"ONE YEAR CROSS SECTIONAL STUDY OF PATIENTS WITH ORGANOPHOSPHOROUS COMPOUND POISONING - PREDICTING THE NEED FOR VENTILATOR SUPPORT"

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ENDORSEMENT

This is to certify that the dissertation entitled **"ONE YEAR CROSS SECTIONAL STUDY OF PATIENTS WITH ORGANOPHOSPHOROUS COMPOUND POISONING – PREDICTING THE NEED FOR VENTILATOR SUPPORT"** is a bonafide research work done by **(REG NO. BG0116001).**

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LIST OF ABBREVIATIONS USED

ACh	-	Acetylcholine
AChE	-	Acetyl cholinesterase
APACHE II	-	Acute Physiology and Chronic Health Evaluation II
CNS	-	Central nervous system
CPK	-	Creatine phosphokinase
DEF	-	Tribufos
DFP	-	Disopropylophosphate flourdate
DDVP	-	Dichlorvas
DNA	-	Deoxyribonucleic acid
DPDA	-	Dipropyldopamine hydrobromide
EPN	-	Ethyl p-nitrophenyl phenylphosphonothionate
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
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
OMPA	-	Octamethyl pyrophosphoramide
OP	-	Organophosphorus
p	-	Probability
P=O	-	Phosphorous oxygen bond
P2AM	-	Pralidoxime
PAM	-	Pralidoxime
POP	-	Peradenya Organophosphorus Poisoning
RBCs	-	Red blood cells
SChE	-	Serum cholinesterase
SGOT	-	Serum glutamic oxaloacetic transaminase
SGPT	-	Serum glutamic pyruvic transaminase
TOCP	-	Triorthocresylphosphate
TEPP	-	Tetraethyl pyrophosphate
U/L	-	Units per liter
WHO	-	World Health Organization

ABSTRACT

Background and objectives

Oraganophosphorous compound poisoning is an important indication for emergency ICU admission in most hospitals throughout India. This study has been undertaken to identify the factors which help in predicting the need for ventilator support in patients with organophosphorus poisoning.

Methodology

The present one year hospital based cross sectional study was done on a total of 100 patients admitted with organophosphorus compound poisoning in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from January 2017 to December 2017. On admission clinical examination of the patients was done using POP score and GCS score. Estimation of serum cholinesterase levels was done at the time of admission, on third day and fifth day. Treatment required and the need for ventilator support was predicted with the help of these parameters.

Results

Maximum number of patients were in age group ≤30 years (30%). There was male preponderance with a M;F of 1.78:1. Poisoning was common with farmers (36%). The most commonly consumed compound was chorpyrifos (24%). Majority of the patients (63%) presented between 3 to 6 hours of consumption. Vomiting and breathlessness were the most common symptoms while miosis, and bradycardia were the commonly observed signs. POP score and GCS score revealed mild intoxication in 54% and 42% of the patients. Majority of the patients (64%) showed

an increasing trend of serum cholinesterase. Respiratory failure was the most common complication seen in 8% of the patients. Out of 100 cases, 37% required ventilator support. Mortality was noted in 12% of the patients

Conclusion and interpretation

There is a statistically significant correlation between POP score, GCS score with ventilator support, with complications and outcome. Serum cholinesterase levels showed significant correlation with ventilator support and outcome (on serial estimation only but not first day levels). Also correlation of time of consumption to arrival at hospital to ventilatory support and correlation of large initial bolus doses of atropine required at arrival to need for ventilatory support was statistically significant.

Keywords Organophosphorus compound poisoning; Peradeniya Organophosphorus Poisoning (POP) Scale; Glasgow Coma Scale (GCS); Serum cholinesterase;

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INTRODUCTION

Organophosphorous compounds were first discovered more than 100 years ago and are at present the predominant group of insecticides employed globally for pest control. Examples of organophosphates include insecticides (malathion, parathion, diazinon, fenthion, dichlorvas, chlorpyrifos, ethion), nerve gases (soman, sarin, tabun, VX), ophthalmic agents (echothiophate, isoflurophate), and antihelmintics (trichlorfon).[1,2]

Organophosphorous compound poisoning is found to be a leading cause of death in agricultural countries globally[3-4]. WHO estimates that nearly 3 million pesticide poisoning occur worldwide and cause more than 2,20,000 deaths.[6]

OP compound poisoning is an important indication for emergency admission in most hospitals throughout India[6.] Hospital based statistics suggest that nearly half of the admissions to emergency with acute poisoning are due to Organophosphorous compounds[8].

India being predominantly an agricultural country pesticides and insecticides are abundantly being used[9.]OP compounds are easily available in shops and have resulted in a gradual increase in suicidal & accidental poisoning. Nearly 90% of the poisoning are suicidal with a fatality rate of >10%, 8-10% accidental and <1% Homicidal. Occupational exposure accounts for 1/5th of accidental poisoning with fatalities of <1%.[7]

The organophosphorous compounds are the organic derivatives of phosphorous containing acids. Organophosphorous compounds combine with

esteratic sites of acetyl cholinesterase, that is phosphorylated & phosphorylated esteratic sites undergo hydrolysis. The phosphorylated enzyme is inactive and thus unable to hydrolyze acetylcholine[13].

The biological effects of OP compound are as a result of accumulation of endogenous acetylcholine at sites of cholinergic transmission. This causes disruption of transmission of nerve impulses in both peripheral & central nervous system. Most organophosphorous compounds are readily absorbed through respiratory, oral mucous membrane, GIT mucous and through intact skin, as they are lipid soluble.[13]

Clinical effects are manifested via activation of autonomic and central nervous system and at nicotinic receptors on skeletal muscles[10,11]. The early causes of death are ventricular arrhythmias, CNS depression, seizures or respiratory failure which may be due to excessive bronchial secretions, bronchospasm, pulmonary edema, aspiration of gastric contents, paralysis of respiratory muscles or apnea associated with depression of the medullary respiratory center[15] .Late mortality is associated with respiratory [12,14]and infections like pneumonia, septicemia or complications related to intensive care management [17]. As a treatment modality for this complication ventilator is required.

The Peradenya Organophosphorous Poisoning (POP) scale assesses the severity of poisoning based on symptoms at presentation and is simple to use[19]. There are various other factors which predict the outcome in acute organophosphorous poisoning which include Glasgow Coma Scale (GCS), serum cholinesterase (SChE) level and ECG findings (prolongation of QT interval)[16,18].

Various grading systems proposed suggests that most cases can be managed in the ICU. But this cannot be applied to developing countries like India wherein ICU facilities are rather limited[9].

Hence the present study is undertaken to identify the factors, which help in predicting the need for ventilator support and thus helping to reduce mortality by timely institution of ventilator support[9].

OBJECTIVES

1. To study the clinical profile of patients with organophosphorous compound poisoning.

2. To identify the factors which help in predicting the need for ventilator support in patients with organophosphorous compound poisoning.

REVIEW OF LITERATURE

Historical review

The first account of synthesis of a highly potent antiacetyl cholinesterase compound tetraethyl pyrophosphate(TEPP) was given by Philipe de Clermont in 1854. During the past four decades, more than 35,000 different formulations have come into use as pesticides. Of these, organophosphorous insecticides are possibly the most widely used in the world[21, 22]

In the 1930s, "Jamaican ginger paralysis" affected thousands of people in the Caribbean because a popular remedy was adulterated with the organophosphate triorthocresylphosphate (TOCP)[23].

Modern investigations of organ phosphorous compound date from 1932 when Lange and Krugger recorded the synthesis of dimethyl & diethyl phosphofluoridates. They noted that these compounds caused a persistent choking sensation and blurring of vision. This observation led Schrader of I.G. Farben industries to develop organ phosphorous compound, first as agricultural insecticides and later as potential chemical warfare agents. Consequently, during World War II, several toxic compounds were developed and used as nitrogen gases in Germany[20.]

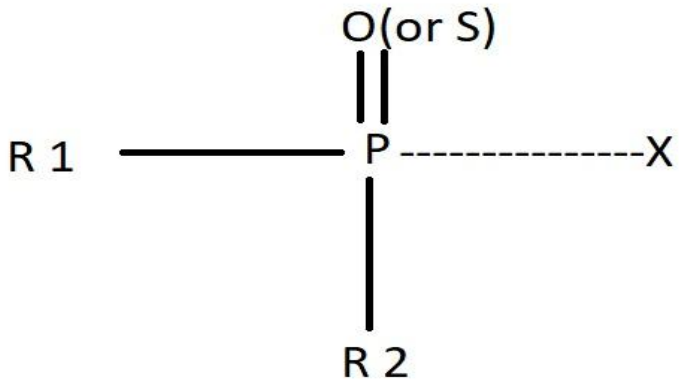
In 1991, these very compounds formed the cornerstone of Iraq's much-dreaded chemical warfare arsenal during the Gulf war.

Organophosphorous compounds first came to India in 1951, to be used as insecticides and in 1962 first Organophosphorous poisoning was reported in India.

CHEMISTRY

Organophosphorous compounds are usually esters /amides of thiol derivatives of phosphoric / phosphonic acids.

The General formula being:



Where,

R1 and R2 are usually simple alkyl or aryl groups.

X known as the “leaving group” may be one of a wide variety of substituted or branched aliphatic, aromatic or heterocyclic groups linked to phosphorous via a bond of some liability usually -O-or -S-.

The double bonded atom may be O or S & the related compound, termed as phosphate or phosphorothioate[7].

CLASSIFICATION

Holmstedt proposed a classification system for organophosphorous compounds that is of pharmacological and toxicological interest.

The compounds are divided into 5 groups with a few relevant examples[20].

Group A: (X: Halogen, Cyanide, and Thiocyanate)

E.g.: Disopropylophosphate flourdate (DFP)

ISO propyl methyl phosphoflouridate (SARIN)

Pinacolyl Methyl phosphoflouridate (SOMAN)

Group B: (X: Alkyl, alkoxy, aryloxy)

E.g.: Forstenon, DDVP, Pyrazoxon

Group C: (X: Thiol or Thiophosphorous Compound)

E.g.: Parathion, Malathion, Azethion, Diazinon, Systox, and Demeton

Group D: (Pyrophosphates and related compounds)

E.g.; TEPP, DPDA, OMPA

Group E: (Quaternary Ammonium Compound)

E.g.: Phospholin

An older more commonly used classification divides these compounds into:

1. Alkyl phosphates (Eg. TEPP, HETP, OMPA, Malathion, Systox, DFP etc).
2. Aryl Phosphates (Eg. Demeton, Parathion, EPN, Chlorothion, Diazinon, etc)

PHARMACODYNAMICS & METABOLISM

These compounds are generally dispersed as aerosols /dusts, consisting of organ phosphorous compound absorbed to an inert finely particulate material. Therefore practically all routes including gastrointestinal tract, skin and mucous membranes following contact with the liquid form, rapidly and effectively absorb these compounds. The lungs also absorb them, after inhalation of the vapors or finely dispersed dusts/ aerosols.

Following absorption they quickly distribute in all tissues, maximum concentration usually being reached in the liver and the kidneys. Lipophilic compounds may reach high concentration in neural and other lipid rich tissues.

Plasma half-life ranges from few minutes to few hours, depending on the compounds and route of administration.

Metabolism occurs primarily by oxidation. Parathion is converted to biologically active compound – "Paroxon" by microcosms in the liver.

Malathion is metabolized to inactive compound more rapidly in higher animals, and consequently is less dangerous to man.

Highly lipid soluble agents such as Chlorfenthion may produce symptoms and signs of cholinergic over activity for an extended period of days to weeks, caused by subcutaneous lipid storage followed by subsequent chronic systemic release afterredistribution. These compounds also cause repeated release after apparently successful management.

Detoxification of the organophosphorous insecticides occurs, either by bio-chemical modification of their structure or by linkage to the binding site without toxicological significance.

Elimination of organophosphorous compounds and their metabolites occur mainly through urine and faeces, with 80-90% of most of compounds being eliminated within 48 hours. A very small portion of compounds and their active forms are excreted unchanged in the urine. Some compounds are known to persist in the body for longer periods.

MECHANISM OF ACTION

Anticholinesterases bind to and inhibit a number of enzymes, yet it is their action on the esterase which is of clinical importance[7].

a. Inhibition of Acetylcholinesterases (AchE)

Acetylcholinesterases (Ach E) are responsible for the hydrolytic cleavage of Acetylcholine (Ach) to choline & acetic acid. Acetylcholine is a neurotransmitter for all postganglionic autonomic fibers, postganglionic parasympathetic fibers, postganglionic sympathetic fibers, neuromuscular junction and some interneuron synapses in the CNS.

A potential reaction causes release of acetylcholine in the presynaptic cleft, most of which is degraded by acetylcholinesterases. Acetylcholine, which is not degraded, binds to the postsynaptic receptors resulting in the generation of an excitatory postsynaptic potential and propagation of the impulse.

Anti-acetylcholinesterase has two sites namely, anionic an esteratic site. Acetylcholine binds to the anionic site on acetylcholinesterases and undergoes hydrolysis in a few seconds. The reversible Anti-acetylcholinesterases combine with acetylcholinesterases at the anionic site and this blocks attachment of the substrate[25].

Irreversible Anti-acetylcholinesterases on the other hand binds to the esteratic site of the acetylcholinesterase and inhibit irreversibly thereby phosphorylating it. This leads to accumulation of acetylcholine at the synapses with initial overstimulation followed by inhibition of synaptic conduction.

Following inhibition, reactivation of the enzyme (acetylcholinesterase) occurs at the rate of 1% per day by slow de novo synthesis of fresh enzyme and also by spontaneous dephosphorylation[26].

The rate of inactivation (phosphorylation) and reactivation (dephosphorylation) depends of the species and the tissue in addition to the chemical group attached accounting for the differences in toxicity[27].

Response to reactivating agents decline with time, a process referred to as 'aging' of the inhibited enzyme. It is a result of loss of an alkyl or alkoxyl group leaving a much more stable monoalkyl or monoalkoxyl phosphoryl acetylcholinesterase[28]. The aged phosphorylated enzyme cannot be reactivated by oximes[29]. In chemical warfare agents like soman, aging occurs rapidly[26].

b. Neuropathy target esterase inhibition:

Neuropathy target esterase inhibition (NTE) followed by its transformation to an aged form is responsible for the organophosphate- induced delayed neuropathy (OPIDN)[30].

PATHOPHYSIOLOGY

Acetylcholine is a neurotransmitter released by the terminal nerve endings of all postganglionic parasympathetic ganglia.

There are two types of this enzyme present in the body; in red blood cells called true cholinesterase and serum pseudocholinesterase[31].

True cholinesteraseare found primarily in nervous tissues and erythrocytes, while pseudo cholinesterase are present in plasma, liver and non-neuronal tissues. Pseudo cholinesterase levels helps to diagnose a case of suspected poisoning[7].

CLINICAL FEATURES

The clinical manifestations of organophosphorous poisoning are a result of cholinergic over activity and can be divided into the effects of over stimulation of the muscarinic, nicotinic and CNS receptors[5].

Muscarinic Receptors	**Nicotinic Receptors**	**Central Receptors**
CVS	**Muscles**	
Bradycardia	Fasciculations	Altered Consciousness
Hypotension	Weakness	Respiratory Depression
GIT	Paralysis	Cheyne– Stokes Respiration
Salivation	Cramps	Dysarthia
Nausea	**CVS**	Tremors
Vomiting	Tachycardia	
Abdominal Pain	Hypertension	
Diarrhea		
Tenesmus		
Feacal Incontinence		
RS		
Bronchorrhea		
Wheezing		
Cough		
Eye		
Miosis		
Lacrimation		
Skin		
Moist		

The clinical diagnosis is based on:

a. History of exposure

b. The presence of several of the above symptoms and signs.

The time interval between the exposure and onset of symptoms and signs varies with the route and degree of exposure. The interval maybe within 5 minutes after massive ingestion and is almost always less than 12 hours. The severity of manifestation varies with the degree of poisoning.

Nambaet al[32] have made a classification of organophosphorous poisoning insecticide which is modified from Grob et al[33] is as follows: -

Latent poisoning: -

No clinical manifestations are seen. Diagnosis based on estimation of serum cholinesterase activity, which is inhibited by 10 to 50%

Mild poisoning: -

The patient complains of fatigue, headache, dizziness, nausea, vomiting, excessive sweating, salivation, abdominal cramps or diarrhea. Serum cholinesterase levels are 20-50% of normal values.

Moderate poisoning: -

The patient complains of generalized weakness, difficulty in talking, muscular fasciculations and miosis. Serum cholinesterase levels are 10-20% of normal values.

Severe poisoning: -

Marked miosis, loss of pupillary reflex to light, muscular fasciculations, flaccid paralysis, and secretions from the mouth and nose, rales in the lungs, respiratory difficulty and cyanosis are seen in patients with severe poisoning. Serum cholinesterase levels are lower than 10% of normal values.

However, this proposed grading has proved unworkable in clinical practice because of many varied clinical criteria in different grades, as well as the difficulty in remembering and applying them in acute clinical situation[5 ,14].

The second classification was proposed by Bardin et al[14] and is as follows: -

Grade 0 Positive history

No signs of organophosphorous poisoning.

Grade 1 Mild secretions

Few fasciculations

Normal level of sensorium.

Grade 2 Copious secretions,

Generalized fasciculations,

Rhonchi, crepitations,

Hypotension (systolic BP <90mmHg)

Disturbed level of consciousness, not stuporous

Grade 3 Stupor,

PaO2 < 50mmHg,

Chest roentgenogram abnormal

This study by Bradin et al showed that patients with grade 3 manifestations on admission were associated with increased requirement for mechanical ventilator. The presence of other complications and increased days of ICU stay have been observed in the above patients.

Following organophosphorous poisoning three well-defined clinical phases[7] are seen:

1. Initial acute cholinergic crisis.
2. The intermediate syndrome
3. Delayed Polyneuropathy: OPIDN-Organophosphorous Induced Delayed Neuropathy. In addition chronic organophosphate induced neuropsychatric disorder (COPIND) can occur.

1. Acute cholinergic phase:

This is the initial phase of acute poisoning resulting in muscarinic and nicotinic effects. The accumulation of acetylcholine at the muscarinic site produces an increase in secretions. Bronchorrhea, salivation, sweating, bradycardia, vomiting and an increase in gastro-intestinal motility (abdominal tightness and cramps) are seen. In the eye, organophosphorous agents cause the diagnostic miosis which results in blurring of vision. The effects of increased acetylcholine at nicotinic sites. Eg: neuromuscular junction, cause muscle fasciculation. Inhibition of acetylcholinesterase

in the brain leads to headache, insomnia, giddiness, confusion and drowsiness. After severe exposure, slurred speech, convulsions, respiratory depression and coma occur.

The mechanism of action of paralysis is depolarization and desensitization blocks induced by acetylcholine at the neuromuscular junctions. Death is likely during this initial cholinergic phase due to effects on the heart like bradycardia, arrhythmias; respiratory failure and depression of vital centers in the brain. Bradycardia may be severe and may progress to heart block. The cholinergic phase usually lasts 24 to 48 hours and constitutes a medical emergency that requires treatment in an ICU[2] .

2. Intermediate syndrome

Senanayake and Karallieda first coined the term "Intermediate syndrome" in 1987[34]. After recovery from the cholinergic crisis, but before the expected onset of delayed polyneuropathy, some patients develop a muscle paralysis, which is described as Intermediate syndrome. This phenomenon has been reported in 20-68% of the patients[35].

