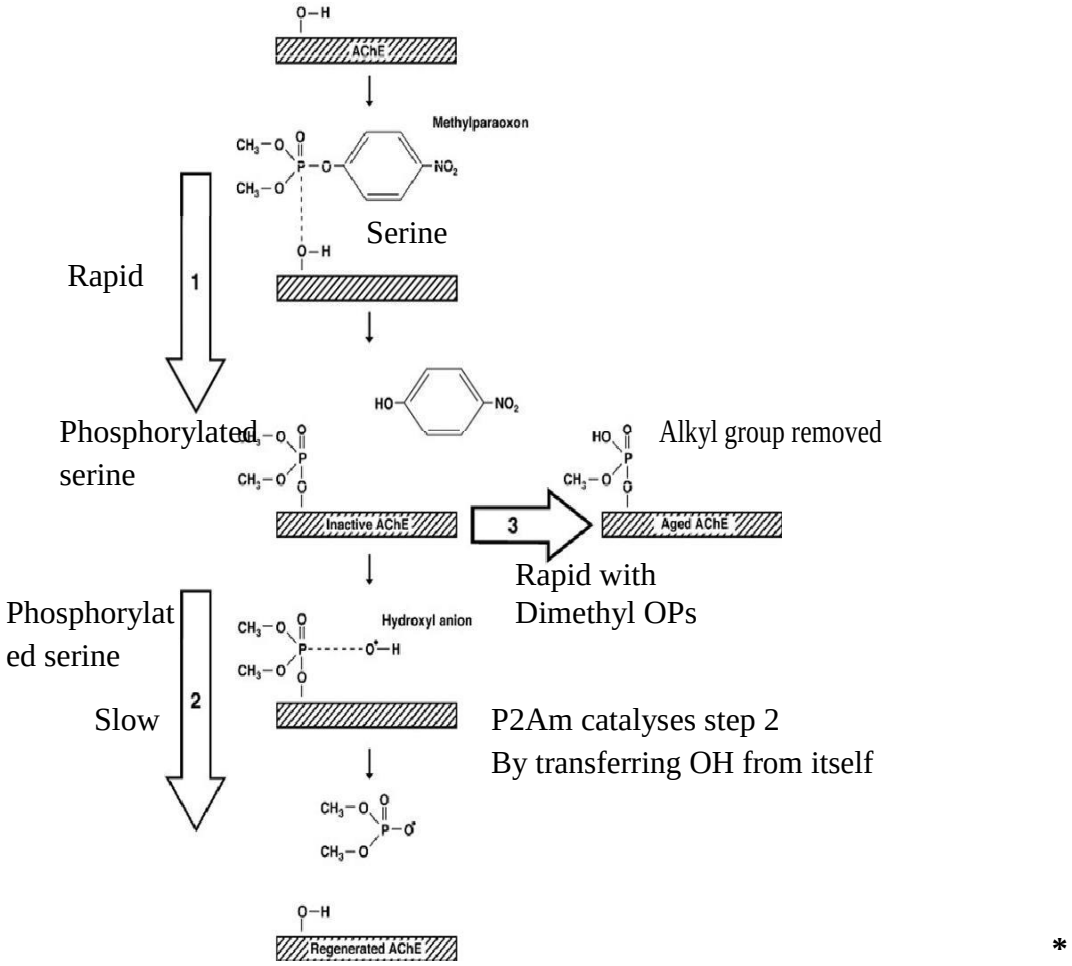


Figure 3: Interactions between OP compound and AChE enzyme.

OPs can be absorbed cutaneously, or they can be ingested or inhaled. Onset and duration of action depend on the nature and type of compound, the degree and route of exposure, the mode of action of the compound, lipid solubility, and rate of metabolic degradation.

Clinical manifestations:

The signs and symptoms of acute organophosphate poisoning are due to the effects caused by excess acetylcholine (cholinergic syndrome); they can manifest at different times. Signs and symptoms can be divided into three groups:

- Muscarinic effect: parasympathetic.
- Nicotinic effect: sympathetic and motor.
- Central nervous system effect: M1 muscuranic receptor stimulation.

The clinical syndrome of organophosphorus poisoning can be classified into

1. Acute manifestations: The cholinergic phase (occurs within 0-24 hrs.).
2. Intermediate syndrome: Due to persistent nicotinic effects of acetylcholine excess (Develops 24 to 96 hours after resolution of acute cholinergic symptoms and may persist for 4 to 18 days)
3. Chronic manifestations: Delayed neurotoxicity due to axonal degeneration. (Occurs 2-3 weeks after exposure to large doses and recovery may take upto 12 months.)

According to the degree of the severity of poisoning, the following signs and symptoms can occur during the acute phase [11,12]:-

*Mild: anorexia, headache, dizziness, weakness, anxiety, substernal discomfort, fasciculations of the tongue and eyelids, miosis, and impairment of visual acuity.

*Moderate: nausea, salivation, bronchorrhoea, lacrimation, abdominal cramps, diarrhoea, vomiting, sweating, hypertension or hypotension, and muscular fasciculations.

*Severe: miosis or mydriasis, non-reactive pupils, dyspnoea, respiratory depression, pulmonary oedema, cyanosis, loss of sphincter control, convulsions, coma, bradycardia or tachycardia, cardiac ischaemia, cardiac dysrhythmias, hypokalaemia, and hyperglycaemia. Acute pancreatitis has also occurred. Muscular paralysis may involve the respiratory muscles.

Muscarinic effects by organ systems include the following:

Cardiovascular - Bradycardia, hypotension

Respiratory - Rhinorrhea, bronchospasm, bronchorrhea, cough

Gastrointestinal - Increased salivation, nausea and vomiting, abdominal pain, diarrhea, and fecal incontinence

Genitourinary - Urinary incontinence

Ocular - Blurred vision, miosis

Glands - Increased lacrimation, increased sweating

Nicotinic signs and symptoms include muscle fasciculations, cramping, weakness, and diaphragmatic failure. Autonomic nicotinic effects include hypertension, tachycardia, pupillary dilation, and pallor.

CNS effects include anxiety, restlessness, confusion, ataxia, seizures, insomnia, dysarthria, tremors, and coma.

Neurological manifestations:

Type I - Acute paralysis secondary to persistent depolarization at the neuromuscular junction.

Type II (intermediate syndrome) - Intermediate syndrome was described in 1974 by Wadia et al [13], with an incidence from 8-49%. It develops 24-96 hours after resolution of acute cholinergic poisoning symptoms and manifests commonly as paralysis and respiratory distress. This syndrome involves proximal muscle groups, with relative sparing of distal muscle groups. Various degrees of cranial nerve palsies also are observed. Neuromuscular transmission defect and toxin-induced muscular instability play a role in intermediate syndrome. Intermediate syndrome persists for 4-18 days, can require intubation, and can be complicated by infections or cardiac arrhythmias.

Type III - Organophosphate-induced delayed polyneuropathy (OPIDP) occurs 2-3 weeks after exposure to large doses of certain OPs. Distal muscle weakness with relative sparing of the neck muscles, cranial nerves, and proximal muscle groups characterize OPIDP. Recovery can take up to 12 months.

Death in severe poisoning is likely due to effects on hear (bradycardia, arrythmias and hypotension), respiration (central or peripheral respiratory failure) and on the brain (depression of vital centres)[9].

The mechanism of cardiac toxicity is unclear and the following have all been postulated:

1. A direct toxic effect on the myocardium
2. Overactivity of cholinergic or nicotinic receptors causing haemodynamic alteration
3. Hypoxia
4. Acidosis
5. Electrolyte abnormalities
6. High dose atropine therapy. (used as treatment for organophosphate poisoning).

Management:

The following are the major aspects in the management of patients with acute organophosphorus poisoning. Despite the large number of pesticide poisoning cases occurring worldwide every year, the current evidence base in the management of acute organophosphorus poisoning is small [28].

1. Asses breathing and circulation.
2. Ensure adequate airway – suction of copious secretions and vomitus.
3. Ensure adequate oxygenation and ventilation.
4. Anticholinergic drugs – atropine.
5. Hemodynamic resuscitation.
6. Removal of contaminated clothing (in cases of accidental exposure or spilling over body).
7. Skin and mucus membranes decontamination.
8. Control of convulsions.
9. Monitoring of heart rate, blood pressure, oxygenation and level of consciousness.

10. Gastric lavage.

11. Oximes.

12. Active cooling and sedation.

Gastric decontamination

1. Ipecacuanha has been used to induce emesis in poisoned patients. It takes 20-30min to work and vomiting may last for more 30min[23]. If loss of consciousness occurs during this time, intubation may be needed in an unconscious vomiting patient. Since patients with pesticide poisoning may suddenly deteriorate, ipecacuanha is contraindicated.

   Forced emesis in hospital using other techniques is ineffective[23].

Gastric lavage is a routine practice in most cases of organophosphorus poisoning by oral route. The efficacy of gastric lavage falls rapidly with time since ingestion[24]. By the time most patients arrive in hospital, the majority of pesticide will have passed into the small bowel, out of the reach of gastric lavage. Some diluted solvent may be left in the stomach – this will smell of 'pesticide' if sucked out with a NG tube. The volume of fluid in the stomach will appear large in cholinergic poisoning due to the secretion of fluid into the bowel[25]. However most of the hospital practices gastric lavage even up to 4-6 hours after thy intake of poison.

3. Activated charcoal is routinely advocated to "adsorb" ingested poison.

There is currently no evidence that either single or multiple dose regimens of activated charcoal result in clinical benefit[26,27].The practice of giving charcoal rests upon usual practice and is now being evaluated in an RCT (ISRCTN02920054).

Giving fluids:

There have been no studies on the effects of giving IV fluids in ill patients with OP poisoning. However, due to the cholinergic effects, these patients lose a great deal of fluid into their gastrointestinal tract and lungs, and onto their skin as sweat, resulting in intravascular fluid depletion. Some also develop severe diarrhea that results in fluid and potassium loss[14].

There is no evidence that giving fast IV fluid to patients with bronchorrhoea is dangerous as long as atropine is being given simultaneously to dry the lung secretions.

Oximes

Oximes reactivate the acetylcholinesterase by removing the phosphoryl group (reaction 2, figure1). Pralidoxime is the enzyme which is used most commonly worldwide. It occurs in two common forms: Pralidoxime chloride (2- PAM, molecular weight 173) and Mesylate(molecular weight 232).

In the following situations however reactivation of inhibited acetylcholinesterase will be absent or limited.

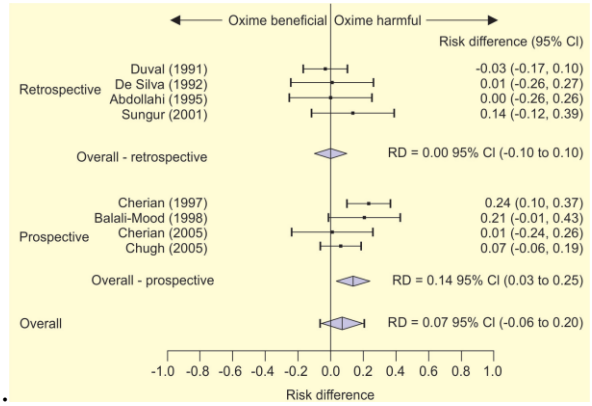
1. Poor affinity of the OP-AchE complex.
2. Insufficient dose or duration of treatment.
3. Persistence of the OP within the patient and hence rapid reinhibition of newly reactivated enzyme.
4. Ageing of the inhibited acetylcholinesterase.(reaction 3, figure 1).

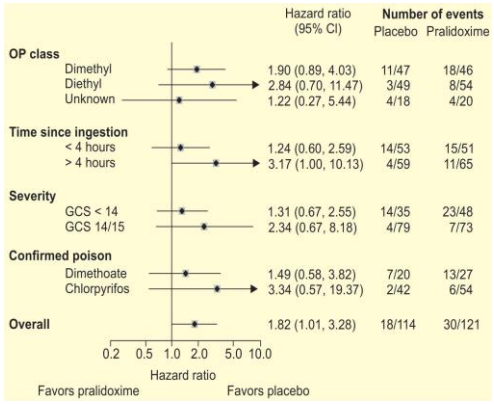
The clinical benefit of oximes for OP pesticide poisoning is not clear, being limited by the type of OP, poison load, time to start of therapy, and dose of oxime [29,30]. Current World Health Organisation guidelines recommend giving a 30 mg/kg loading dose of

pralidoxime over 10–20 min, followed by a continuous infusion of 8–10 mg/kg per hour until clinical recovery or 7 days, whichever is later [30,31]. However assessment of the primary outcomes in a recent meta-analysis indicated either a null effect or a tendency of harm with oxime therapy[32].

The lack of current prospective randomized controlled trials, with appropriate patient stratification, mandates ongoing assessment of the role of oximes in organophosphate poisoning.

Association between oxime therapy and mortality. Forest plot representation using the random effects model depicting association between oxime therapy and mortality. The vertical straight line denotes null effect. The individual points denote the risk difference (RD) of each study, and the lines on either side the 95% confidence intervals (CI). Combined estimates as well as individual estimates for the retrospective group, as well as the prospective study group are represented. Oxime therapy was not associated with a significant increase in mortality [38]





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Active cooling and sedation:

Hyperthermia is a serious complication in hot and humid wards. A febrile patient should receive the minimum amount of atropine needed to control muscarinic signs, sedation if there is excessive agitation and muscle activity, and active cooling.

Reduce agitation with diazepam. Restraining a non-sedated agitated patient to the bed is associated with complications, including death. Such patients struggle against their bonds and generate excess body heat, which may result in hyperthermic cardiac arrest[14].

Diazepam is preferred over haloperidol because large doses of haloperidol may be required in patients receiving atropine. Haloperidol is also non-sedating, associated with disturbances of central thermoregulation and prolongation of the QT interval, and pro-convulsant. Diazepam may also have other advantages because animal studies suggest that it reduces damage to the central nervous system[33] and diminishes central respiratory failure[34].

Anticholinergic drugs:

Atropine is a specific antidote for the treatment of poisoning with organophosphorus and carbamate insecticides and organophosphorus nerve agents.

Atropine is the best-known member of a group of drugs known as muscarinic antagonists, which are competitive antagonists of acetylcholine at muscarinic receptors. It has no effect at the nicotinic receptors. This naturally occurring tertiary amine was first isolated from the *Atropa belladonna* plant by Mein in 1831. Atropine earlier enjoyed widespread use in the treatment of peptic ulcer, today it is mostly used in resuscitation, anaesthesia, and ophthalmology. It may also be used to counteract adverse parasympathomimetic effects of pilocarpine, or neostigmine administered in myasthenia

gravis. The first report of atropine being used as an antidote for acetylcholinesterase inhibitors was by Fraser in 1870, he used atropine to counteract the effect of physostigmine on the pupil. Sanderson in 1961 described the effect of intraperitoneally administered atropine given alone, or combined with oximes, on the survival of rats poisoned by organophosphates[11].

Textbooks list many features of the cholinergic syndrome [11,12] . However, in practice mainly the following five are used in routine assessment: miosis, excessive sweating, poor air entry into the lungs due to bronchorrhoea and bronchospasm, bradycardia, hypotension and fasciculations. If none of these signs are present, then the patient does not yet have clinical cholinergic poisoning and does not require atropine. Fasciculations are due to the depolarizing blockade at the neuromuscular junction as stated earlier, atropine does not reverse fasciculations. However, it is possible that these signs will occur later, for example as a pro-poison (thion) OP is converted to the active oxon form, as a fat-soluble OP such as fenthion leaches out of fat stores into the blood, or if the patient has presented soon after the ingestion. Careful observation is required to look for the development of cholinergic signs.

Giving atropine before oxygen:

Although it is preferable that oxygen is given early to all ill patients, delay should not occur in giving atropine if oxygen is unavailable.

Some reports suggest that atropine should not be given to a cyanosed patient until oxygen has been given - to reduce the risk of atropine inducing ventricular tachycardias[11]. While apparently sensible, such advice risks preventing doctors working in small rural hospitals from giving life-saving atropine treatment, since many do not have oxygen.

Furthermore, in treatment of more than 800 patients receiving atropine, many of whom received atropine before oxygen, no patient had a cardiac arrest within minutes of giving atropine[14]. The primary evidence for an increased risk of a ventricular dysrhythmia from giving atropine to a cyanosed patient consists of very few patients. Since atropine dries secretions and reduces bronchospasm, its administration should reduce cyanosis. There is no good evidence that giving atropine to a cyanosed patient causes harm [14].

Use of atropine

Basic pharmacology and animal work suggests that early antagonism of pesticide toxicity should be associated with better outcomes [15,16]. Although there are few studies on the subject, there is some evidence that patients in the developing world often die soon after admission[17]. Full and early atropinisation is an essential and simple part of early management. Delayed atropinisation can result in death from central respiratory depression, bronchospasm, bronchorrhoea, severe bradycardia and hypotension[25]. Animal work suggests that these early deaths may be primarily due to central cholinergic stimulation[15].

The initial half-life of distribution of atropine is about 1 minute[11, 22]. After intravenous dosing, atropine distributes rapidly with only 5% remaining in the blood compartment after five minutes. Studies in anesthetized patients indicate that the peak effect is seen within three minutes of an IV injection[23].

There is therefore no need to wait for more than five minutes before checking for a response and giving another bolus dose if no response has occurred. After intravenous dosing, atropine elimination fits a two-compartment model with an intrinsic clearance of 5.9-6.8 ml/kg/min and a plasma half-life of 2.6-4.3 hours in the elimination phase[11]. In an emergency situation, it may be necessary to give atropine before intravenous or

intraosseous access can be established. The endotracheal route has therefore been used, when vascular access was not available with success. The best regimen for the administration of atropine has not been established [11]. A study performed in

Bangalore, India, found that a regimen of bolus loading doses followed by an infusion improved outcome compared to repeated bolus doses [19] however, this study used historical controls which risks inflating benefit compared to

RCTs [20,21]. Although the benefit of infusions is not yet proven, the use of bolus loading doses followed by an infusion may save time, require less observation, produce less fluctuation in plasma atropine concentrations, and make weaning easier [11].

It is preferred to give low doses of atropine to start with and then rapidly escalate the dose. An alternative approach is to start with much bigger doses, to ensure rapid atropinisation, and then wait for the atropine levels to fall. Because of the dangers of over-atropinisation, however, the former practice offer more control by starting with low doses. It is difficult to distinguish patients who needed very large doses of atropine from those who required just a few milligrams[18].

There are various recommendations about the use of atropine in the treatment of organophosphorus poisoning. Eddleston et al *obtained thirty three different recommendations for atropinisation from clinical toxicology textbooks and electronic sources, national formularies, and international textbooks of internal medicine.

The following is the atropine recommendations in text books, handbooks, and online databases of clinical toxicology as published by Eddleston et al in there study on comparison of recommended regimens of atropine[18].

Dose recommended in the literature for atropine in treatment of acute OP poisoning.

Table 2: Atropine dose recommendations in literature.

Source		Edition/ year	recommendation	Maximum dose 24 hrs	Atropine dose per hour
Harrisons book of Medicine	text	19th/2015	The field-loading dose is 2, 4, or 6 mg, with retreatment every 5–10 minutes	>480 mgs	20-30mgs/hour
Davidsons book of Medicine	text	22nd/2014	0.6–2 mg IV, repeated every 10–25 mins	288 mgs	12 mgs/hour
British national		46th/2003	2mgs repeated every 5-10	576 mgs	24 mgs/hour
Formulary			Minutes		
Oxford textbook		4th/2003	2 mgs repeated every 10-30	288 mgs	12 mgs/hour
Of			Minutes		
Medicine					
WHO treatment		1999	1-2 mgs repeated every 5-	576 mgs	24 mgs/hour
Guide			10 minutes		
Poisindex OP		2003	2-5 mgs every 10–30	720 mgs	30 mgs/hour
Poisoning			Minutes		
Ford		1st/2001	1-2 mgs repeated every 5	Large Doses	
			minutes doubling the dose.	(100s of mgs)	
Reigart		5th/1999	If GCS normal: 2-4 mg	Large doses.	96 mgs/hour
			every 15 min.		
			If GCS reduced: 4-8 mg		
			every 5-15 minutes.		
Fernando		2nd/1998	2-10 mg, then 2 mg	298 mgs.	12 mgs/hour
			Repeated every 10-15		
			Minutes		
Goodman Gilman s book of pharmacological therapeutics		2011	2-4 mg ,then 2mg every 5-10 minutes	More than 200mg	8-9mg/hour
SACTRC		2007	1.5-3 mg ,and doubling dose every five minute still signs of atropinisation fallowed by continuous infusion 10-20% of atropinized dose hourly		1.5-3mg /hour infusion

He calculated the time needed to achieve initial atropinisation by using these regimens on his 22 patients enrolled in the study. The patients required a mean of 23.4 mg (standard deviation 22.0, range 1–75 mg) atropine to clear the lungs, raise the pulse above 80 bpm, and restore systolic blood pressure to more than 80 mmHg. Textbook recommendations varied markedly — atropinisation of an average patient, requiring the mean dose of 23.4 mg, would have taken 8 to 1380 mins; atropinisation of a very ill patient, requiring 75 mg, would have taken 25 to 4440 mins. Thus there is great variation in recommendations for adequate dose of atropine. Thus there is a need to review the evidence for atropine administration and to produce and disseminate a simple guideline that will be useful for doctors faced by this severe form of poisoning across the world. Given the paucity of existing evidence, clinical studies looking to determine the optimal dosing regimen of atropine that rapidly and safely achieves atropinisation in these patients are required.

In another study South Asian Clinical Toxicology Research Collaboration (SACTRC) works in partnership with local physicians in Sri Lanka to conduct clinical trials in a number of clinical research centers. In this context we can observed considerable variation in the use of atropine within Sri Lanka. The recommended dosing protocol for symptomatic patients in the titrated atropine cohort has been described more fully . It consists of an initial bolus of 1.5 to 3 mg of atropine with doses doubling every 5 minutes until atropinisation is achieved. Clearing of chest on auscultation was used as the primary endpoint of atropinization. Following that an infusion is given with a rate that is estimated from the size of the initial dose required

to achieve atropinisation. This is typically in the range of 1 to 2 mg/hour. The infusion dose is subsequently adjusted up or down depending on the presence of signs of under atropinisation (chest signs, sweating, bradycardia and miosis) or over atropinisation (absent bowel sounds, fever, tachycardia, mydriasis and confusion). The '*ad hoc*' dose cohort received intermittent boluses, infusion or a combination of bolus and infusion as decided by the treating doctors.

During the study 272 symptomatic patients with anticholinesterase poisoning requiring atropine were admitted to the three hospitals. Outcomes of death and ventilation were analyzed for all patients, 226 patients were prospectively assessed for atropine toxicity. At baseline patients in the titrated dose cohort had clinical signs consistent with greater toxicity. This in part may be due to ingestion of more toxic OPs. They received less pralidoxime and atropine and were less likely to develop features of atropine toxicity such as delirium (1% vs 17%), hallucinations (1% vs 35%) or either (1% vs 35%) and need for patient restraint (3% vs 48%) compared with the '*ad hoc*' dose regime. After adjusting for the pesticides ingested, there was no difference in mortality and ventilator rates between protocols[52].

SHREE B M PATIL MEDICAL COLLEGE & HOSPITAL VIJAYAPUR 2-3mg IV repeated every 10-15 minutes until the therapeutic end point is reached i.e. until pulmonary secretions dried(reflected by improved oxygenation and ease of breathing(or ease of ventilation) .Fallowed by decreasing dosage or increase duration.

Criteria for atropinisation

Patients die acutely from respiratory or circulatory failure, the former of which is exacerbated by bronchospasm and bronchorrhoea. All respond to atropine treatment.

There are no comparative studies of markers for adequate atropinisation[18]. In particular, since current endpoints do not include a CNS endpoint, it is possible that atropinisation is not reversing the CNS cholinergic syndrome, which may have significant effect on preventing early death from OP poisoning [15]. In practice, air entry on chest auscultation, heart rate, and blood pressure are the main parameters used for adequate atropinisation.

Furthermore, both dilated pupils and tachycardia can result from stimulation of nicotinic ACh receptors, and tachycardia result from low total peripheral resistance with a partially compensatory high cardiac output[35].

In summary the target end point to atropine therapy[14]:

a) Clear chest on auscultation with no wheeze

b) Heart rate >1000 beats/min

c) Pupils no longer pinpoint

d) Dry axillae

e) Systolic blood pressure >80 mmHg

Atropine toxicity

There has been a tendency among clinicians to advocate over-atropinisation to reduce the risk of them being under-atropinised. However, atropine toxicity has its risks and complications. Hyperthermia is a particularly serious complication in the hot wards of the developing world. Initial CNS stimulation leads to severe agitation. Agitated patients in ambient temperatures greater than 35C, not sweating because of atropine, can become very hot. Hyperthermia resulting from the high ambient temperatures is exacerbated by intense muscle activity due to atropine-induced agitation and failure of sweating, and sometimes if the patient is an alcoholic, then the situation is worsened due to delirium tremens. Endpoints such as pulse rates >120/min and totally dilated pupils suggest that the patients are being given too much atropine. A further problem with fast heart rates is ischaemic heart disease in elderly patients. There are reports of patients with fairly mild poisoning who died in ICU from myocardial infarctions after being given atropine to keep their heart rate at 120–140 bpm[18]. Other complication of atropine usage includes paralytic ileus, urinary retention, and precipitation of glaucoma and hypersensitivity reactions[11].

Glycopyrronium bromide (glycopyrrolate)

Glycopyrronium bromide has been used instead of atropine because it is thought to have fewer adverse effects on the central nervous .system. There are no randomized controlled trials comparing glycopyrronium bromide (glycopyrrolate) versus placebo, but it is unlikely that such a trial would be considered ethical unless glycopyrronium bromide and placebo were administered in addition to atropine. One

small randomized controlled trial found no significant difference in mortality or ventilation rates between glycopyrronium bromide and atropine, but it may have lacked power to detect clinically important differences[37].

Conclusion:

The above review shows that there is a lacuna in information on the requirement of atropine in the management of acute organophosphorus poisoning. Although there are reports of patient being managed with both low and high dose atropine there are no recommendations or human studies or any formulated protocol in the treatment of organophosphorus poisoning.

This study is designed in two parts of comparison to be analyses of the current dosing practice of atropine being used in the department of medicine

The data will be used to develop a dosing protocol

MATERIALS AND METHODS

SOURCE OF DATA : Data of Patients who are enrolled in the study, collected from patients fulfilling inclusion and exclusion criteria attended both in ICU(Intensive care unit) and Emergency ward with h/o acute organophosphorus poisoning were considered for study at tertiary care hospital ,Shri B.M. PATIL Medical College, Hospital and Research Centre, Vijayapura during period of Jan 2016 to September 2017. These cases taken are not overlapped with other postgraduate students.