The cardinal feature of this syndrome is muscle weakness affecting predominantly the proximal limb muscle and neck flexors. Motor cranial nerve palsies (III to VII and X) also occur. Respiratory muscle weakness leading to respiratory failure could lead to a fatal outcome. Deep tendon reflexes are usually depressed. The intermediate syndrome occurs after recovery from the cholinergic crisis within 24 hours to 96 hours but before the expected onset of the delayed neuropathy, which occurs 2 to 3 weeks after the poisoning.[4]

Complete recovery occurs within 4 to 18 days, if adequate ventilator support is provided. The agents commonly responsible are fenthion, monocrotophos, dimethoate, diazinon and methylparathion[36].

3. Delayed Polyneuropathy

The neuropathy develops following latent periods of 2-4 weeks after the cholinergic crisis. The cardinal symptoms are distal muscle weakness, calf pain preceding the weakness and in some cases paraesthesia in the distal parts of the limbs. Weakness initially appears in the leg muscles causing foot drop, followed by small muscles of the hands. Later it may extend proximally and even involve the truncal muscles. Deep tendon jerks are absent. The prognosis of patients with mild neuropathy is good but those with severe neuropathy are usually left with persistent deficits that are claw hand, foot drop, persistent atrophy, spasticity and ataxias.

Delayed Polyneuropathy is common following exposure to organophosphorous compounds, which have weak anticholinesterase activity Eg. Triorthocresylphosphate.

The occurrence of Delayed Polyneuropathy appears to follow phosphorylation and subsequent aging of an enzyme in axons called as neuropathy target esterase (NTE).The function of this enzyme is not clear yet. It is however present in the brain, spinal cord and the peripheral nervous system. NTE is a membrane bound protein with high esterasecatalytic activity. This enzyme also undergoes ageing[37]. The agents commonly responsible are mepafox and chloropyrifos[38,39,40,41].

Chronic Organophosphate Induced Neuropsychiatric Disorder (COPIND)

Behavioral effects have been documented following acute or chronic organophosphorous poisoning. These include:

a. Impairment of vigilance, information processing, psychomotor speed and memory.

b. Poor performance and perception of speech.

c. Increased tendency to depression, anxiety and irritability.

d. A tendency to faster frequencies and higher voltages in EEG.

Extra pyramidal manifestations (dystonia, rest tremors, cogwheel rigidity and chorea-athetosis)[41] may occur four to forty days after organophosphorous poisoning. Recent studies suggest that Parkinson's disease is a more common in patients who report to have had previous exposure to pesticides[42].

Other effects of organophosphorous intoxication

Altered immunity to infection

In 1974, Bellin and Chow[43]suggested that organophosphorous agents might have an effect on the human immune system. Casaliet al[44] demonstrated that parathion suppressed both the primary IgM and IgG response to sheep erythrocytes in mice.

Newcombe[45] showed an increased incidence of lymphoproliferative disorders associated with impaired natural killer cell and cytotoxic T-cell function.

Murray et al[46] reported influenza like symptoms in 23 patients after occupational exposure to organophosphorous compounds.

Changes in metabolism and endocrine activity

In animal experiments, changes in the diurnal pattern of plasma ACTH have been reported following organophosphorous poisoning.[47] Nicotinic receptors also function in brain pathway that increases the release of several pituitary hormones including vasopressors, ACTH and prolactin. In man, nonketotic hyperglycemia may occur[48,49].

Effects on the CVS

Kiss and Fazekas[50] reported QT prolongation along with ST-segment, T-wave anomalies and other forms of arrhythmias. Recurrent ventricular tachycardia with the torsade de pointes phenomenon was seen.

Complete atrioventricular block may occur[51].QTc prolongation indicates poor prognosis and a higher incidence of respiratory failure[52].

Respiratory system

Respiratory arrest is a common terminal manifestation of OP poisoning. It can be recalled that muscarinic action produces increased bronchial secretions and bronchoconstriction. On the other hand nicotinic action produces intercostal and other respiratory muscle weakness leading to respiratory paralysis[55].

Effects on Reproduction

There is a report of termination of pregnancy following organophosphorous poisoning during the first trimester[53]. In experimental animals, organophosphorous poisoning during pregnancy causes pre and post natal death and congenital abnormalities like vertebral deformities, limb defects, polydactyly and cleft palate[55].

Effects on other systems

- Eyes: myopia and pigmentary degeneration of retina.
- Joints: arthritis
- Interference with mitochondrial oxidative metabolism[32,55].

Changes in metabolism and endocrine activity

Transient hyperglycaemia and glycosuria are often found in severe OP poisoning. Absence of ketone bodies differentiates it from diabetic coma, except for coma in diabetic patients due to hyperosmolarity from excessive blood glucose[55].

GI effects

After ingestion of organophosphate compound, the common initial symptoms may be increased salivation, nausea, vomiting, abdominal tightness and cramps. Other muscarinic manifestations include diarrhea, tenesmus and fecal incontinence[55].

Temperature Regulation

Several studies have noted derangement of temperature regulation in the form of hypothermia (incidence 7%). Some patients may experience fever lasting for many days, a biphasic response[32,55].

Vocal card paralysis

In few patients vocal card paralysis was reported within 2 days[32,55].

DIAGNOSIS

Acute cholinergic crisis[55-58]

- History of ingestion of the compound
- Signs and symptoms
- Inhibition of cholinesterase activity
- Improvement after atropine and oxime therapy

Organophosphate poisoning is generally diagnosed clinically based on the characteristic symptoms and the history of exposure to OP agents. When diagnosis is not evident, a depressed serum or RBC cholinesterase level (<50%)is helpful. If OP poisoning is suspected, therapy should never be withheld pending for confirmation of lab values[55-58].

Intermediate syndrome

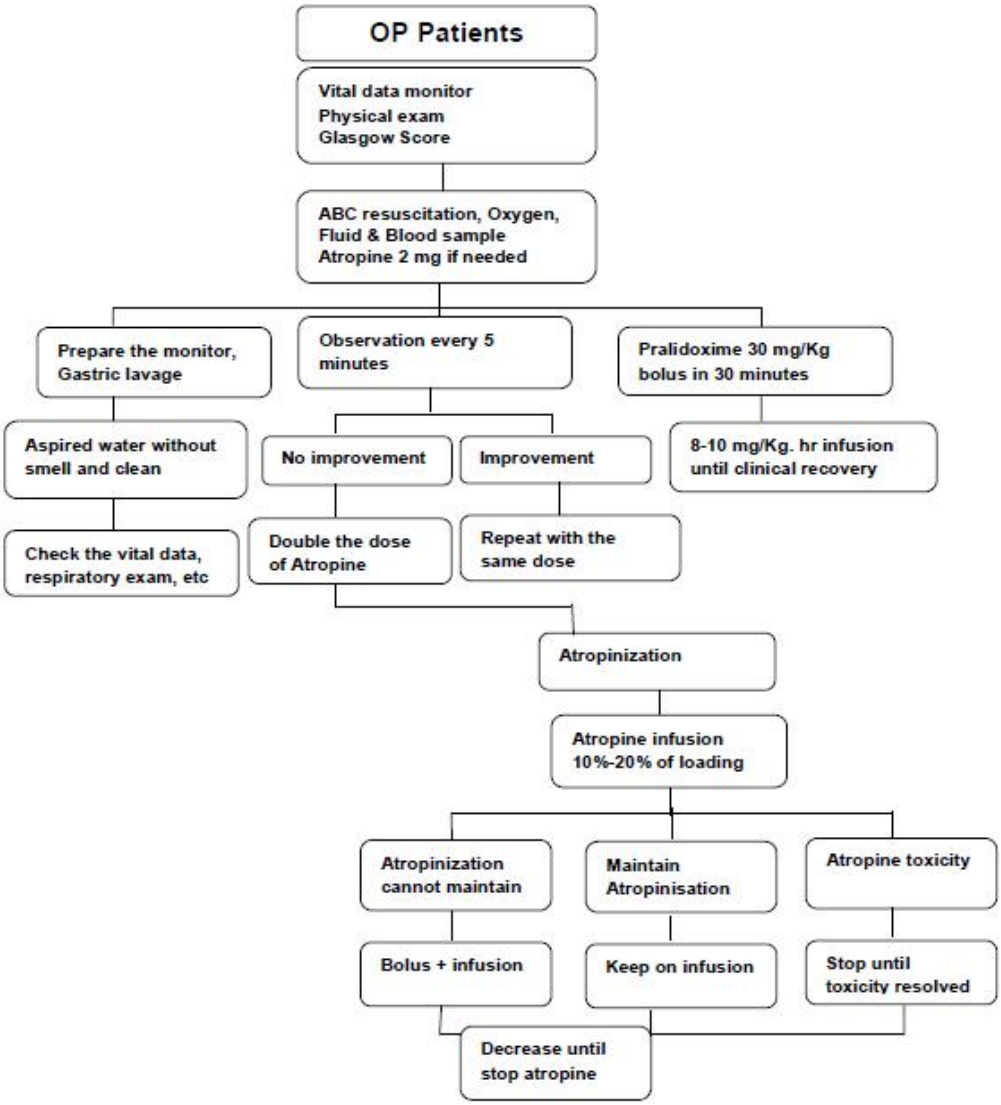
The diagnosis is clinical and should be suspected when a patient who is recovering from the cholinergic crisis develops respiratory difficulty. The presence of muscle weakness in the absence of muscle fasciculations and other cholinergic features differentiates it from cholinergic crisis. The early onset of muscle weakness distinguishes the IMS from the delayed polyneuropathy, which appears 2-3 weeks after poisoning[55-58].

Delayed polyneuropathy

History of intoxication with OP agents and the time of onset and distribution of muscle weakness differentiate from other causes of acute polyneuropathy[55-58].

TREATMENT

Figure 2.Treatment Protocol for OP Poisoned Patient[59]



All patients should be managed as emergencies in hospital.

A. Acute Cholinergic Crisis:

Treatment is based on the following principles:

a) Minimizing further absorption of the insecticide.

b) Pharmacologically countering the effects of the poison.

c) Maintaining vital functions.

Successful management requires rapid and simultaneous implementation of the above principles.

Decontamination

First aid measures should include removal of patient from the contaminated environment, removal of contaminated clothes and washing of the skin and eyes. The person is to be washed with copious amount of water and soap (OPs are hydrolyzed in an aqueous solution at high ph)[59].

Gastric lavage is most effective within 30 minutes of ingestion but is advised also at the time of admission after taking necessary precautions to protect the airway. If the patient is semiconscious/unconscious Ryle's tube aspiration can be done. Activated charcoal may be administered to reduce further absorption from the stomach.

Attention should not be diverted from ABC of Cardiopulmonary resuscitation. Seizures are to be controlled by appropriate measures. Two IV lines should be secured and blood samples for hematological and biochemical analysis should be collected.

ECG should be recorded. Laboured breathing, sweating, pin point pupil would suggest OP/carbamate poisoning[59].

Atropine:

Treatment with anticholinergic medication is still the mainstay of treatment and should be started as soon as the airway has been secure.

Atropine acts as a physiological antidote, effectively antagonizing the muscarinic-receptor-mediated action. It has virtually no effect against the peripheral neuromuscular dysfunction and subsequent paralysis induced by organophosphorous agent.

Atropine should be started immediately and should not be withheld even if oxygen is not available. There is no substantial evidence that giving atropine to a cyanosed patient would cause harm.[59]

A recommended dose is 2-4 mg intravenous, repeated at interval of 5-10 minutes initially and continued until signs of atropinisation (dry mucous membrane, dilated pupils, flushing of skin and a heart rate of > 100 beats/ minute) appear. Atropine therapy should be maintained until there is complete recovery.

Infusion of atropine are used in some centers in dose of 0.02-0.08 mg/kg/hr[60]. Infusion of atropine has produced significant reduction in mortality in some centers when compared to conventionally intermittent therapy[61]. A heart rate exceeding 140 beats/minute should be avoided. ST-segment abnormalities in the ECG may be induced by large doses of atropine. These may be corrected with propronalol, eliminating any need to reduce the rate of administration of atropine. Atropine crosses

the blood brain barrier and may cause severe toxic effects such as convulsions, psychosis and coma[62].

Criteria of Atropinisation

There are no comparative studies on markers for adequate atropinisation. Since patients usually die from respiratory or circulatory failure; air entry on chest auscultation, heart rate and blood pressure were given more importance than dilatation of pupil, rise in body temperature, dryness of mouth/skin.[59,63]

Target ends points for atropine therapy:

- Clear chest on auscultation with no wheeze
- Heart rate > 80 beats / min
- Pupil no longer pin point
- Dry axilla
- Systolic blood pressure > 80 mm of Hg.[59]

Atropine toxicity

Confusion, agitation, hyperthermia, ileus, tachycardia etc would suggest over atropinisation which would necessitate discontinuation of the atropine infusion, followed by frequent observation. When they settle down the infusion is to be started at 70- 80% of the previous rate. Hyperthermia is a serious complication in hot wards which needs prevention. To avoid preservative toxicity atropine should be reconstituted in normal saline and used[59].

Transfer to ICU

Atropine and resuscitation measures are to be continued and vitals recorded while transferring to ICU. Patients with the following criteria may need ventilator support[64].

- History of intake of large amount of poison
- Copious secretions
- Disturbed level of consciousness
- Signs of hypoventilation or respiratory obstruction by secretions

Hence intubation and ventilation facilities should be available in the ICU. A monitor should be connected to the patient and vital parameters including oxygen saturation are to be observed and recorded in OP observation sheet[59].

Guidelines for ventilator support[59]

I. Respiratory Gas Tensions
   i. Direct indices
      a. Arterial Oxygen Tension < 50 mm Hg on room air
      b. Arterial CO_2 Tension > 50 mm Hg in the absence of metabolic alkalosis
   ii. Derived Indices
      a. PaO_2 / FiO_2< 250 mm of Hg
      b. PA –aO_2 (Pulmonary arterial – alveolar O_2 gradient) > 350 mm of Hg
      c. Vd / Vt> 0.6

II. Clinical – Respiratory rate (RR) > 35 breaths / min

III. Mechanical Indices

i. Tidal Volumes <5 ml/kg

ii. Vidal capacity <15 ml/kg

iii. Maximum inspiratory force <- 25 cm of H_2O

Glycopyrrolate:

This is a quaternary ammonium compound can be used as an alternative to atropine.

The advantages of Glycopyrrolate over atropine are: -

a) Better control of secretions[65] .

b) Less tachycardia[66].

c) Fewer CNS side effects[67].

Hence it's use is recommended when there is copious secretion as an adjunct to atropine or when features of atropine toxicity like delirium etc are confused with CNS effects of poison or when atropine is not available. 7.5 mg of glycopyrrolate in 200ml of saline is started as infusion and is titrated to the desired effects of dry mucus membranes[59] .It has also been given at a dose of 0.2mg IM stat and repeated 6thhrly if required.

Oximes:

The observation that oximes reactivates phosphorylated AchE more rapidly than spontaneous hydrolysis led to the development of Pralidoxime (Pyridine-2-aldoxime methyl chloride, PAM) and later Obidoxime.

The reactivating action of pralidoxime is most marked at the skeletal neuromuscular junction. It acts by reactivation of the inhibited phosphorylated enzyme to free the active form. Its dose is 1 gm 8 hourly given intravenous in 250ml normal saline over 30 minutes. It has no muscarinic effect. It has a short half-life of 1.2 hours when given intravenous[68] and does not cross the blood brain barrier[20].

PAM should be administered as early as possible, at least within 4-36 hours as regeneration of AchE depends primarily on the life span of the erythrocytes when aging of the enzyme has occurred[26].

PAM is available as the chloride iodide, mesylate and methyl sulfate salts. The chloride salt is more stable than iodide in dry state and is preferred for intramuscular use.

The major pharmacological action of oximes is to reactivate AchE by removal of phosphate group bound to the esteritic site[5]. This action occurs shortly after poisoning and inhibition of the enzymes, after which the enzyme ages and becomes more firmly bound to esteratic site[69].Oximes should be given as soon as possible before aging takes place. They are most effective if given within 6 hours of poisoning, but beneficial response is seen upto 24 hours of poisoning.

The therapeutic effects of oximes seemed to depend on the plasma concentrations of the organophosphorous agent with the benefit being, minimal at high concentrations of organophosphorous in the blood. Pralidoxime does not cross the blood-brain barrier whereas obidoxime does.

Paradoxically high doses of pralidoxime may cause neuromuscular block and other effects including inhibition of AchE[20].

High frequencies of cardiac arrhythmias were observed in patients who received high cumulative doses of atropine and Obidoxime.

Ventilation

This is the most useful advancement made in the management of OP poisoning. Indications for ventilation have already been discussed. Regular and close observation during the initial course will guide us regarding when to ventilate. Succinylcholine is to be avoided (since it requires serum ChE for its metabolism). Non-depolarising neuromuscular blocking agents require higher doses to show effect[59].

Diazepam:

Some reports have indicated that benzodiazepines are useful as antidotes in poisoning by anticholinesterases[70]. This appears to counteract some aspects of CNS derived symptoms and also increase therapeutic effects of atropine and PAM.

Diazepam is used to treat convulsions after organophosphorous poisoning and in the support of ventilatory care.

Fluoride:

Fluoride and atropine combination produces a greater antidote effect than atropine alone[26].

It was noted that increased cholinesterase levels were observed in workers in a plastic factory handling fluoride compounds.

Magnesium:

Kiss and Fazekas[50] reported that ventricular premature contractions were successfully eliminated with intravenous magnesium sulfate. Magnesium was thought to counteract direct toxic inhibitory effect of organophosphates on sodium-potassium ATPase.

Phenothiazines:

The use of phenothiazines in the management of organophosphorous poisoning is controversial. Diazepam has proven to be satisfactory and popular alternative[7].

Respiratory stimulants:

Respiratory stimulants should not be used in the treatment of organophosphorous poisoning in humans, particularly, in view of the bronchospasm, neuromuscular block and convulsions that are associated with intoxication[71].

Other measures:

Dialysis of blood against activated charcoal (hemoperfusion) is effective in demeton-S-methyl sulphoxide; dimethoate and parathion poisoning[72].Prompt improvements have been reported following repeated injections of purified human cholinesterase [25].

Corticosteroids, camphor, potassium chloride and vitamin C have been used with varying degree of success. However, all these regimens need further evaluation[7].

Management of Intermediate Syndrome[7]

Prompt and effective management of respiratory insufficiency is the cornerstone of treatment of Intermediate syndrome.

Patients should be observed for early signs of respiratory failure and facilities for ventilatory care should be made available. Frequent blood-gas analyses are useful in monitoring and weaning from ventilatory support.

Diazepam in 10 mg intravenous doses may be useful in anxious or restless patients on ventilator.

Management of Delayed Neuropathy[7]

No specific drug therapy has proved useful. The muscle weakness benefits from regular exercise and physiotherapy.

Complications

Complications resulting from organophosphorous poisoning occur in about 43% of cases with acute intoxication[73,75].

Death can often occur early (within 24hours) in untreated cases and upto 10 days in hospital with optimal management[74].

Early deaths are due to CNS depression, seizures, and ventricular arrhythmias (Eg. Torsade de pointes) or respiratory failure due to excessive bronchial secretions, pulmonary edema, aspiration pneumonia, respiratory muscle paralysis or respiratory center depression[15].

Late mortality is caused by respiratory failure[12, 75]associated with infection (pneumonia, septicemia) or ventilator related complications.

There are various studies in which respiratory failure was the commonest complication encountered following acute organophosphorous poisoning[76,77].

The pathogenesis is multifactorial and related to aspiration of gastric contents, excessive secretions in the airways, pulmonary infections, pneumonia, septicemia and development of ARDS[12].

Respiratory consequences of muscarinic overstimulation including rhinorrhoea, bronchorrhea, bronchoconstriction and laryngeal spasm may contribute to respiratory failure. These are often combined with nicotinic effects such as respiratory muscle weakness and paralysis (including paralysis of tongue and nasopharynx).

Central depression of respiratory center occurs following cholinergic overstimulation of synapses in the brain stem and is a prominent cause of hypoxia, respiratory failure and death in the early period of acute organophosphorous poisoning[15].