Period and type of study :

Comparative experimental study conducted during the period of January 2016 to September 2017 .

Study design:

The patients were randomly divided into two groups according group chosen by time of enrollment. The sealed box contained equal numbers of cards with either "group **A**"(**Shri B M Patil medical college and hospital**) or "group **B**"(**SOUTH ASIAN CLINICAL TOXICOLOGY RESEARCH COLLABORATION)** and a study code written on them. These two groups were then given atropine with different dosing regimens as described in detail in the following text and in Fig. 2 and all patients followed up till discharge and data analyzed.

INCLUSION CRITERIA.

1. History of organophosphate poisoning (within 48 hours of poisoning and above age 18yrs.) or Signs of organophosphate poisoning (at least one of the following four signs- bronchorrhea, miosis, fasciculation,

bradycardia) and

2. Low serum cholinesterase level(less than 25% of normal) with Moderate or severe poisoning (Namba scale)

EXCLUSION CRITERIA

1. Admission after 48 hours of poisoning
2. Carbamate or other poisoning. Patients with mild poisoning assessed by the Namba scale36 (appendix 1).
3. Patients with known systemic illness like malignancy, chronic lung disease, renal or -hepatic failure.
4. Pregnancy.

Using the formula

With anticipated mean difference of and hospital stay (days) between two study groups as 1.2 days and anticipated standard deviation of 1.7 days, minimum sample size of one group is 54,with 90% power and 5% level of significance.

Total sample size = 54+54 =108 Subject Enrollment

Study subjects were selected on the basis of history and physical examination. Specially trained hospital medical officers were recruited from the medical wards for monitoring the study patients and collecting clinical data. Written informed consent was sought from all enrolled subjects, and in those who were unable to do so through being too unwell or unconscious, it was sought from their attending relatives. Of these patients, those who recovered were asked if they wished to continue in the study. The patients were treated according to allocated groups A or B as shown in Fig. 2.Study doctors were unblinded as to which study group the patients were in as the nature of

the treatments with frequent reviews and dose/interval adjustments made blinding difficult. Baseline data including age, sex, religion, occupation,education, marital status, circumstances of poisoning, type of poison used, nature of poisoning and treatment prior to hospitalization including prior gastric lavage

(E.g. in primary health care centers or Private hospital or any other centers) were recorded for all patients by conducting direct interviews with the patients and attending relatives. Data on timing and dose of atropinization and atropine toxicity were recorded in an atropinization observation chart as developed by Eddleston et al[18]. Once the target number (n=50%) had been recruited in both groups, a meeting of the investigators was held to review the adequacy of enrollment to date and it was agreed that recruitment be ceased.

All consecutive patients with organophosphorus poisoning or clinically suspected organophosphorus poisoning who fulfilled the inclusion criterion, admitted to the Shri .B.M.Patil Medical college hospital Vijaypura between 1st January 2016 and 30 September 2017 were enrolled into the study. Data was prospectively collected from the inpatient records during their treatment and patients were followed up till discharge from the hospital. based On the data collected a dosing regimen was evolved for the treatment of acute organophosphorus poisoning. The treatment was carried out as per the current practices in the hospital. All the decisions regarding the treatment the patient was made by the treating team.

Statistical analysis

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean, standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries. Chi-square (χ^2)/ Freeman-Halton Fisher exact test was employed to determine the significance of differences between groups for categorical data. The difference of the means of analysis variables between two independent groups was tested by unpaired t test. If the p-value was < 0.05, then the results were considered to be statistically significant otherwise it was considered as not statistically significant. Data were analyzed using SPSS software v.23.0. and Microsoft office.

CONSORT Flow Diagram

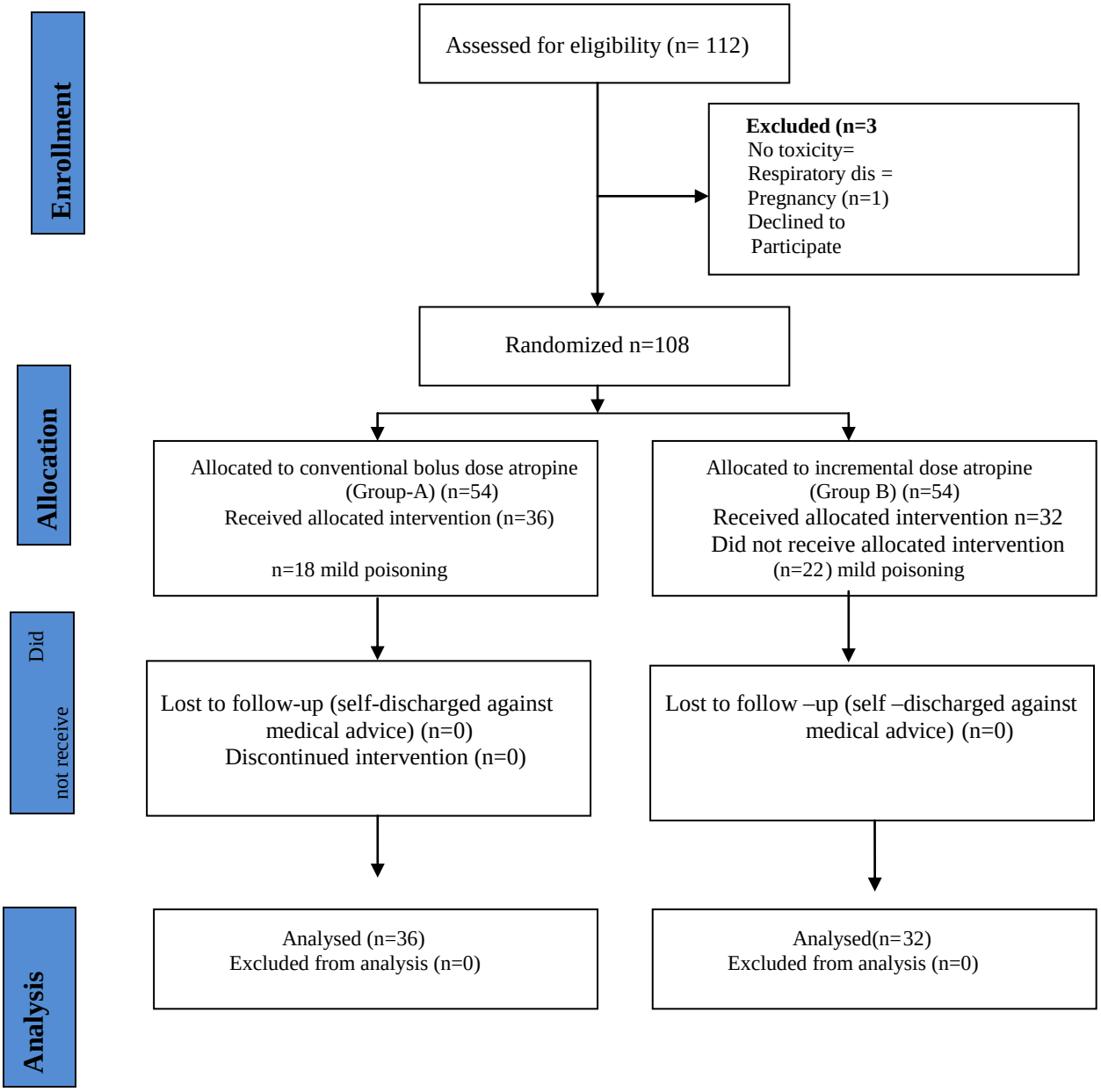


Fig. 1 Consort flow diagram of trial

Clinical assessment:

*

Daily assessment and documentation of the hourly atropine requirement, atropine toxicity, in hospital events, complications, onset of intermediate syndrome, requirement for mechanical ventilation, tracheostomy and duration of mechanical ventilation were documented according to the clinical proforma (Appendix 2) by the investigator till the patient was discharged from the hospital for both parts of the study.

Assessment of severity of poisoning

The severity of poisoning was assessed by the Namba scale[36] (appendix 1). The poisoned patients were divided into three categories, mild, moderate and severe. Only patients with moderate or severe poisoning were included in the study.

Diagnosis of intermediate syndrome:

Intermediate syndrome was defined as proximal muscle weakness of Grade 3 or less, 72 hrs after poisoning with or without requirement ofmechanical ventilation.

Markers used to asses' atropine toxicity

Confusion, delirium, pyrexia, absent bowel sounds or urinary retention were used mainly in the diagnosis of atropine toxicity. Other possible reasons for the mentioned features were excluded.

Outcome measures

1. Duration of hospital stay was calculated from the time of admission to the discharge of the patient from the hospital, measured in days.
2. Duration of ICU stay: from admission to discharge from medical ICU measured in days.

   The patients are transferred from the medical ICU on the basis of the following criterion

   a. Off ventilatory support for 24 hours

   b. Hemodynamically stable
3. Need for ventilation

The need for ventilation was based on the following guidelines

   a. Respiratory rate of more than 30/min.

   b. Shallow breathing

   c. Oxygen saturation of < 90% or PaO_2 < 60 mmHg or PCO_2 > 50 mm Hg.

4. Duration of ventilation: Time till mechanical ventilation was discontinued. This included weaning time.
5. Intermediate syndrome:

   Presence or absence of intermediate syndrome was assessed in each case according to the criterion laid down earlier.

6. Infections:

   Most common infections in these patients viz. Aspiration pneumonia,

ventilator associated pneumonia, thrombophlebitis, blood stream infection, catheter related infections, skin and tracheostomy site infection and exposure keratitis was looked for in each patient.

7. Other hospital related morbidities like occurrence of acute renal failure, pancreatitis, hepatic dysfunction, arrhythmias, cardiac arrest and atropine toxicity was also documented.

8. Mortality

   Death occurring due to any cause was considered to be directly or indirectly due to the poisoning.

Patients lost to follow up

These were patients who were taken away by the relatives due to monetary constraints, futility of the situation etc. These patients were counted as "dead" for the analysis.

TREATMENT FOLLOWED

Data was prospectively collected from the inpatient records during their treatment and patients were followed up till discharge from the hospital. Based on the data collected a dosing regimen was evolved for the treatment of acute organophosphorus poisoning. The treatment was carried out as per the current practices in the hospital. All the decisions regarding the treatment of the patient was made by the treating team.

Method of implementation of the study protocol:

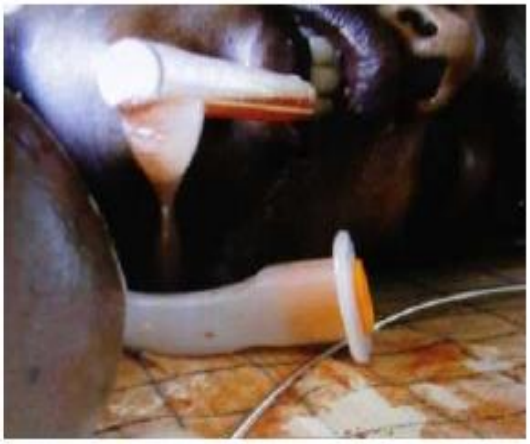
On arrival to the emergency room, the patients were screened and enrolled into the study. The stabilization was carried out in the emergency ward and patients were monitored using continuous ECG monitor, pulse oximetry and blood pressure. Atropine was used for the initial atropinisation as decided in group protocol. After achieving the target blood pressure and heart rate the patients were given planned atropine infusion as per the protocol.

Initially the atropine infusion was administered by using microdrip intravenous sets or infusion pump in the emergency room. Once stable, the patients were shifted to the ward or ICU. The instructions regarding the study protocol was documented for administration by the nursing staff and they were instructed regarding the same. In the ICU, patients were monitored continuously. In the event of bradycardia, patient was given atropine bolus dose by the nursing staff, to achieve the target heart rate as planned. The reason for change in the dose of atropine planned was documented. The infusion rate was decreased in the event of a tachycardia. Vital parameters were recorded continuously in the ICU and at least hourly in the ward on the first day. The decision for intubation and shifting to ICU if required was taken by the treating physician.

The patients were examined daily and the hourly dose administered, the hospital events and complications were documented by the researcher. Data was also collected from the inpatient hospital records. Patients were also examined regularly by the treating physicians and all the decisions regarding the daily management was taken by them. The decisions regarding duration of ventilation, tracheostomy and

antibiotic usage was also decided by them. The patients were assessed daily till discharge from the hospital.

The algorithm for treatment used in the study is shown in Fig. 2. Overall, patient management was the responsibility of the ward doctors and study- specific treatments (A, B and oxime) were given by the study doctors working Closely with the ward doctors or ICU in charge or in emergency room. The initial treatment was establishment of a clear airway and adequate ventilation. High-flow oxygen was given if necessary and airways were maintained using a Guedel airway. The patients were kept in the left lateral position, with head down and neck extended to minimize aspiration. As most patients were clinically dehydrated, circulation was supported by giving 500–1,000 ml normal saline (10–20 ml/kg) over the first 10–20 min. Measures were taken to reduce absorption of OPC through skin and mucus membranes, i.e. washing with water and soap where available and removal of clothes contaminated with OPC. Vital signs and Glasgow Coma Score (GCS) were recorded at baseline and throughout the admission. In persons with suspected mild poisoning, a 'test dose' of intravenous parenteral atropine sulphate (1 mg in adults) was given (Fig. 2). If signs of atropinization occurred rapidly, poisoning was considered to be mild and such individuals were not included for analysis in the study (Fig. 1). Both groups received parenteral atropine, as described in the following text, following predefined algorithms, during which they were monitored at least every 3 h-6hrs.



OROPHARYNGEAL AIRWAY USED

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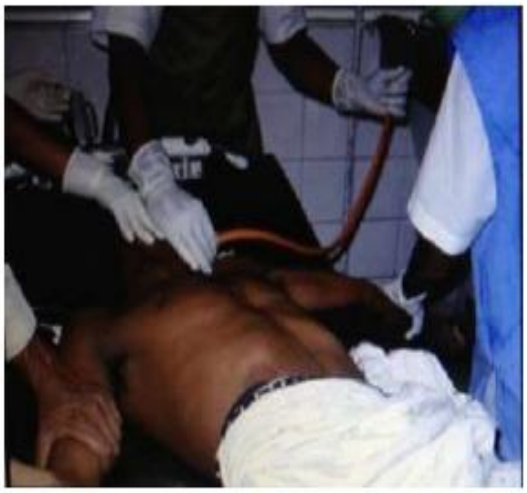


GASTRIC LAVAGE

ACTIVATED CHARCOAL

Management of acute Organophosporus poisoning

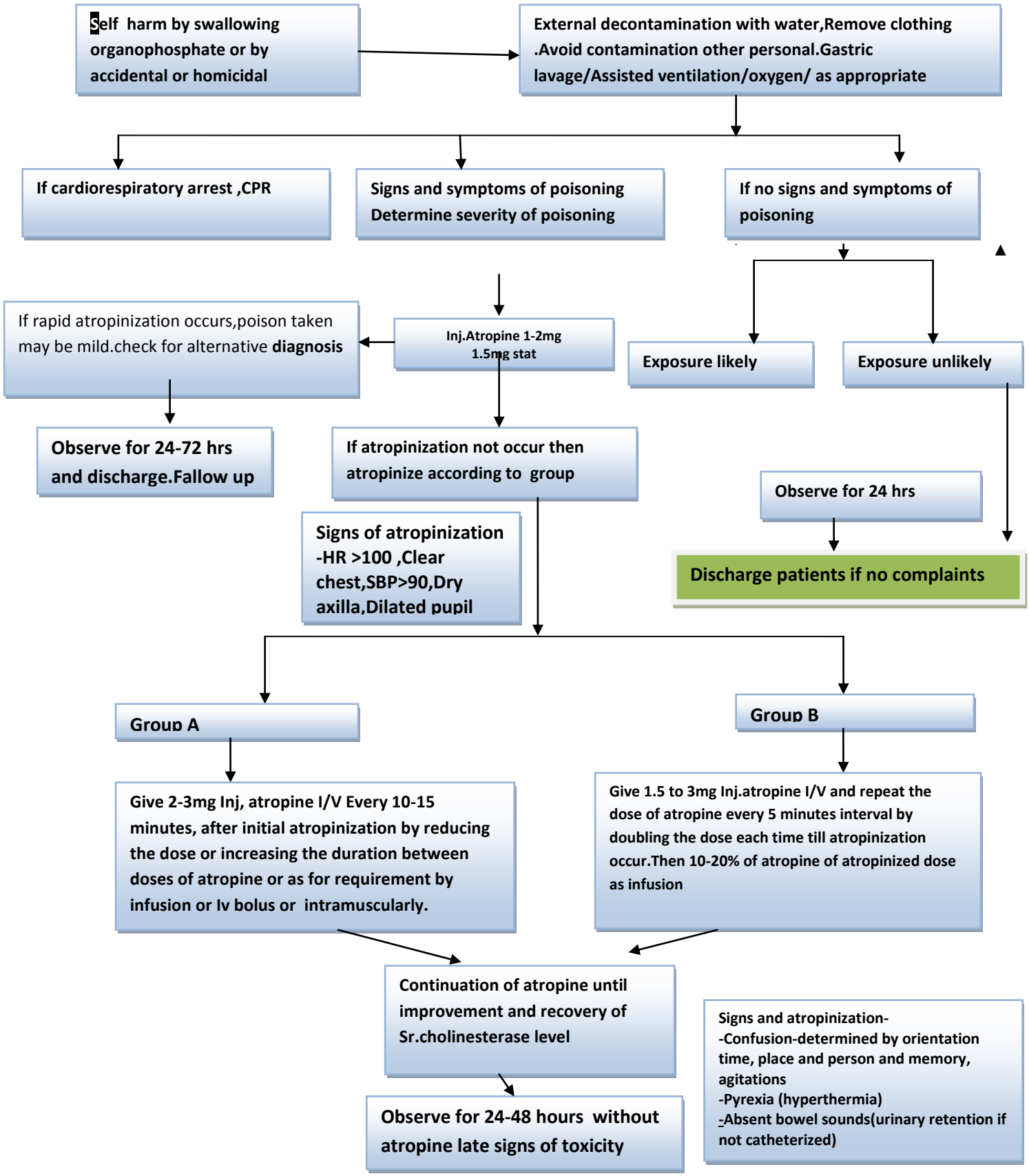


Fig :2

Algorithm for the approach to the management of poisoning due to organosphosphate compounds.

Common treatment followed to both group

The severity of poisoning was assessed by the Namba scale[36.](appendix 1). The poisoned patients were divided into three categories, mild, moderate and severe. Gastric lavage and adequate ventilation maintained, The stabilization was carried out in the emergency ward and patients were monitored using continuous ECG monitor, pulse oximetry and blood pressure.clinically assessed dehydration corrected with Intravenous fluids, Oxygenation and catheterization before atropine was made. Pam (oximes) had been given both groups. Treatment with pralidoxime was repeated as a bolus at the same dose and rate as the initial dose every 8 h for 48 h in all surviving patients as per standard practice. Evidence of pralidoxime toxicity including tachycardia, muscular rigidity, neuromuscular blockade, hypertension, laryngospasm and mild hepatitis was recorded and managed by reducing the subsequent dose of pralidoxime and/or atropine as appropriate. Atropinization fallowed according to group decided by treating team. Other supportive treatment had been made as for requirement, like eye care, oral care, endotracheal tube care,, Tracheostomy did in some patients who had longer period of ventilator supports.

Diagnosis of intermediate syndrome(IMS):

Intermediate syndrome was defined as proximal muscle weakness of Grade 3 or less, 72 hrs. After poisoning with or without requirement of mechanical ventilation. All patients were monitored for early signs of IMS due to OPC poisoning. Signs of IMS were weakness of neck flexion, difficulty in lifting the head off the pillow, use of accessory muscles of respiration, nasal flaring, tachypnea, sweating, cranial nerve palsies and proximal limb muscle weakness with retained distal muscle strength. IMS

was managed by supportive measures including intubation and ventilation if required**.** The patients were discharged after a minimum of 24 h of observation post- cessation atropine if they had no residual features of OPC poisoning.

Markers used to asses' atropine toxicity

Confusion, pyrexia, absent bowel sounds or urinary retention were used mainly in the diagnosis of atropine toxicity. Other possible reasons for the mentioned features were excluded. Daily assessment and documentation of the hourly atropine requirement, atropine toxicity, in hospital events, complications, onset of intermediate syndrome, and requirement for mechanical ventilation, tracheostomy and duration of mechanical ventilation were documented according to the clinical proforma (Appendix 2) by the investigator till the patient was discharged from the hospital for both parts of the study.

PSYCHIATRIC EVALUATION: Patients evaluation by a psychiatrist done in both groups.

Group A

The number of patients in groups received increasing doses of Inj.atropine 2- 3mg every 10-15 minutes depending on severity of signs and symptoms as decision made by treating clinician. This was repeated every 10 to 15 min until signs of atropinization were clinically evident(11 thesis bangl)((clear chest on auscultation with resolution of bronchorrhea, heart rate of >100 beats per minute, systolic blood pressure >90 mmHg, dry axillae and pupils >2mm in diameter). Inj.atropine 1cc= 0.6mg, that means 2-5cc of atropine had been repeated every 10-15 minutes until signs of atropinization ,this is

followed by either intravenous infusion or bolus or intramuscular route for maintenance every hourly according to clinically assessed and the subsequent dosing with atropine injections was individualized either by decreasing the dose or increasing the duration in between doses as per the preference of the treating clinician, provided features of atropinization were still present. If one or all of these features were absent, the dose or frequency of atropine was increased as per the preference of the treating clinician. Atropinization was maintained for at least 24 h until clinical recovery, i.e. Resolution of all features of cholinergic crisis listed earlier.

Following the initial atropinization, patients had to be reassessed for the five features of atropinization every 15 minutes. When atropinization could not be achieved (bronchospasm or bradycardia, sweating and miosis), further bolus atropine was administered. This dose was decided by the treating clinician and the maintenance infusion or Intrvenous or intrmascular was subsequently adjusted by of total bolus dose in the initial loading. After atropinzation, patients were observed at least every hour for 6 hours. If atropine toxicity developed (confusion, pyrexia, absent bowel sounds; all three should be present), atropine was stopped and patients were closely monitored. Intravenous diazepam was administered in case of agitation. Atropine could be restarted at 70-80% of previous rate after toxicity resolved. Patients were also carefully monitored for development of intermediate syndrome (broken neck sign, use of accessory muscles for respiration, nasal flaring, tachypnea, sweating, cranial nerve palsies, and proximal muscle weakness)

Prescribed deviation in the atropine dosing

In case of a decline in heart rate to less than 100 bpm on day 1 and 60 bpm on day 2 onwards 2-3cc i.e. 1-2mg atropine intravenous boluses/ Intramuscular to be repeated every 15 minutes. In case of persistent tachycardia HR > 120bpm atropine infusion can be lowered by 1 mg/h or assessed clinically.

Group B

It consists of an initial bolus of 1.5 to 3 mg of atropine with doses doubling every 5 minutes until atropinisation is achieved. Clearing of chest on auscultation was used as the primary endpoint of atropinization. Following that an infusion is given with a rate that is estimated from the size of the initial dose required to achieve atropinisation.This is typically in the range of 1 to 2 mg/hour. Incremental dose was defined as 1.8–3 mg atropine by intravenous (IV) infusion, repeating the dose every 5 minutes interval doubling the dose each time to the point atropinization occurs, followed by 10–20% of atropine required for atropinization,every hour by IV infusion. Bolus dose was defined as (2–5 mg of atropine every 10–15 minutes) followed by maintenance using reduced doses or increasing the timeduration in between the doses

They were given more rapidly incremental doses of bolus atropine, i.e. a first dose of 1.8–3.0 mg (3–5 ampoules) depending on severity as assessed by the treating clinician followed by another 5 min later double this dose if needed. This was repeated every 5 min until all clinical signs of atropinization were clearly evident. After initial atropinization, patients were maintained on an atropine infusion, using 10% of the atropine required to load the patient given per hour (e.g. if atropine required for

atropinization was 15 mg, 1.5 mg was Infused each hour by mixing the amount of atropine required for 24 h with 1,000 ml normal saline and giving it at a rate of 40 micro drops per minute as a continuous infusion). After initial atropinization, patients were reviewed and assessed for the five features of atropinization (given earlier for group A) every 15 min. At each review, if the features of atropinization were not present, a further bolus of atropine was given (the amount decided by the treating clinician) and the infusion rate was also increased by 0.6 mg/h until the features appeared. Once this had occurred, patients were observed at least every hour (more frequently if between 3 and 5mg/h of atropine was being given) for the first 6 h to check that the atropine infusion rate was sufficient and that there were no signs of atropine toxicity (the presence of all three of confusion, hyperthermia (axillary temperature >99°F) and absent bowel sounds). Thereafter, patients were seen at 2-to 3-h intervals if no complications were present. When atropine toxicity occurred, atropine infusion was stopped temporarily and the patient was checked after 30 min to see whether the features of toxicity had settled. If not, the treatment was withheld with further reviews every 30 min until atropine toxicity had resolved. Atropine was then restarted at 70– 80% of the previous rate. The patient was observed frequently to ensure that the new infusion rate had reduced the signs of atropine toxicity without permitting the reappearance of cholinergic signs. This infusion was discontinued after a minimum of 24 h once all features of cholinergic crisis had resolved.

Initials XXX	Study number ipd RGN HR >100/mn	Date of admission XXX	Pupil size	Dry axilla	Systolic BP>100 mm of hg	Bowel sounds	CONFUSED A/D/N/I	FEVER>37.5 C	ATROPINE INFUSION Bolus? given	
		Clear lungs								BOLUS
Time										
22.3	52	Crepts+	Pin point	No	90/60	I	No	No		2.4
22.35	60	Crepts+	Pin poin	No	90/60	I	No	No		4.8
22.4	82	+/-occ	Pin poin	yes	100/60	N	No	No		4
22.5	100	**WHEEZE**	2mm	Yes	-	D	No	No		2
23	104	Clear	3mm	Yes	-	D	No	No	2	infusion
23.15	102	Clear	3-4mm	Yes	-	D	No	No	2	infusion
23.3	102	Clear	3-4mm	Yes	-	D	No	No	2	infusion
0.3	98	Clear	3-4mm	Yes	100/70	D	No	No	2	infusion
1.3	85	Clear	3-4mm	Yes	-	D	No	No	2	infusion
2.3	72	**WHEEZE**	3-4mm	Yes	-	N/D	No	No		2
2.35	96	Clear	3-4mm	yes	-	D	No	No	2.4	infusion
2.45	98	Clear	3-4mm	Yes	-	D	No	No	2.4	infusion
4	102	Clear	3-4mm	yes	-	D	No	No	2.4	infusion

An observation chart group B recording the initial atropinisation of an organophosphorus-poisoned patient

Atropinisation was reached at 23.00, 30 min after the first atropine dose was given; a total of 13.4 mg of atropine was required. After 10 min, doubling doses were no longer used because there was a clear response to therapy with the pulse climbing above 80 beats/min and the chest sounding better. After a further 1.5 hours, the pulse rate started to drop but it was not until it had dropped below 80 beats/min and wheeze had become audible in the chest that another 2 mg bolus was given to atropinise the patient again. The atropine infusion rate was also increased and the patient remained stable for the next few hours.

A/D/N/I, absent/decreased/normal/increased; crepts, crepitations; syst. BP, systolic blood pressure. **Clinical features in bold type indicate that atropine is required. Dashes indicate that no BP reading was taken [9].**

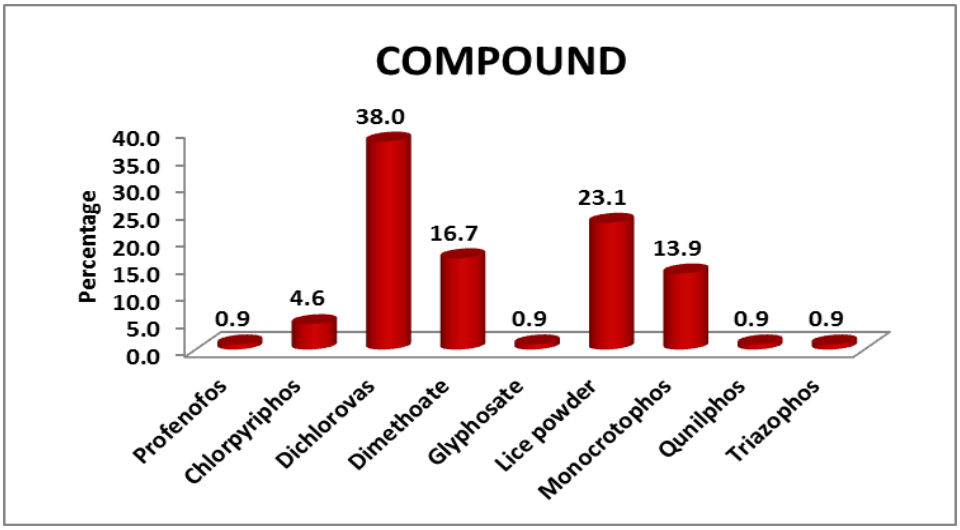
Notes:

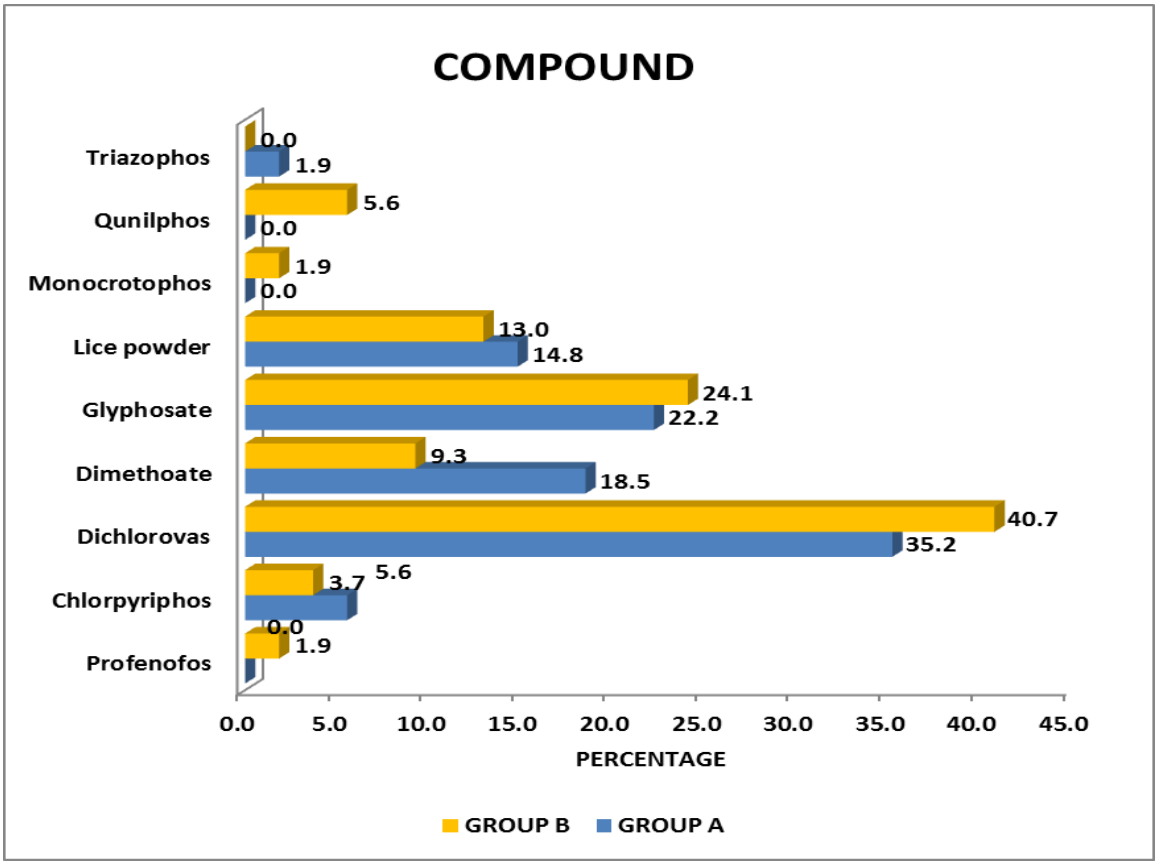
1. The aim of atropine therapy is to clear the chest and reach the end-points for all five parameters.
2. There is no need to aim for a heart rate of 120–140 beats/min. This suggests atropine toxicity rather than simple reversal of cholinergic poisoning. Such high heart rates will cause particularly severe complications in older patients with pre-existing cardiac disease – myocardial infarctions may result. However, tachycardias are also caused by hypoxia, agitation, alcohol withdrawal, pneumonia, hypovolaemia, and fast oxime administration. Tachycardias are not a contraindication for atropine if other features suggest under-atropinisation.
3. Aspiration will commonly result in focal crepitations. Attempt to distinguish such crepitations from the more general crepitations of bronchorrhoea.
4. Splashes of organophosphorus into the eye will produce intense miosis that may not respond to atropine therapy. However, symmetrical is likely to be due to systemic effects of the ingested pesticide.

OBSERVATIONS AND RESULTS

Features on enrollment there were 54 patients enrolled in group A and 54 in Group B. Demographic history and pre-enrollment management for both Groups are shown in Table 1. There were no significant differences between the two groups. There was a wide range of times to hospitalization because some patients were treated initially in a peripheral Health Care centers and/or district hospital or any private hospital and then referred to Shri. B.M.Patil medical college and hospital, while others were admitted directly to this hospital. Of the 108 study subjects who took a known substance, Dichorovas 38%,malathion 23.1% , Dimethoate 16.7,monocrotophos 13.9%,chlorpyriphos .6%,profenofos 0.9%,/ qunilphos 0.9%,Triazophos 0.9%,glyphosate 0.9%taken. Table of clinical feature shows the clinical features of OPC poisoning present on enrollment. There were no differences between the two Groups.

FIGURE 1: DISTRIBUTION OF OP COMPOUND





Type of compound poisoned: In present study we observed that Dichlorovas, Lice powder are more consumed distribution is given in table percentage shown in above table …

Features on Enrollment

A total of 54 patients admitted to hospital during the study period out of which 36 subjects with moderate to severe organophosphorus poisoning were enrolled in the study according to the inclusion criterion. 15 patients with organophosphorus poisoning had only mild poisoning and hence were not taken for analysis 3 patients were not enrolled due to discharge against medical advice

In study group B totally 54 subjects of which 32 patients analysed for moderate severe poisoning.22 patients are mild poisoned not taken for allocation.

AGE: The average age of the patients was 29.2 group A years and 29.7 in group B with 85% of our patients being less than 40 years old

TABLE 1: DISTRIBUTION OF AGE BETWEEN STUDY GROUPS

AGE (Yrs)	GROUP A		GROUP B		p value
	N	%	N	%	
<20	10	18.5	8	14.8	0.763
20-29	24	44.4	27	50.0	
30-39	11	20.4	13	24.1	
40-49	5	9.3	2	3.7	
≥50	4	7.4	4	7.4	
Total	54	100.0	54	100.0	

FIGURE 2 : DISTRIBUTION OF AGE BETWEEN STUDY GROUPS

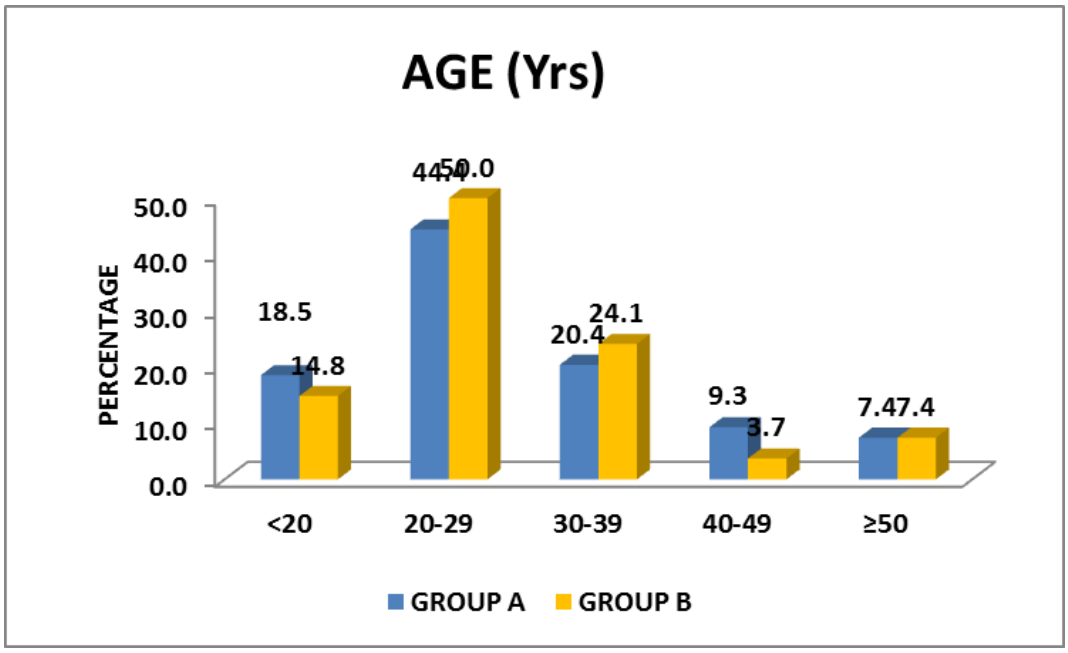
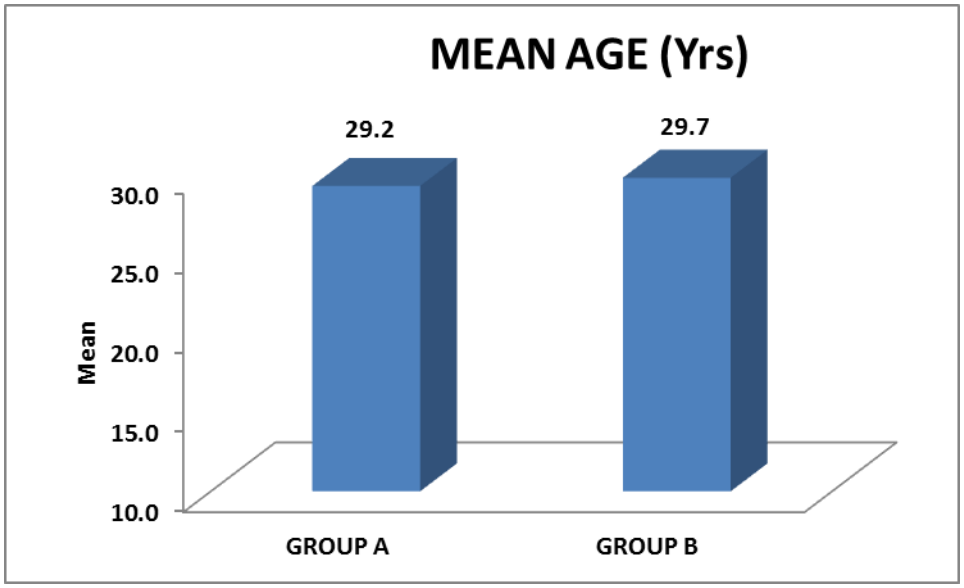


TABLE 2: MEAN AGE BETWEEN STUDY GROUPS

PARAMETERS	GROUP A		GROUP B		p value
	Mean	SD	Mean	SD	
Age	29.2	10.5	29.7	12.3	0.102

FIGURE 3 : MEAN AGE BETWEEN STUDY GROUPS

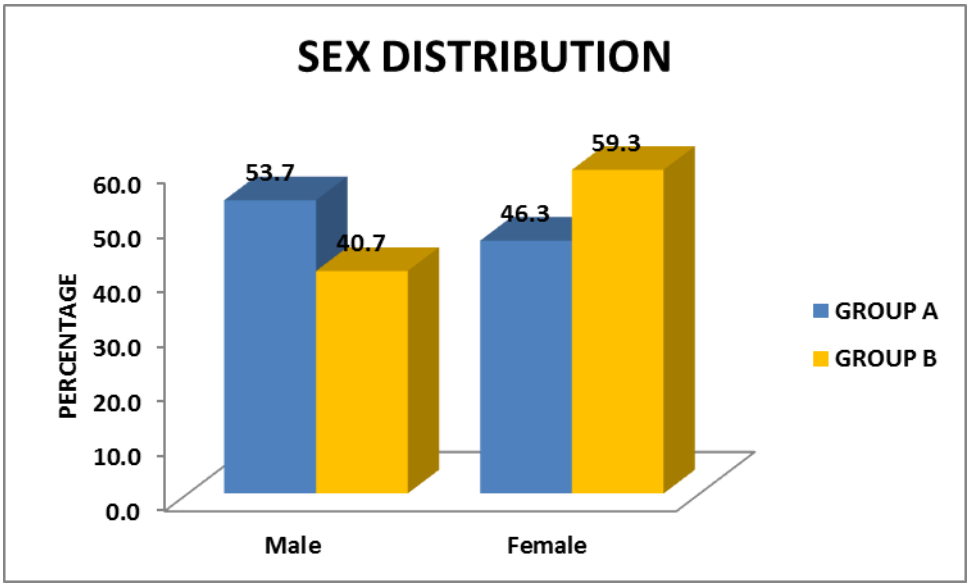


SEX DISTRIBUTION: Figure shows reference of sex distribution. There were were 29 (53.7)males and 25(46.3) females in group A and 22(40.&) males and 32(59.3) female in B group .

TABLE 3 : DISTRIBUTION OF SEX BETWEEN STUDY GROUPS

SEX	GROUP A		GROUP B		p value
	N	%	N	%	
Male	29	53.7	22	40.7	0.177
Female	25	46.3	32	59.3	
Total	54	100.0	54	100.0	

FIGURE 4 : DISTRIBUTION OF SEX BETWEEN STUDY GROUPS



Occupation: There are agriculture related activities were more seen in both group included in the study 31.5% and 27.8% are farmers and 24.1 and 40.7%. whereas students are 25.9% and 16.7% as distribution in A and B groups respectively as major one others are less as shown in fig.

TABLE 4 : DISTRIBUTION OF OCCUPATION BETWEEN STUDY GROUPS

OCCUPATION	GROUP A		GROUP B		p value
	N	%	N	%	
Bussiness	4	7.4	3	5.6	0.439
Farmer	17	31.5	15	27.8	
Housewife	13	24.1	22	40.7	
Service	3	5.6	1	1.9	
Student	14	25.9	9	16.7	
Unemployed	3	5.6	4	7.4	
Total	54	100.0	54	100.0	

FIGURE 5 : DISTRIBUTION OF OCCUPATION BETWEEN STUDY GROUPS

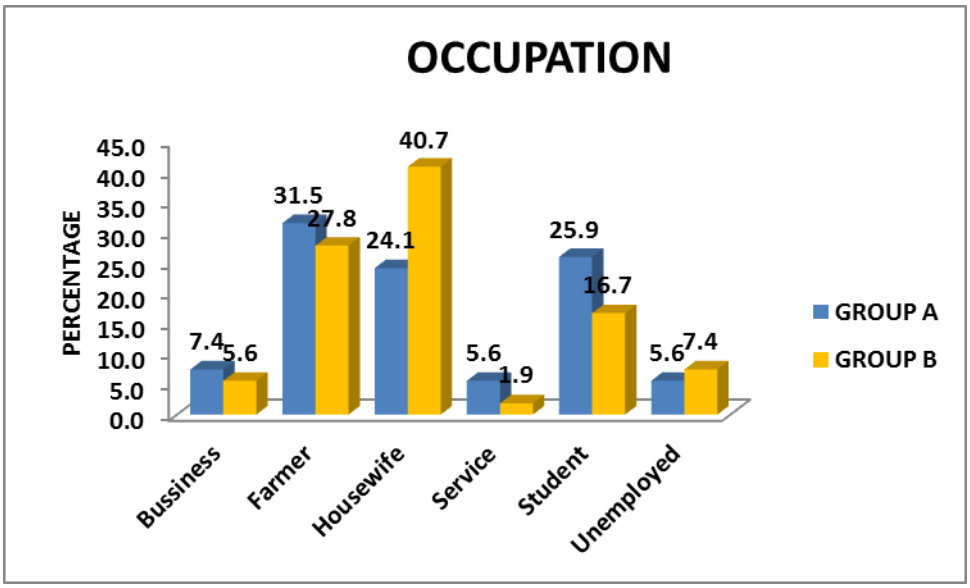
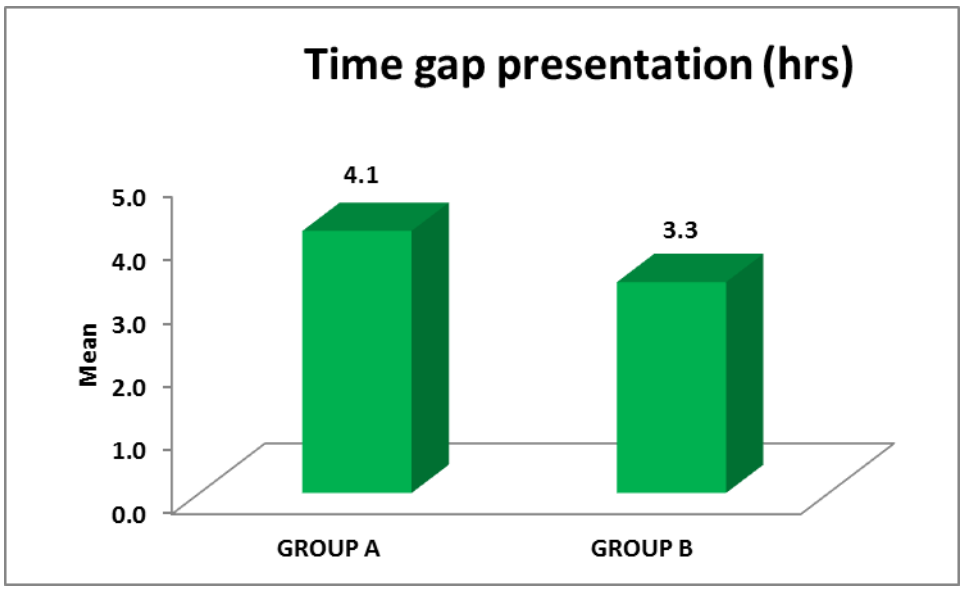


TABLE 5 : TIME GAP PRESENTATION (Time interval from consumption of organophosphorus compound to arrival at hospital)

We observed that most of the patients arrived between 3 to 6 hours of consumption, only few patients arrived after 6 hour

PARAMETERS	GROUP A		GROUP B		p value
	Mean	SD	Mean	SD	
Time gap presentation (hrs)	4.1	2.8	3.3	1.6	0.928

FIGURE 6 : MEAN TIME GAP PRESENTATION BETWEEN STUDY GROUPS

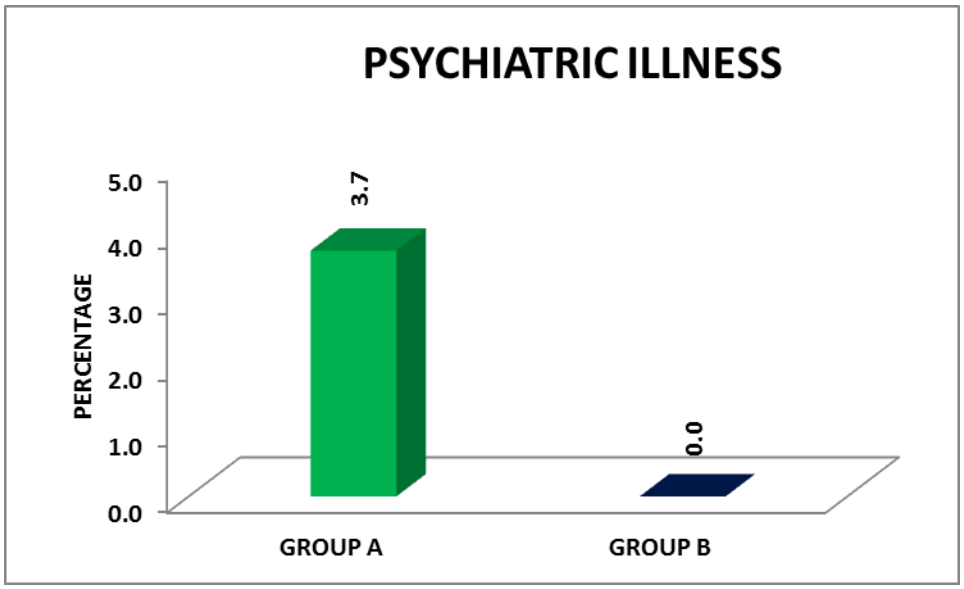


PSYCHIATRIC EVALUATION: In only In group A 10 (27.8%) patients evaluation by a psychiatrist revealed any factors for which any specific treatment was needed , the rest of the suicide attempts were either accidental or due to an impulsive act secondary to an acute stress situation

TABLE 6 : DISTRIBUTION OF PSYCHIATRIC ILLNESS BETWEEN STUDY GROUPS

	GROUP A		GROUP B		p value
	N	%	N	%	
PSYCHIATRIC ILLNESS	2	10	0	0.0	<0.001*

FIGURE 7 : DISTRIBUTION OF PSYCHIATRIC ILLNESS BETWEEN STUDY GROUPS



In only 10 (27.8%) patients evaluation by a psychiatrist revealed any Factors for which any specific treatment was needed, the rest of the suicide Attempts were either accidental or due to an impulsive act secondary to an acute stress situation (Figure).

TABLE 7 : DISTRIBUTION OF ALCOHOL BETWEEN STUDY GROUPS

ALCOHOL	**GROUP A**		**GROUP B**		**p value**
	N	**%**	**N**	**%**	
NO	42	77.8	44	81.5	0.633
YES	12	22.2	10	18.5	
Total	54	100.0	54	100.0	

CLINICAL FEATURES: The clinical features at admission are given in table.