Peripheral neuromuscular block producing respiratory muscle weakness and paralysis as well as the recently described intermediate neuropathy[34] contributes to the development of respiratory insufficiency at a later stage.

Sudden cardiovascular collapse is often the first indication of unsuspected or incipient respiratory failure, a presentation that is associated with a high mortality[12].

The development of pneumonia is the most important cause of delayed respiratory failure after organophosphorous poisoning and occurs in upto 43% of the patients[12,73,75]. Upto 80% of patients with pneumonia had respiratory failure; majority of these could be diagnosed within 96 hours of poisoning[12].

Inadequate or delayed atropinisation appears to be one of the principle reasons for the development of pneumonia[75] and emphasizes the importance of skilled medical assessment and treatment at an early stage after poisoning.

Prevention[7]

Preventive measures should be considered at all the levels of the chain of insecticide movement through the environment-formulation manufacture, mixing application and disposal. Psychiatric counseling should be done for prevention of second episode and general counseling and drug therapy for depression.

Strict guidelines should be adopted during transport and storage to prevent contamination of food, clothing, drugs, toys, cosmetics and furnishing.

METHODOLOGY

This study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2017 to December 2017.

Study design and duration

A one year hospital based cross sectional study.

Study period

The present study was conducted from January 2017 to December 2017.

Place

The present study was conducted in Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum a tertiary care teaching hospital attached to Jawaharlal Nehru Medical College, Belgaum.

Source of Data

The study comprised of patients with organophosphorus compound poisoning admitted in the wards and ICUs of the Department of General Medicine

Sample size

A total of 100 patients with organophosphorus compound poisoning were studied.

Sampling procedure

The total number of organophosphorus poisoning cases in the last three years was 404 according to the Department of Medical Records, KLES Dr. Prabhakar Kore Hospital and Research Centre, Belgaum. Considering 80% of the annual average number of cases with poisoning during the last three years, the sample size was determined as 107 cases. Hence 100 cases of organophosphorus compound poisoning were included in the study.

Selection criteria

Inclusion

- Patients above 18 years of age
- Patients with history of organophosphorous compound ingestion and coming within 24 hours of consumption to hospital.

Exclusion

- Patients with mixed poisoning (organophosphorous compound and other compounds)
- Patients with chronic lung diseases.
- Patients with lung disorder secondary to cardiac disease.

Ethical clearance

Prior to the commencement, the ethical clearance was obtained from Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belgaum.

Informed Consent

All the patients fulfilling selection criteria were explained about the nature of study and a written informed consent was obtained before enrollment (Annexure I). In case of patients with altered mental status, the relatives of the patients were briefed about the nature of study and written informed consent was obtained.

Method of collection of Data

Demographic data such as age and sex were recorded. Patients / relatives were interviewed for chief complaints and past history. History of organophosphorus compound including type of organophosphorus compound, quantity consumed, route of exposure, intention of consumption, were noted as reported by either the patient or informant. A thorough physical, clinical and systemic examination was carried out based on Peradeniya Organophosphorus Poisoning (POP) Score and Glasgow Coma Score (GCS). These findings were recorded on a predesigned and pretested proforma (Annexure II).

Investigations

The selected patients underwent the following investigations:

- Serum cholinesterase levels
- Electrocardiogram

Study variables

The patients were evaluated for following study variables:

1. PeradeniyaOrganophosphorus Poisoning (POP) Scale

The POP scale assesses the severity of the poisoning based on the symptoms at presentation and is simple to use.[17]

- *Peradeniya Organophosphorus Poisoning (POP) Scale Clinical criteria Score*

Variables	Findings	Score
Respiratory Rate	Normal (<20 /minute)	0
	Tachypnoea (>20 / minute)	1
	Tachypnoea (>20 / minute with central cyanosis)	2
Heart rate	Normal (>60 /minute)	0
	Bradycardia (41-60 /minute)	1
	Bradycardia (<40 /minute)	2
Level of consciousness	Conscious and rationale	0
	Impaired response to verbal commands	1
	No response to verbal command	2
Fasciculation	None	0
	Present - Generalised or continuous	1
	Both – Generalised and continuous	2
Pupil size	> 2 mm	0
	< 2 mm	1
	Pin point	2
Seizures	Absent	0
	Present	1

Interpretationof POP score

- Mild poisoning - A score of 0 to 3
- Moderate poisoning - A score of 4 to 7
- Severe poisoning - A score of 8 to 11

2. Glasgow Coma Score (GCS)

The severity of organophosphorous poisoning was also determined by the Glasgow Coma Score (GCS)

Variables	Findings	Score
Eye Opening	To eye opening	1
	To speech	2
	To pain	3
	Spontaneously	4
Best verbal response	None	1
	Incomprehensible sounds	2
	Inappropriate words	3
	Patient confused	4
	Patient oriented	5
Best motor response	None	1
	Extensor response to painful stimulus	2
	Flexion to painful stimulus	3
	Withdraws from pain	3
	Localizes to pain stimulus	5
	Obeys commands	6

Interpretation of GCS Score

- Mild poisoning: A score of 12 to 15
- Moderate poisoning : A score of 8 to 11
- Severe poisoning: A score of ≤7

Serum cholinesterase levels

The estimation of serum cholinesterase levels was done by PCHE method using Flex reagent cartridge manufactured by Roche company, Model- COBAS 6000. Pseudo cholinesterase levels were estimated at the time of admission and during follow up of the patient on the third day and fifth day of hospital stay. The serum cholinesterase levels between 3165 to 6333 U/L were regarded as normal.[98]The trend of change in serum cholinesterase levels was also noted.

Ventilatory support

The treatment required and the need for ventilator support was estimated on admission with the help of these parameters. Follow- up was done on Day 3 and Day 5 to assess the accuracy of our parameters for the need for ventilator support

Complications

Patients were monitored for complications during the follow-up in the hospital.

Outcome

Patients were evaluated for the outcome as survived and expired

Statistical analysis

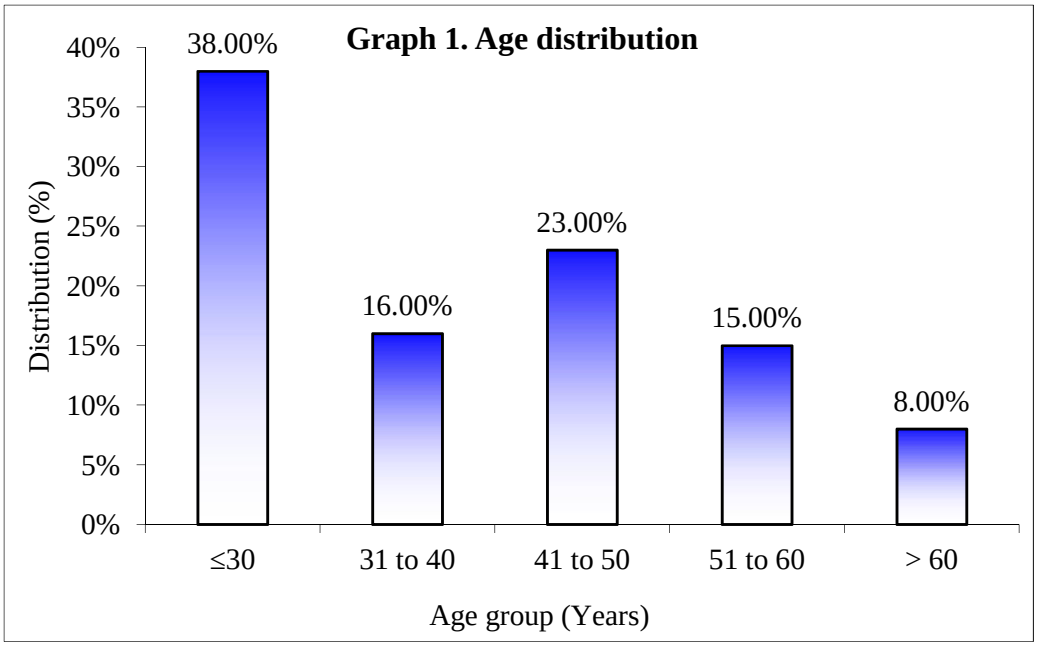
Thedataobtained was coded and entered into Microsoft Excel Worksheet (Annexure III). Data was analysed using SPSS statistical software version 20.0 The categorical data was expressed as rates, ratios and proportions and comparison was done using either chi-square test or Fisher's exact test. The continuous data was expressed as mean ± standard deviation (SD) and comparison was done using independent sample 't' test. A probability value ('p' value) of less than or equal to 0.05 was considered as statistically significant.

OBSERVATIONS AND RESULTS

The present one year hospital based study titled “One year cross sectional study of patients with organophosphorous compound poisoning – predicting the need for ventilator support” was carried out in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. During the study period from January 2017 to December 2017, a total of 100 patients admitted with organophosphorous compound poisoning were studied. The findings/observations and final results are tabulated as below.

Table 1. Age distribution

Age group (Years)	Distribution (n=100)	
	Number	Percentage
≤30	38	38
31 to 40	16	16
41 to 50	23	23
51 to 60	15	15
> 60	8	8
Total	**100**	**100.00**

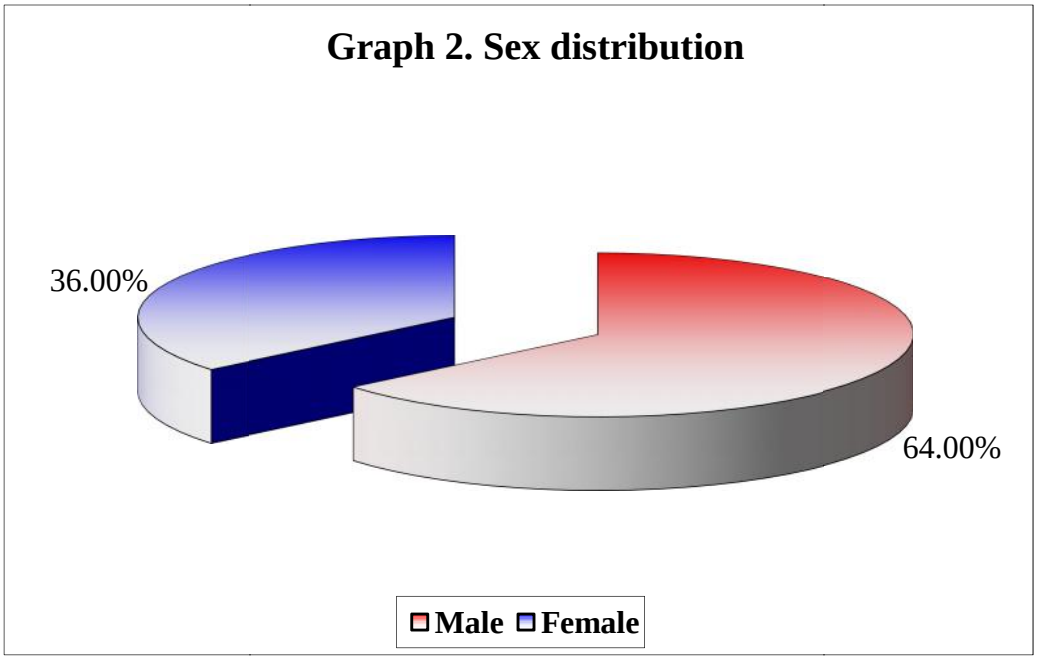


Patients age ranged from 18 to 72 years. Maximum number of caseswere in the age group of ≤30 years i.e; 38 patients (38%), between 41-50 years 23 cases (23%), 31-40 years 16 cases (16%), 51-60 years 15 cases (15%)and only 8 cases (8%) in the age group >60 years.

Inference: More number of cases were in the age group of ≤ 30 years

Table 2. Sex distribution

Sex	Distribution (n=100)	
	Number	Percentage
Male	64	64
Female	36	36
Total	**100**	**100.00**

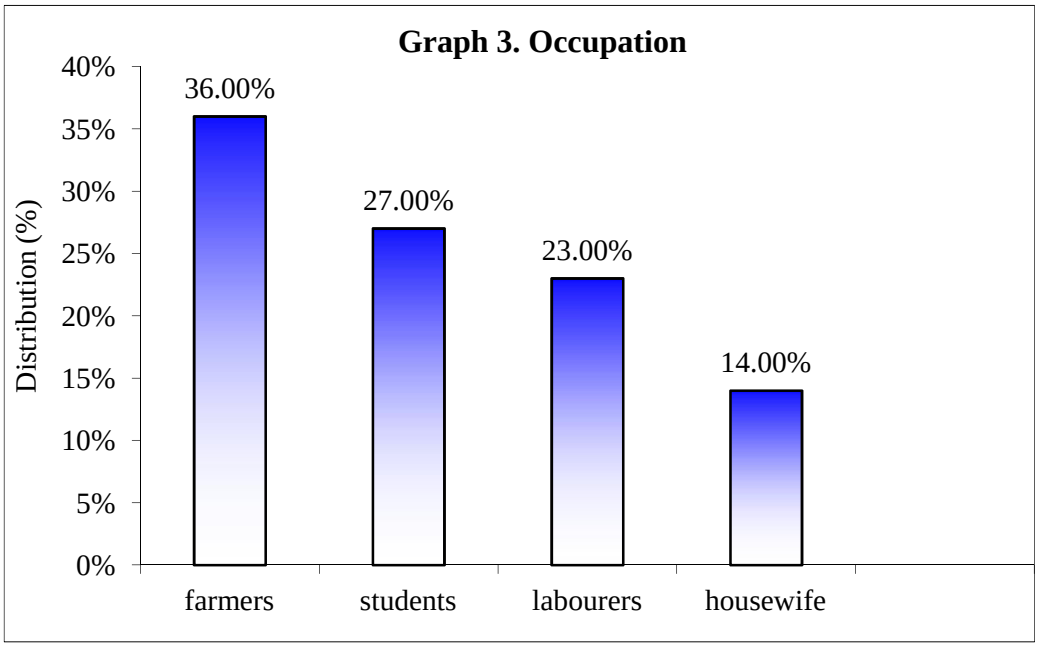


Out of 100 patients, 64 were males (64%) and 36 patients (36%) were females. accounting to a M:F ratio of 1.78:1

Inference: Male preponderance was observed.

Table 3. Occupation

Occupation	Distribution (n=100)	
	Number	**Percentage**
Farmers	36	36
Students	27	27
Labourers	23	23
Housewife	14	14
Total	**100**	**100.00**



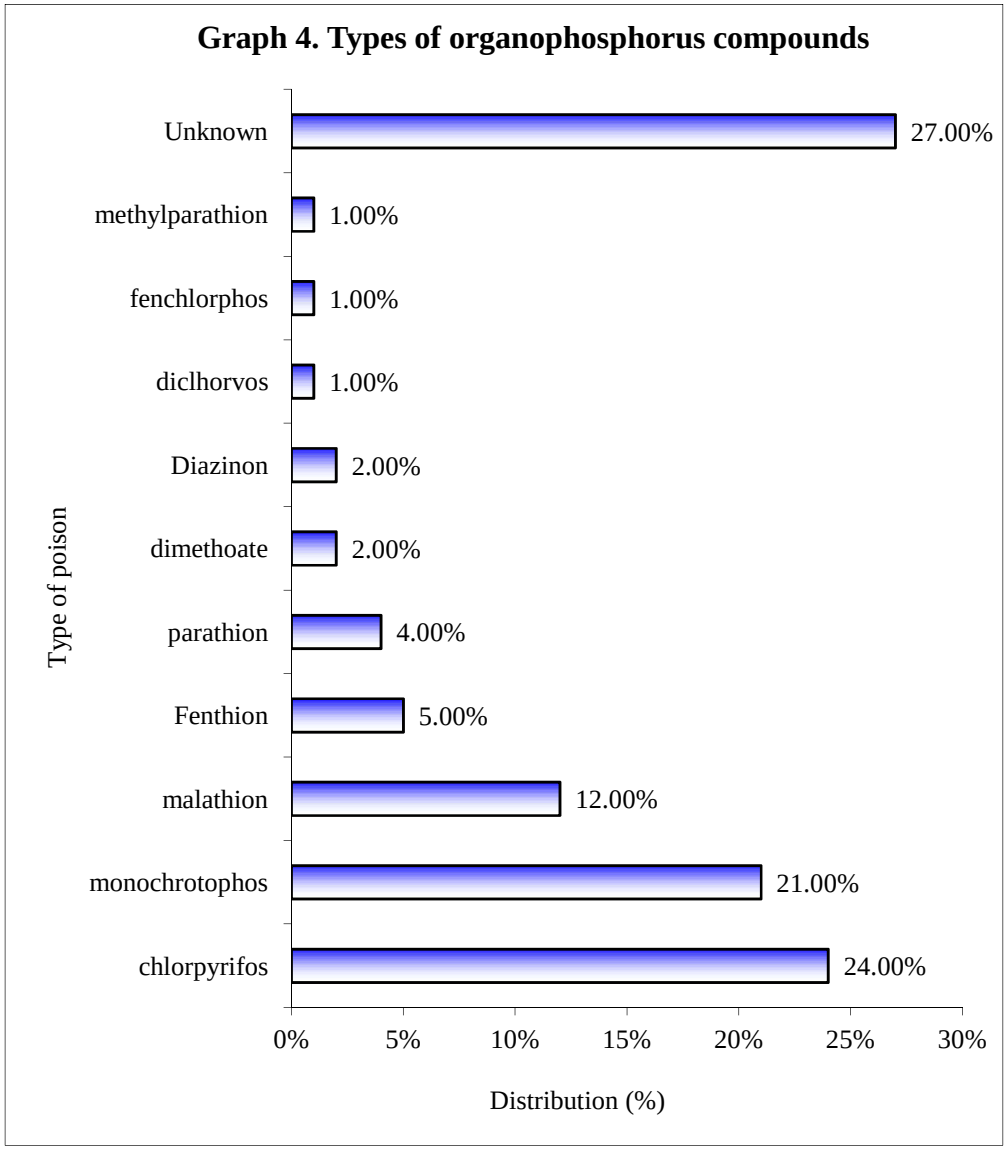
Out of 100 patients studied, the commonest occupation with poisoning was seen in farmersi.e; 36 cases (36%), followed by students 27 cases (27%), labourers23 cases (23%) and housewives 14 cases (14%).

Inference: Poisoning was more common with farmers.

Table 4. Types of organophosphorous compunds

Type of poison	Distribution (n=100)	
	Number	**Percentage**
Chlorpyrifos	24	24
Monochrotophos	21	21
Malathion	12	12
Fenthion	5	5
Parathion	4	4
Dimethoate	2	2
Diazinon	2	2
Dichlorvas	1	2
Fenchlorophos	1	1
Methylparathion	1	1
Unknown	27	27
Total	**100**	**100.00**

Graph 4. Types of organophosphorus compounds



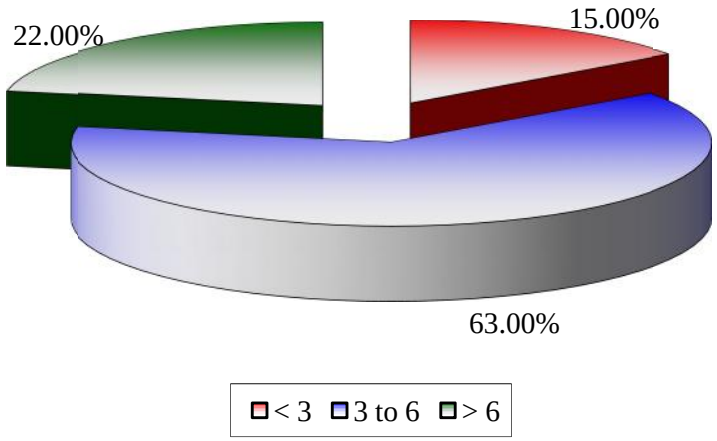
In the present study we observed that maximum number of patients had consumed chlorpyriphosi.e;24 patients (24%), followed by21 patients (21%) monocrotophos, 12 patients (12%) malathion, 5 patients (5%) fenthion, 4 patients (4%) parathion, 2 patients (2%) dimethoate, 2 patients (2%) diazinon, 1 patient (1%) dichlorovas, 1 patient (1%) fenchlorophos, 1 patient(1%) methylparathion and in 27patients (27%) the compound was unknown.