TABLE 8 : DISTRIBUTION OF CLINICAL FEATURES BETWEEN STUDY GROUPS ON ADMISSION

CLINICAL FEATURES	GROUP A		GROUP B		p value
	N	%	N	%	
GIT (nausea, vomiting, diarrhoea, abdominal cramps, etc.)	49	90.7	51	94.4	0.462
RESPIRATORY (wheeze, crepitations)	1	1.9	1	1.9	-
CNS (anxiety, tension, headache, Drowsy or altered sensorium, convulsion, coma, irregular breathing)	9	16.7	14	25.9	0.24
Eyes (miosis, blurred vision)	40	74.1	47	87.0	0.089
Micturation	1	1.9	3	5.6	0.308

FIGURE 8 : DISTRIBUTION OF CLINICAL FEATURES BETWEEN STUDY GROUPS

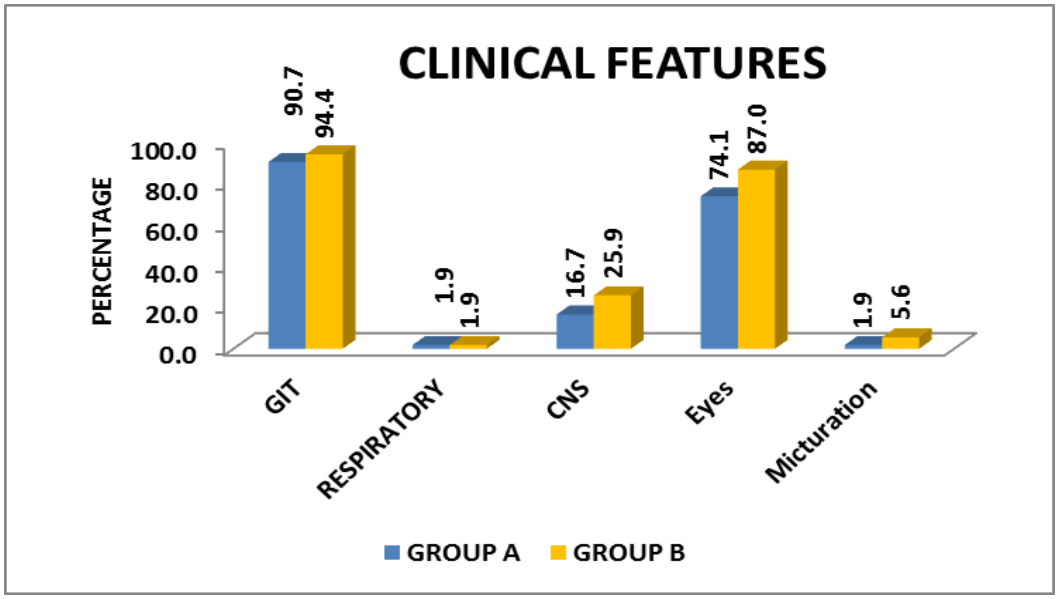
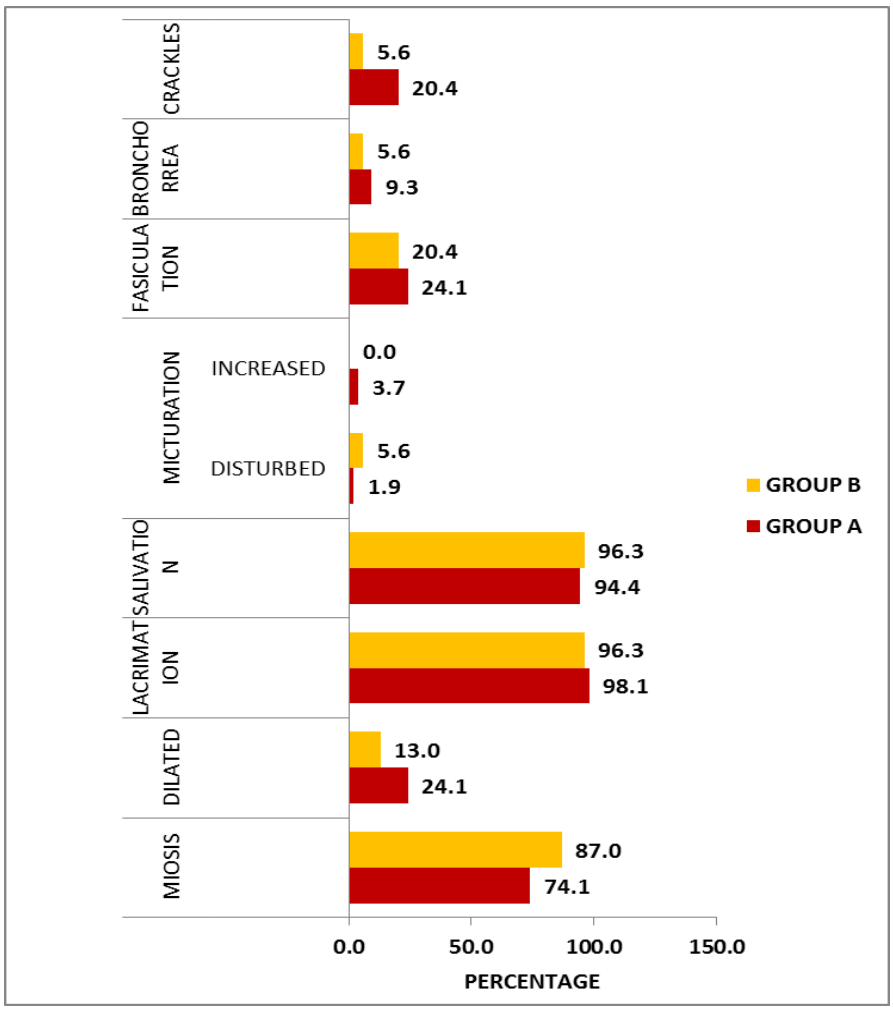


TABLE 9 : DISTRIBUTION OF PARAMETERS BETWEEN STUDY GROUPS

PUPILS		GROUP A		GROUP B		p value
		N	%	N	%	
MIOSIS		40	74.1	47	87.0	0.089
DILATED		13	24.1	7	13.0	0.137
LACRIMATION		53	98.1	52	96.3	0.558
SALIVATION		51	94.4	52	96.3	0.647
MICTURATION	DISTURBED	1	1.9	3	5.6	0.308
	INCREASED	2	3.7	0	0.0	0.223
FASICULATION		13	24.1	11	20.4	0.643
BRONCHORREA		5	9.3	3	5.6	0.348
CRACKLES		11	20.4	3	5.6	0.022*

FIGURE 9 : DISTRIBUTION OF PARAMETERS BETWEEN STUDY GROUPS

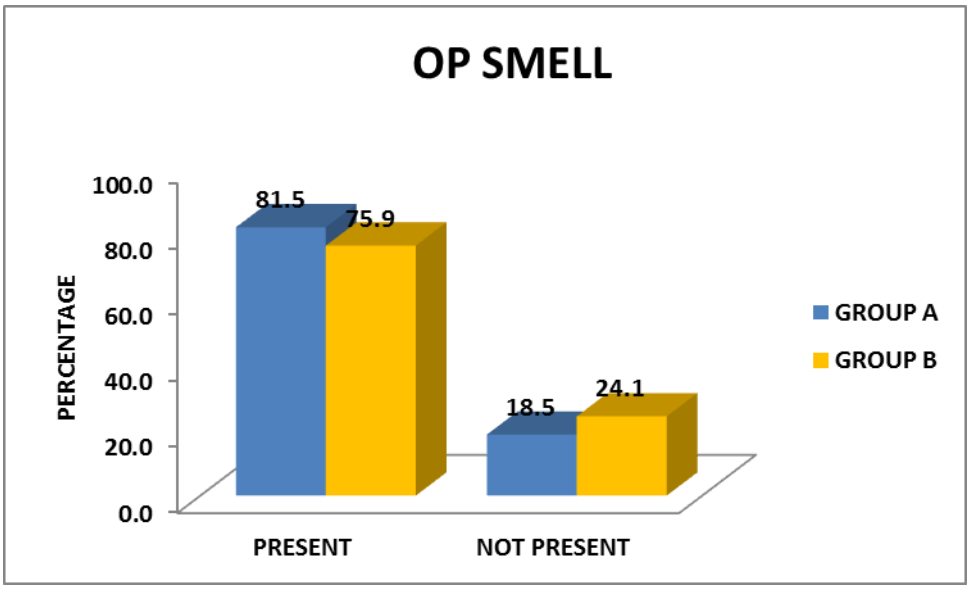


OP smell: All patients admitted with history of ingestion of OP compound got its smell almost more than 80% patients presents with OP smell

TABLE 10 : DISTRIBUTION OF OP SMELL BETWEEN STUDY GROUPS

OP SMELL	GROUP A		GROUP B		p value
	N	%	N	%	
PRESENT	44	81.5	41	75.9	0.481
NOT PRESENT	10	18.5	13	24.1	
Total	54	100.0	54	100.0	

FIGURE 10 : DISTRIBUTION OF OP SMELL BETWEEN STUDY GROUPS



SERUM CHOLINESTERASE: out of 36 patients in group A and 32 patients in group B

In our study of group A estimation of serum cholinesterase on Day 1 revealed 12 patients (33.3%) had ≤ 2000 Units/L, 2 patients (5.6%) in the range of 2000-4000 , 18 patients (50%) in the range of 4000 – 5000 and only 4 patients (11.1%) had in the range of >8000 u/l. Similarly estimation of serum cholinesterase on 5th day and at discharge is shown in the above table.

In group B 11(34.4) less than 2000 u/l,1(3.1%)2000-4000 u/l,17(53.1%) had between 4000-8000 u/l,3(9.4%) had more than 8000u/l.

TABLE 11 : DISTRIBUTION OF SERUM CHOLINESTERASE DAY1 BETWEEN STUDY GROUPS

SR CHOLENESTERASE DAY1 in U/L	GROUP A		GROUP B		p value
	N	%	N	%	
<2000	12	33.3	11	34.4	0.957
2000-4000	2	5.6	1	3.1	
4000-8000	18	50.0	17	53.1	
>8000	4	11.1	3	9.4	
Total	36	100.0	32	100.0	

FIGURE NO 11: DISTRIBUTION OF SERUM CHOLINESTERASE DAY1 BETWEEN STUDY GROUPS.

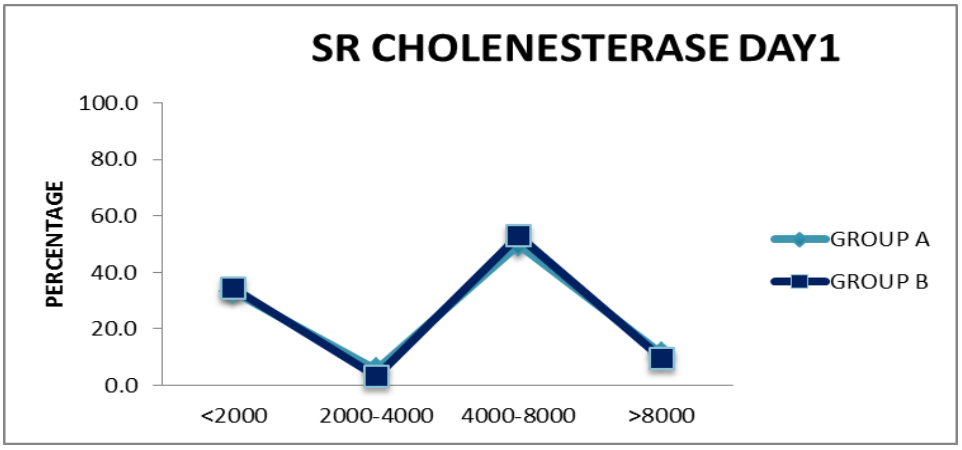


TABLE 12 : Serial estimation of serum cholinesterase during hospital stay varies with

group wise From day 2 to day 5 ,all estimations not available due to hospital stay duration or rejected cases.

SR CHOLENESTERASE (MEAN±SD)In U/L	DAY1	DAY2	DAY3-5
GROUP A	3864.9±562.1	1934.0±1062.2	3042.9±1082.4
GROUP B	2536.0±742.1	2164.1±992.3	1888.6±1061.5

FIGURE 12 : SERIAL ESTIMATION OF SERUM CHOLINESTERASE

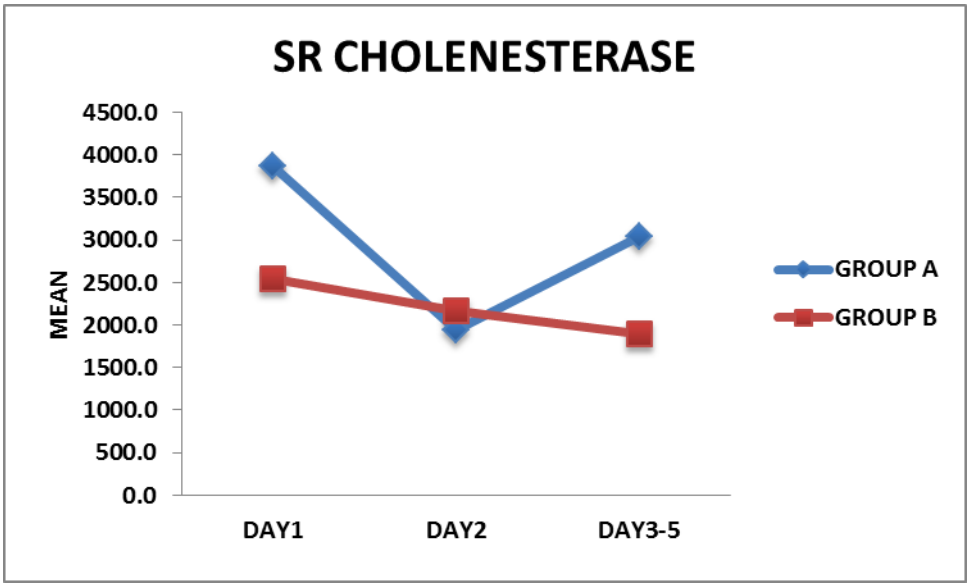


TABLE 13 : DISTRIBUTION OF OP DETECTED BETWEEN STUDY GROUPS

OP DETECTED	GROUP A		GROUP B		p value
	N	%	N	%	
DETECTED	52	96.3	51	94.4	0.647
NOT DETECTED	2	3.7	3	5.6	
Total	54	100.0	54	100.0	

FIGURE 13 : DISTRIBUTION OF OP DETECTED BETWEEN STUDY GROUPS

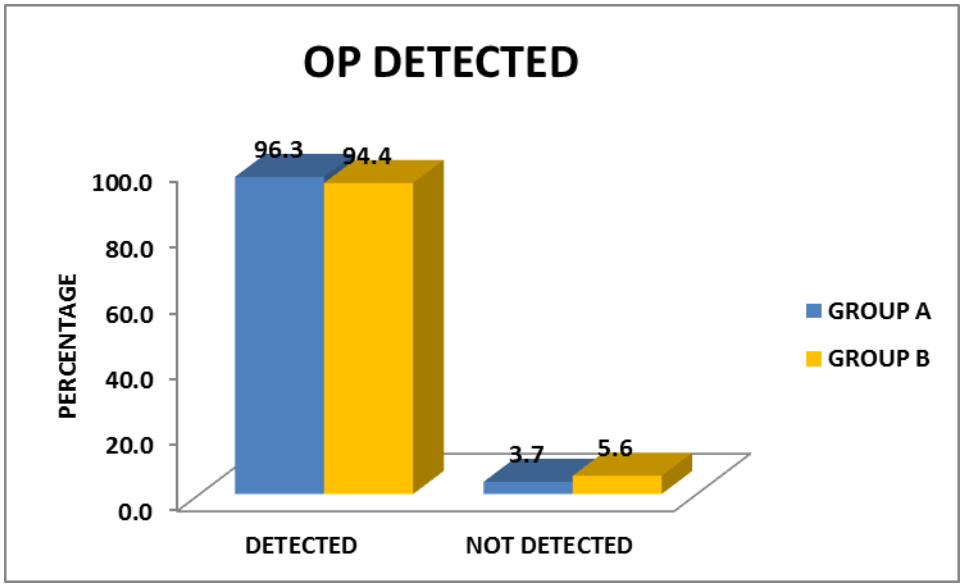


TABLE 14 : ECG : **DISTRIBUTION OF ECG BETWEEN STUDY GROUPS ON ADMISSION**

ECG	GROUP A		GROUP B		p value
	N	%	N	%	
BRADYCARDIA	20	13.0	30	37.0	0.001*
NORMAL	10	42.6	18	29.6	
TACHYCARDIA	24	44.4	6	33.3	
Total	54	100.0	54	100.0	

Note: * significant at 5% level of significance ($p<0.05$)

INTUBATION AT ADMISSION: Most of subjects who had on ventilator had intubated on admission or within six hours of admission, there are 10 patients in group A and 8 patients in group of analyzed patients. **:** On admission 13.9% in A group and 6.3 % in group were received endotracheal intubation and ventilation

TABLE 15 : DISTRIBUTION OF INTUBATION AT ADMISSION BETWEEN STUDY GROUPS

INTUBATION AT ADMISSION	GROUP A		GROUP B		p value
	N	%	N	%	
NO	31	86.1	30	93.8	0.301
YES	5	13.9	2	6.3	
Total	36	100.0	32	100.0	

PRE HOSPITALISATION TREATMENT

In group A 37% and in B 29.6 patients presented to the hospital after gastric lavage at a local hospital. About 36% in A and of patients had received atropine in some form before admission to the hospital. Only 1(4%) patient had received treatment with pralidoxime before admission and 1 patient was intubated at a local hospital and referred here for further treatment.

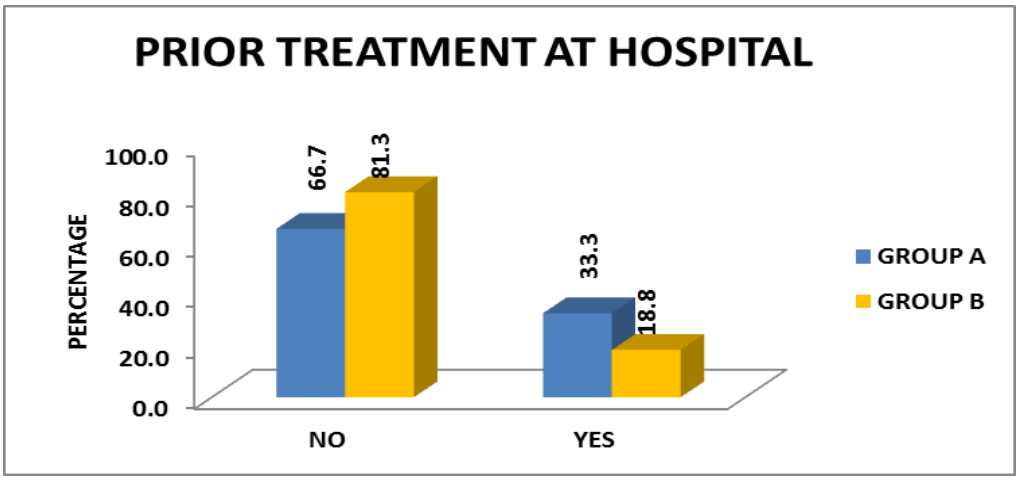
TABLE 16 : DISTRIBUTION OF PRIOR GASTRIC LAVAGE BETWEEN STUDY GROUPS

PRIOR GASTRIC LAVAGE	GROUP A		GROUP B		p value
	N	%	N	%	
NO	34	63.0	38	70.4	0.414
YES	20	37.0	16	29.6	
Total	54	100.0	54	100.0	

TABLE 17 : DISTRIBUTION OF PRIOR TREATMENT ATROPINE AT HOSPITAL BETWEEN STUDY GROUPS

PRIOR TREATMENT AT HOSPITAL	GROUP A		GROUP B		p value
	N	%	N	%	
NO	24	66.7	26	81.3	0.174
YES	12	33.3	6	18.8	
Total	36	100.0	32	100.0	

FIGURE 14 : DISTRIBUTION OF PRIOR TREATMENT ATROPINE AT HOSPITAL BETWEEN STUDY GROUPS



ATROPINE DOSING

INITIAL ATROPINISATION

In group **A**, the mean atropine required for initial atropinisation of a patients was 19.9 mg (range 0-52mg, SD 9.12).The mean time requires for atropinization was 5.8h i.e. 348 minutes. The bar diagram below shows the required doses for initial atropinisation in all 36 patients enrolled into the study. Minimum time taken for atropinization was 2hrs and maximum 18hrs from 7.2mg to 43.2mg .Total mean atropine given initial 24hrs was 52.1 in 24hrs.

TABLE 18 : ATROPINE DOSE BETWEEN STUDY GROUPS AMONG 36 SUBJECTS IN GROUP A AND 32 SUBJECT IN GROUP B ANALYSED FOR MODERATE TO SEVERE POISONING

ATROPINE DOSE	GROUP A		GROUP B		p value
	Mean	SD	Mean	SD	
0 -2 hrs	6.2	2.7	18.2	8.8	<0.001*
3 -4 hrs	6.0	3.5	3.6	2.8	0.003*
5 -6hrs	5.9	3.2	5.2	3.1	0.343
7 -8 hrs	6.0	3.7	6.5	3.2	0.552
9 - 24hrs	29.4	17.4	33.8	11.6	0.227
DAY1 total	52.1	22.7	67.3	18.1	0.003*
DAY 2	23.6	18.5	31.5	11.8	0.047*
DAY 3	16.9	14.5	23.1	13.6	0.091
DAY 4	12.7	14.1	19.3	14.8	0.101
DAY 5	10.8	17.2	17.7	11.9	0.209
DAY 6	11.5	13.2	19.3	9.6	0.175
DAY 7	8.7	13.5	13.3	10.3	0.484
DAY8	14.7	19.9	25.8	4.2	0.522

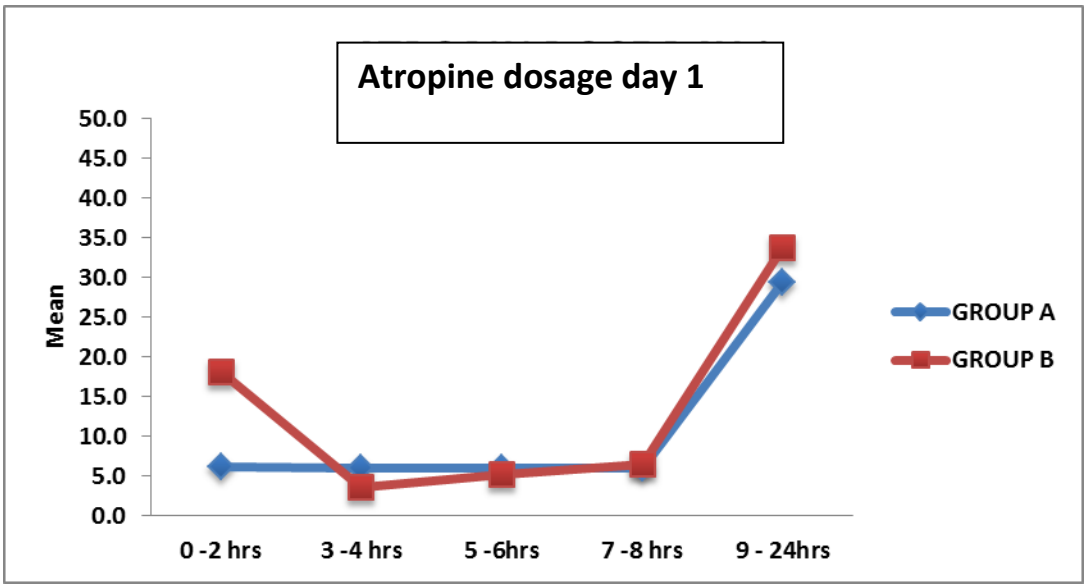
Note: * significant at 5% level of significance ($p<0.05$)

In group **B,** the mean atropine required for initial atropinisation of a patient was 18.2 mg (range 9-30mg, 9.12).The mean time requires for atropinization was 28.1minutes i.e. less than one hour . Mean atropine in day 1(24hrs) was 67.3mg. The bar diagram below shows the required doses for initial atropinisation in all 32 patients enrolled into the study

The mean total dose requirement of atropine for treatment on day 1 was 67.3 mgs. Figure shows the mean hourly requirement of atropine during the

first 12 hours of treatment in the hospital

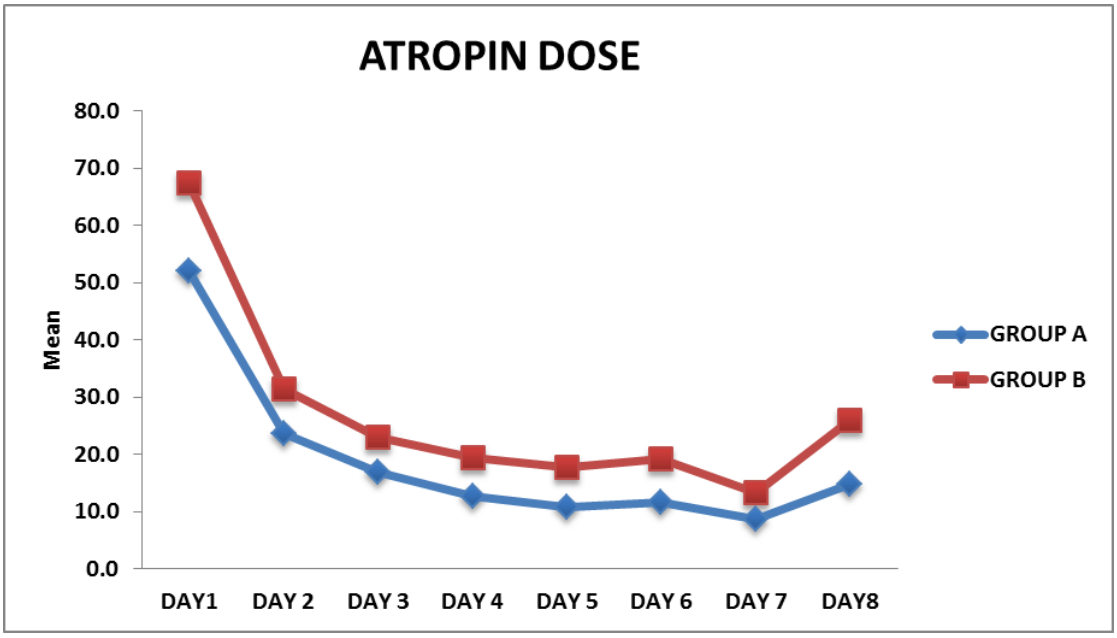
FIGURE 15 : ATROPINE DOSE BETWEEN STUDY GROUPS DAY 1



When we compare atropine dosing and dosage of atropinization, time taken for atropinization was more than 5 hours and mean atropinized dose was 19.9mg in group A and in group B 18.2mg in just 30minutes .In total 24hours mean atropine was 52.2mg in A and 67.3mg in group B respectively on day1. Later bolus doses and infusion after patients had reassessed for reatropinization were not taken for consideration initially.

Time to atropinization and total dose of atropine given to each group for recovered cases only 36 and 32 subjects in A and B groups respectively. Patients who died or self-discharged before completing treatment are not shown as they did not receive a full course of atropine

FIGURE 16 : ATROPINE DOSE BETWEEN STUDY GROUPS DAY 1 TO DAY 8.



	Number	Mean	p value
Time to atropinization (minutes)			
Group A	32	348	<0.001*
Group B	30	28.1	
Amount of atropine (mg)			
Group A	32	106	0.031*
Group B	30	141	

Note: * significant at 5% level of significance ($p<0.05$)

ATROPINE TOXICITY:

In Group A 9 patients had toxicity out of 36 and 5 patients in group B out of 32 The low incidence of atropine toxicity is due to usage of low doses infusion in B than A group and also prompt reduction in the dose required if needed due to regular monitoring.

ANTIBIOTIC USAGE:

The prevalence of use of antibiotics in the management of patients was 88.9% (n=68) and an infection most commonly respiratory or urinary was documented in 86% (n=66) of patients. The antibiotic use increased with increase in the hospital stay. Fever, leucocytosis is a common feature which occurs early in OP poisoning and does not necessarily mean an infection.

INTERMEDIATE SYNDROME

AMONG 36 SUBJECTS IN GROUP A AND 32 SUBJECT IN GROUP B ANALYSED FOR MODERATE TO SEVERE POISONING

Intermediate syndrome developed in 25% In group A of patients and 12.5% in group B. The mean onset time for the development of intermediate syndrome was 4.1 days (range 1-7 days, SD 2.02)

TABLE 19: DISTRIBUTION OF INTERMEDIATE SYNDROME BETWEEN STUDY GROUPS AMONG 36 SUBJECTS IN GROUP A AND 32 SUBJECT IN GROUP B ANALYSED FOR MODERATE TO SEVERE POISONING

INTERMEDIATE SYNDROME	GROUP A		GROUP B		p value
	N	%	N	%	
	7	25	4	12.5	0.048*

Note: * significant at 5% level of significance (p<0.05)

FIGURE 17 : DISTRIBUTION OF INTERMEDIATE SYNDROME BETWEEN STUDY GROUPS

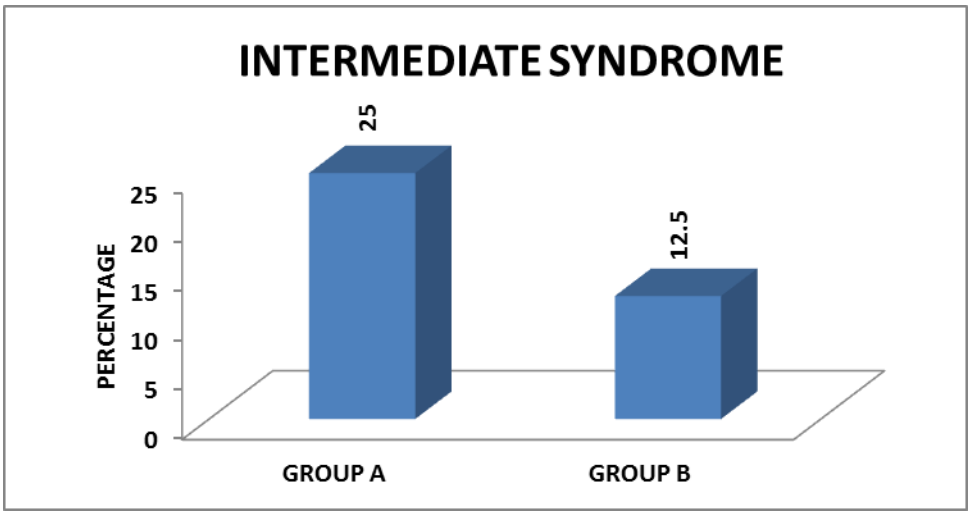
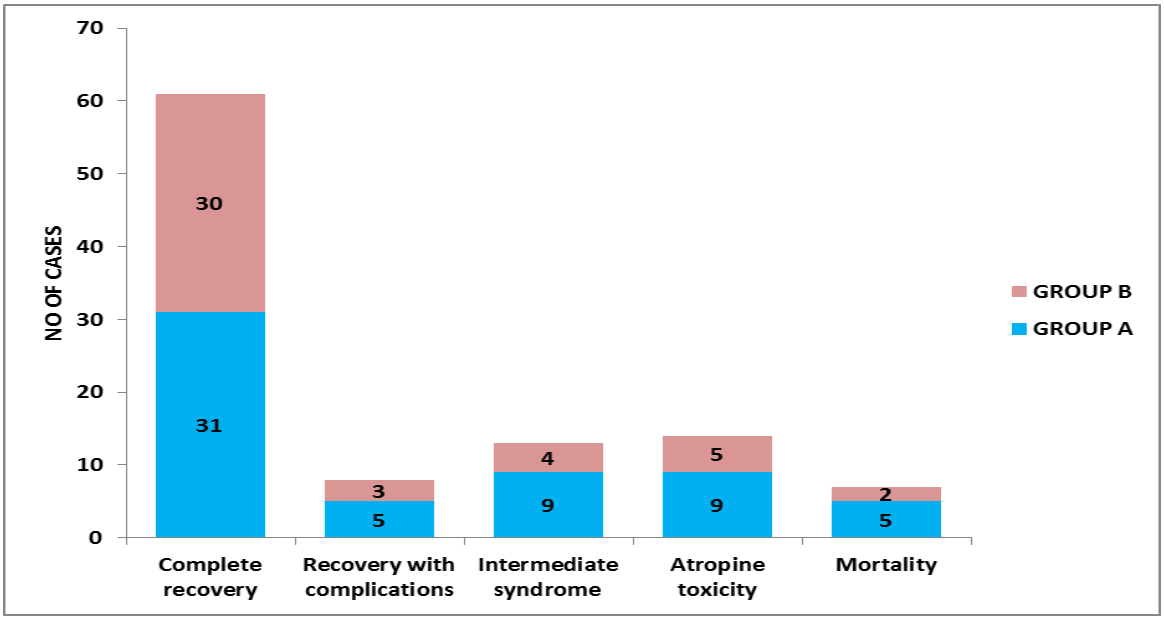


TABLE 20 : OUTCOMES:

Outcome	Group A		Group B		Total	P value
	N	%	N	%	N	
Complete recovery	31	50.8	30	49.2	61	0.301
Recovery with complications	5	62.5	3	37.5	8	0.564
Intermediate syndrome	7	69.2	4	30.8	13	0.048*
Atropine toxicity	9	64.3	5	35.7	14	0.027*
Mortality	4	71.4	2	28.6	7	0.481

FIGURE 18 : TABLE OF OUTCOME



Ventilation (Respiratory support)

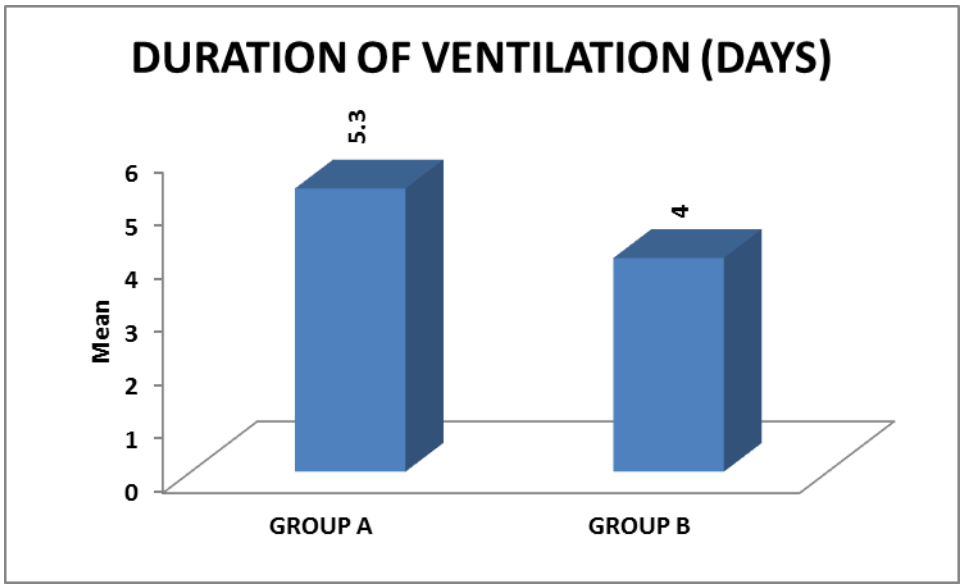
In group A total of 10 (27%) patients needed mechanical ventilation during the treatment. The mean duration of ventilation was 5.34 days (range 1-19 days, SD3.4.Tracheostomy done in one patient 19 days on ventilation .

In group B total of 8(25%)patients needed mechanical ventilation during the treatment. The mean duration of ventilation was 4 days (range 1-19 days, SD1.8.

TABLE 21 : DURATION OF VENTILATION (DAYS)

DURATION OF VENTILATION (DAYS)	GROUP A		GROUP B		p value
	MEAN	SD	MEAN	SD	
	5.3	3.4	4.0	1.8	0.515

FIGURE 19 : DURATION OF VENTILATION



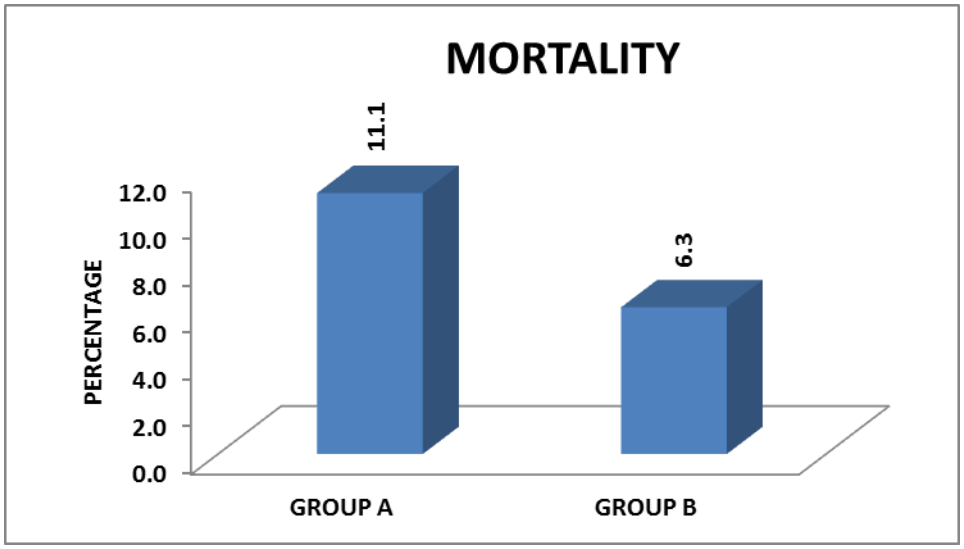
MORTALITY

In group A total of 36 (88.9%) of patients survived and were discharged in a normal state. The all-cause mortality related to poisoning was 4(11.1%) in group A and in group B 30(93.8) recovered and discharged .Total mortality in group B was 2(6.3%)

TABLE 22 : Table of mortality comparison between two groups

OUTCOME	GROUP A		GROUP B		p value
	N	%	N	%	
ALIVE/RECOVERED	32	88.9	30	93.8	0.481
MORTALITY	4	11.1	2	6.3	
Total	36	100.0	32	100.0	

FIGURE 20 :MORTALITY



COMPLICATIONS:

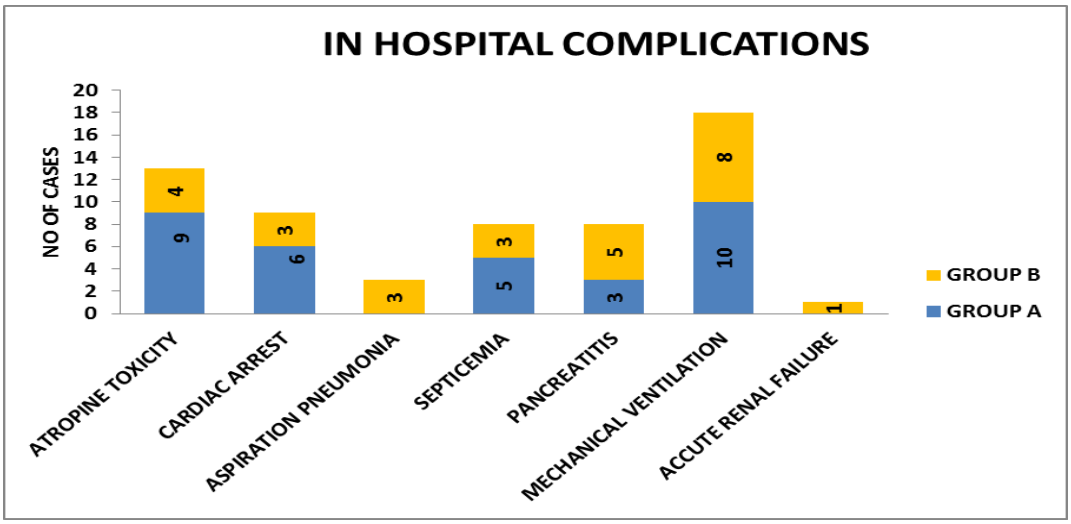
The following complications were observed during the treatment in the hospital .During the treatment period after initial atropinisation and stabilization, when the atropine infusion was given as per the fixed protocol and patient's heart rate, blood pressure, wheeze, secretions were closely monitored.

Table 23 : Distribution Of In Hospital Complications between Study Groupsamong 36 Subjects In Group A And32 Subject In Group B Analyzed For Moderate To Severe Poisoning.

COMPLICATIONS	GROUP A		GROUP B		p value
	N	%	N	%	
ATROPINE TOXICITY	9	25.0	4	12.5	0.027*
CARDIAC ARREST	6	16.7	3	9.4	0.043*
ASPIRATION PNEUMONIA	0	0.0	3	9.4	<0.001*
SEPTICEMIA	5	13.9	3	9.4	0.657
PANCREATITIS	3	8.3	5	15.6	0.657
MECHANICAL VENTILATION	10	27.8	8	25.0	0.796
ACCUTE RENAL FAILURE	0	0.0	1	3.1	<0.001*

Note: * significant at 5% level of significance ($p<0.05$)

FIGURE 21: DISTRIBUTION OF COMPLICATIONS BETWEEN STUDY GROUPS



DISCUSSION

Atropine is the only universally accepted specific treatment in the management of anticholinesterase poisoning. Despite this universal acceptance, there areno data to guide administration with a consequent wide variation in recommendations for dosing [18] .

Published literature is very sparse in the area of guidelines for adequate atropinisation in OP poisoning. Over several years the department of medicine at Shri B M Patil medical college hospital, Vijayapura as well SACTRC has found that keeping the heart rate at or around 100 beats per minute and ensuring absence of crackles on lung auscultation, are the two most important end points for adequate atropinisation. Pupillary size and assessment of central nervous stimulation have not been found useful (unpublished communication). Therefore heart rate, dry axillae, and crackles were used as the main indicators of adequate atropinisation, in this study.

In the study we found that rapid atropinization followed by atropine infusion greatly reduced mortality when compared to standard treatment with boluses of atropine 4(11.1% in group A) vs. 2 (6.3% in group B)

It required a shorter time to atropinization and there was a lower incidence of atropine toxicity, although the total dose of atropine administered was a little greater. There was also less IMS with this regimen and outcome as recovery is better.

1. When we compare group A,4 (11.1%) of patients died, compared to 2(6.3%) in group B. In other study done previously had same outcome where 45 cases organophosphorus poisoning study showed that continuous infusion significantly reduced mortality compared to intermittent boluses (23.5% to

8.8%; p<0.05) [39]

2. The similar study conducted on 131 samples shows that rapid incremental dose atropinization followed by atropine infusion reduces mortality and morbidity from OPC poisoning and shortens the length of hospital stay and recovery advantage over conventional incremental bolus doses alone(24.7% versus 8%, p<0.05)[40]
3. The study conducted on 56 patients shows frequency of atropine toxicity in the rapid incremental regimen followed by infusion (1.8%) was considerably lower than conventional regimen (48%) [41].
4. The overall mortality rate from OPC poisoning in this study was 8.7.%. The present study found that those with lower GCS on enrolment had much higher mortality as found previously [42].Intravenous high-dose atropine is an established lifesaving component of the initial management of OPC poisoning. However, there remains much discussion as to the optimal dosing regimen and numerous variations are in use around the world. In spite of an increasing frequency of suicidal attempts with OPCs, few studies have been undertaken to rationalize therapy for OPC poisoning and there remains great scope for reducing the high mortality through optimizing therapy.
5. The relatively few formal trials in OPC poisoning that have been done make it clear that early antagonism of OPC toxicity is associated with better outcome[39] .Full and early atropinization is ideal as delayed atropinization can result in avoidable death from central respiratory depression,bronchospasm, bronchorrhoea, severe bradycardia and/or hypotension [43].

In group A, one patients died before atropinization was achieved. It is likely that the accelerated regimen employed in arm B of this study was responsible for the difference in mortality and complications. Conventional bolus dose atropine treatment of OPC poisoning, the most frequently used regimen, was postulated and found in this study to be associated with delay in stabilization of poisoned patients, more atropine toxicity and ultimately more fatality

DURATION OF HOSPITAL STAY IN DAYS	**GROUP A**		**GROUP B**		**p value**
	N	**%**	**N**	**%**	
≤5	18	50.0	10	31.3	0.481
>5	18	50.0	22	68.8	
Total	36	100.0	32	100.0	

TIME TO DEATH IN HRS	**GROUP A**		**GROUP B**		**p value**
	N	**%**	**N**	**%**	
≤24	1	0.0	0	3.1	0.606
>24	3	5.6	2	9.4	
Total	36	100.0	32	100.0	

The time to atropinization of group A was much (6.5 times) longer than that of group B ($p<0.001$) although the average requirement of atropine was found to be a little greater in group B. With treatment B, the rapidly incremental dosing regimen, atropinization could be achieved within a very short time. Although there are few studies on the subject, there is some evidence that patients in the developing world often die soon after admission [44] The rapid and effective stabilization and treatment of pesticide poisoned patients on admission should reduce the number of early deaths,

improve the prognosis for surviving atients over the first few days and reduce the number and severity of long-term sequelae.

6. The study which compares two groups method ,the conventional bolus dose atropine treatment of OPC poisoning with incremental dose atropine treatment followed by atropine infusion. In clinical practice, dosage regimens are usually designed according to severity of poisoning and to the signs of atropinization[45]

   In the present study, persons with mild poisoning were not analyzed; otherwise, no discrimination was made with respect to severity, with all moderate–severe patients being included and strictly randomized. Due to there being no accepted grading scales for the severity of OPC poisoning, there was no attempt to formally grade severity of poisoning in enrolled patients.

   Enrolled patients age(50% in A and 44.4% in B) were less than 30 years of age and the two groups were broadly similar in terms of demographics, prehospital treatment received, time to reach hospital and presenting features. A majority (90%) of the subjects took substances which were known and most (91.7%) poisonings were self-inflicted. In India OPCs are the commonest chemical agents used for attempted suicide [1]

7. The time interval between exposure to OPC and onset of symptoms of poisoning varies with the route and degree of exposure[46]. Following massive ingestions, the symptoms appear within several minutes[44]With smaller amounts,in most instances, symptoms appear within 30 min of exposure and almost always in less than 12 h [46].In our series,the earliest presentation of an individual with features of toxicity was within 30 min of ingestion of OPC,

although 25% took more than 4 h to reach the hospital. Delay in discovery and transport to the hospital as well as differences in treatment-seeking behaviour can cause differences in presentation and increased mortality. Local effects on eyes and respiratory tract may appear within minutes. After ingestion of OPC, the initial symptoms may be gastrointestinal or may be referable to any of the other organs affected [46.] In the present series, pupillary and glandular manifestations were almost invariably present.

8. **IMS** occurs due to dysfunction of the post-synaptic neuromuscular junction[47] It is a neurological complication affecting many patients with OPC poisoning during conventional treatment or on recovery from acute cholinergic crisis.In the present study, IMS developed in 14 patients among whom the offending OPC agent was identified in eight of these four ingested malathion, two ingested dichlorvos and two ingested dimethoate. Regimen B was found to be associated with a lower risk of developing IMS ($p<0.05$). The pathogenesis of IMS is not understood but it is thought to be due to persistent inhibition of acetylcholinesterase. Theoretically, therefore, its incidence should be unaffected by atropine but may be reduced by effective oxime therapy, although evidence for this is lacking.

The difference in incidence of IMS between the two groups in the present study suggests that an alternative mechanism may be contributing. No clinical trial which was adequately powered to explore this has yet been undertaken. A difference in IMS between the two groups may alternatively reflect an imbalance between the two groups, although there were no other

indications of this from the analysis.

9. Ventilation support was needed for 18 patients out of 68. Of these 12, survived. In other studies, the survival rate in OPC poisoning requiring mechanical ventilation varied between 13% and 50% in a variety of settings[48-50] Ventilator support was required in significantly fewer patients treated with regimen B. Again, this was probably due to earlier reversal of cholinergic features by rapid atropinization. The cause of respiratory failure in OPC poisoning is multifactorial, including bronchorrhea, bronchospasm, weakness of respiratory muscles and centrally mediated respiratory depression.
10. To examine safety, the occurrence of atropine toxicity also compared between the treatment groups. Patients treated with conventional bolus dose were found to be more at risk of developing atropine toxicity (25% vs 12.5%, $p<0.05$). Previous studies have found similar rates with bolus regimens (26%)[51].This was probably due to repeated bolus dosing with atropine and also due to failure to reduce the dose of atropine quickly. A recent trial comparing ad hoc bolus doses with titrated doses found ad hoc dosing to result in more and higher doses of atropine, more atropine toxicity and longer hospital stays [52].In conventional bolus dosing regimens, after initial atropinization, it is not clear as to how frequently to follow up the patient and how quickly and how much to reduce the dose of atropine. The lower incidence of atropine toxicity in group B patients was probably partly due to the continuous infusion of atropine producing little fluctuation of blood levels.

CONCLUSION

The results of this study shows the following-

1. Use of atropine for organophosporus poisoning given by individualized incremental bolus doses followed by continuous infusion has several advantages over conventional incremental bolus doses alone.
2. Early atropinization reduces mortality and atropine toxicity which leads to better hospital outcome and recovery.
3. Accurate and frequent monitoring is require in conventional incremental bolus dosing regimens for atropinization and toxicity.
4. More studies are required to predict and study causes of wide variability in atropine requirements.

LIMITATIONS

1. The study is done on a small number of patients.
2. The study was not designed to compare outcomes such as mortality.
3. Many patients enrolled into the study were referred from a peripheral hospital and treatment received there might have had some influence on the requirement of atropine.
4. All patients did not receive the same standard of care as some of them were managed in the general wards due to non availability of beds in the ICU.
5. The study involved only the moderate to severe organophosphorus poisoned patients. Many of them requiring mechanical ventilation were also sedated, this may have had masked the mild features of atropine toxicity.

SUMMARY

In the study role of atropine in acute organophosphorus poisoning shows early atrpopinization by incremental bolus doses followed by continuous infusion had better outcomes in terms of recovery than that of conventional incremental bolus doses alone. Early stabilization will leads to less mortality. Conventional bolus dosing regimens require more frequent follow-up to monitor for atropinization and atropine toxicity and this is impractical in very busy resource-limited settings. Adoption of a regimen that results in rapid atropinization will likely save significant numbers of lives. Recommended regimens must be simple and easily used by training physicians. With incremental bolus doses followed by infusion ,where there is early atropinization and stabilization in a very short time. In this sustained blood levels of atropine with little chance of fluctuation and ultimately less toxicity and lower mortality.

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APPENDIX-I

Severity grading of organophosphorus poisoning by Namba et al [36]

Severity score	symptoms	Serum BuChE levels (%)
Mild	Dizziness, nausea, vomiting, diarrhea, abdominal pain, salivation and wheezing.	20 – 50 %
Moderate	All of the above and weakness, inability to walk, fasciculations, dysarthria, miosis.	10 – 20 %
Severe	All of the above and coma, flaccid paralysis, pulmonary edema, respiratory distress.	< 10 %

APPENDIX 2

Data abstraction form – Organophosphate poisoning study

PATIENT SERIAL NUMBER:

Name: Hospital Number:

Age: Sex: State:

Name of Pesticide: Type: Dimethyl / Diethyl / Unknown

Amount consumed(ml) : Source:

Date/Time of consumption:

Pre-hospital treatment:

Gastric lavage given: Yes / No Atropine: Yes / No

Oximes: Yes / No If yes dose: Time given:

Cardiac/Respiratory arrest: Yes / No Ventilation: Yes / No

At admission: (Circle features present at admission)

Bradycardia	Tachycardia	Arrhythmia
Hypotension	Hypertension	Ventricular Fib
Miosis	Mydriasis	Tachypnea
Paradoxical respiration	Respiratory arrest	Aspiration pneumonia
Seizures	Acute renal failure	Leukocytosis
Electrolyte abnormality	GCS	Pseudo chol

In hospital data (circle features observed)

Cardiac arrest	Arrhythmias	Hypotension
Ventricular fibrillation	Aspiration pneumonia	Nosocomial pneumonia
Nosocomial UTI	Septicaemia	Acute renal failure
Hepatic dysfunction	Pancreatitis	Atropine toxicity

Organisms identified & site:

Days	1	2	3	4	5	6	7
Atropine dose planned (mg)							
Additional dose							
Total dose							

Day1	0-2 hrs	3-4 hrs	5-6 hrs	7-8 hrs	9-24 hrs
Atropine dose planned (mg)					
Additional dose					
Total dose					

Intermediate syndrome: Yes / No If yes onset time:

Ventilation: Yes / No If yes duration (days):

Tracheostomy: Yes / No If yes day of tracheostomy (after poisoning):

Duration of ICU stay (days): Duration of hospital stay (days):

ICU outcome: Dead / Alive / Discharged at request/ PVS

Hospital outcome: Dead / Alive / Discharged at request / PVS

Total cost (Rs.)

ANNEXURES

ETHICAL COMMITTEE CERTIFICATE

B.L.D.E.UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR – 586103
INSTITUTIONAL ETHICAL COMMITTEE No/58/2015
20/11/15

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 17-11-2015 *at* 03 pm *scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.*

Title "comparative study of current dosing practice of atropine in organophosphorus poisoning at BLDE university Shri, B.M. Patil Medical college with protocol of administration practised at south asian clinical toxicology research collaboration"

Name of P.G. Student : Dr Supriya A Singh
Dept of Medicine

Name of Guide/Co-investigator : Dr. R.C. Bidri,
Professor

DR.TEJASWINI VALLABHA
CHAIRMAN

CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before E.C. for Scrutinization
1)Copy of Synopsis/Research Project
2)Copy of informed consent form.
3)Any other relevant documents.

CONSENT FORM

To voluntarily agree to take part in this study I must sign on the line below: If chose to take part in this study I may withdraw at any time I am not giving up any ofmy legal rights, by signing this form. My signature below indicates that I have read orhave read to me this entire consent form including the risks and benefits and had allquestions answered, I will be given a copy of this consent form.

Signature or thumb impression of the subject _____

Name _____ Date: _____

Signature or thumb impression of authorized representative _____

Name _____

Relation to the subject: Date: _____

Signature or thumb impression of the witness

Name_____ Date: _____

Signature of the investigator

Name _____ Date: _____

PROFORMA

Organophosphorus poisoning study data collection sheet

Case No/ Inpatient Number:

Name :

Age /Sex :

Address :

DOA : DOD:

Occupation :

Religion : Caste :

Socio-economic class :

History:

1. Informant Patient / Relative / Others

2. Type of OP compound consumed

3. History of consumption of Alcohol/ Kerosene/ Any other poison

4. Route of exposure

5. Quantity consumed

6. Time interval from ingestion to hospitalization(Time gap at presentation)

7. Intention Homicidal / Suicidal / Accidental

Complaints at presentation (Clinical features)	Yes	No
Vomiting		
Smell of Op		

Diarrhea		
Sweating		
Breathlessness		
Muscle twitching		
Convulsions		
Micturation		
Bronchorrhea		
Bradycardia		
Hypotension		
Consciousness		
Any other complaints		

Past history	Yes	No
Consumption of poison		
Psychiatric illness		
Ischemic heart disease		
Diabetes mellitus		
Hypertension		
Any other		

Family history

Personal history

Treatment history

On Examination :Smell /vitals/view

Smell

Vital signs

Temperature :

Pulse :

Respiratory rate :

Blood pressure

Pupil

1.General condition
2. Decubitus
3. Built
4. Nutrition
5. BMI
6. Waist circumference
7. Hip circumference
8. Waist:Hip ratio
9. Pulse /min
10. Peripheral pulses
11. B.P mmHg
12. Respiratory rate /min / SP O2
13. JVP
14. Pallor
15. Icterus
16. Cyanosis
17. Clubing
18. Edema
19. Lymph Nodes
20. Markers of op poisoning

Systemic examination

Respiratory system :

Cardiovascular system :

Per abdomen :

Central nervous system

Diagnosis

Investigations

Duration of stay in hospital

Requirement of mechanical ventilation **Yes/No**

If Yes, duration of mechanical ventilation

Outcome Survived / Expired

Treatment

Intubation at admission **Yes/No**

ATROPINE DOSE (mg)

Day 1		0-1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	12-24
Atropine dose infusion														
Additional Total														

	Day 1	Day 2	Day 3	Day 4
Infusion/Bolus				
Additional				
Total				

Atropine toxicity Y/N , HR- , Dose of atropine

Serum cholinesterase

Intermediate syndrome

Duration of ventilation

Time to regain consciousness

Complications

Antibiotics used

Infection - URTI / LRTI /others

Fever

Premorbid condition-

Outcome -- alive / dead.