Inference : In 27 patients (27%) the compound was unknown. In remaining patients the commonest compound observed was chlorpyriphosin 24 patients (24%).

Table 5. Time interval from consumption of organophosphorus compound to arrival at hospital

Time (hours)	Distribution (n=100)	
	Number	Percentage
< 3	15	15
3 to 6	63	63
> 6	22	22
Total	**100**	**100.00**

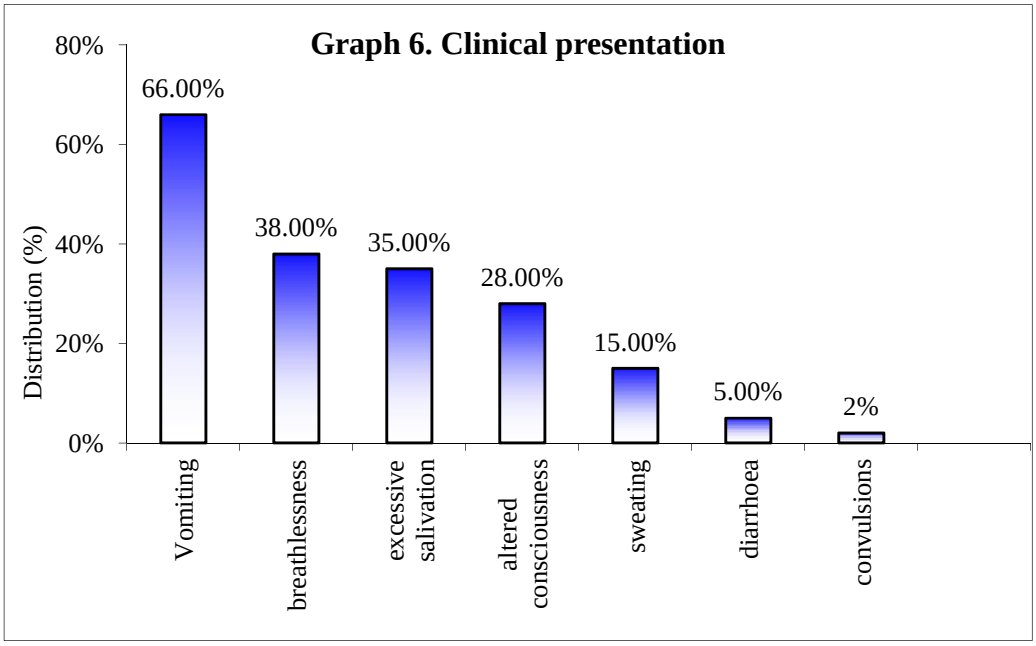
Graph 5. Time interval from consumption of organophosphorus compound to arrival at hospital



We observed that majority of the patients i.e;63 (63%) arrived between 3 to 6 hours of consumption or organophosphorous compound, 22 patients (22%) arrived after 6 hours and 15 patients (15%) arrived within 3 hours.

Table 6. Clinical presentation

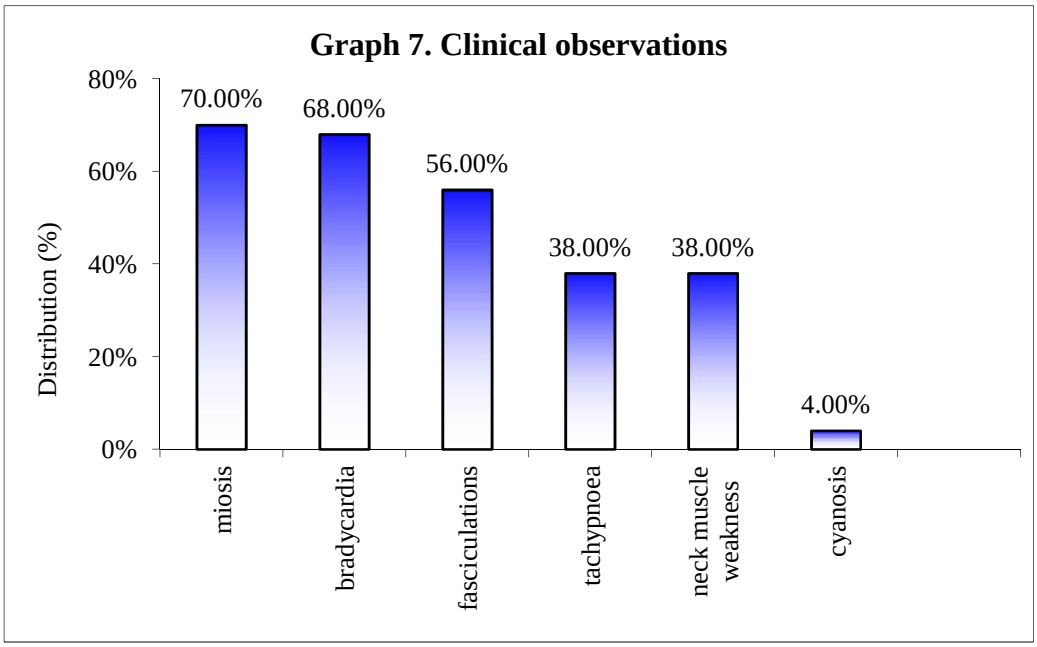
Presenting Symptoms	Distribution (n=100)	
	Number	Percentage
Vomiting	66	66
Breathlessness	38	38
Excessive Salivation	35	35
Altered consciousness	28	28
Sweating	15	15
Diarrhoea	5	5
Convulsions	2	2



In our present study, majority of the patients presented with one or the other symptoms of poisoning. Majority i.e; 66 patients (66%) had vomiting, followed by breathlessness 38 patients (38%), excessive salivation 35 patients (35%), altered consciousness 28 patients (28%), sweating 15 patients (15%),diarrhoea 05 patients (5%) and seizures 2 patients (2%).

Table 7. Clinical observations

Clinical Signs	Distribution (n=100)	
	Number	Percentage
Miosis	70	70
Bradycardia	68	68
Fasciculations	56	56
Tachypnoea	38	38
Neck muscle weakness	38	38
Cyanosis	04	04



In majority of our patients following clinical observations were made- miosis 70 patients (70%), bradycardia 68 patients (68%), fasciculations 56 patients (56%), tachypnoea 38 patients (38%), neck muscle weakness 38 patients (38%) and cyanosis 4 patients (4%).

Table 8.Clinical presentation based on Peradeniya organophosphorus poisoning (POP) score

Variables	Findings	Distribution (n=100)		Score
		Number	Percentage	
Respiratory Rate	Tachypnoea	40	40	1-2
	Normal	60	60	0
	Total	**100**	**100.00**	
Heart rate	Bradycardia	68	68	1-2
	Tachycardia	10	10	0
	Normal	22	22	0
	Total	**100**	**100.00**	
Level of consciousness	Conscious and rationale	72	72	0
	Impaired response to verbal commands	13	13	1
	No response to verbal command	15	15	2
	Total	**100**	**100.00**	
Fasciculation	None	44	44	0
	Present - Generalised or continuous	37	37	1
	Both - Generalised and continuous	19	19	2
	Total	**100**	**100.00**	
Pupil size	> 2 mm	30	30	0
	< 2 mm	42	42	1
	Pin point	28	28	2
	Total	**100**	**100.00**	
Seizures	Present	2	2	1
	Absent	98	98	0
	Total	**100**	**100.00**	

The clinical presentation based on POP scoring system is depicted in the above table.

Table 9. Severity of poisoning based on POP score

Severity	POP score	Distribution (n=100)	
		Number	Percentage
Mild	≤3	54	54
Moderate	4 to 7	28	28
Severe	8 to 11	18	18
	Total	**100**	**100.00**

In our study we observed that 54 patients (54%) had mild intoxication, 28 patients (28%) had moderate intoxication and 18 patients (18%) had severe intoxication.

Table 10. Severity of poisoning based on GCS score

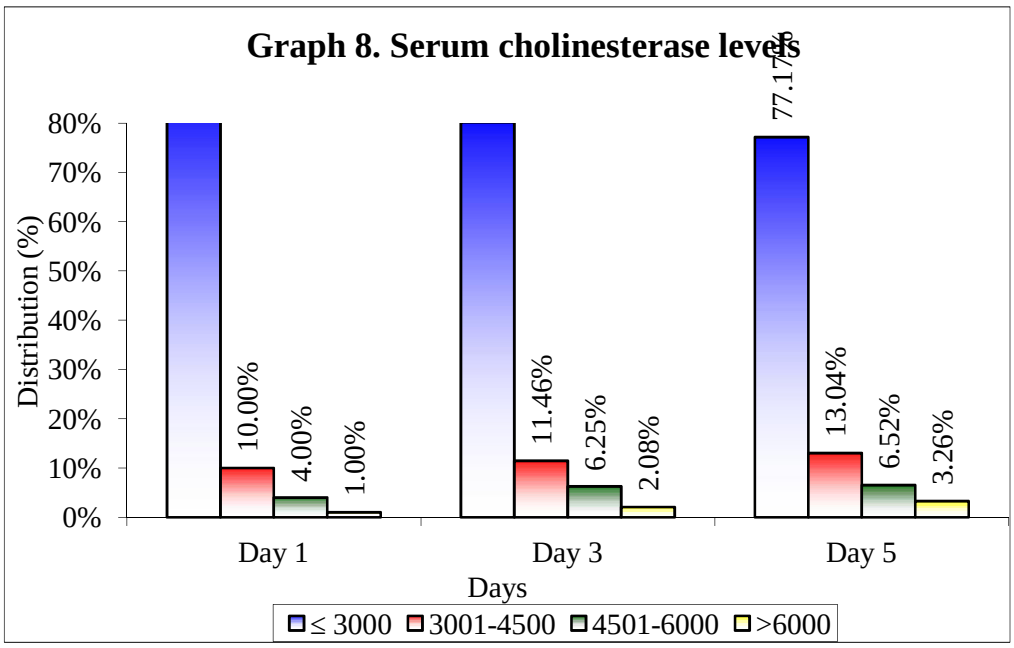
Severity	GCS score	Distribution (n=100)	
		Number	Percentage
Mild	12-15	42	42
Moderate	8 to 11	36	36
Severe	≤7	22	22
	Total	**100**	**100.00**

Similarly we observed that based on GCS score,42 patients (42%) had mild intoxication, 36 patients (36%) had moderate intoxication and 22 patients (22%) had severe intoxication.

Laboratory Parameters

Table 11. Levels of serum cholinesterase on Day1, Day3 and Day 5

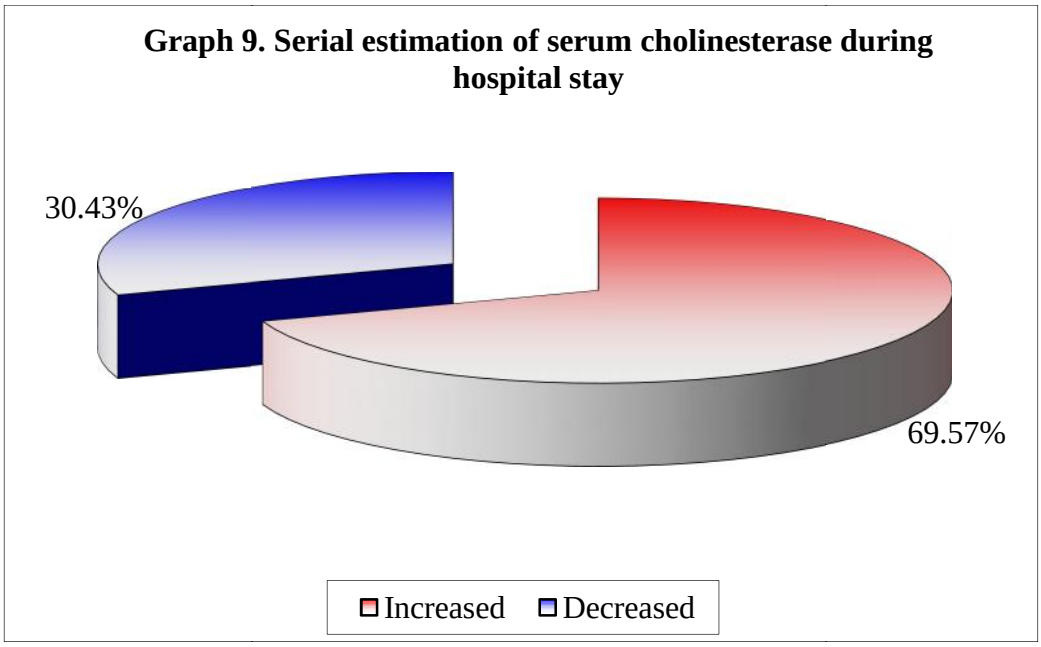
Days	Serum cholinesterase levels (U/L)	Distribution	
		Number	Percentage
1st Day (n = 100)	≤ 3000	85	85
	3001-4500	10	10
	4501-6000	4	4
	>6000	1	1
	Total	**100**	**100.00**
3th Day(n = 96)	≤ 3000	77	80.20
	3001-4500	11	11.46
	4501-6000	6	6.25
	>6000	2	2.08
	Total	**96**	**100.00**
5th Day(n = 92)	≤ 3000	71	77.17
	3001-4500	12	13.04
	4501-6000	6	6.52
	>6000	3	3.26
	Total	**92**	**100.00**



In our study estimation of serum cholinesterase on Day 1 revealed 85 patients (85%) had ≤ 3000 Units/L, 10 patients (10%) in the range of 3001 – 4500, 04 patients (4%) in the range of 4501 – 6000 and 01 patients (1%) had >6000. Similarly estimation of serum cholinesterase on 3th day and 5th day is shown in the above table.

Table 12. Serial estimation of serum cholinesterase during hospital stay

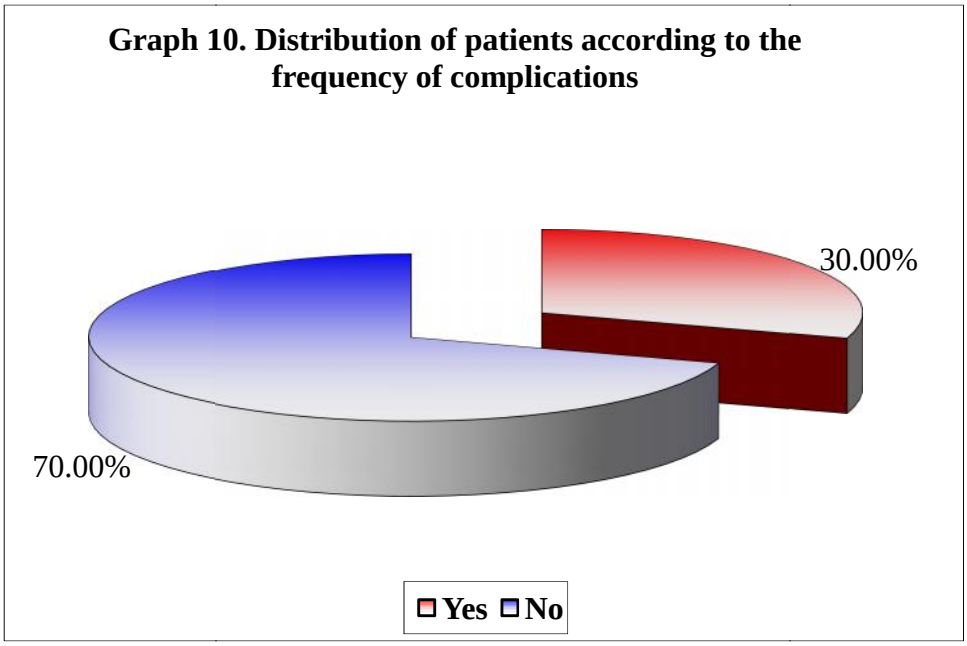
Serum cholinesterase levels	Distribution (n=92)	
	Number	**Percentage**
Increased	64	69.57
Decreased	28	30.43
Total	**92**	**100**



In the present study we observed that 64 patients (69.57%) had an increasing trend of serum cholinesterase levels and 28 patients (30.43%)had a decreasing trend. However in 8 patients it was not possible to study the changing trend as somepatients got discharged and some expired during serial estimation.

Table 13. Various complications

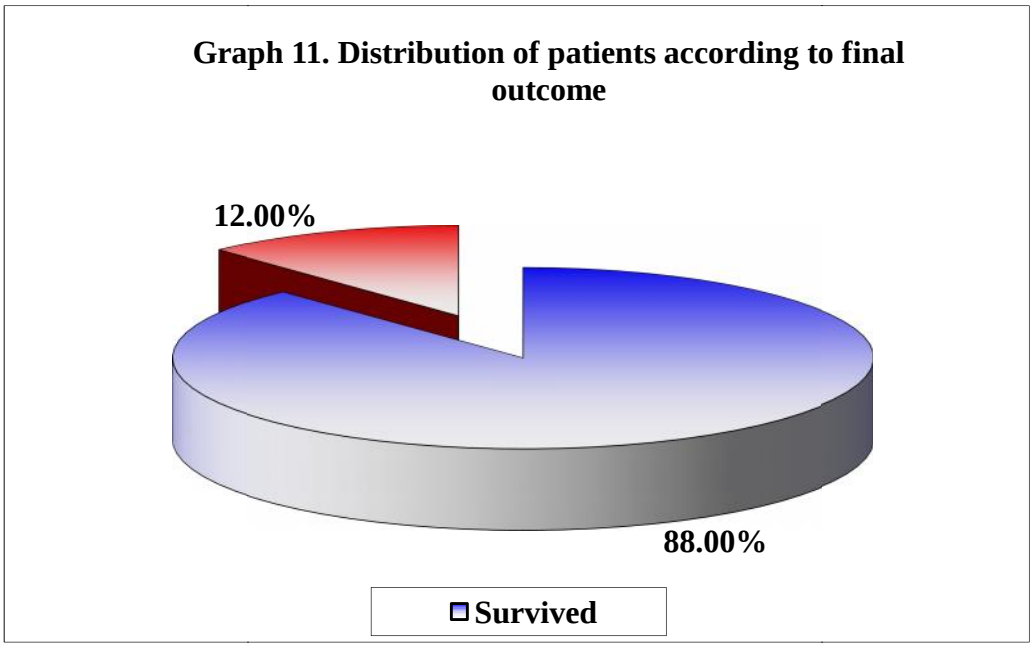
Complications (n=30)	Distribution	
	Number	Percentage
Respiratory failure	8	26.67
Psychosis	7	23.33
Hypotension	5	16.67
Acute renal failure	4	13.33
Acute respiratory distress syndrome	2	6.67
Cardiac arrhythmias	2	6.67
Convulsions	2	6.67
Total	**30**	**100.00**



A total of 30 patients had various complications, respiratoy failure was the commonest complication observed in 8 patients (26.67%), psychosis 7 patients (23.33%), hypotension 5 patients (16.67%), acute renal failure 4 patients (13.33 %), acute respiratory distress syndrome 2 patients (6.67%), cardiac arrythmias 2 patients (6.67%) and convulsions 2 patients (6.67%).