COMMONLY AVAILABLE OP BRANDS


73
2015
sphenic acid
Rf = 0.8
ock
idea!
GLYPHOSATE 41% S.L.
Roundup
Herbicide
117
2014
282
2015
Rf=0.8
QL
JAI KISAN
CROP CARE
ZAILO
CYPERMETHRIN 5% +
CHLORPYRIPHOS 50% EC INSECTICIDE
जाइलो
CHLORPYRIPHOS,Rf
जेईलो
MARKETED BY
ZUARI AGRI SCIENCES LTD.
METHYL PARATHION
DHANUMAR
मिथाइल पैराथियान
धानुमार
R-0
Dhanuka Agritech Limited
AN ISO 9001:2000 COMPANY


Classic Super
(INSECTICIDE)
RF -0.8
CHEMINOVA
idea!
73
2015
Phosphenic acid
Rf = 0.8
i-Lock
GLYPHOSATE 41% S.L.
Roundup
Herbicide
117
2014
282
2015
Rf=0.8
QL
JAI KISAN
CROP CARE
ZAILO
CYPERMETHRIN 5% +
CHLORPYRIPHOS 50% EC INSECTICIDE
जाइलो
CHLORPYRIPHOS,R
जेईलो
MARKETED BY
ZUARI AGRI SCIENCES LTD.


NUVAN
MONOSAAN 36
INSECTICIDE
VARDAAN
CHLORPYRIPHOS – RF-08
Classic Supe
(INSECTICIDE)


TAFGOR
CHEMINOVA
ROGOR
GHATAK
INSECTICIDE
Insecticide
Crysta

GROUP A

S.No	Name of Patient	D0A	D0D	ATROPINE R	CHOLENESTRSE R	Compound	Time gap presentation	CLINICAL FEATURES	GENERAL PHYSICAL EXAM				PUPILS							
									pulse/mn	BP mmhg	RR/mn	TEMP IN F	Miosis	Dilated	LACRIMATION	SALIVATION	MICTURATIO	FASICULATIO	RONCHORRE	CRACKLES
1	Shrimanth Bhimaray Nidagundi	1/2/2016	1/8/2016	7.2mg	8120	Tataphen	2hrs27min	Irrelevant talking ,profuse sweating	120	120/80	16	98.6	YES	NO	YES	NO	NORMAL	ABSENT	ABSENT	ABSENT
2	Mahadev Basappa Nyangond	1/5/2016	1/11/2016	3.6mg	195	Dichlorovas	5hr40mins	vomiting	122	130/80	16	98.6	NO	YES	YES	YES	NORMAL	PRESENT	ABSENT	ABSENT
3	Sangeeta Kumar Madavi	1/9/2016	1/13/2016	69.6mg	4664	Dimethoate	6hrs20mins	vomiting,abdominal discomfort	82	104/70	14	98.6	NO	YES	YES	YES	NORMAL	PRESENT	ABSENT	ABSENT
4	Tippana B Pujari	1/20/2016	1/25/2016	14.4mg	3890	Dimethoate	4hrs	vomiting,abdominal discomfort	80	160/90	18	98.6	NO	YES	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
5	Nagappa Sharanappa Hugar	2/8/2016	2/13/2016	4.8mg	7298	Dichlorovas	3hrs50mins	vomiting	110	150/92	22	98.6	YES	NO	YES	YES	NORMAL	PRESENT	ABSENT	ABSENT
6	Mallamma Bhimanna Pujari	2/13/2016	2/18/2016	54mg	8699	Lice powder	5hrs	vomiting	80	110/70	14	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
7	Priya Raju Bandiwaddar	2/28/2016	3/6/2016	19.8mg	241	Monocrotophos	5hrs	vomiting	120	130/70	14	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	PRESENT
8	Sanganagouda Malakanagouda patil	3/11/2016	3/20/2016	13.2mg	563	Dichlorovas	11hrs	drowsy,under influence alcohol	130	160/80	20	97.6	YES	NO	YES	YES	NORMAL	ABSENT	PRESENT	PRESENT
9	Rukma Vittal Lokapur	3/13/2016	3/18/2016	4.8mg	4766	Lice powder	2hrs10min	vomiting,abdominal discomfort	112	110/80	16	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
10	Sahadev Pandu Pandre	3/30/2016	3/31/2016	34.8mg	404	Dimethoate	4hrs	vomiting,giddiness,altered sensorium	102	90/64	20	98.6	YES	NO	YES	YES	NORMAL	ABSENT	PRESENT	PRESENT
11	Sudharani Dondiba Bhosale	4/5/2016	4/7/2016	38.4mg	8248	Monocrotophos	4hrs	vomiting,abdominal discomfort	86	120/70	18	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
12	Vinod Shamray Bairshetty	4/17/2016	4/17/2016	3.6mg	8144	Dichlorovas	5hrs30min	vomiting,loss of consciousness,incontinence	170	80/50	30	94	NO	YES	YES	YES	INCREASED	ABSENT	PRESENT	PRESENT
13	Rajabai Ranjansab Wadaf	5/19/2016	5/23/2016	38.4mg	9144	Lice powder	5hrs30min	vomiting	102	136/80	18	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
14	Mudukappa Shamrao Hosamani	5/22/2016	5/31/2016	24mg	928	Dichlorovas	1hrs30min	vomiting	86	140/90	22	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
15	Dareppa Chandram Motagi	6/3/2016	6/7/2016	48mg	6879	Monocrotophos	1hrs40min	vomiting	96	130/80	18	98.6	NO	YES	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
16	Sunil Siddalingappa Jatti	8/1/2016	8/8/2016	60mg	726	Dichlorovas	1hrs5min	vomiting,abdominal pain	86	120/70	18	98.6	NO	NO	YES	YES	NORMAL	PRESENT	ABSENT	ABSENT
17	Siddu Gurappa Jangamashetti	8/11/2016	8/20/2016	48mg	935	Dichlorovas	2hrs20min	vomiting,abdominal discomfort	120	150/100	18	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
18	Somilibai Pandu Rathod	8/12/2016	8/13/2016	60mg	7901	Monocrotophos	4hrs	vomiting	102	160/90	20	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
19	Gururaj Pandappa Hadimani	8/21/2016	8/30/2016	52.8mg	873	Dichlorovas	2hrs	vomiting,loose stools	60	120/70	16	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
20	Anasuya Muttanagouda Patil	10/29/2016	10/30/2016	21.6mg	371	Dichlorovas	4hrs	breathlessness.urine incontinence vomiting	108	130/80	16	98.6	YES	NO	YES	YES	INCREASED	PRESENT	ABSENT	PRESENT
21	Guranna Amarappa Awaradi	1/22/2017	1/30/2017	73.2MG	105	Dimethoate	4hrs	Vomiting	120	126/70	20	98.6	NO	Yes	NO	YES	NORMAL	ABSENT	PRESENT	PRESENT
22	Ramesh Shivanand Kamgond	1/30/2017	2/6/2017	60MG	187	Dichlorovas	1h	vomitimg,abdominal discomfort	92	160/90	20	98.6	YES	NO	YES	YES	NORMAL	PRESENT	ABSENT	ABSENT
23	Anjana Praveen Pujari	2/1/2017	2/4/2017	18mg	4484	Lice powder	2 hrs30mins	vomiting	86	100/70	16	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
24	Suresh Shrimantappa Pattanashetti	3/29/2017	4/2/2017	26.4mg	7557	Dichlorovas	7hrs 30 min	drowsy	102	110/80	18	98.6	YES	NO	YES	YES	NORMAL	PRESENT	ABSENT	PRESENT
25	Anappa Beerappa Dalawai	4/22/2017	4/24/2017	7.2mg	7268	Dimethoate	2hrs	vomiting	104	130/80	16	98.6	NO	YES	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
26	Dundappa Siddappa Talewad	5/2/2017	5/5/2017	8.4mg	7783	Dimethoate	3hrs 15min	vomiting	90	124/80	24	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
27	Annapurna Ashok Dashavanth	5/9/2017	5/12/2017	10.8mg	3972	Monocrotophos	5 hrs	giddiness,vomiting	118	140/90	16	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
28	Malashree Basavaraj Harijan	5/9/2017	5/12/2017	36mg	6523	Dichlorovas	4 hrs	vomiting	120	110/70	16	98.6	NO	YES	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
29	Iranagoud Sharanagoud Borawat	5/11/2017	5/14/2017	24mg	4458	Dichlorovas	1 hrs 40min	vomiting,drowsy	90	130/80	16	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
30	Surekha Mudakappa Chandokoli	5/17/2017	5/20/2017	7.2mg	4635	Dimethoate	3 hrs 30min	vomiting	112	120/80	18	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
31	Aravind Gundanna Dodamani	5/18/2017	5/21/2017	14.4mg	115	Dimethoate	4 hrs 15min	unconscious,convulsions,frothingfrom mouth	146	150/110	40	98.6	YES	NO	YES	YES	NORMAL	PRESENT	PRESENT	PRESENT
32	Bhimray Ningappa Biradar	5/19/2017	6/6/2017	27.6mg	224	Monocrotophos	6 hrs	vomiting,under influence of alcohol	98	100/70	30	98.6	YES	NO	YES	YES	NORMAL	PRESENT	ABSENT	PRESENT
33	Pooja G Betageri	5/27/2017	5/29/2017	8.4mg	6446	Dichlorovas	6 hrs 27min	vomiting	60	120/70	14	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
34	Bandenamaj Gani Kamalapur	6/6/2017	6/8/2017	26.4mg	6670	Chlorpyriphos	1 hrs	vomiting,abdominal discomfort	80	110/70	16	98.6	NO	YES	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
35	Shivappa Shankarappa Pujari	6/13/2017	6/17/2017	40.2mg	5770	Dichlorovas	3hrs	vomiting ,pain in abdomen	54	120/70	14	98.6	NO	YES	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
36	Mahadevi Sindhu Naik	6/17/2017	6/21/2017	84.0mg	5279	Dichlorovas	5 hrs	fever ,vomiting,abdominal discomfort	64	110/70	14	99	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
37	Yallawwa Hanamanth Malakari	6/17/2017	21/16/17	40.4mg	8861	Chlorpyriphos	6 hrs	vomiting ,pain in abdomen	90	110/70	16	98.6	YES	NO	YES	NO	NORMAL	ABSENT	ABSENT	ABSENT
38	Revansidda Sabu Kottalagi	7/3/2017	7/5/2017	50.4mg	2356	Dimethoate	4 hrs	vomiting under influence of alcohol	92	136/80	20	98.6	YES	NO	YES	YES	NORMAL	PRESENT	ABSENT	ABSENT
39	Roopashree Somanath Hadapad	7/4/2017	7/10/2017	32.4mg	6058	Lice powder	2 hrs 30min	drowsy	120	110/70	14	98.6	YES	NO	YES	YES	NORMAL	PRESENT	ABSENT	PRESENT
40	Sapna Dasharath Kattimani	7/5/2017	7/7/2017	21.6mg	5208	Lice powder	1 hrs 30min	vomiting	100	120/70	16	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
41	Basagond Mallappa Biradar	7/5/2017	7/7/2017	22.8mg	6907	Chlorpyriphos	2 hrs	vomiting	92	130/80	16	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
42	Parasappa Kallappa Lokure	7/6/2017	7/10/2017	54.2mg	6875	Dichlorovas	16 hrs	vomiting	90	110/70	18	98.6	NO	YES	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
43	Kamalabai Shantappa Kadakal	7/6/2017	7/14/2017	25.2mg	6562	Lice powder	2hrs 10min	vomiting,abdominal discomfort	86	110/80	18	98.6	NO	YES	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
44	Naveen Mohanray Kulkarni	7/12/2017	7/14/2017	14.4mg	3185	Dichlorovas	3 hrs 30min	vomiting	64	128/90	14	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
45	Neha Malasiddappa Patil	7/17/2017	7/22/2017	20.4mg	6020	Lice powder	2hrs 30min	vomiting	60	100/90	14	98.6	YES	NO	YES	YES	NORMAL	PRESENT	ABSENT	ABSENT
46	Meerasab Charlsaib Shaikh	7/19/2017	7/21/2017	15.6mg	4817	Monocrotophos	1 hrs	vomiting,abdominal discomfort	80	110/80	14	98.6	YES	NO	YES	YES	NORMAL	PRESENT	ABSENT	ABSENT
47	Poornima Hanamanth Walikar	7/25/2017	7/29/2017	39.6mg	6485	Lice powder	1hrs 15min	vomiting	110	110/70	16	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
48	Hanamanth Pandu Jamakhandi	8/4/2017	8/8/2017	44.4mg	6218	Dichlorovas	3 hrs 20min	vomiting	80	120/80	14	98.6	NO	YES	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
49	Savita Manohar Rathod	8/14/2017	8/22/2017	10.8mg	141	Lice powder	8 hrs 30min	vomiting,abdominal discomfort	92	140/70	18	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
50	Shobha Mahadev Gheradi	8/25/2017	8/29/2017	12mg	5600	Monocrotophos	3 hrs	vomiting	92	130/90	14	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
51	Soumya Gurabasappa Busannal	9/3/2017	9/6/2017	7.2mg	2497	good night	1 hrs 15min	vomiting	50	130/80	14	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
52	Manayya Kenchappa Kembhavi	9/6/2017	9/8/2017	9.6mg	1663	Dimethoate	6 hrs	vomiting	110	130/80	16	98.6	YES	NO	YES	YES	NORMAL	ABSENT	ABSENT	ABSENT
53	Shilpa Mallikarjun Hundekar	9/11/2017	9/22/2017	49.2mg	5657	Lice powder	8 hrs 45min	nconscious,frothing from mouth bladder incontinen	98	150/80	30	98.6	YES	NO	YES	YES	DISTURBED	ABSENT	ABSENT	PRESENT
54	Wasim Ismail Awati	9/11/2017	9/17/2017	26.4mg	5654	Lice powder	8 hrs 14min	vomiting	102	130/90	14	98.6	YES	NO	YES	NO	NORMAL	ABSENT	ABSENT	ABSENT

OP SMELL PRESENT OR NOT	PSYCHIATRIC ILLNESS	SYSTEMS RESPRATORY	CVS	PA	CNS	Sr cholenesterase day1	Investigations											PRIOR GASTRIC LAVAGE YES OR NO	PRIOR TREATMENT AT HOSPITAL	Intubation at admission	Inj.Atroin first 24hrs	OXIMES INJ,PAM 1gm	Intermediate syndrome	Duration of ventilation	Antibiotics used	Complications	OP DETECTED OR NOT	Outcome Alive/Dead/DAMA
							CBC			RBS	RFT	LFT				X RAY	ECG											
							WBC	HB	PLT			SGPT	SGOT	SR BIL	AMYLASE													
PRESENT	NO	NAD	NAD	NAD	NAD	8120	14460	16.2	2.73	128mg/dl	NORMAL	60	39	0.5	18	NORMAL	TACHYCARDIA	NO	NO	NO	7.2mg	YES	NO		Inj Cefera T 1 gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	195	13080	11.6	1.55	102	NORMAL	20	23	0.5	22	NORMAL	TACHYCARDIA	NO	NO	NO	3.6mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	4664	18800	12	3.95	142	NORMAL	10	16	0.5	24	NORMAL	NORMAL	YES	YES	NO	69.6mg	YES	NO		Inj Cefera 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	3890	13500	15.9	2.4	146	NORMAL	27	37	0.5	14	NORMAL	NORMAL	YES	YES	NO	14.4mg	YES	NO		Inj Cefera 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	7298	8380	15.5	2.15	67	NORMAL	57	32	0.5	64	NORMAL	TACHYCARDIA	NO	NO	NO	4.8mg	YES	NO		Inj Monocef 1gm		DETECTED	ALIVE
PRESENT	YES	NAD	NAD	NAD	NAD	8699	13280	11.8	2.4	140	NORMAL	19	22	0.5	97	NORMAL	NORMAL	NO	NO	NO	54mg	YES	NO		Inj Monocef 1gm		DETECTED	ALIVE
PRESENT	NO	CREPTS	NAD	NAD	NAD	241	5350	16	4.75	150	NORMAL	14	16	0.4	16	NORMAL	TACHYCARDIA	YES	YES	YES	19.8mg	YES	YES	7 DAYS	Inj Tazomac 4.5gm	CARDIAC ARREST	DETECTED	DEATH
PRESENT	NO	CREPTS	NAD	NAD	DRWOSY	563	21520	15.9	0.48	135	HIGH	22	51	0.5	500	NORMAL	TACHYCARDIA	NO	NO	YES	13.2mg	YES	YES	10DAYS	Inj Tazomac 4.5gm	ASPIRATION PNEUMONIA	DETECTED	DEATH
PRESENT	NO	NAD	NAD	NAD	NAD	4766	12800	12	3.41	104	NORMAL	9	19	0.5	52	NORMAL	TACHYCARDIA	NO	NO	NO	4.8mg	YES	NO		Inj Cefera 1gm		DETECTED	ALIVE
PRESENT	NO	CREPTS	NAD	NAD	ALTERED SENSORIUM	404	14790	9.3	1.9	100	NORMAL	24	38	0.5	110	NORMAL	TACHYCARDIA	NO	NO	YES	34.8mg	YES	yes	2DAYS	Inj Taxim 1gm		DETECTED	AMA
PRESENT	NO	NAD	NAD	NAD	NAD	8248	7290	9.9	2.76	98	NORMAL	12	21	0.3	12	NORMAL	NORMAL	NO	NO	NO	38.4mg	YES	NO		Inj Ceffrony1		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	UNCONSCIOUS	8144	10500	16.4	3.26	214	NORMAL	114	128	0.8	122	NORMAL	TACHYCARDIA	YES	YES	YES	3.6mg	YES	NO	1DAY	Inj Ceftrimax T 1.5GM	CARDIAC ARREST	DETECTED	DEATH
PRESENT	NO	NAD	NAD	NAD	NAD	9144	11500	11.4	2.1	123	NORMAL	123	56	0.6	123	NORMAL	TACHYCARDIA	YES	YES	NO	38.4mg	YES	NO		Inj Zoxem S 1.5GM		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	928	19400	18.4	3.73	91	NORMAL	53	30	0.9	77	NORMAL	NORMAL	NO	NO	NO	24mg	YES	NO		Inj CEFERA 1GM		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	6879	8310	15.3	1.72	84	NORMAL	11	19	1.2	128	NORMAL	NORMAL	YES	YES	NO	48mg	YES	NO		Inj Ceftrimax 1.5gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	726	28000	15.8	3.3	136	NORMAL	117	108	1.2	240	NORMAL	NORMAL	NO	NO	NO	60mg	YES	NO		Inj Monocef SB 1.5gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	935	9110	14.4	1.55	79	NORMAL	18	45	1	79	NORMAL	NORMAL	NO	NO	NO	48mg	YES	NO		Inj Resiclav 1.2mg		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	7901	12730	14.4	2.46	319	NORMAL	20	19	0.5	38	NORMAL	TACHYCARDIA	NO	NO	NO	60mg	YES	NO		Inj Ceftrimax 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	873	9760	16.2	2.38	79	NORMAL	19	22	0.5	28	NORMAL	BRADYCARDIA	NO	NO	NO	52.8mg	YES	NO		Inj Resiclav 1.2mg		DETECTED	ALIVE
PRESENT	NO	CREPTS	NAD	NAD	UNCONSCIOUS	371	43180	16.9	4.32	378	NORMAL	54	94	0.8	128	NORMAL	TACHYCARDIA	NO	NO	YES	21.6mg	YES	YES	2 DAYS	Inj Tazomac 4.5gm	CARDIAC ARREST	DETECTED	DEATH
PRESENT	NO	RHONCHI	NAD	NAD	NAD	105	13290	9.9	3.26	80	NORMAL	14	28	0.5	28	NORMAL	TACHYCARDIA	YES	YES	NO	73.2MG	YES	NO		Inj Monocef 1gm		DETECTED	ALIVE
ABSENT	NO	NAD	NAD	NAD	NAD	187	20620	16.6	3.25	128	NORMAL	14	24	0.4	14	NORMAL	NORMAL	NO	NO	NO	60MG	YES	NO		Inj Cefera 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	4484	16260	13.5	1.62	108	NORMAL	12	16	0.6	22	NORMAL	NORMAL	NO	NO	YES	18mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	CREPTS	NAD	NAD	DROWSY	7557	18030	15.8	3.16	128	NORMAL	14	13	1.1	11	NORMAL	TACHYCARDIA	NO	NO	NO	26.4mg	YES	YES	2DAYS	Inj Tazomac 4.5gm	ATROPINE TOXICITY	DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	7268	13510	14.9	1.77	121	NORMAL	13	27	1.6	14	NORMAL	TACHYCARDIA	YES	YES	NO	7.2mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	7783	11390	14.1	2.61	103	NORMAL	27	22	0.5	10	NORMAL	TACHYCARDIA	YES	YES	NO	8.4mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	DROWSY	3972	11750	8.2	5.15	81	NORMAL	10	23	0.5	30	NORMAL	TACHYCARDIA	YES	YES	NO	10.8mg	YES	NO		Inj Taxim 1gm		NOT DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	6523	9000	10.9	3.59	106	NORMAL	22	34	0.8	28	NORMAL	TACHYCARDIA	YES	YES	NO	36mg	YES	NO		Inj Taxim 1gm		NOT DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	4458	5830	15.5	2.36	125	NORMAL	13	14	0.7	108	NORMAL	NORMAL	NO	NO	NO	24mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	CREPTS	NAD	NAD	NAD	4635	5640	9.8	2.29	80	NORMAL	13	26	0.4	12	NORMAL	TACHYCARDIA	NO	NO	NO	7.2mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	CREPTS	NAD	NAD	UNCONSCIOUS	115	29570	18	3.85	126	HIGH	20	25	0.5	126	NORMAL	TACHYCARDIA	NO	NO	YES	14.4mg	YES	yes	2DAYS	Inj Tazar 4.5gm	NOSOCOMIAL PNEUMONIA	DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	224	13020	11.9	3.38	195	NORMAL	31	71	1	228	ABNOMAL	NORMAL	NO	NO	YES	27.6mg	YES	YES	19DAYS	Inj Piprex T 4.5gm	ASPIRATION PNEUMONIA	DETECTED	AMA
ABSENT	YES	NAD	NAD	NAD	NAD	6446	10130	10.8	3.18	121	NORMAL	10	13	0.5	14	NORMAL	BRADYCARDIA	NO	NO	NO	8.4mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	6670	9880	14.4	2.36	110	NORMAL	21	16	0.7	22	NORMAL	NORMAL	YES	YES	NO	26.4mg	YES	NO		Inj Cefera 1gm		DETECTED	ALIVE
ABSENT	NO	NAD	NAD	NAD	DROWSY	5770	8610	12.9	2.55	116	NORMAL	15	17	0.9	80	NORMAL	BRADYCARDIA	YES	YES	NO	40.2mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	5279	6000	13.6	2.42	95	NORMAL	18	21	0.7	50	NORMAL	BRADYCARDIA	YES	YES	NO	84.0mg	YES	NO		Inj Cefera 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	8861	6440	10.8	1.74	90	NORMAL	12	26	0.5	26	NORMAL	NORMAL	NO	NO	NO	40.4mg	YES	NO		Inj Cefera 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	2356	6470	14.2	3.76	78	NORMAL	14	28	0.8	18	NORMAL	NORMAL	NO	NO	NO	50.4mg	YES	NO		Inj Cefera 1gm		DETECTED	AMA
PRESENT	NO	CREPTS	NAD	NAD	DROWSY	6058	26140	14	3.41	178	NORMAL	14	22	0.5	128	NORMAL	TACHYCARDIA	NO	NO	YES	32.4mg	YES	yes	5DAYS	Inj Tazomac 4.5gm	ATROPINE TOXICITY	DETECTED	ALIVE
ABSENT	NO	NAD	NAD	NAD	NAD	5208	14970	11.9	3.15	102	NORMAL	128	104	0.2	24	NORMAL	TACHYCARDIA	NO	NO	NO	21.6mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	6907	4250	14.3	2.93	82	NORMAL	15	18	0.5	62	NORMAL	NORMAL	NO	NO	NO	22.8mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	6875	14220	15	1.63	101	NORMAL	18	35	0.6	16	NORMAL	NORMAL	YES	YES	NO	54.2mg	YES	NO		Inj Acszone 1.5gm		DETECTED	ALIVE
ABSENT	NO	NAD	NAD	NAD	NAD	6562	11720	11.7	2.86	77	NORMAL	44	20	0.4	28	NORMAL	NORMAL	YES	YES	NO	25.2mg	YES	NO		Inj Acszone 1.5gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	3185	11190	13.8	2.49	90	NORMAL	41	28	0.5	16	NORMAL	BRADYCARDIA	NO	NO	NO	14.4mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	6020	16590	11.8	2.32	158	NORMAL	11	18	0.4	11	NORMAL	BRADYCARDIA	NO	NO	NO	20.4mg	YES	NO		Inj Cefera 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	4817	8520	14.7	2.47	93	NORMAL	14	19	1.8	202	NORMAL	NORMAL	NO	NO	NO	15.6mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
ABSENT	NO	NAD	NAD	NAD	NAD	6485	14500	10.4	3.02	128	NORMAL	127	22	0.4	22	NORMAL	TACHYCARDIA	NO	NO	NO	39.6mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	DROWSY	6218	12810	14.2	2.5	111	NORMAL	24	28	0.5	34	ABNOMAL	NORMAL	YES	YES	NO	44.4mg	YES	NO		Inj Cefera T 1 gm		DETECTED	ALIVE
ABSENT	NO	NAD	NAD	NAD	NAD	141	16200	15.1	2.56	192	NORMAL	13	22	0.5	18	NORMAL	NORMAL	YES	YES	NO	10.8mg	YES	NO		Inj Cefera T 1 gm		DETECTED	ALIVE
ABSENT	NO	NAD	NAD	NAD	NAD	5600	24200	13.7	5.41	175	NORMAL	88	12	0.5	44	NORMAL	NORMAL	NO	NO	NO	12mg	YES	NO		Inj Cefera T 1 gm		DETECTED	ALIVE
ABSENT	NO	NAD	NAD	NAD	NAD	2497	11530	13	2.01	81	NORMAL	10	17	0.6	46	NORMAL	BRADYCARDIA	NO	NO	NO	7.2mg	YES	NO		Inj Taxim 1gm		DETECTED	ALIVE
PRESENT	NO	NAD	NAD	NAD	NAD	1663	19680	14.2	2.49	183	NORMAL	17	19	0.8	17	NORMAL	TACHYCARDIA	YES	YES	NO	9.6mg	YES	NO		Inj Tazomac 4.5gm		DETECTED	ALIVE
PRESENT	NO	CREPTS	NAD	NAD	UNCONSCIOUS	5657	9710	12.9	2.79	112	NORMAL	10	21	0.4	118	NORMAL	NORMAL	NO	NO	YES	49.2mg	YES	yes	3DAYS	Inj Tazar 4.5gm	ATROPIN TOXICITY	DETECTED	ALIVE
ABSENT	NO	NAD	NAD	NAD	NAD	5654	17300	15	3.3	76	NORMAL	12	18	0.7	20	NORMAL	TACHYCARDIA	YES	YES	NO	26.4mg	YES	NO		Inj Cefera 1gm		DETECTED	ALIVE