Table 14. Final Outcome of patients

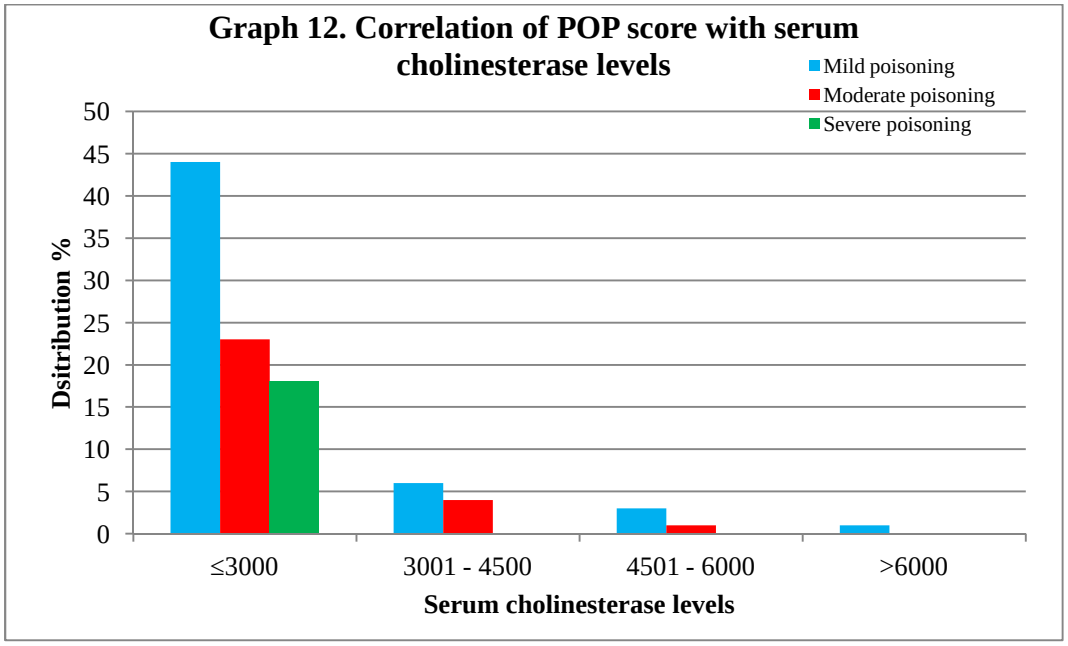
Final Outcome	Distribution (n=100)	
	Number	Percentage
Survived without complications	69	69
Survived with complications	19	19
Expired	12	12
Total	**100**	**100.00**



In the present study 88 patients (88%) survived in which 69 patients (69%) did not develop any complications, 19 patients (19%) survived inspite of developing complications and remaining 12 patients (12%) expired.

Table 15. Correlation of POP scorewith first day serum cholinesterase levels

Serum cholinesterase levels (U/L)	Severity of poisoning (POP score) n=100					
	Mild		Moderate		Severe	
	No	%	No	%	No	%
≤ 3000	44	51.76	23	27.06	18	21.18
3001-4500	6	60.00	4	40.00	0	0.00
4501-6000	3	75.00	1	25.00	0	0.00
>6000	1	100.00	0	0.00	0	0.00
Total	**54**	**54**	**28**	**28**	**18**	**18**
Chi-square=8.9863 P = 0.0112*						



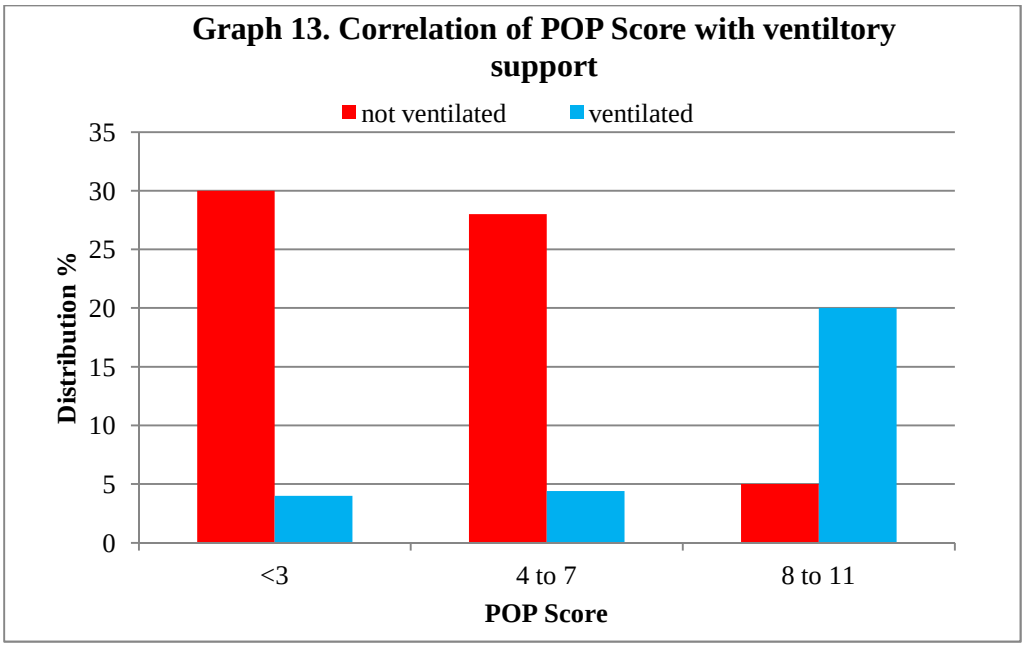
P= 0.0112

In the present study correlation of POP score with first day serum cholinesterase levels revealed, total of 54 patients had mild intoxication, 28patients had moderate intoxication and 18 patients had severe intoxication. The severity of intoxication is positively correlated with lower serum cholinesterase levels, p value= 0.0112 being statistically significant.

Table 16. Correlation of POP score with ventilatory support

POP Score	Ventilatory support (n=100)			
	Without		With	
	Number	Percentage	Number	Percentage
≤ 3	30	88.23	4	11.77
4-7	28	58.33	20	41.67
8-11	5	27.78	13	72.22
Total	**63**	**63**	**37**	**37**
Chi-square=19.3172 P = 0.0001*				

p=0.0001



In our present study comparison of POP score with ventilatory support revealed 63 patients (63%) did not require ventilatory support, remaining 37 patients (37%) required ventilatory support. However in patients with higher POP scorebetween 8-11, only 5 patients (27.78%) did not require ventilator whereas 13 patients (72.22%) required ventilator, p value = 0.0001 being statistically significant.

Table 17. Correlation of POP score with complications

POP Score	Complications (n=100)			
	Without		With	
	Number	Percentage	Number	Percentage
≤3	32	94.12	2	2.13
4 to 7	34	70.83	14	31.82
8 to 11	4	22.22	14	77.78
Total	**70**	**70**	**30**	**30**
Chi-square=28.9998 P = 0.0001*				

p=0.0001

The above table depicts the percentage of complications with the severity of poisoning based on POP score, p value = 0.0001 being statistically significant.

Table 18. Correlation of POP score with final outcome

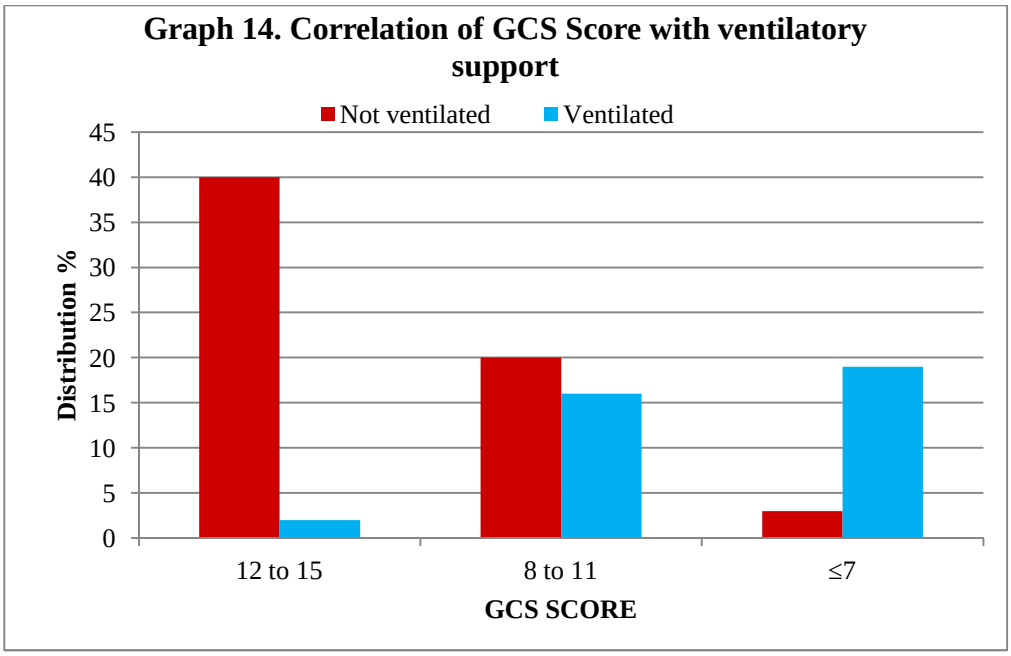
POP Score	Final Outcome (n=100)			
	Survived		Expired	
	Number	Percentage	Number	Percentage
≤ 3	33	97.06	1	2.94
4-7	45	93.75	3	6.25
8-11	10	55.56	8	44.44
Total	**88**	**88**	**12**	**12**
Chi-square=22.0884 P = 0.0001*				

p=0.0001

In the present study POP score was correlated with final outcome of patients that is higher POP Score had higher mortality, p value =0.0001being statistically significant.

Table 19. Correlation of GCS score with ventilatory support

GCS Score	Ventilatory support (n=100)			
	Without		With	
	Number	Percentage	Number	Percentage
12-15	40	95.24	2	4.76
8-11	20	55.56	16	44.44
≤7	3	13.64	19	86.36
Total	**63**	**63**	**37**	**37**
Chi-square=42.5805 P = 0.0001*				



p=0.0001

In the present study with 100 patients, requirement of ventilatory support, is positively correlated with lower GCS score.p value = 0.0001being statistically significant.

Table 20. Correlation of GCS score with complications

GCS Score	Complications (n=100)			
	Without		With	
	Number	Percentage	Number	Percentage
12-15	39	92.86	3	7.14
8-11	28	77.78	8	22.22
≤7	3	13.64	19	86.36
Total	**70**	**70**	**30**	**30**
Chi-square=44.7672 P = 0.0001*				

p=0.0001

The above table depicts the percentage of complications with the severity of poisoning based on GCS score, p value =0.0001 being statistically significant.

Table 21. Correlation of GCS score with final outcome

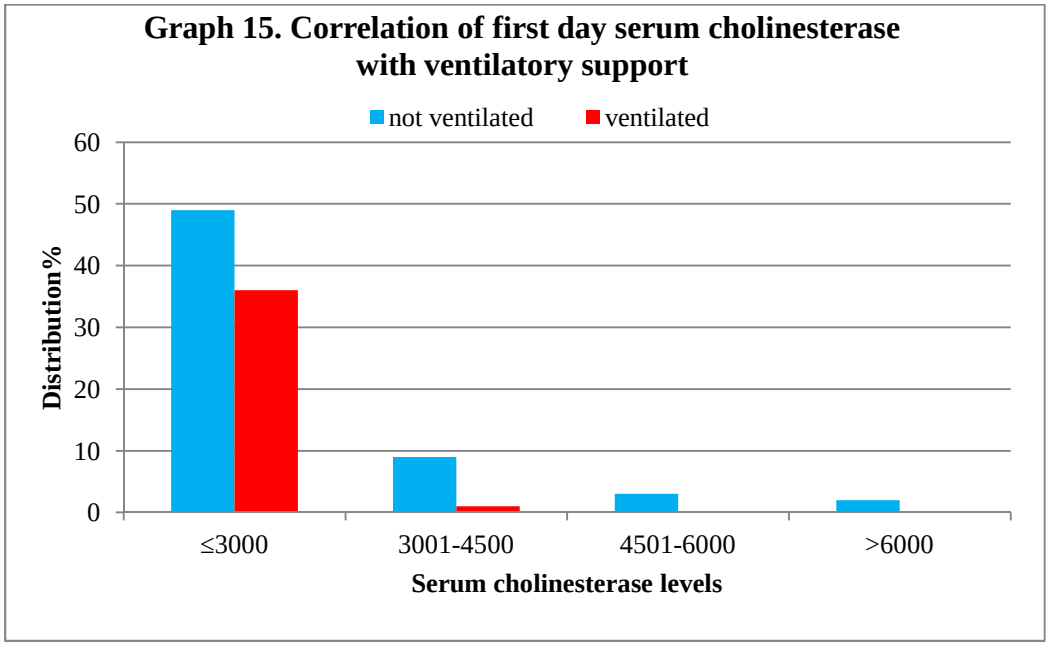
GCS Score	Final outcome (n=100)			
	Survived		Expired	
	Number	Percentage	Number	Percentage
12-15	42	100.0	0	0.00
8-11	34	93.75	2	6.25
≤ 7	12	55.56	10	44.44
Total	**88**	**88**	**12**	**12**
Chi-square=30.4606 P = 0.0001*				

p=0.0001

In the present study we observed that lower the GCS Score, overall mortality of patients was higher, p value =0.0001being statistically significant.

Table 22. Correlation of first day serum cholinesterase with ventilatory support

Serum cholinesterase levels (U/L)	Ventilatory support (n=100)			
	Without		With	
	Number	Percentage	Number	Percentage
≤ 3000	49	57.65	36	42.35
3001-4500	9	90.00	1	10.00
4501-6000	4	100.00	0	0.00
>6000	1	100.00	0	0.00
Total	**63**	**63**	**37**	**37**
Chi-square=0.9511 P = 0.6211				

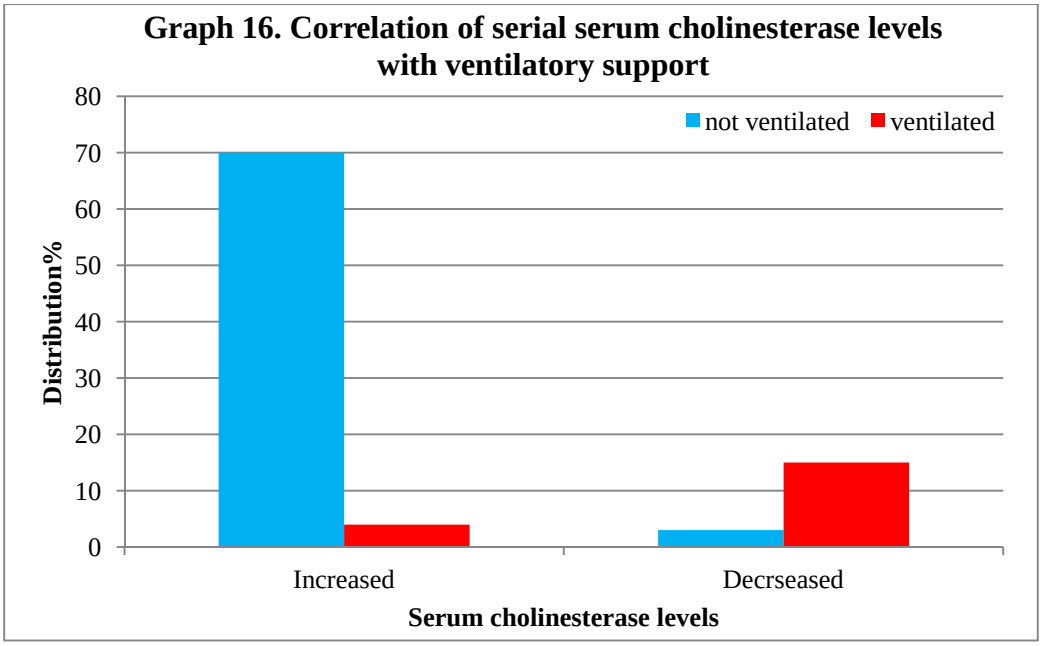


p=0.6211

In the present study of 100 patients, requirement of ventilator support is directly correlated with lower first day serum cholinesterase levels as depicted in above table, however thep value = 0.6211 was found to be statistically insignificant.

Table 23.Correlation of serial estimation of serum cholinesterase with ventilatory support.

Serum cholinesterase levels	Ventilatory support			
	Without		With	
	Number	Percentage	Number	Percentage
Increased (n = 64)	54	84.38	10	15.62
Decreased (n = 28)	4	14.29	24	85.71
Total (n = 92)	**58**	**63.04**	**34**	**36.96**
Chi-square with Yates's correction = 2.1675 P = 0.0141*				



p=0.014

Above table depictsserial estimation of serum cholinesterase levelswhich revealed thatin patients with increasing trend of serum cholinesterase requirement ofventilatory support was less as compared to patients decreasing trend of serum cholinesterase, p value =0.014being statistically significant.

Table 24. Correlation of first day serum cholinesterase with complications

Serum cholinesterase levels (U/L)	Complications (n=100)			
	Without		With	
	Number	Percentage	Number	Percentage
≤ 3000	57	67.06	28	32.94
3001-4500	9	90.00	1	10.00
4501-6000	3	75.00	1	25.00
>6000	1	100.00	0	0.00
Total	**70**	**70**	**30**	**30**
Chi-square=2.6524 P = 0.2653				

p=0.2653

The above table depicts the percentage of complications with first day serum cholinesterase levels, however p value =0.2653 being statistically insignificant.

Table 25. Correlation of first day serum cholinesterase with final outcome

Serum cholinesterase levels (U/L)	Final outcome (n=100)			
	Survived		Expired	
	Number	Percentage	Number	Percentage
≤ 3000	75	88.24	10	11.77
3001-4500	8	80.00	2	20.00
4501-6000	4	100.00	0	0.00
>6000	1	100.00	0	0.00
Total	**88**	**84.71**	**12**	**15.29**
Chi-square=1.2922 P = 0.5241				

p=0.5241

In the present study first day serum cholinesterase levels was correlated with final outcome that is patients with lower cholinesterase levels had higher mortality, however p value =0.5241was statistically insignificant.

Table 26. Correlation of serial estimation of serum cholinesterase with final outcome

Serum cholinesterase levels	Final outcome			
	Survived		Expired	
	Number	Percentage	Number	Percentage
Increased (n = 64)	63	98.44	1	1.56
Decreased(n = 28)	20	71.43	8	28.57
Total (n= 92)	**83**	**90.22**	**9**	**9.78**
Chi-square with Yates's correction = 25.7777 P = 0.0001*				

p=0.0001

In the present study we observed 92 patients with changing trends of serum cholinesterase levels. In patients with increasing trend there was only 1 death observed (1.56%)whereas in patients with decreasing trend 8 patients (28.57%) expired, p value =0.0001 being statistically significant.

Table 27. Correlation of time to hospitalization with ventilatory support

Time lag to treatment (hours)	Ventilatory support (n=100)			
	Without		With	
	Number	Percentage	Number	Percentage
<3	11	73.33	1	26.67
3-6	42	63.49	20	36.51
>6	10	54.54	16	45.45
Total	**63**	**63**	**37**	**37**
Chi-square=1.3681 P = 0.0320*				

P value=0.0320

In our present study, we observed that in patients arriving within 3 hours of consumption of organophosphorous compound, only 1 required ventilator support as compared to patients arriving between 3 to 6 hours (20 patients ventilated) and patients arriving>6 hours (16 patients ventilated), p value= 0.032 being statistically significant.

Table 28.Correlation ofinitial bolus dose of atropine with ventilatory support

Atropine dose (in mg)	Ventilatory support (n=100)			
	Without		With	
	Number	Percentage	Number	Percentage
<10	8	100.00	0	0.00
10-35	36	72.00	14	28
35-60	15	60.00	10	40.00
>60	4	23.53	13	76.47
Total	**63**	**63**	**37**	**37**
Chi-square=8.1590 P = 0.0430*				

p=0.0430

Similar attempt was made to study the outcome of patients with initial bolus dose of atropine, we observed that patients who received higher bolus dose of atropine >60mg at arrival required ventilator support more as compared to those who received lower bolus dose of atropine at arrival, p value= 0.0430 being statistically significant.