GROUP B

S.No	Name of Patient	Regd No(Ipd No)	AGE	SEX	ADRESS	DOA	DOD	Compound	Time gap presentation	CLINICAL FEATURES	SR CHOLINESTERASE	INJ ATROPINE 1ST 24HRS	VITALS				PUPILS				
													PULSE/mn	BPmmhg	RR /mn	TEMP in F	MIOSIS	DILATED	LACRIMATI	SALIVATION	MICTURATI
1	Shantabai Somanagouda Patil	886	48	F	MUDDEBIHAL	1/9/2016	1/16/2016	MONOCRPTOPHOS	4 hrs 50min	Abdominal discomfort,vomiting	462	55.8mg	64	120/80	16	98.9	YES	NO	YES	YES	NORMAL
2	Vitthal Sangappa Jinjarawad	1764	55	M	VIJYAPURA	1/18/2016	1/25/2016	DICHLOROVAS	2 hrs 20min	Vomiting	423	97.2mg	60	170/80	16	98.6	YES	NO	YES	YES	NORMAL
3	Bagappa Amasidha Pujari	2298	34	M	VIJYAPURA	1/21/2016	1/23/2016	DICHLOROVAS	2 hrs 40min	Vomiting	7837	63mg	60	110/70	14	98.6	YES	NO	YES	YES	NORMAL
4	Rajashree Basavaraj Gadad	2889	25	F	INDI	1/26/2016	1/30/2016	DICHLOROVAS	2 hrs	DROWSY,FROTHING FROM MOUTH,N	6823	66mg	62	130/70	16	98.6	YES	NO	YES	YES	NORMAL
5	Gourawwa Jettappa Keshapur	7338	28	F	MUDDEBIHAL	3/3/2016	3/18/2016	CHLORGUARD	4hrs 45min	Vomiting	105	89.4mg	96	100/70	14	98.6	YES	NO	YES	YES	NORMAL
6	Nikita Ashok Hosamani	8447	22	F	INDI	3/13/2016	3/18/2016	LICE POWDER	2hrs	Vomiting,Abdominal discomfort	1833	70.2mg	80	122/70	14	98.6	YES	NO	YES	YES	NORMAL
7	Iramma Mallappa Kumbar	8789	28	F	YADAGIR	3/16/2016	3/21/2016	LICE POWDER	6hrs 30min	Vomiting	3920	68.4mg	64	110/70	14	98.6	YES	NO	YES	YES	NORMAL
8	Preeti Dyamagond Koli	11247	18	F	VIJYAPURA	4/4/2016	4/8/2016	LICE POWDER	2 hrs 30min	Vomiting	5151	64.2mg	60	110/70	14	98.6	YES	NO	YES	YES	NORMAL
9	Anita Raju Pawar	12505	28	F	VIJYAPURA	4/14/2016	4/19/2016	LICE POWDER	1 hrs	Vomiting,Abdominal discomfort	5892	49.8mg	92	150/90	16	98.6	YES	NO	YES	YES	NORMAL
10	Shilpa Shasappa Chalawadi	13525	19	F	BAGEWADI	4/22/2016	4/30/2016	DICHLOROVAS	3 hrs	Vomiting	8355	69mg	60	90/60	18	98.6	YES	NO	YES	YES	NORMAL
11	Kashinath Mallappa Sanakki	13651	25	M	SINDAGI	4/24/2016	4/29/2016	DICHLOROVAS	4 hrs 40min	Vomiting	5407	64.8mg	86	130/80	16	98.6	YES	NO	YES	YES	NORMAL
12	Ayesha Mustaq Sayad	14738	35	F	INDI	5/2/2016	5/4/2016	LICE POWDER	6 hrs	Vomiting	250	84.6mg	86	130/80	16	98.6	YES	NO	YES	YES	NORMAL
13	Venkatesh Suresh Pattar	14755	28	M	VIJYAPURA	5/2/2016	5/4/2016	DIMETHOATE	2 hrs 16mi n	Vomiting	6191	72mg	78	110/70	16	98.6	YES	NO	YES	YES	NORMAL
14	Sanganna Paramappa Bekinal	19501	35	M	BAGEWADI	6/12/2016	6/19/2016	MONOCRPTOPHOS	3 hrs	Vomiting	8353	88.2mg	64	110/70	16	98.6	YES	NO	YES	YES	NORMAL
15	Mallikarjun Lakshaman Tenalli	20263	23	M	INDI	6/18/2016	6/21/2016	MONOCRPTOPHOS	3 hrs 51min	Vomiting	6361	70.2mg	60	120/80	14	98.6	NO	YES	NO	YES	NORMAL
16	Ningappa Channamalla Almel	20287	26	M	INDI	6/18/2016	6/25/2016	CHLORPYRIFOS	2 hrs	Vomiting ,Abdomainal discomfort	137	82.8mg	96	140/80	18	98.6	YES	NO	YES	YES	NORMAL
17	Laxmi Chanamallappa Manehalli	24241	27	F	BAGEWADI	7/24/2016	7/29/2016	LICE POWDER	2 hrs 21 min	Vomiting	4158	70.2mg	74	110/70	16	98.6	YES	NO	YES	YES	NORMAL
18	Parvati Basanagouda Biradar	27021	28	F	BAGEWADI	8/15/2016	8/20/2016	MONOCRPTOPHOS	2 hrs 25min	Vomitimg,sweating	5235	72mg	88	140/90	14	98.6	YES	NO	YES	YES	NORMAL
19	Grimialla Bhimashappa Kakhadaki	27492	27	M	BAGEWADI	8/19/2016	8/22/2016	DICHLOROVAS	2 hrs 52min	Vomiting	7343	68.4mg	110	120/70	14	98.6	YES	NO	YES	YES	NORMAL
20	Chandru Shranappa Halli	27975	18	M	SINDAGI	8/22/2016	8/26/2016	LICE POWDER	7 hrs 42 min	Vomiting	3575	79.2mg	84	110/70	16	98.6	YES	NO	YES	NO	NORMAL
21	Chandappa Jatteppa Pujari	35150	60	M	SINDAGI	10/21/2016	10/29/2016	DIMETHOATE	5 hrs 34min	Vomiting	5724	86.4mg	64	106/80	14	98.6	YES	NO	YES	YES	NORMAL
22	Bheemanna Yallappa Bajantri	35229	40	M	VIJYAPURA	22/10/206	10/28/2016	DICHLOROVAS	2 hrs 35min	Vomiting,Unconscious	765	88.2Mmg	114	124/68	18	98.6	YES	NO	YES	YES	NORMAL
23	Annapurna Govind Jadhav	35158	38	F	SINDAGI	10/22/2016	10/24/2016	MONOCRPTOPHOS	4 hrs 52min	Vomiting,sweating,bowel,bladder inc	6650	47.4mg	56	90/60	14	98.6	YES	NO	YES	YES	DISTURBED
24	Shantu Basavaraj Bidari	35559	18	M	INDI	10/25/2016	10/30/2016	DICHLOROVAS	1 hrs 45min	Vomiting	6850	83.4mg	80	120/80	16	98.6	YES	NO	YES	YES	NORMAL
25	Renukabai Danappa Ganjal	36070	25	F	INDI	10/30/2016	11/5/2016	DICHLOROVAS	3 hrs 45min	Vomiting ,Abdomainal discomfort	5645	70.2Mmg	120	110/80	16	98.6	NO	YES	YES	YES	NORMAL
26	Parvati Butale Savilkar	36417	36	F	INDI	11/2/2016	11/9/2016	DIMETHOATE	2 hrs	Vomiting ,sweating	1747	80.4mg	98	130/70	16	98.6	YES	NO	YES	YES	NORMAL
27	Shivakanta Bherrappa Shirshyad	36935	30	F	INDI	11/6/2016	I5/11/16	DICHLOROVAS	2 hrs 23min	Vomiting ,bladder incontinence	7826	66.0mg	68	110/70	16	98.6	YES	NO	YES	YES	DISTURBED
28	Danesh Rajashekar Hosamani	7706	25	M	INDI	3/9/2017	2I/03/17	DICHLOROVAS	5 hrs 34min	Vomiting,sweating	152	72mg	120	130/80	20	98	NO	YES	NO	NO	NORMAL
29	Shaila Yallappa Honamatti	12312	19	F	SINDAGI	4/18/2017	4/22/2017	LICE POWDER	7 hrs 42 min	Vomiting,frothing from mouth	3863	33.6mg	118	130/88	20	98.6	YES	NO	YES	YES	NORMAL
30	Geeta Santosh Pujari	14634	26	F	INDI	5/8/2017	5/16/2017	DICHLOROVAS	2 HRS 28MIN	Breathlessness,vomiting,unconscious	158	81mg	140	110/70	24	98.6	YES	NO	YES	YES	NORMAL
31	Mallamma Basanagouda Meti	14709	30	F	MUDDEBIHAL	5/9/2017	5/12/2017	DICHLOROVAS	4 hrs	vomiting	5497	24mg	118	130/80	16	98.6	NO	YES	YES	YES	NORMAL
32	Parvati Ningappa Mang	15079	36	F	SANGALI	5/11/2017	5/19/2017	QUNILPHOS	1hrs30min	Vomiting,frothing from mouth	244	57.6mg	100	130/90	18	98.6	YES	NO	YES	YES	NORMAL
33	Shridevi Doddappa Kembhavi	17415	27	F	YADAGIRI	5/31/2017	6/5/2017	CHLORPYRIFOS	5 hrs 34min	vomiting,drowsy	125	85.8mg	100	130/80	16	98.6	NO	YES	YES	YES	NORMAL
34	Akshata Premanand Tangadgi	18210	20	F	INDI	6/6/2017	6/10/2017	DICHLOROVAS	4 hrs	vomiting	5368	32.4mg	130	130/70	18	98.6	YES	NO	YES	YES	NORMAL
35	Kumar Duragappa Bandiwaddar	18825	21	M	KALABURAGI	6/12/2017	6/22/2017	DIMETHOATE	4 hrs	vomiting	3286	69.6mg	64	110/70	16	98.6	YES	NO	YES	YES	NORMAL
36	Shaila Santosh Gennur	19484	31	F	BAGEWADI	6/16/2017	6/17/2017	DICHLOROVAS	3hrs	vomiting,abdominal pain	107	48mg	120	130/80	16	98.6	YES	NO	YES	YES	NORMAL
37	Basavanth Appasaheb Bomanalli	19593	24	M	VIJYAPURA	6/17/2017	26.06/17	MONOCRPTOPHOS	2 hrs 40min	Breathlessness,vomiting,frothing fror	414	78mg	60	120/80	34	98.6	YES	NO	YES	YES	NORMAL
38	Kashinath Shivanna Biradar	19702	28	M	MUDDEBIHAL	6/18/2017	6/21/2017	DICHLOROVAS	4hrs 45min	vomiting	5267	68.4mg	116	120/70	16	98.6	YES	NO	YES	YES	NORMAL
39	Suvarna Shivanand Bellubki	21300	19	F	VIJYAPURA	6/30/2017	7/5/2017	LICE POWDER	2hrs15min	vomiting	6652	52.2mg	62	130/70	16	98.6	YES	NO	YES	YES	NORMAL
40	Trupti Rudragouda Biradar	21593	19	F	INDI	7/2/2017	7/6/2017	LICE POWDER	2hrs15min	Vomiting,Abdominal discomfort	4160	57.6mg	76	110/70	16	98.6	YES	NO	YES	YES	NORMAL
41	Ashwini Manjunath Kuri	21736	22	F	VIJYAPURA	7/3/2017	7/9/2017	LICE POWDER	4HRS30MIN	UNCONSCIOUS	1407	23.4mg	120	90/60	20	98.6	YES	NO	YES	YES	NORMAL
42	Nagaraj Mallappa Hilli	24182	28	M	INDI	7/23/2017	7/28/2017	DICHLOROVAS	5hrs	Vomiting,Unconscious	226	68.4mg	52	150/90	12	98.6	YES	NO	YES	YES	NORMAL
43	Ningamma Shankarappa Koluragi	24300	35	F	INDI	7/24/2017	7/27/2017	DIMETHOATE	1hhrs45min	vomiting	5180	60mg	64	110/70	14	98.6	YES	NO	YES	YES	NORMAL
44	Appasi Balu Biradar	24981	32	M	VIJYAPURA	7/29/2017	8/2/2017	DICHLOROVAS	4hrs	vomiting,drowsy	668	48mg	90	140/80	18	98.6	NO	YES	YES	YES	NORMAL
45	Geeta Natikar	25034	19	F	INDI	7/30/2017	8/5/2017	DICHLOROVAS	2hrs24min	Vomiting,Abdominal discomfort	4257	57.6mg	140	90/30	16	94	YES	NO	YES	YES	NORMAL
46	Shantabai Basagond Biradar	25739	65	F	VIJYAPURA	8/4/2017	8/7/2017	DICHLOROVAS	2hrs	vomiting,bowel incontinence	5992	28.8mg	86	160/100	14	98.6	NO	YES	YES	YES	NORMAL
47	Ninganagouda Guralingappa Chanagound	26735	20	M	SINDAGI	8/12/2017	8/17/2017	DICHLOROVAS	4hrs30min	vomiting,drowsy	6227	48mg	102	130/70	18	98.6	YES	NO	YES	YES	NORMAL
48	Aisha Monalasab Attar	28803	24	F	VIJYAPURA	8/29/2017	9/1/2017	DICHLOROVAS	1hrs54min	vomiting	6770	33.6mg	82	130/70	16	98.6	YES	NO	YES	YES	NORMAL
49	Sangeeta Sangi Jadhav	29443	21	F	VIJYAPURA	9/3/2017	9/7/2017	LICE POWDER	1hrs52min	Abdominal discomfort,vomiting	2560	54.6mg	110	160/90	18	99.3	YES	NO	YES	YES	NORMAL
50	Appasab Mallappa Biradar D	30780	35	M	VIJYAPURA	9/13/2017	9/20/2017	ROGAR	2hrs21min	vomiting	1016	72.6mg	92	120/70	14	98.6	YES	NO	YES	YES	NORMAL
51	Sangeeta Sunil Rathod	30784	20	F	VIJYAPURA	9/13/2017	9/20/2017	ROGAR	2hrs	vomiting	914	55.8mg	102	150/90	18	98.6	YES	NO	YES	YES	NORMAL
52	Rachappa Gurappa Sabarad	31265	80	M	VIJYAPURA	9/17/2017	9/19/2017	ROGAR	3hrs15min	Vomiting,frothing ,unconsciouness	104	90mg	50	170/90	24	98.6	YES	NO	YES	YES	NORMAL
53	Malleshi Sahebagouda Biradar	31680	34	M	KALABURAGI	9/20/2017	9/22/2017	MONOCRPTOPHOS		giddiness,blurring of vision	135	66mg	52	140/80	16	98.6	YES	NO	YES	YES	DISTURBED

			OP SMELL	PAST HISTORY OF ILLNES			PSYCHIATRIC ILLNESS	FAMILY HISTORY		PERSONAL H/O				HABITS			MENSTRUAL H/O	RS	CVS	PA	CNS	SR CHOLINESTERASE	INVESTIGATION				
																								CBC	HB	PLT	RBS	RFT
FASICULATIC	BRONCHOPH	CRACKLES		HTN	DM	OTHERS		HTN	DM	DIET	APPETITE	SLEEP	B/B	SMOKING	ALCOHOL	TOBACCO							WBC				
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	MENOPAUSE	NAD	NAD	NAD	NAD	462	21500	13	2.84	112	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	EPILEPSY	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	YES	NO	NO		NAD	NAD	NAD	NAD	423	9030	13.4	1.93	123	NORMAL
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO		NAD	NAD	NAD	NAD	7837	7590	14.2	2.01	83	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	DROWSY	6823	5950	11.3	1.5	89	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	105	15410	12.9	2.58	133	NORMAL
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	1833	11500	7	3.43	217	NORMAL
PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	3920	8500	13.8	2.55	128	NORMAL
PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	5151	16670	13.3	2.22	101	NORMAL
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	5892	16400	11.7	3.51	120	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	8355	13610	13.8	2.59	196	NORMAL
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	DECREASED	INCREASED	NORMAL	NO	YES	NO		NAD	NAD	NAD	NAD	5407	13900	14.6	2.65	120	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	250	18670	12.7	3.61	218	NORMAL
PRESENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	YES	NO		NAD	NAD	NAD	NAD	6191	12220	15.9	2.68	86	NORMAL
PRESENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	YES		NAD	NAD	NAD	NAD	8353	8950	14.7	2.52	92	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO		NAD	NAD	NAD	NAD	6361	8020	15.8	2.12	74	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO		NAD	NAD	NAD	NAD	137	12580	13.5	3.29	106	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	4158	13600	11.3	3.03	140	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	5235	8900	11.6	2.78	85	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	YES	NO		NAD	NAD	NAD	NAD	7343	8910	13	4.04	50	NORMAL
PRESENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO		NAD	NAD	NAD	NAD	3575	7800	14	2.83	157	NORMAL
PRESENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	YES	NO	YES		NAD	NAD	NAD	NAD	5724	9500	15.2	2.41	75	NORMAL
PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	YES	YES		CREPTS	NAD	NAD	UNCONSCIOS	765	8490	14.4	1.77	133	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	DISTURBED	NO	NO	NO	PREMENOPA	NAD	NAD	NAD	NAD	6650	8600	10.7	2.75	92	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	YES	YES	YES		NAD	NAD	NAD	NAD	6850	11440	15.3	3.7	120	NORMAL
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	5645	9040	12.1	2.74	96	NORMAL
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	1747	18870	12.7	2.35	254	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	DISTURBED	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	7826	9510	12.7	3.46	78	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	YES	NO		NAD	NAD	NAD	NAD	152	13270	16	2.93	69	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	3863	16490	10.6	2.65	99	NORMAL
PRESENT	CREPTS	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	CREPTS	NAD	NAD	UNCONSCIOS	158	12290	10.9	2.94	174	NORMAL
ABSENT	NORMAL	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	5497	12740	12.7	3.27	109	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	244	16750	12.1	3.28	122	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	DROWSY	125	18060	13.1	3.66	84	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	5368	8140	10.7	2.8	122	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO		NAD	NAD	NAD	NAD	3286	17340	17.7	3.4	107	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	107	21610	11.4	3.12	94	NORMAL
PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO		CREPTS	NAD	NAD	NAD	414	18000	15.4	2.84	166	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	YES	NO		NAD	NAD	NAD	NAD	5267	7130	15.4	2.52	86	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	DECREASED	DECREASED	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	6652	17620	12.6	3.77	102	NORMAL
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	4160	10940	11.7	2.16	129	NORMAL
PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	UNCONSCIOS	1407	25900	12.7	3.29	114	NORMAL
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO		NAD	NAD	NAD	NAD	226	20360	14.7	3.34	109	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO		NAD	NAD	NAD	NAD	5180	11730	11.9	4.24	112	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	YES	NO		NAD	NAD	NAD	DROWSY	668	7350	13	2.45	87	NORMAL
PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	DROWSY	4257	41880	11.9	1.63	257	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	DECREASED	NORMAL	NO	NO	NO	MENOPAUSE	NAD	NAD	NAD	NAD	5992	24510	12.3	3.48	107	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO		NAD	NAD	NAD	NAD	6227	9800	14.8	2.7	98	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	6770	11540	13.5	3.31	91	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	DROWSY	2560	16250	11.9	3.67	114	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	DECREASED	INCREASED	NORMAL	NO	YES	NO		NAD	NAD	NAD	DROWSY	1016	14040	13.7	2.26	128	NORMAL
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	NO	NO	NORMAL	NAD	NAD	NAD	NAD	914	10780	11.2	2.1	112	NORMAL
ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	MIXED	NORMAL	NORMAL	NORMAL	NO	YES	YES		CREPTS	NAD	NAD	UNCONSCIOS	104	12120	4.9	3.2	138	NORMAL
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	ABSENT	ABSENT	ABSENT	VEG	NORMAL	NORMAL	NORMAL	NO	NO	NO		NAD	NAD	NAD	NAD	135	10490	13.9	2.78	112	NORMAL

						PRIOR GASTRIC LAVAGE	PRIOR TREATMENT AT HOSP	INTUBATION AT ADMISSION	INJ ATROPINE 1ST 24HRS	OXIMES INJ PAM	DURATION OF VENTILATION	ANTIBIOTICS USED	INTERMEDIATE SYNDROME	COMPLICATIONS	OP DETECTED OR NOT	Outcome Alive/Dead/DAMA
LFT				X RAY	ECG											
SGPT	SGOT	SR BILI	SR AMYLASE													
13	21	0.5	29	NORMAL	BRADYCARDIA	YES	YES	NO	55.8mg	YES		INJ KEPHAZONE 1gm			DETECTED	ALIVE
18	24	0.5	15	NORMAL	BRADYCARDIA	NO	NO	NO	97.2mg	YES		INJ TAXIM 1gm			DETECTED	ALIVE
26	33	0.5	58	NORMAL	BRADYCARDIA	NO	NO	NO	63mg	YES		INJ CEFERA 1gm			DETECTED	ALIVE
13	11	0.6	123	NORMAL	BRADYCARDIA	NO	NO	NO	66mg	YES		INJ TAXIM 1gm			DETECTED	ALIVE
14	19	0.8	123	NORMAL	NORMAL	NO	NO	NO	89.4mg	YES		INJ ACSZONE 1.2gm			DETECTED	ALIVE
36	49	1.1	22	NORMAL	NORMAL	NO	NO	NO	70.2mg	YES		INJ O2 100ml			DETECTED	ALIVE
12	31	0.5	21	NORMAL	BRADYCARDIA	NO	NO	NO	68.4mg	YES		INJ CEFFRONY 1gm			DETECTED	ALIVE
20	27	0.5	90	NORMAL	BRADYCARDIA	NO	NO	NO	64.2mg	YES		INJ CEFERA 1gm			DETECTED	ALIVE
12	16	0.5	18	NORMAL	NORMAL	NO	NO	NO	49.8mg	YES		INJ BIOCET 1gm			DETECTED	ALIVE
11	16	0.5	75	NORMAL	BRADYCARDIA	YES	YES	NO	69mg	YES		INJ CEFTRIAXONE 1gm			DETECTED	ALIVE
20	24	0.5	82	NORMAL	NORMAL	NO	NO	NO	64.8mg	YES		INJ MONOCEF 1gm		ATROPINE TOXICITY	DETECTED	AMA
12	22	0.6	24	NORMAL	NORMAL	NO	NO	NO	84.6mg	YES		INJ TAXIM 1gm			DETECTED	ALIVE
26	15	0.5	98	NORMAL	NORMAL	NO	NO	NO	72mg	YES		INJ CEFERA 1gm			DETECTED	ALIVE
13	18	0.8	207	NORMAL	BRADYCARDIA	NO	NO	NO	88.2mg	YES		INJ ZOXEM S 1.5gm		ATROPINE TOXICITY	DETECTED	ALIVE
16	38	0.6	88	NORMAL	BRADYCARDIA	YES	YES	NO	70.2mg	YES		INJ ZOXEM S 1.5gm			DETECTED	ALIVE
32	21	0.8	53	NORMAL	NORMAL	NO	NO	NO	82.8mg	YES		INJ RESICLAV 1.2gm			DETECTED	ALIVE
41	22	0.6	144	NORMAL	BRADYCARDIA	YES	YES	NO	70.2mg	YES		Inj Zoxem S 1.5gm			DETECTED	ALIVE
13	18	0.7	54	NORMAL	NORMAL	NO	NO	NO	72mg	YES		Inj Cefera 1gm			DETECTED	ALIVE
14	18	0.5	135	NORMAL	TACHYCARDIA	NO	NO	NO	68.4mg	YES		Inj Ceftriaxone 1gm			DETECTED	ALIVE
22	19	0.7	75	NORMAL	NORMAL	YES	YES	NO	79.2mg	YES		Inj Monocef SB 1.2gm			DETECTED	ALIVE
12	26	0.7	34	NORMAL	BRADYCARDIA	NO	NO	NO	86.4mg	YES		Inj Cefera T 1gm			DETECTED	ALIVE
58	65	0.5	248	NORMAL	TACHYCARDIA	NO	NO	YES	88.2Mmg	YES	5 days	Inj Taxim 1gm		ASPIRATION PNEUMONIA	DETECTED	ALIVE
28	58	0.8	63	NORMAL	BRADYCARDIA	YES	YES	NO	47.4mg	YES		Inj Ceftriaxone 1gm			DETECTED	ALIVE
17	21	0.5	56	NORMAL	NORMAL	NO	NO	NO	83.4mg	YES		Inj Ceffrony 1gm			DETECTED	ALIVE
24	36	0.5	66	NORMAL	TACHYCARDIA	YES	YES	NO	70.2Mmg	YES		Inj Minoclav 1.2gm			DETECTED	ALIVE
10	20	0.5	53	NORMAL	NORMAL	NO	NO	NO	80.4mg	YES		Inj Cefera 1gm			DETECTED	ALIVE
23	17	0.5	62	NORMAL	BRADYCARDIA	NO	NO	NO	66.0mg	YES		INJ CeferaT 1.5gm			DETECTED	ALIVE
32	18	1.1	124	NORMAL	TACHYCARDIA	YES	YES	NO	72mg	YES		INJ Acszone 1gm			DETECTED	ALIVE
17	22	0.5	109	NORMAL	TACHYCARDIA	YES	YES	NO	33.6mg	YES		INJ Monocef 1GM			DETECTED	ALIVE
21	36	0.5	122	NORMAL	TACHYCARDIA	NO	NO	YES	81mg	YES	8 days	INJ Tazomac 4.5gm		ATROPINE TOXICITY	DETECTED	ALIVE
15	14	0.5	124	NORMAL	TACHYCARDIA	YES	YES	NO	24mg	YES		INJ Cefera 1Ggm			NOT DETECTED	ALIVE
14	17	0.4	84	NORMAL	TACHYCARDIA		NO	NO	57.6mg	YES		INJ Acszone 1.5gm			DETECTED	ALIVE
15	20	0.4	86	NORMAL	TACHYCARDIA	YES	YES	NO	85.8mg	YES		INJ Taxim 1gm		ATROPINE TOXICITY	DETECTED	ALIVE
11	16	0.9	98	RT CONSOLIDATION	TACHYCARDIA	NO	NO	NO	32.4mg	YES		Inj Taxim 1gm		PNEUMONIA	DETECTED	ALIVE
19	18	0.6	144	RT CONSOLIDATION	BRADYCARDIA	NO	NO	NO	69.6mg	YES		INJ Cefera 1 gm		ATROPINE TOXICITY	DETECTED	ALIVE
15	24	0.5	156	NORMAL	TACHYCARDIA	YES	YES	NO	48mg	YES		INJ Cefera T 1.5gm			DETECTED	ALIVE
31	47	2	156	NORMAL	BRADYCARDIA	NO	NO	NO	78mg	YES		Inj Cefera 1gm			DETECTED	ALIVE
10	16	1.1	120	NORMAL	TACHYCARDIA	NO	NO	NO	68.4mg	YES		INJ Taxim 1gm			DETECTED	ALIVE
21	15	0.6	84	NORMAL	BRADYCARDIA	NO	NO	NO	52.2mg	YES		Inj Minoclav 1.2gm			DETECTED	ALIVE
12	15	0.5	23	NORMAL	NORMAL	NO	NO	NO	57.6mg	YES		INJ RESICLAV 1.2gm			DETECTED	ALIVE
10	26	0.5	123	NORMAL	TACHYCARDIA	YES	YES	NO	23.4mg	YES		INJ MICROTAZ 4.5gm			DETECTED	ALIVE
30	31	0.7	123	NORMAL	BRADYCARDIA	NO	NO	YES	68.4mg	YES	4days	INJ TAXIM 1gm			DETECTED	ALIVE
25	22	0.5	53	NORMAL	BRADYCARDIA	NO	NO	NO	60mg	YES		Inj Cefera 1gm			NOT DETECTED	ALIVE
12	20	0.6	45	NORMAL	NORMAL	YES	YES	NO	48mg	YES		INJ TAXIM 1gm			DETECTED	ALIVE
11	26	0.4	69	NORMAL	TACHYCARDIA	NO	NO	YES	57.6mg	YES	3days	INJ TAZOMAC 4.5gm			DETECTED	ALIVE
36	40	o.6	135	NORMAL	NORMAL	NO	NO	NO	28.8mg	YES		INJ CEFERA T1.5gm			DETECTED	ALIVE
45	28	0.5	120	NORMAL	TACHYCARDIA	YES	YES	NO	48mg	YES		INJ RESICLAV 1.2 gm			NOT DETECTED	ALIVE
23	45	0.6	123	NORMAL	NORMAL	NO	NO	NO	33.6mg	YES		INJ TAXIM 1gm			DETECTED	ALIVE
13	45	0.5	56	NORMAL	TACHYCARDIA	NO	NO	YES	54.6mg	YES	5 days	Inj Cefera 1gm			DETECTED	ALIVE
17	24	0.5	22	NORMAL	NORMAL	NO	NO	YES	72.6mg	YES	4DAYS	Inj Cefera 1gm		CARDIAC ARREST	DETECTED	DEATH
15	45	0.6	56	NORMAL	TACHYCARDIA	NO	NO	NO	55.8mg	YES		Inj Cefera 1gm			DETECTED	ALIVE
9	18	0.7	84	CONSOLIDATION	BRADYCARDIA	NO	NO	YES	90mg	YES	3DAYS	INJ Tazomac 4.5gm		CARDIAC ARREST/PNEUMONIA	DETECTED	DEATH
17	31	0.4	158	NORMAL	BRADYCARDIA	YES	YES	YES	66mg	YES	2DAYS	Inj Cefera 1gm		CARDIAC ARREST	INHALATION	AMA

TOTAL SUBJECTS IN GROUP A 54 OF WHICH 36 SUBJECTS ANALYSED FOR MODERATE TO SEVERE POISONING N=36

		ipd			Compound	Time gap presentation	CLINICAL FEATURES	SERUM CHOLENESTRSE R			initial atropinization 24hrs in	Atropinized dosage	Time taken for atropinization in hrs	atropine toxicity	Intubation at admission	duration of ventilation in days	Intermediate syndrome	outcome		DEATH OF TIME
								DAY	DAY2	DAY3-5								ALIVE	DEATH	DEATH <24HR
1	Mahadev Basappa Nyangond		1/5/2016	1/11/2016	Dichlorovas	5hr40mins	vomiting	195		131	102	28.8	8		NO					
2	Sangeeta Kumar Madavi		1/9/2016	1/13/2016	Dimethoate	6hrs20mins	vomiting,abdominal discomfort	4664			69.6	24	3	YES	NO					
3	Priya Raju Bandiwaddar		2/28/2016	3/6/2016	Monocrotophos	5hrs	vomiting	241	257	380	64.8	28.8	2		YES	7	yes		died	
4	Mallamma Bhimanna Pujari		2/13/2016	2/18/2016	Lice powder	5hrs	vomiting	8699			54	30.6	7		NO					
5	Sanganagouda Malakanagouda patil		3/11/2016	3/20/2016	Dichlorovas	11hrs	drowsy,under influence alcohol	563			66	13.2	4		YES	10	yes		died	
6	Sahadev Pandu Pandre		3/30/2016	3/31/2016	Dimethoate	4hrs	vomiting,giddiness,altered sensorium	404			34.8	9	6		YES	3	yes			
7	Sudharani Dondiba Bhosale		4/5/2016	4/7/2016	Monocrotophos	4hrs	vomiting,abdominal discomfort	8248			39.6	12	5		NO					
8	Vinod Shamray Bairshetty		4/17/2016	4/17/2016	Dichlorovas	5hrs30min	vomiting,loss of consciousness,incontinence	8144			9	9	2	YES	YES	1			died	
9	Tippana B Pujari		1/20/2016	1/25/2016	Dimethoate	4	vomiting,abdominal discomfort	3890		4926	54.6	12	2		no					
10	Mudukappa Shamrao Hosamani		5/22/2016	5/31/2016	Dichlorovas	1hrs30min	vomiting	928	2940	3610	39.6	14.4	4		NO					
11	Sunil Siddalingappa Jatti		8/1/2016	8/8/2016	Dichlorovas	1hrs5min	vomiting,abdominal pain	726		3160	60	20.6	6		NO					
12	Siddu Gurappa Jangamashetti		8/11/2016	8/20/2016	Dichlorovas	2hrs20min	vomiting,abdominal discomfort	935	2765	3072	49.2	21	3		NO					
13	Gururaj Pandappa Hadimani		8/21/2016	8/30/2016	Dichlorovas	2hrs	vomiting,loose stools	873			52.8	27	12		NO					
14	Anasuya Muttanagouda Patil		10/29/2016	11/3/2016	Dichlorovas	4hrs	breathlessness.urine incontinence vomiting	371			56.4	24	3	YES	YES	2			died	
15	Guranna Amarappa Awaradi		1/22/2017	1/30/2017	Dimethoate	4hrs	Vomiting	105	370		105	28.8	8	YES	NO					
16	Ramesh Shivanand Ka ond		1/30/2017	2/6/2017	Dichlorovas	1h	vomiti ,abdominal discomfort	187		1119	60	14.4	3		NO					
17	Anjana Praveen Pujari		2/1/2017	2/4/2017	Lice powder	2 hrs30mins	vomiting	4484		4639	28.8	7.2	4		no					
18	Aravind Gundanna Dodamani		5/18/2017	5/21/2017	Dimethoate	4 hrs 15min	unconscious,convulsions,frothingfrom mouth	115	119		84	23.4	20	YES	YES	2				
19	Bhimray Ningappa Biradar		5/19/2017	6/6/2017	Monocrotophos	6 hrs	vomiting,under influence of alcohol	224	418	966	96	21.6	4	YES	YES	19	yes			
20	Suresh Shrimantappa Pattanashetti		3/29/2017	4/2/2017	Dichlorovas	7hrs 30 min	drowsy	7557			26.4	3.6	2	YES	yes	2				
21	Naveen Mohanray Kulkarni		7/12/2017	7/14/2017	Dichlorovas	3 hrs 30min	vomiting	3185			15.6	7.6	4		NO					
22	Mahadevi Sindhu Naik		6/17/2017	6/21/2017	Dichlorovas	5 hrs	fever ,vomiting,abdominal discomfort	5279			84	43.2	9		NO					
23	Yallawwa Hanamanth Malakari		6/17/2017	21/16/17	Chlorpyriphos	6 hrs	vomiting ,pain in abdomen	8861			42	23.4	16		NO					
24	Revansidda Sabu Kottalagi		7/3/2017	7/5/2017	Dimethoate	4 hrs	vomiting under influence of alcohol	2356			63	43.2	8		NO					
25	Parasappa Kallappa Lokure		7/5/2017	7/7/2017	Chlorpyriphos	2 hrs	vomiting	6907			51.6	9	5		NO					
26	Kamalabai Shantappa Kadakal		7/6/2017	7/14/2017	Lice powder	2hrs 10min	vomiting,abdominal discomfort	6562	4992		32.8	9	4		NO					
27	Neha Malasiddappa Patil		7/17/2017	7/22/2017	Lice powder	2	vomiting	6020		4076	20.4	7.2	4		no					
28	Poornima Hanamanth Walikar		7/25/2017	7/29/2017	Lice powder	1hrs 15min	vomiting	6485			39.6	24	6		NO					
29	Hanamanth Pandu Jamakhandi		8/4/2017	8/8/2017	Dichlorovas	3 hrs 20min	vomiting	6218			44.4	28.8	8		NO					
30	Savita Manohar Rathod		8/14/2017	8/22/2017	Lice powder	8 hrs 30min	vomiting,abdominal discomfort	141			56.4	24	7		NO					
31	Soumya Gurabasappa Busannal		9/3/2017	9/6/2017	Lice powder	1 hrs 15min	vomiting	2497			28.8	7.2	4		NO					
32	Manayya Kenchappa Kembhavi		9/6/2017	9/8/2017	Dimethoate	6 hrs	vomiting	1663			43.2	21.6	6		NO					
33	Shilpa Mallikarjun Hundekar		9/11/2017	9/22/2017	Lice powder	8 hrs 45min	conscious,frothing from mouth bladder incontine	5657		3302	56.4	36	4	YES	YES	3				
34	Wasim Ismail Awati		9/11/2017	9/17/2017	Lice powder	8 hrs 14min	vomiting	5654			50.4	21.6	3		NO					
35	Roopashree Somanath Hadapad		7/4/2017	7/10/2017	Lice powder	2 hrs 30min	drowsy	6058		4471	32.4	9	5		YES		yes			
36	Somilibai Pandu Rathod		8/12/2016	8/13/2016	Monocrotophos	4hrs	vomiting	7901			61.2	28.8	4	YES	NO					
											52.07777778	19.91	5.69							
							SERUM CHOLENESTERASE DAY1	DAY1	DAY 3-5	%										
												19.91	**5.8**	**9**	**10**	**5.3**	**5**		**death total 4**	**1**

	DURATION OF HOSPITAL STAY<5 DAYS		INITIAL ATROPINIZATION AND DOSAGE																													
DEATH>24	<5DAYS	>5DAYS	DAY1/24hrs	0 -2 hrs	3 -4 hrs	5 -6hrs	7 -8 hrs	9 - 24hrs	day1 total	DAY 2	DAY 3	DAY 4	DAY 5	DAY 6	DAY 7	DAY8									GROUP A CHOLENESTERASE				WBC 11000	SEPICEMIA	SR AMYLASE >140	PANCREATITIS
	18	18		9.6	9.6	4.8	4.8	73.2	**102**	15.6	16.8	6																YES			
				12	12	6	7.2	32.4	**69.6**	18	4.8	4.8	2.4													**DAY 1**	**DAY2-DAY5**	**PERCENTAGE**	YES			
				3.6	13.2	3.6	8.4	36	**64.8**	30	14.4	9.6	3.6	4.8											**SER CHOLENESTERASE**							
				7.2	9	7.2	7.2	23.4	**54**	41.4	16.8	6	1.2												**<2500**				YES		97	
				7.2	9.6	4.8	9	35.4	**66**	27.6	45.6	61.2	63.6	38.4											**2500-5000**				YES		500	
				3.6	3.6	7.2	9	11.4	**34.8**	13.2	9.6	1.2													**>5000**				YES		110	
				4.8	4.8	6	7.2	16.8	**39.6**	14.4	3.6	3.6																			12	
				7.2	0.6	1.8			**9**																						122	
				7.2	4.8	14.4	1.2	27	54.6	14.4	3.6	2.4	0.6																YES		11	
				7.2	6	2.4	2.4	21.6	**39.6**	38.4	33.6	26.4	18	10.2	6														YES		77	
				7.2	7.2	6	9.6	30	**60**	67.2	19.2																		YES		240	
				9	7.2	4.8	8.4	19.8	**49.2**	16.8	30	26.4	4.8	7.2	4.8																79	
				8.4	3.6	2.4	3.6	34.8	**52.8**	30	27.6	22.8		3.6																	28	
				7.2	8.4	8.4	13.2	19.2	**56.4**																				YES		128	
				9	9	7.2	10	69.8	**105**	36	10.2	8.6	3		0.6														YES		28	
				4.8	9.6	1.2	1.2	54	**60**	25.2	24	12	2.4	2.4															YES		14	
				6	1.2	3.6	3.6	14.4	**28.8**																				YES		22	
				3.6	9.6	7.2	3	60.6	**84**	3.6	6																		YES		126	
				9.6	9	3	14.4	60	**96**	49.2	32.4	36	33.6	30	36	28.8	28.8	21	14.4	14.4	9.6	9	3.6	3.6	2.4					YES		228	
				3.6	3.6		3.6	15.6	**26.4**	2.4	2.4	3.6																	YES		11	
				1.2	1.2	2.4	1.2	9.6	**15.6**																						16	
				4.8	10.8	14.4	10.8	50.4	**84**	51.6	37.2	13.2																			50	
				7.2	2.4	3.6	1.2	27.6	**42**	22.8	60	7.2																			26	
				10.8	3.6	9.6	3.6	35.4	**63**	4.8	4.8	3.6																			18	
				6	1.2	2.4	7.2	34.8	**51.6**	14.4	9.6	2.4																	YES		16	
				3.6	2.4	2.4	1.2	13.2	**32.8**	16.8	20.4	7.2	7.2	3.6	3.6																28	
				2.4	1.2	3.6	2.4	10.8	**20.4**	3.6	3.6	1.2																	YES		14	
				7.2	2.4	8.4	3.6	18	**39.6**	24	18	9.6	2.4	3.6	1.2	0.6													YES		22	
				7.2	9.6	7.2	9.6	10.8	**44.4**	10.8	3.6																		YES		34	
				4.8	9.6	7.2	2.4	32.4	**56.4**	13.2	10.8	6	2.4																YES		18	
				1.2	4.8	9.6	6	7.2	**28.8**	4.8	6	3.6	0.6																		46	
				7.2	5.4	9.6	1.2	19.8	**43.2**																				YES		17	
				7.2	4.8	5.4	9.6	29.4	**56.4**	15.6	3.6																				118	
				3.6	3.6	4.8	4.8	33.6	**50.4**	79.2	33.6	34.8																	YES		20	
				1.2	4.8	6	7.2	13.2	**32.4**	10.8	4.8	10.8	15.6																YES		128	
				8.4	6	9.6	10.8	26.4	**61.2**	14.4	6																		YES		38	
																														5	**>140 =3**	
3	18	18							**52.077**																							

TOTAL SUBJECTS IN GROUP B 54 OF WHICH 32 SUBJECTS ANALYSED FOR MODERATE TO SEVERE POISONING N=32

	NAME	ipd	DOA	DOD	Compound	Time gap presentation		SR CHOLINESTERASE			INITIAL ATROPINE IN 24 HRS	Atropinized dosage	Time taken for atropinization in hrs	atropine toxicity	Intubation at admission	duration of ventilation in days	Intermediate syndrome	outcome		TIME OF DEATH		DURATION OF HOSPITAL STAY		INITIAL ATROPINIZATION AND DOSAGE GROUP A				
								DAY1	DAY2	DAY3-4								ALIVE	DEATH	DEATH<24	DEATH>24	< 5 DAYS	>5 DAYS	DAY1/24hrs	0 -2 hrs	3 -4 hrs INFUSION	5 -6hrs	7 -8 hrs
																						10	22					
1	Shantabai Somanagou		1/9/2016	1/16/2016	MONOCRP	4 hrs 50mir	Abdominal	462	1253		55.8	14.4	30												14.4	5.4	5.4	5.4
2	Vitthal Sangappa Jinjar		1/18/2016	1/25/2016	DICHLORO	2 hrs 20mir	Vomiting	423	645	1461	97.2	13.2	30												13.2	15	15	15
3	Rajashree Basavaraj Ga		1/26/2016	1/30/2016	DICHLORO	2 hrs	DROWSY,FI	6823	2001		66	33.6	40												33.6	3.6	3.6	3.6
4	Gourawwa Jettappa Ke		3/3/2016	3/18/2016	CHLORGUA	4hrs 45min	Vomiting	105	175	432	66	18.6	35	yes											18.6	5.4	5.4	5.4
5	Nikita Ashok Hosaman		3/13/2016	3/18/2016	LICE POWD	2hrs	Vomiting,A	1833	546		70.2	13.2	30												13.2	5.4	5.4	5.4
6	Ayesha Mustaq Sayad		5/2/2016	5/4/2016	LICE POWD	6 hrs	Vomiting	250	421		84.6	37.2	35	yes											37.2	7.2	7.2	5.4
7	Sanganna Paramappa		6/12/2016	6/19/2016	MONOCRP	3 hrs	Vomiting	8353	7515	7321	88.2	30	40												30	5.4	5.4	5.4
8	Ningappa Channamalla		6/18/2016	6/25/2016	CHLORPYR	2 hrs	Vomiting ,A	137	491	1190	82.8	14.4	25												14.4	4.8	4.8	7.2
9	Chandru Shranappa Ha		8/22/2016	8/26/2016	LICE POWD	7 hrs 42 mi	Vomiting	3575	4564		79.2	13.2	30												13.2	3.6	4.8	6
0	Chandappa Jatteppa P		10/21/2016	10/29/2016	DIMETHOA	5 hrs 34mir	Vomiting	5724	5696		86.4	30	25	yes											30	6	9	12
11	Bheemanna Yallappa B		22-Oct	10/28/2016	DICHLORO	2 hrs 35mir	Vomiting,U	765	335	100	88.2	14.2	30		yes	5	yes								14.2	3.6	4.8	7.2
12	Annapurna Govind Jad		10/22/2016	10/24/2016	MONOCRP	4 hrs 52mir	Vomiting,sv	6650	5973		47.4	9	20												9	1.2	4.8	9.6
13	Parvati Butale Savilkar		11/2/2016	11/9/2016	DIMETHOA	2 hrs	Vomiting ,s	1747	1747		80.4	14.2	25												14.2	5.4	5.4	5.4
14	Shivakanta Bherrappa		11/6/2016	I5/11/16	DICHLORO	2 hrs 23mir	Vomiting ,b	7826	2506		66	9	25												9	1.2	4.8	9.6
15	Danesh Rajashekar Ho		3/9/2017	2I/03/17	DICHLORO	5 hrs 34mir	Vomiting,sv	152	483	1597	72	30	30												30	2.4	3.6	3.6
16	Shaila Yallappa Honam		4/18/2017	4/22/2017	LICE POWD	7 hrs 42 mi	Vomiting,fr	3863	5357		33.6	9	25												9	1.2	2.4	2.4
17	Geeta Santosh Pujari		5/8/2017	5/16/2017	DICHLORO	2 HRS 28M	Breathlessr	158	447		81	14.2	30		yes	8	yes								14.2	1.8	7.2	7.2
18	Parvati Ningappa Mang		5/11/2017	5/19/2017	QUNILPHO	1hrs30min	Vomiting,fr	244	654		57.6	14.2	30												14.2	2.4	4.8	9.6
19	Shridevi Doddappa Ker		5/31/2017	6/5/2017	CHLORPYR	5 hrs 34mir	vomiting,d	125	119	72	85.8	30	40	yes											30	6	9.6	9.6
20	Kumar Duragappa Ban		6/12/2017	6/22/2017	DIMETHOA	4 hrs	vomiting	3286	2469	3286	69.6	14.2	35												14.2	1.8	3.6	7.2
21	Basavanth Appasaheb		6/17/2017	26.06/17	MONOCRP	2 hrs 40mir	Breathlessr	414	1800		78	21	25												21	2.4	4.8	9.9
22	Ashwini Manjunath Ku		7/3/2017	7/9/2017	LICE POWD	4HRS30MIN	UNCONSCI	1407	786		23.4	9	15												9	0.6	1.2	1.2
23	Nagaraj Mallappa Hilli		7/23/2017	7/28/2017	DICHLORO	5hrs	Vomiting,U	226	995		68.4	30	30		yes	4									30	1.8	2.4	3
24	Appasi Balu Biradar		7/29/2017	8/2/2017	DICHLORO	4hrs	vomiting,d	668	824		48	13.2	30												13.2	1.8	1.8	3.6
25	Sangeeta Sangi Jadhav		9/3/2017	9/7/2017	LICE POWD	1hrs52min	Abdominal	2560	1946		54.6	13.2	25		yes	3									13.2	2.4	2.4	3.6
26	Ninganagouda Guralin		8/12/2017	8/17/2017	DICHLORO	4hrs30min	vomiting,d	6227	4603		48	14.2	25												14.2	1.8	2.4	3.6
27	Aisha Monalasab Attar		8/29/2017	9/1/2017	DICHLORO	1hrs54min	vomiting	6770	6902		33.6	9	15												9	1.8	2.4	2.4
28	Appasab Mallappa Bira		9/13/2017	9/20/2017	ROGAR	2hrs21min	vomiting	1016	2315		72.6	14.2	30		yes	4	yes		died						14.2	3.6	9.6	9.6
29	Sangeeta Sunil Rathod		9/13/2017	9/20/2017	ROGAR	2hrs	vomiting	914	631	1538	55.8	13.2	25												13.2	2.4	4.8	9.6
30	Rachappa Gurappa Sab		9/17/2017	9/19/2017	ROGAR	3hrs15min	Vomiting,fr	104	700		90	30	30	yes	yes	3	yes		died						30	6	12	
31	Malleshi Sahebagouda		9/20/2017	9/22/2017	MONOCRPTOPHOS		giddiness,b	135	899		66	9	15		yes	2									9	1.2	2.4	9.6
32	Geeta Natikar		7/30/2017	8/5/2017	DICHLORO	2hrs24min	Vomiting,A	4257	2140		57.6	30	25		yes	3									30	1.8	3.6	3.6
											67.4	**20.3**	**26.9**	**5**	**8**	**4**	**4**		**2**	**0**	2	10	22					
								DAY 1	DAY2-DAY5	PERCENTAGE																		
							DAY 1 SER CHOLENESTERASE																						
							<2500																					
							2500-5000																					
							>5000																					

9 - 24hrs	day1 total	DAY 2	DAY 3	DAY 4	DAY 5	DAY 6	DAY 7	DAY8	GROUP A CHOLENESTERASE				WBC 11000	SEPICEMIA	240	PANCREATITIS
													YES		79	
25.2	**55.8**	19.2	7.2	10.8						**DAY 1**	**DAY2-DAY1**	**PERCENTAGE**			28	
39	**97.2**	26.4	26.4	16.8	8.4	6.6	3.6		**SER CHOLENESTERASE**						128	
21	**66**	43.2	29.4	4.8					**<2500**				YES		28	
31.2	**66**	43.2	43.2	34.2	28.8	28.8	28.8		**2500-5000**						14	
40.6	**70.2**	43.2	28.8	27.6	4.2				**>5000**				YES		22	
27.3	**84.6**	18													126	
42	**88.2**	37.8	23.4	18	14.4	9.6	3.6						YES		228	
50.8	**82.8**	43.2	43.2	28.8	28.8	28.8	12.6								11	
51.6	**79.2**	18	9.6	8.4											16	
29.4	**86.4**	38.2	30	13.2	10.8										50	
63.2	**88.2**	38.4	26.4	22.8	21	9.6									26	
22.8	**47.4**	28.8	9.6										YES		18	
42	**80.4**	39.6	28.8	72	10.8										16	
41.4	**66**	38.4	28.8	16.8									YES		28	
32.4	**72**	43.2	14.4	9.6	28.8	28.8	28.8	28.8					YES		14	
18.6	**33.6**	9.6	7.2	12									YES		22	
50.6	**81**	43.2	43.2	34.2	28.8	28.8	13.2						YES		34	
26.6	**57.6**	28.8	14.4	13.2	21	18	6						YES		18	
30.6	**85.8**	35.3	28.8	16.8									YES		46	
42.8	**69.6**	30	27.6	26.4	6								YES		17	
39.9	**78**	47.4	33	15									YES		118	
11.4	**23.4**	14.4	0.6										YES		20	
31.2	**68.4**	43.2	49.8	19.2									YES		128	
27.6	**48**	28.8	14.4	4.8											38	
33	**54.6**	28.8	21	9	3.6								YES		56	
26	**48**	9.6	7.2	3.6											120	
18	**33.6**	12	3.6	3.6											123	
35.6	**72.6**	41.4	43.2	43.2	43.2	14.4	9.6	22.8					YES		22	
25.8	**55.8**	14.4	15	8.4											56	
42	**90**	43.2	4.8										YES		84	
43.8	**66**	21.6													158	
18.6	**57.6**	36	28.8	28.8	7								YES		69	
	67.4															
													>11000=	3	>140=5	5
													<11000=			
													TOTAL 6			