DISCUSSION

In the present study of 100 patients with organophosphorus compound poisoning predicting the need for ventilator support was done by correlating with POP score, GCS, serum cholinesterase levels and other variables.

All 100 patients who presented with organophosphorus compound poisoning were with suicidal bid.This is similar to a study done by Khazi MA et al[78]. This maybe because in agricultural countries like India, organophosphorus compounds have easy availability and low cost. However studies done in countries like Japan, have shown that accidental exposure was a major cause of poisoning [24,32].

In our study patients age ranged from 18 to 72 years. Maximum cases were in the age group of ≤30 years i.e. 38 patients (38%). This is similar to study done by Rajeev et al[9],Banday et al[79],Rehiman et al[80]and Tripathi et al[81].

When sex was taken into consideration we observed 64 males (64%) and 36 females (36%) were present in our study group. There was a male preponderance with a M:F ratio of 1.78:1. This observation is similar to studies done by Rajeev et al[9], Banday et al[79] and Tripathi et al[81]. In contrast to this another study by Rehiman et al.[80] observed more number of females in their study population.

An attempt was made to find poisoning with various professions and we observed that poisoning was common with farmers i.e;36 patient (36%) followed by students 27 (27%), labourers 23 (23%) and housewives 14(14%). This is almost similar to study by Khazi MA et al[78] , Shah Harsh D et al[82] and Banday et al.[79] where farmers constituted 32% ,78% and 47% of the study population respectively.

In contrast Rajeev et al[9] observed that poisoning was common with students in their study group.

We tried to analyse our patients with various types of organophosphorus compound they had consumed which is depicted in table 4. We observed chlorpyrifos was the commonest compounds in 24patients (24%), monochrotophos 21 patients (21%) and malathion 12 patients (12%). This is almost similar to study by Kumar CU et al.[91] and Shah Harsh D et al.[82]where most commonly consumed compounds were monocrotophos and chlorpyrifos. However in another study by Rehiman et al.[94] commonest compound consumed was parathion and dichlorvos and Rajeev et al[9] observed that commonest compound was methylparathion in 50% of their patients.

Similarly we tried to analyze the time of consumption of organophosphorous compound to arrival at the hospital. 15 patients (15%) arrived to hospital in <3 hours of consumption,63 patients (63%) arrived between 3 to 6 hours of and remaining 22 patients (22%) arrived after 6 hours of consumption. A study by Rajeev et al.[9] observed that most of their patients arrived at the hospital within the mean time interval of 4 hours.

Patients presented with various symptoms of organophosphorus compound poisoning. Majority had vomiting in 66 patients (66%), followed by breathlessness 38 patients (38%), excessive salivation 35 patients (35%), altered consciousness 28 patients (28%), sweating 15 patients (15%), diarrhoea 05 patients (5%) and only 2 patients (2%) had seizures. Studies done by Venkateshwarlu N et al.[83] and Tripathi et al[81] also observed symptoms of vomiting, breathlessness, increased salivation and sweating in their study population. In comparison study by Kavya et al[84] found abdominal pain and increased bodily secretions in their patients.

We also observed various clinical signs in the patients. Miosis was the commonest finding in 70 patients (70%) followed by bradycardia 68 patients (68%), fasciculations 56 patients (56%), tachypnoea 40 patients (40%), neck muscle weakness 38 patients (38%) and cyanosis 4 patients (4%). Khazi MA et al[78] also observed almost similar clinical findings in their patients.

We made an attempt to study clinical presentation based on POP score which revealed severe intoxication in 18 patients (18%), moderate intoxication in 48 patients (48%) and mild intoxication in 34 patients (34%). This is depicted in table 8 (POP score) and table 9 (severity). However study by Tripathi et al.[81] observed mild intoxication in 65% of their patients, 22.5% cases had moderate intoxication and only 12.5% cases had severe intoxication.

Similarly attempt of observing severity of poisoning based on GCS score revealed severe intoxication in 22 patients (22%), moderate intoxication in 36 patients (36%) and mild intoxication in 42 patients (42%). Tripathi et al.[81] in their study observed mild intoxication in 67.5% of the cases, moderate intoxication in 15% cases and severe intoxication in 17.5% of the cases.

An attempt was made to study various laboratory parameters in our patients. Serum cholinesterase estimation done on serial days (day1, day3 and day5) revealed various levels on different days which is depicted in table 11. Studies by Kumar et al[87] and Kumar CU et al.[86] have also attempted similar estimation of serum cholinesterase levels in their study

We observed the serial estimation of serum cholinesterase levels revealed increasing trend in 64 patients (69.57%) and decreasing trend in 28 patients (30.43%)

during their stay in hospital. In the remaining 8 patients it was not possible to study the trend as some got discharged and some expired during serial estimation. Kumar et al[87] and Kumar CU et al.[86] also observed in majority of their patients increasing trend of serum cholinesterase estimation.

We observed various complications of OP compound poisoning in 30 patients. Respiratory failure was the most common seen in 8 patients (26.67%), psychosis 7 patients (23.33%), hypotension 5 patients (16.67%), acute renal failure 4 patients (13.33%),acute respiratory distress syndrome, cardiac arrhythmias 2 patients (6.67%) and convulsions 2 patients(6.67%). Study by Banday et al.[79]observed episodic convulsions in 13.5% patients and hypotension in 11.3% of the patients. Another study by Venkateshwarlu N et al.[83]observed that majority of the patients had pulmonary edema seen in 46% of their study population.

Final outcome of patients in our study revealed88 patients (88%) survived in which 69 patients (69%) did not develop complications, 19 patients (19%) survived inspite of developing complications and 12 patients (12%) expired. Studies by Rajeev et al.[9] and Rehiman et al[80] had a mortality rate of 16% and 14% respectively. This is in sharp contrast to study done by Banday et al.[2]who observed a higher mortality rate of 33%.

Similarly we tried correlation of POP score with first day serum cholinesterase levels, which revealed 54 patients (54%) had mild intoxication, 28 patients (28%) had moderate intoxication, and 18 patients (18%) had severe intoxication. The severity of intoxication is significantly correlated with lower serum cholinesterase levels (p=0.0112,statistically significant). Studies done by Rehiman et al.[80] and Shah Harsh D et al[82]also found significant correlation in their study. However Kumar et al.[80]did

not find significant correlation with POP score and serum cholinesterase levels in their study group.

An attempt was made to correlate between POP score and need for ventilator and we observed out of 100 cases in our study, 63 patients (63%) did not require ventilator support, remaining 37 patients (37%) however required ventilator support. In patients whose POP score was higher between 8-11, only 5 patients (27.78%)did not require ventilator whereas 13 patients (72.22%) required ventilator, (p value=0.0001, statistically significant). Study by Rajeev et al[9]. , Tripathi et al[81]. found significant correlation between POP score and ventilator support.

We attempted correlation between POP score with complications and we observed in patients with higher POP score between 8-11, 14 patients (77.78%) developed complications and only 4 patients (22.22%) did not have complications. In patients with POP score between 4-7, 14 patients had complications (31.82%) whereas 34 patients (70.83%) did not have complications and patients with POP score ≤3, only 2 patients (2.13%) had complications and 32 patients (94.12%) did not have complications (p value= 0.0001, statistically significant). Tripathi et al[81]. observed similar findings in their study.

Similarly attempt was made to correlate POP score with final outcome of patients. In patients with POP score between 8 to 11, 10 patients (55.56%) survived and 8 (44.44%) expired, in POP score between 4 to 7, 45 patients (93.75%) survived and 3 (6.25%) expired and in POP score ≤3, 33 patients (97.06%) survived and only 1 patient (2.94%) expired (p value=0.0001; statistically significant). Similar observations were made by Rajeev et al[9]. , Tripathi et al[81]. in their study group.

We observed correlation of GCS score with need for ventilator support which revealed a significant correlation between lower GCS score and need for ventilator support which is depicted in table 19. (p value=0.0001, statistically significant). Rajeev et al[9].,Tripathi et al[81] made similar observations in their study group.

Attempt was made to correlate between GCS score with complications. In patients GCS score ≤7, 19 patients (86.36%) had complications and 3 patients (13.64%) did not have complications. In GCS score between 8 to 11, 8 patients (22.22%) had complications and 28 patients (77.78%) did not have complications whereas in GCS score between 12 to 15, only 3 patients (7.14%) had complications and 39 patients (92.86%) did not have complications (p=0.0001, statistically significant). Tripathi et al.[81] observed similar findings in their study.

In comparison of GCS score with final outcome of patients we observed lower the GCS score, overall mortality was higher as depicted in table 21(p value=0.0001, statistically significant). Rajeev et al[9]., Tripathi[81] et al. also found similar correlation between lower GCS score with higher mortality rates in their study group.

We observed correlation of first day serum cholinesterase levels with need for ventilator support which is depicted in table 22. Patients with lower serum cholinesterase levels required ventilator support more as compared to higher levels of serum cholinesterase on day one. However the p value = 0.6211 was statistically insignificant. Goswamy et al.[91] and Rajeev et al.[9] found statistically significant correlation in their study population. However study by Kumar et al.[90]did not find significant correlation between first day serum cholinesterase levels and requirement of ventilator support.

When we did correlation of serial estimation of serum cholinesterase with requirement of ventilator support we observed that patients with increasing trend of serum cholinesterase levels required ventilator support less as compared to patients with decreasing trend of serum cholinesterase which is depicted in table 23 (p=0.014, statistically significant). Study by Kumar CU et al.[86] and Kumar et al.[90] found significant correlation between serial estimation of serum cholinesterase and requirement of ventilator support.

Correlation of first day serum cholinesterase levels with complications revealed more complications in patients with lower levels serum cholinesterase of ≤3000 U i.e; 28 patients (32.94%) whereas in serum cholinesterase levels between 3001-4500U, one patient (10%) and in serum cholinesterase levels between 4501-6000U ,also only one patient(25%) developed complication.

However the p value = 0.2653 was statistically insignificant. Different authors have not correlated serum cholinesterase levels with complications.

Attempt was made to correlate first day serum cholinesterase levels with final outcome. We observed patients with lower first day serum cholinesterase levels had higher mortality which is depicted in table 25. However p value = 0.5241was statistically insignificant. Study done by Khazi MA et al[78], Shah D et al.[82] and Kavya et al.[84] found significant correlation between low levels of serum cholinesterase and increased mortality. In contrast to this Kumar et al.[90] did not observe significant correlation in their study population.

We also attempted correlation of serum cholinesterase estimation on serial days with final outcome which showed 64 patients (69.57%) had increasing trend of serum cholinesterase levels and 28 patients (30.43%) had decreasing trend. In patients with increasing trend only 1 patient (1.56%) expired whereas in patients with decreasing trend, 8 patients (28.57%) expired. (p=0.0001; statistically significant). Studies by Yun et al.[85], Kumar CU et al.[86]and Kumar et al.[90] observed similar correlation with increased mortality in patients with decreasing trend of serum cholinesterase levels.

We attempted correlating time of consumption to arrival of patients at hospital to the requirement of ventilator support and we observed that in patients arriving <3 hours of consumption, only 1 patient (6.67%) required ventilator support as compared to 20 patients (31.75%) requiring ventilator support arriving between 3 to 6 hours and 16 patients (62.5%) requiring ventilator support coming after 6 hours of consumption (p = 0.0320;statistically significant).Rajeev et al.[9] , Banday at al.[79] and Goel et al.[76] also made similar observations in their study group.

Finally we tried to correlate initial bolus dose of atropine given at arrival of patients to the requirement of ventilator support and observed that patients requiring more bolus doses of atropine i.e; >60mg at arrival required ventilator support more as compared to patients received lower bolus doses of atropine (p=0.0430; statistically significant). Similar observations were made by Shah Harsh D et al.[82], Singh et al.[93] and Kumar et al.[90]in their study population.

In our present study with a small sample size of 100 patients, we observed a significant correlation between POP score and first day serum cholinesterase levels, POP score with need for ventilator support, POP score with complications and POP

score with final outcome. Similarly our observation was a significant correlation between GCS score and need for ventilator support, GCS score with complications and GCS score with final outcome.

We also observed a significant correlation between serum cholinesterase levels and need for ventilator support and with final outcome. This was observed only on serial estimation but not on first day serum cholinesterase levels. However correlation between first day serum cholinesterase levels and complications was statistically insignificant.

Further we observed a significant correlation between time of arrival of patients to hospital and need for ventilator support. Finally we observed a significant correlation between initial bolus doses of atropine given at arrival and need for ventilator support.

Few studies have shown lack of relationship between serum cholinesterase levels and clinical outcome. This could be possibly because of serum cholinesterase levels not being a reliable biomarker in assessing the clinical severity of organophosphorus compound poisoning.

This can be explained by a proposed mechanism that tissue concentration of cholinesterase (true cholinesterase), which is thought to be more closely related to the clinical manifestations of organophosphorus compound poisoning is poorly correlated with plasma levels of enzyme (pseudocholinesterase / serum cholinesterase).

The organophosphate inhibition of these two enzymes (true cholinesterase and pseudocholinesterase) may be different.

And it is also possible that toxic manifestations of some of the organophosphorus compounds are independent of cholinergic mediated mechanism.

So we feel it is prudent to study with a large sample size and estimation of true cholinesterase enzyme levels and to find other mechanisms of toxicity which are independent of enzyme inhibition.

CONCLUSION

In the present study of 100 patients we observed various clinical features of organophosphorus compound poisoning. POP score, GCS score and serum cholinesterase levels were correlated with need for ventilator support and also with complications and final outcome.

From the present study we observed the following facts:

- In all our patients, poisoning was due to suicidal bid.
- Males patients were more compared to females.
- The most common age group was ≤30 years.
- Poisoning was most common with farmers.
- Chlorpyrifos was the commonest compound.
- Vomiting and breathlessness were the most common symptoms while miosis, and bradycardia were the commonly observed signs.
- Most common complication observed was respiratory failure.
- Serial estimation of serum cholinesterase levels (day 1, day 3 and day 5) showed an increasing trend in majority of patients.
- We found a statistically significant correlation between POP score and first day serum cholinesterase levels, POP score with ventilator support, with complications and with final outcome. Similarly correlation of GCS score with ventilator support, with complications and with final outcome was statistically significant.
- Even comparison of serum cholinesterase levels with ventilator support and final outcome had a significant correlation (on serial estimation only but not

on first day levels). Similarly the correlation between first day serum cholinesterase and complications was statistically insignificant.

- Also correlation of time of consumption to arrival at the hospital to ventilator support and correlation of initial bolus doses of atropine given at arrival to need for ventilator support was statistically significant.
- Most of patients did not require ventilator support.
- Finally majority of the patients survived and mortality was seen in 12 patients.

We feel it would be worth studying other variables like age, sex, type of compound, quantity and toxicity of different compounds with the need for ventilator support which was not possible in our present study because of a small sample size of 100 patients. Hence a larger sample size maybe required to address these issues.

SUMMARY

The present study of 100 patients with organophosphoruscompound poisoning admitted in Department of medicine, KLES Dr.PrabhakarKore Hospital and Medical Research Centre, Belgaum during the study period from January 2017 to December 2017, was undertaken to predict the need for ventilator support.

The results observed were significant correlations between POP score and GCS score with need for ventilator support and also with complications and final outcome. There was a statistically significant correlation between serum cholinesterase levels with need for ventilator support and final outcome (on serial estimation only but not on first day levels). Finally we found a significant correlation between time of consumption to arrival at hospital andinitial bolus doses of atropine given at arrival to need for ventilator support.

However we did not study the correlation of variables like age, sex, type of compound, quantity and toxicity of different compounds with the need for ventilator support.

BIBLIOGRAPHY

1. Kalkan S, Erdogan A, Aygoren O, Capar S, Tuncok Y. Pesticide poisonings reported to the drug and poison information center in Izmir, Turkey. Vet Hum Toxicol 2003;45:50-2.
2. Siva PV, Padma VSA, Sarma DVHS, Reddy SM. Activity of Serum Cholinesterase in Organo - Phosphorus poisoning cases: A prospective study. J Pharmaceutical Biomed Sci 2012;20(03):1-3.
3. Singh S, Wig N, Chaudhary D et al : Changing pattern of acute poisoning in adults : experience of a large north west Indian hospital (1970-1989). JAPI 1997 ; 45 : 194-197.
4. Malik GM, Mubarik M, Romshoo GJ: Organophosporous poisoning in the Kashmir valley 1994 to 1997. NEJM 1998 ; 338: 1078-1079
5. Philip G. Bardin. Organophosphorous and carbamate poisoning. Archieves of internal medicine 1994; 154 : 1433-1441
6. Kavya S.T., Srinivas V, Chandana, Madhumati R, "Clinical Profile of patients with Organophosphorous Poisoning in an Intensive Care Unit in a tertiary hospital" International Journal of Clinical Cases and Investigations 2012. Volume 4 (ISSUE 3), 24:31,1ST OCTOBER 2012.
7. Karalliedde L, Senanayake N : Organophosporous insecticide poisoning. British Journal of anaeasthesia 1989; 63; 736-750.
8. Singh S, Sharma N. Neurological syndromes following organophospate poisoning. Neurology India 2000 ; 48 (4) : 308-13.
9. Rajeev H., Arvind M.N. "Study of clinical and biochemical parametrs in predicting the need for ventilator support in organophosphorous compound

poisoning". Journal of Evolution of Medical and dental sciences 2013; Vol2, Issue 49,December 09; Page: 9555-9570.

10. Siva PV, Padma VSA, Sarma DVHS, Reddy SM. Activity of Serum Cholinesterase in Organo - Phosphorus poisoning cases: A prospective study. J Pharmaceutical Biomed Sci 2012;20(03):1-3.
11. Rusyniak DE, Nanagas KA. Organophosphate poisoning. Semin Neurol 2004;24(2):197-204.
12. Tsao TC, Jwang Y, Lan R, Sheieh W, Lee C. Respiratory failure in acute organophosphorous and carbamate poisoning. Chest 1990; 98 : 631-636.
13. Koelle GB. Pharmacology and toxicology of organophosphorous and carbamates. In: clinical and experimental toxicology of organophorphates and carbamates. Ballantyn B, Marrs T, eds. Butterworth Hunmann, Oxford 1992; 33-37.
14. Bardin PG, Van Eeden SF, Joubert JR. Intensive care management of acute organophosphorous compound: a 7-year experience in the west cape. South aferican medicine journal 1987; 72 : 593-597.
15. Steward WC, Anderson Ea. Effects of cholinesterase inhibition when injected into the medulla of the rabbit. Journal of Pharmacological Experimental Therapy 1968; 162: 309-317.
16. Sam KG, Kondabolu K, Pati D, Kamath A, Pradeep Kumar G, Rao PG. Poisoning severity score, APACHE II and GCS: effective clinical indices for estimating severity and predicting outcome of acute organophosphorus and carbamate poisoning. J Forensic Leg Med 2009;16(5):239-47.

17. Bardin PG, Van Eeden SF. organophosphorous poisoning: grading the severity and comparing treatment between atropine and glycopyrolate. Critical care Medicine 1990; 18 : 956-960.
18. Kang EJ, Seok SJ, Lee KH, Gil HW, Yang JO, Lee EY, et al. Factors for determining survival in acute organophosphate poisoning. Korean J Intern Med 2009;24(4):362-7.
19. Senanayake N, de Silva HJ, Karalliedde L. A scale to assess severity in organophosphorus intoxication: POP scale. Hum Exp Toxicol 1993;12:297-9.
20. Taylor P. Anticholinesterase agents In: Gilman AG, Goodman LS, Rall TW, Murad F eds. The pharmacological basis of therapeutics. New York: Mac Millan 1985; 110-129
21. Hayes M.M, Vander Westhuizen N.G, Gelfanol M., 1978; "Organophosphate Poisoning in Rhodesia". S. Afr. Med. J., 54:230 – 234.
22. Jeyaratnam J., 1990: "Acute Poinsoning: A major health problem", World health stat. Q, 93:139-145.
23. Morgan J.P: The Jamaica ginger Paralysis, JAMA 1982;248:1864-1867.
24. Mutalik GS, Wadia RS and Pai VR. Poisoning by diazinon an organophosphorous insecticide. Journal Of Indian medical association 1962; 38:67-71.
25. Hayes WJ. Organophosphorous insecticide. In : Hayes WJ, ed. Pesticides studied in man. Batlimore, Williams and Wilkins, 1982; 284-413
26. Karalliedde L, Organophosphorous poisoning and anesthesia, Anaesthesia 1999:54: 1073-1088.

27. Johnson MK. Inhibition, reactivation and aging of cholinesterases. Organophosphorous Winter meeting, Hannibal House, London, 1992.
28. Maroni M. Review of toxicological properties and biotransformation of organophosphorous esters. In : WHO Manual Of Analytical maethods. Cremona : WHO collaboration center for occupational health 1985 ; 3-39.
29. Davies DR, Green AL. The kinetics of reactivation by oximes of cholinesterase inhibition by Organophosphorous compounds. Biochemical Journal 1956; 63: 529-535.
30. Johnson MK. Lauwerys R. Protection by some carbamates against the delayed neurotoxic effects of diesopropylphosphorofluoriddate. Nature 1969; 222: 1066-1067.
31. Moss DW, Hunderson DR, Kachmar JF, Exzymes. In: Tietz NW, ed. Textbook of clinical Chemistry. Philadelphia, WB Saunders Co; 1986: 619-774.
32. Namba T, Nolte CT, Jackrel J, Grob D. Poisoning due to organophosphorous insecticide. American Journal of Medicine 1971; 50 : 475-492.
33. Grob D, John RJ. Treatment of anticholinesterase intoxication with oximes. JAMA 1958; 166:1855.
34. Senanayake N, Karalliedde L. neurotoxic effects of Organophosphorous insecticide. NEJM 1987; 316, 716-763.
35. Leon-S-Fidas E, Pradilla G. et el: Neurological effects of Organophosphorous pesticide. BMJ 1996 ; 313 : 690-691.
36. Surjit singh and Sharma. Neurological syndromes following Organophosphorous poisoning. Neurology India 2000; 48: 308-313.

37. Johnson MK, Lauwerys R. Protection by some carbamates against the delayed neurotoxic effects of diisoproylphosphoroflouride . Nature 1969; 222: 1066-1067.
38. Bidstrup PL, Bonnell JA, Beckett AG. Paralysis following poisoning by a new Organophosphorous insecticide(mipafox). BMJ 1953; 1:1068-1072.
39. Hiersons R, Johnson MK. Clinical and toxicological investigations of a case of delayed neuropathy in man after acute poisoning by an Organophosphorous pesticide. Archieves of Toxicology 1978; 40:279-284.
40. Jederzyowsksa H, Rowinska-Marcincka K, Hoppe B. Neuropathy due to phtosol (Agritox) a report of a case. Acta Neuropathologica 1980; 49;163-168.
41. Senanyake N, Sanmuganathan PS. Extrapyramidal manifestations complicating Organ phosphorous insecticide poisoning. Human and experimental Toxicology 1995; 14: 600-604.
42. Mingon A, Board P, Blackburn AC, Mellick GD, Le Counteur DG. Parkinsons disease, pesticide and Gluthathion transferase polymorphism. Lancet 1998; 352: 1344-1346.
43. Bellin JS, Chow I. Biochemical effects of chronic low level exposure to pesticides. Research communications in chemical pathology and pharmacology 1974; 9:325-327.
44. Casale GP, Cohen SD, Dicapua Ra. The effect of Organophosphorous induced cholinergic stimulation on the antibody response to sheep erythrocytes in inbred mice. Toxicology and Applied Pharmacology 1983; 68:198-205.
45. Newcombe DS. Immune surveillance, Organophosphorous exposure and lymphoma genesis. Lancet 1992; 339:539-541.

46. Murray VS, Wesiman HM, Dawling S, Morgan I, House IM. Health effects of Organ phosphorous sheep dips. BMJ 1992; 305: 1090.
47. Cevin M, Leeb JE, Wishnow RM, et al. Effects of low-level administration of dichlorvas on adrenocorticotrophic hormone secretion, adrenal cholesteryl ester and steroid metabolism. Biochemical Pharmacology 1980; 29: 635-641.
48. Haubenstock A. More on the triad pf pancreatitits, hyperamylasemia and hyperglycemia. Journal of American Medical Associtition 1963; 249: 1563.
49. Akthar N, Kayani SA, Ahmad MM, Shahab M. Insecticide induced changes in secretary activity of the thyroid glands in rats. Journal of applied pharmacology 1996; 16 :397-400.
50. Kiss Z, Fazekas T. Organophosphorous and Torsade de pointes ventricular tachycardia. Journal of the royal society of medicine 1983; 76: 984-985.
51. Wren C, Carson PHM, Sanderson JM. Organophosphorous poisoning and complete heart block. Journal of royal society of medicine 1981; 74;688-689.
52. Chuang FR, Jang SW, Lin JL, Chern MS, Chen JB, Hsu KT. QTc prolongation indicates a poor prognosis in patients with Organophosphorous poisoning. American journal of emergency medicine 1996; 14: 451-453.
53. Gadoth N, Fisher A. Late onset of neuromuscular block in Organophosphorous poisoning. Annals of internal medicine 1978; 88: 654-655.
54. Hantson P, Hainaut P, Vander Stappen M, Mahieu p. Regulation of body temperature after acute Organophosphorous poisoning. Canadian journal of anaesthesia 1996; 43: 755.
55. Yatendra S, Joshi SC, Singh M, Joshi A, Kumar J. Organophosphorus Poisoning: An Overview. International J Health Sci Res 2014;4(8):245-57.

56. Carlton FB, Simpson WM, Haddad LM. Pesticides. In: Haddad LM, Shannon MW, Winchester JF, eds. *Clinical Management of Poisoning and Drug Overdose.* 3rd ed., Philadelphia: W. B. Saunders Company; 1998. p. 836-45.
57. Cherian AM, Peter JV, Samuel J. Effectiveness of P2AM (PAM-pralidoxime) in the treatment of organophosphorus poisoning. A randomized, double-blind, placebo-controlled trial. J Assoc Phys India 1997;45:22-4.
58. Singh G, Avasthi G, Khurana D, Whig J, Mahajan R. Neurophysiological monitoring of pharmacological manipulation in acute OP poisoning. Electroencephalogr Clin Neurophysiol 1998;107(2):140-8.
59. Sundaray NK, Ratheesh Kumar J. Organophosphorus Poisoning: Current management guidelines. Medicine Update 2010;20:420-5.
60. Du toit PW, Muller Fo, Van Tonder WN, Ungerer MJ. Experience with the intensive care management of Organophosphorous insecticide poisoning. South African medical journal 1981; 60: 227-229.
61. Taturi J, Roberts J. Organophosphorous poisoning. Annals of emergency medicine 1987; 16: 193-202.
62. Rumach BH. Anticholinergic poisoning. Treatment with physostigmine. Pediatrics 1973; 52: 449-551.
63. Eddleston M, Dawson A, Karalliedde L, Dissanayake W, Hittarage A, Azher S, et al. Early management after self poisoning with an Organophosphorus or carbamate pesticide –a treatment protocol for junior doctors. Crit Care 2004;8(6):R391-7.
64. Chugh SN, Aggarwal N, Dabla S, Chhabra B. Comparative Evaluation of "Atropine Alone" and "Atropine with Pralidoxime (PAM)" in the Management of Organophosphorus Poisoning. JIACM 2005;6(1):33-7.

65. .Mirakhur RK,Dundee JW. Glycopyrrolate: pharmacology. Anesthesia 1983; 38: 1195-1203.
66. Mc Cubbin TD, Brow JH, Dewar KMS, Jones LJ, SpenceAA. Glycopyrrolate as a premedicant. Comparison with atropine. British journal of anesthesia 1979; 51: 885-889.
67. Proakis AG, Harres GB. Comparative penetration of glycopyrrolate and atropine across the blood bran barrier in anesthetized dogs. Anesthesiology 1978; 48: 339-344.
68. Sedill FR, Groff Wa. Intramuscular and intravenous administration of small does of Pyridine-2-aldoxime methyl chloride in man. Journal of pharmaceutical science 1971; 60: 1224-1228.
69. Durham WF, Hayes WJ. Organophosphorous poisoning and its therapy. Achieves of environmental health 1962; 5: 21-47.
70. Johnson DD, Wibcox CW. Studies on the mechanism of protective and antidote actions of diazepam in Organophosphorous poisoning. European journal of pharmacology 1975; 34: 127-132.
71. Lherman Y, Gutamn H. the use of respiratory stimulants in Organophosphorous intoxication. Medical hyothesis 1988; 26: 267-269.
72. 61. Luzhniknov EA, Yaroslawsky AA, Molodenkov MN, Shurkalin BK, Ewsur NG, Barswkow UF. Plasma perfusion through charcoal in methyl parathion poisoning. Lancet 1977; 1: 38-39.
73. Zweiner RJ, Ginsburg CM. Organophosphorous and carbamate poisoning in infants and children. Pediatrics 1988; 121-126.

74. De Condole CA, Douglas WW, Evans CL, et al. The failure of respiration in death by anticholinesterase poisoning. British journal of pharmacology 1953; 8: 446-475.
75. Bardin PG, Van Eeden SF, Joubert JR. Intensive care management of acute organophosphorous compound: a 7-year experience in the west cape. South aferican medicine journal 1987; 72 : 593-597.
76. Goel A, S Joseph , Dutta TK. Organophosphate Poisoning: Predicting the need for ventilatory support. JAPI 1998; 46: 786-90
77. G Avasthi, G Singh. Serval neuro-electrophysiological studies in acute Organophosphate Poisoning- correlation with clinical finding, serum cholinesterase levels and atropine dosages. JAPI 2000; 48(8): 794-799.
78. Khazi MA, Sainath C, Parvez A. A Cross Sectional Study of estimation of Plasma Pseudo cholinesterase and its Correlation to mortality among organophosphorus poisoning patients. Indian J Basic Applied Med Res 2014;3(3):285-91.
79. Banday T.H., Tathineni B, Desai MS, Naik V. “Predictors of morbidity and mortality in organophosphorous poisoning: A case study in rural hospital in Karnataka, India. North American Journal Of Medical Science 2015; 7: 259-65.
80. Rehiman S , Lohani SP, Bhattarai MD. “Correlation of Serum Cholinesterase Level, Clinical Score at Presentation and Severity of Organophosphorous Poisoning” Journal of Nepal Medicine Association 2008; 47 (170): 47-52
81. Tripathi S. “Prognostic Value of Glasgow Coma Scale, Poisoning Severity score and Serum Acetylcholinesterase levels in Organophosphorous

Poisoning". Journal of Evolution of Medical and Dental Sciences 2014;Vol3, Issue 13, March31; Page: 3415-3422.

82. Shah Harsh D et al. Acute Organophosphorus Poisoning And Clinical Admission Score Association Among Patients Admitted In Emergency Ward Of A Tertiary Teaching Hospital Of Medical College. JPBMS, 2012, 17(08).
83. Venkateshwarlu N, Gandiah P, Prabhakar KK, Indira G, Sivarajappa P. Significance of Serum Cholinesterase Levels in Patients of Organophosphorus Poisoning. International J Recent Trends Sci Technol 2013;9(2):270-4.
84. Kavya S.T, Srinivas V, Chandana, Madhumati R. Clinical Profile of Patients with Organophosphorus poisoning in an intensive care unit in a tertiary hospital. International Journal of Clinical cases and investigations 2012. Volume 4(Issue 3), 24:31.
85. Yun HW, Lee DH, Lee JH, Cheon YJ, Choi YH. Serial serum cholinesterase activities as a prognostic factor in organophosphate poisoned patients. Hong Kong Med J 2012;19(2):92-7.
86. Kumar CU, Kishan PV, Chandrashekhar E, Usharani P. The utility of serial serum cholinesterase as a prognostic marker in organophosphorus compound poisoning. International J Basic Clin Pharmacol2014;3(3):529-33.
87. Weissmann-Brenner.A,David A, Vidan A, Hourvitz A.:Organophosphate poisoning:A Multihospital Survey.IMAJ 2002;4:573-576.
88. Aygun D, Doganay Z, Altintop L, Guven H, Onar M, Deniz T, et al. Serum acetylcholinesterase and prognosis of acute organophosphate poisoning. J Toxicol Clin Toxicol. 2002;40(7):903–10.

89. Zawar S D et al. Correlation between Plasma cholinesterase Level and clinical severity of acute orgonophophate and carbamate poisoning. JAPI. 2001;149:91
90. Kumar RG, Rama Rao PSV, Nrushen P. A study on serum cholinesterase levels as a prognostic marker in organophosphorus poisoning.Asian Pacific Journal of Health Sciences, 2017; 4(1):91-99.
91. Goswamy R, Chaudhuri A, Mahashur AA. Study of respiratory failure in organophosphate and carbamate poisoning. Heart Lung. 1994;23(6):466-72.
92. Basu A, Das AK, Chandrashekar S: organophosphate poisoning - A clinical profile J. Assoc Physicians India:1988:36:24.
93. Singh S, Batra Y.K.: "Is atropine alone sufficient in acute severe Organophosphorus poisoning?: mt. J. Clin. Pharmacol Ther. 1995:93 (11): 628 630
94. Singh S, Sharma N. Neurological syndromes following organophospate poisoning. Neurology India 2000 ; 48 (4) : 308-13.
95. Surjit singh and Sharma. Neurological syndromes following Organophosphorous poisoning. Neurology India 2000; 48: 308-313.

ANNEXURE I – CONSENT FORM

TITLE OF RESEARCH STUDY: ONE YEAR CROSS SECTIONAL STUDY OF PATIENTS WITH ORGANOPHOSPHORUS COMPOUND POISONING- PREDICITNG THE NEED FOR VENTILATOR SUPPORT

Principal Investigator:

Guide:

Introduction and Purpose

Organophosphorus compound poisoning is one of the commonest poisonings seen in India. This study intends to determine the clinical and biochemical parameters that help in predicting the need for ventilator support and thus reduce mortality rate.

Procedure:

If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood samples for the necessary investigations.

Risk and Benefits:

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations. It may cause swelling, pain, redness (rarely happens) at the site from where the blood is drawn.

You may not be benefitted by these investigations but you will be part of this study which is going to be useful to others in the future.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study.

If you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment.

Privacy and Confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy: Does not apply to this research

Financial incentives for participation:

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results:

The results of the study would be forwarded to the KLE University, Belagavi as part of requirement towards the completion of MD degree, review and publishing.

In case of the queries during study or in future you may contact following persons,

If you have any doubts regarding your rights as a participant you may contact:

3. Dr. Ganga Pilli,
   Chairman,
   J N M C Ethical Committee
   for Human Research
   9448863866

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me and has been explained to me in my vernacular language and all my questions have been answered. I will be given a copy of this consent form.

Signature / Left Thumb print of the Participant or legally authorized representative:

Participant Name: ……

Signature / Left thumb impression of the participant: ……..

Name of the legally authorized representative / guardian: ……

Signature / Left thumb impression: ……...

Witness Name: ----------

Signature / Left thumb impression: ……...

Investigator Name and signature: ……...

Date:

Place:

ANNEXURE II – PROFORMA

TITLE: ONE YEAR CROSS SECTIONAL STUDY OF PATIENTS WITH ORGANOPHOSPHORUS COMPOUND POISONING- PREDICITNG THE NEED FOR VENTILATOR SUPPORT

Case No. :

Name :

Age / sex :

Inpatient Number :

Address :

Occupation :

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

7. Intention Homicidal / Suicidal / Accidental

Complaints at presentation	Yes	No
Vomiting		
Breathlessness		
Sweating, salivation		
Diarrhea		
Convulsions		

Any other complaint

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

Family history

Personal history

Treatment history

Physical examination

Temperature :

Pulse :

Respiratory rate :

Blood pressure :

Pupils :

Systemic examination

Respiratory system :

Cardiovascular system :

Per abdomen :

Central nervous system :

POP score

GCS score

Diagnosis

Complications

Requirement of mechanical ventilation Yes/No

Outcome Survived / Expired

ANNEXURES III - MASTER CHART

Serial number	In patient number	Age (Years)	Sex	Occupation	Type of poison consumed	Route of exposure	Quantity consumed (mL)	Time interval from ingestion to hospitalization (Hours)	Initial bolus dose of atropine given	Intention	Clinical presentation							Clinical observation						Examination												Investigations			Complications	Various Complications	Mechanical ventilation	Outcome
																								Vitals					Systemic examination						Serum cholinesterase (U/L)						
																									BP			CNS												
											Vomiting	Breathlessness	Excessive salivation	Altered sensorium	Sweating	Diarrhoea	Seizures	Miosis/ pupil size(POP Score)	Bradycardia	Fasciculation (POP Score)	Tachynoea	Neck muscle weakness	Cyanosis	Pulse rate (/Minute)	Systolic (mm Hg)	Diastolic (mm Hg)	Temperature (^{0}F)	Respiratory rate (/Minute)	Mental status (POP score)	Respiratory system	Cardiovascular system	Per abdomen	GCS Score	POP Score	1	2	3				
1	715145	55	M	L	C	ORAL	100	3	65	su	P	P	A	P	P	A	A	2	P	2	P	A	A	39	110	70	99	43	1	Tc	SB	N	8	8	677	490	400	P	RF	Y	S
2	710529	50	F	L	U	ORAL	U	6	40	su	P	A	A	P	A	A	A	1	A	1	A	A	A	96	26	82	98.2	18	1	N	N	N	13	2	2315	2525	2980	A	A	Y	S
3	708021	24	M	s	MC	ORAL	80	6	30	su	A	A	P	A	A	A	A	1	P	1	A	A	A	37	100	60	98.6	20	0	N	SB	N	14	4	1885	2125	2230	A	A	n	S
4	714555	18	F	s	U	ORAL	50	5	45	su	P	P	A	A	A	A	A	2	P	2	P	A	A	52	110	56	99	40	0	Tc	SB	N	13	6	775	1890	2177	P	Psy	n	S
5	718731	72	F	f	U	ORAL	100	8	72	su	P	P	A	P	P	P	P	2	P	2	P	P	P	39	86	50	99	50	2	Tc	SB	N	6	10	114	89	53	P	CV	Y	E
6	708020	22	F	s	C	ORAL	10	3	8	su	A	A	A	A	A	A	A	A	A	A	A	A	A	108	120	80	98.6	20	0	N	T	N	14	0	5233	6167	6280	A	A	n	S
7	718687	52	F	H	DM	ORAL	80	4	55	su	P	P	P	P	A	A	A	2	P	2	P	P	A	49	90	60	98	48	2	Tc	SB	N	9	8	228	143	116	P	HT	Y	S
8	718682	36	M	f	C	ORAL	50	8	56	su	P	P	P	P	P	A	A	2	P	2	P	P	A	57	130	86	98.6	42	1	Tc	SB	N	10	7	563	1072	1178	A	A	Y	S
9	715573	35	M	f	ML	ORAL	100	9	45	su	P	P	P	P	A	A	A	1	P	2	P	A	A	64	110	70	98.2	48	1	N	N	N	7	5	667	506	224	P	ARF	n	S
10	725683	63	M	f	MC	ORAL	100	8	32	su	P	P	P	A	A	A	A	2	P	2	P	P	A	39	106	70	98	37	0	Tc	SB	N	7	8	721	850	889	P	RF	Y	E
11	779793	24	M	s	U	ORAL	U	2	6	su	A	A	A	A	A	A	A	A	A	A	A	A	A	80	120	76	98	22	0	N	N	N	13	0	2899	2955	3370	A	A	n	S
12	780520	21	F	L	m	ORAL	50	4	40	su	P	A	A	A	A	A	A	1	P	1	P	P	A	58	110	70	98.4	45	0	Tc	SB	N	11	4	871	756	655	A	A	Y	S
13	780764	30	M	f	C	ORAL	20	7	15	su	A	A	A	A	A	A	A	A	A	A	A	A	A	82	130	80	98.6	16	0	N	N	N	12	0	2750	2455	2100	A	A	n	S
14	781569	28	F	L	Fn	ORAL	75	1	40	su	P	P	A	A	A	A	A	1	P	1	P	P	A	56	120	70	98.6	34	0	Tc	SB	N	10	4	145	112	62	P	HT	Y	S
15	781685	40	F	f	C	ORAL	U	4	10	su	A	A	A	A	A	A	A	A	A	A	A	A	A	78	134	96	98	20	0	N	N	N	14	0	2922	3133	3422	A	A	n	S
16	782013	25	M	s	DV	ORAL	U	2	26	su	A	A	A	A	A	A	A	1	A	A	A	A	A	96	106	70	98.4	15	0	N	N	N	12	1	1150	1429	1655	A	A	n	S
17	782294	25	M	L	MC	ORAL	100	2	62	su	P	P	P	P	P	A	A	2	P	2	P	P	A	52	100	60	98.2	36	2	Tc	SB	N	7	8	762	461	354	P	Psy	Y	S
18	782468	24	F	s	U	ORAL	10	4	20	su	A	A	A	A	A	A	A	1	P	A	A	A	A	58	104	56	98	24	0	N	SB	N	13	2	2649	2880	3343	A	A	n	S
19	782973	56	M	f	m	ORAL	10	6	15	su	P	A	A	A	A	A	A	1	P	A	A	A	A	58	126	82	98	19	0	N	SB	N	13	2	2553	2012	-	A	A	n	S
20	783189	18	M	L	C	ORAL	U	4	30	su	P	A	A	A	A	A	A	1	P	A	A	A	A	56	110	84	98.6	20	0	N	SB	N	12	2	2490	2770	2988	A	A	n	S
21	783401	22	M	s	MC	ORAL	100	3	65	su	P	P	P	P	P	P	A	2	P	2	P	P	A	44	106	70	99	36	1	Tc	SB	N	7	8	506	421	189	P	RF	Y	E
22	783460	63	F	H	U	ORAL	20	2	12	su	A	A	A	A	A	A	A	A	A	1	A	A	A	90	124	82	98.2	20	0	N	N	N	14	1	3311	3400	4051	A	A	n	S
23	783523	35	M	f	C	ORAL	15	2	6	su	A	A	A	A	A	A	A	A	A	A	A	A	A	100	120	74	98.6	22	0	N	N	N	14	0	2551	2666	2877	A	A	n	S
24	784454	24	F	s	U	ORAL	U	5	20	su	P	A	A	A	A	A	A	A	A	1	A	A	A	88	102	66	98.6	17	0	N	N	N	11	1	2371	2556	2781	A	A	n	S
25	784667	55	M	f	m	ORAL	U	8	40	su	P	P	P	P	A	A	A	1	P	1	P	A	A	55	132	80	98.4	34	1	Tc	SB	N	11	5	1955	2188	2470	A	A	Y	S

Serial number	In patient number	Age (Years)	Sex	Occupation	Type of poison consumed	Route of exposure	Quantity consumed (mL)	Time interval from ingestion to hospitalization (Hours)	Initial bolus dose of atropine given	Intention	Clinical presentation							Clinical observation						Examination												Investigations			Complications	Various Complications	Mechanical ventilation	Outcome
																								Vitals					Systemic examination							Serum cholinesterase (U/L)						
																									BP			CNS													
											Vomiting	Breathlessness	Excessive salivation	Altered sensorium	Sweating	Diarrhoea	Seizures	Miosis/ pupil size(POP Score)	Bradycardia	Fasciculation (POP Score)	Tachynoea	Neck muscle weakness	Cyanosis	Pulse rate (/Minute)	Systolic (mm Hg)	Diastolic (mm Hg)	Temperature (^{0}F)	Respiratory rate (/Minute)	Mental status (POP score)	Respiratory system	Cardiovascular system	Per abdomen	GCS Score	POP Score	1	2	3				
26	784786	44	F	H	U	ORAL	50	1	12	su	A	A	A	A	A	A	A	1	A	A	A	A	A	98	124	76	98.2	19	0	N	N	N	12	1	2688	2850	2988	A	A	n	S
27	784805	35	M	f	MC	ORAL	50	4	16	su	A	A	A	A	A	A	A	A	P	1	P	P	A	58	130	84	99	28	0	Tc	SB	N	10	2	2642	2900	3050	A	A	n	S
28	784976	26	M	f	U	ORAL	100	5	62	su	P	P	P	P	P	A	A	2	P	2	P	P	A	45	110	60	99	32	2	Tc	SB	N	7	8	416	375	214	P	CA	Y	S
29	785533	43	M	L	Pr	ORAL	U	4	12	su	A	A	A	A	A	A	A	A	A	1	A	A	A	77	120	70	98.6	20	0	N	N	N	14	1	2991	3302	-	A	A	n	S
30	785657	25	M	L	C	ORAL	10	6	8	su	A	A	A	A	A	A	A	A	A	A	A	A	A	90	100	70	98.6	18	0	N	N	N	14	0	6288	6422	6533	A	A	n	S
31	786114	18	F	s	U	ORAL	20	8	40	su	P	P	P	P	A	A	A	1	P	1	P	P	A	56	96	60	98.6	40	1	Tc	SB	N	11	5	2054	1775	1344	A	A	n	S
32	786605	67	F	H	U	ORAL	U	5	15	su	A	A	A	A	A	A	A	A	P	1	A	A	A	58	140	90	98.4	15	0	N	SB	N	13	2	2756	2895	2910	A	A	n	S
33	786632	26	M	s	Fn	ORAL	U	4	12	su	A	A	A	A	A	A	A	A	A	A	A	A	A	102	106	70	98	17	0	N	T	N	14	0	2440	2610	2895	A	A	n	S
34	788085	25	F	s	U	ORAL	10	8	10	su	A	A	A	A	A	A	A	A	P	A	A	A	A	58	100	64	98.6	16	0	N	SB	N	14	1	4866	5022	5131	A	A	n	S
35	788281	38	F	H	U	ORAL	50	6	40	su	P	A	A	A	A	A	A	1	P	1	A	P	A	38	130	86	98.6	32	0	N	SB	N	12	4	566	452	225	P	RF	Y	S
36	788414	48	F	H	MC	ORAL	10	2	12	su	A	A	A	A	A	A	A	A	A	A	A	A	A	106	146	70	98.4	18	0	N	T	N	14	0	2590	2772	2822	A	A	n	S
37	788452	45	M	f	C	ORAL	20	6	42	su	P	P	P	P	A	A	A	1	P	1	A	P	A	54	124	80	98.2	38	1	N	SB	N	10	5	1556	1880	2050	A	A	Y	S
38	788803	45	M	f	m	ORAL	100	4	66	su	P	P	P	P	P	P	A	2	P	2	P	P	P	42	110	60	99	48	2	Tc	SB	N	6	9	562	335	265	P	HT	Y	E
39	788946	35	F	f	MC	ORAL	70	5	64	su	P	P	P	P	P	A	A	2	P	1	P	P	A	48	120	80	98.6	39	2	Tc	SB	N	7	8	446	335	287	P	Psy	Y	S
40	789082	34	M	f	U	ORAL	U	1	10	su	A	A	A	A	A	A	A	A	A	A	A	A	A	110	124	74	98.6	20	0	N	T	N	13	0	3628	3772	3929	A	A	n	S
41	789283	45	F	H	U	ORAL	10	6	15	su	A	A	A	A	A	A	A	1	A	A	A	A	A	90	116	70	98.2	16	0	N	N	N	14	0	2922	2411	2280	A	A	n	S
42	789415	45	M	f	C	ORAL	U	8	62	su	P	P	P	P	P	A	A	2	P	1	P	P	A	40	144	90	98.4	36	1	Tc	SB	N	9	6	993	690	434	A	A	Y	S
43	789604	34	M	L	MC	ORAL	20	6	32	su	P	A	A	A	A	A	A	A	P	A	A	A	A	56	112	72	98.2	19	0	N	SB	N	10	1	2371	2434	2661	A	A	n	S
44	790502	40	F	H	U	ORAL	U	6	45	su	P	A	P	A	A	A	A	1	P	1	P	A	A	50	120	72	98.6	33	0	Tc	SB	N	10	4	445	401	279	A	A	Y	S
45	790537	22	M	s	m	ORAL	50	2	50	su	P	P	P	P	P	A	A	2	P	2	P	P	A	48	108	66	98	38	1	Tc	SB	N	9	8	331	289	254	P	RF	Y	S
46	790758	52	M	f	DM	ORAL	70	4	62	su	P	P	P	P	P	A	A	2	P	2	P	P	A	44	150	90	98.4	38	2	Tc	SB	N	7	8	1045	1680	1966	P	ARF	Y	S
47	790899	22	M	s	MC	ORAL	50	4	44	su	P	P	P	A	A	A	A	2	P	1	A	P	A	58	100	60	98.2	20	0	N	SB	N	11	4	966	750	512	A	A	Y	S
48	791365	52	M	L	C	ORAL	10	4	8	su	A	A	A	A	A	A	A	A	A	A	A	A	A	86	140	90	98.6	18	0	N	N	N	14	0	2544	2665	2882	A	A	n	S
49	791613	45	M	f	m	ORAL	U	5	20	su	A	A	A	A	A	A	A	A	A	A	A	A	A	96	122	80	98.6	19	0	N	N	N	15	0	3349	3500	3622	A	A	n	S
50	791671	59	M	f	FC	ORAL	20	6	30	su	P	A	A	A	A	A	A	1	P	A	A	A	A	55	146	88	98.2	20	0	N	SB	N	12	2	2055	2282	2566	A	A	n	S
51	792571	52	M	f	MC	ORAL	20	4	32	su	P	A	A	A	A	A	A	1	A	1	A	A	A	102	136	76	98.4	20	0	N	T	N	11	2	1988	1086	-	A	A	n	S
52	792593	19	M	s	U	ORAL	100	4	65	su	P	P	P	P	P	A	A	2	P	2	P	P	A	38	100	60	98.4	42	2	Tc	SB	N	7	9	377	354	208	P	ARDS	Y	E
53	792902	46	F	H	U	ORAL	U	5	25	su	P	A	A	A	A	A	A	A	P	A	A	A	A	55	130	70	98.2	19	0	N	SB	N	12	2	1345	1455	1540	A	A	n	S
54	793162	21	M	s	DZ	ORAL	10	7	45	su	P	P	P	A	A	A	A	1	P	1	P	P	A	50	98	62	98.6	28	0	Tc	SB	N	10	4	689	422	295	A	A	Y	S
55	793251	35	M	L	U	ORAL	U	4	14	su	A	A	A	A	A	A	A	A	A	A	A	A	A	80	120	70	98.6	18	0	N	N	N	12	0	3872	3998	4021	A	A	n	S
56	793439	45	M	f	C	ORAL	U	5	30	su	P	A	A	A	A	A	A	1	P	1	A	A	A	54	136	80	99	19	0	N	SB	N	13	3	2267	2420	2560	A	A	n	S

Serial number	In patient number	Age (Years)	Sex	Occupation	Type of poison consumed	Route of exposure	Quantity consumed (mL)	Time interval from ingestion to hospitalization (Hours)	Initial bolus dose of atropine given	Intention	Clinical presentation							Clinical observation						Examination												Investigations			Complications	Various Complications	Mechanical ventilation	Outcome
																								Vitals					Systemic examination							Serum cholinesterase (U/L)						
																									BP			CNS													
											Vomiting	Breathlessness	Excessive salivation	Altered sensorium	Sweating	Diarrhoea	Seizures	Miosis/ pupil size(POP Score)	Bradycardia	Fasciculation (POP Score)	Tachynoea	Neck muscle weakness	Cyanosis	Pulse rate (/Minute)	Systolic (mm Hg)	Diastolic (mm Hg)	Temperature (^{0}F)	Respiratory rate (/Minute)	Mental status (POP score)	Respiratory system	Cardiovascular system	Per abdomen	GCS Score	POP Score	1	2	3				
57	794090	20	F	s	U	ORAL	20	5	25	su	P	A	A	A	A	A	A	1	P	A	A	A	A	58	100	60	98	20	0	N	SB	N	12	2	1211	1500	1977	A	A	n	S
58	794222	66	M	f	C	ORAL	10	4	10	su	A	A	A	A	A	A	A	A	A	A	A	A	A	110	144	78	98.2	20	0	N	T	N	14	0	5223	5741	6250	A	A	n	S
59	794528	21	F	s	MC	ORAL	50	9	50	su	P	P	P	P	A	A	A	2	P	1	P	P	A	42	114	70	98.4	30	1	Tc	SB	N	8	6	650	886	1028	P	Psy	Y	S
60	794563	41	M	f	m	ORAL	U	6	40	su	P	P	A	A	A	A	A	1	P	A	P	P	A	45	130	80	98.4	28	0	Tc	SB	N	7	3	1633	1852	2022	A	A	Y	S
61	794774	42	F	H	MC	ORAL	50	2	26	su	P	A	A	A	A	A	A	1	A	1	A	A	A	95	120	70	98.6	19	0	N	N	N	11	2	2199	2320	2550	A	A	n	S
62	795098	21	F	s	U	ORAL	U	5	20	su	A	A	A	A	A	A	A	1	P	A	A	A	A	84	104	56	98.2	20	0	N	SB	N	13	2	1755	1990	2016	A	A	n	S
63	796281	50	M	f	C	ORAL	U	6	35	su	P	P	A	A	A	A	A	1	P	1	A	A	A	36	136	70	98.4	20	0	N	SB	N	12	4	1456	2075	2220	A	A	n	S
64	796622	32	M	f	Fn	ORAL	100	6	66	su	P	P	P	P	P	A	A	2	P	2	P	P	A	40	120	66	99	34	2	Tc	SB	N	7	8	749	423	388	P	RF	Y	E
65	796703	48	M	L	MC	ORAL	120	8	70	su	P	P	P	P	P	P	P	2	P	2	P	P	P	35	134	70	98.6	48	2	Tc	SB	N	5	10	580	-	-	P	CV	Y	E
66	796808	18	F	s	m	ORAL	10	5	6	su	A	A	A	A	A	A	A	A	A	A	A	A	A	106	100	60	98.6	18	0	N	T	N	13	0	4977	5005	5200	A	A	n	S
67	796825	50	M	f	U	ORAL	U	4	20	su	P	A	A	A	A	A	A	A	A	A	A	A	A	88	120	74	98.6	20	0	N	N	N	14	0	3544	3656	3999	A	A	n	S
68	797261	43	M	f	C	ORAL	30	6	40	su	P	P	A	A	A	A	A	1	P	1	P	P	A	94	106	60	98.4	38	0	Tc	N	N	10	4	882	1224	1435	A	A	Y	S
69	797287	52	M	f	MC	ORAL	U	4	15	su	A	A	A	A	A	A	A	A	P	A	A	A	A	75	146	80	98.2	19	0	N	N	N	13	0	3421	4220	4523	A	A	n	S
70	797359	35	M	L	U	ORAL	10	4	18	su	P	A	A	A	A	A	A	1	A	A	A	A	A	110	120	70	98.4	20	0	N	T	N	13	1	2166	2080	1770	A	A	n	S
71	798406	23	M	s	C	ORAL	30	6	30	su	P	A	A	A	A	A	A	1	P	A	A	A	A	54	110	70	98.6	20	0	N	SB	N	12	2	1550	1820	1920	A	A	n	S
72	798712	21	F	s	U	ORAL	50	5	42	su	P	P	P	A	A	A	A	1	P	1	P	P	A	51	114	80	98.6	34	0	Tc	SB	N	10	4	2226	2551	-	A	A	n	S
73	799777	52	M	f	C	ORAL	100	4	62	su	P	P	P	P	P	A	A	2	P	2	P	P	A	45	140	94	99	38	2	Tc	SB	N	7	8	333	309	215	P	Psy	Y	S
74	801483	21	F	s	m	ORAL	100	4	65	su	P	P	P	P	A	P	A	2	P	1	P	P	A	40	100	60	98.6	40	2	Tc	SB	N	7	8	143	117	89	P	RF	Y	E
75	801865	24	M	s	Pr	ORAL	U	6	45	su	P	P	P	A	A	A	A	1	P	1	A	P	A	55	98	60	98.4	38	0	Tc	SB	N	8	4	779	922	991	P	ARDS	n	S
76	802692	57	F	H	MP	ORAL	20	6	35	su	P	A	A	A	A	A	A	1	P	A	A	A	A	54	140	80	98.2	20	0	N	SB	N	10	2	1654	1880	1974	A	A	n	S
77	802713	35	M	L	MC	ORAL	50	6	48	su	P	P	P	P	A	A	A	2	P	2	P	P	A	39	130	76	98.4	28	1	Tc	SB	N	7	8	1889	2007	2155	P	Psy	Y	S
78	803201	45	M	f	C	ORAL	20	6	30	su	P	A	A	A	A	A	A	1	P	1	A	A	A	52	134	80	98.6	18	0	N	SB	N	11	4	2544	2883	2930	A	A	n	S
79	803641	51	F	H	U	ORAL	U	5	22	su	A	A	A	A	A	A	A	1	A	A	A	A	A	72	130	76	98.6	19	0	N	N	N	11	1	3325	3340	3933	A	A	n	S
80	803688	64	M	f	C	ORAL	10	4	8	su	A	A	A	A	A	A	A	A	A	A	A	A	A	108	140	88	98	16	0	N	T	N	14	0	2350	-	-	A	A	n	S
81	803712	25	M	s	m	ORAL	50	4	30	su	P	A	A	A	A	A	A	1	P	1	A	A	A	40	100	66	98.2	17	0	N	SB	N	11	4	2376	2533	2850	A	A	n	S
82	803945	56	M	f	Fn	ORAL	U	8	40	su	P	P	P	A	A	A	A	2	P	1	A	P	A	45	130	74	98.6	36	0	N	SB	N	7	4	1392	1665	1950	P	ARF	Y	E
83	804111	22	F	s	MC	ORAL	100	5	64	su	P	P	P	P	A	A	A	2	P	2	P	P	A	38	110	82	98.8	42	2	Tc	SB	N	7	9	708	667	431	P	CA	Y	E
84	804576	45	M	L	C	ORAL	10	6	15	su	A	A	A	A	A	A	A	A	P	A	A	A	A	59	120	74	98.6	19	0	N	SB	N	14	1	1220	1422	1560	A	A	n	S
85	804971	61	M	f	U	ORAL	10	9	34	su	P	A	A	A	A	A	A	2	P	1	A	A	A	56	130	82	98.2	16	0	N	SB	N	11	4	1487	1650	1890	A	A	n	S

Serial number	In patient number	Age (Years)	Sex	Occupation	Type of poison consumed	Route of exposure	Quantity consumed (mL)	Time interval from ingestion to hospitalization (Hours)	Initial bolus dose of atropine given	Intention	Clinical presentation							Clinical observation						Examination																Investigations					
																								Vitals					Systemic examination											Serum cholinesterase (U/L)					
																									BP				CNS																	
											Vomiting	Breathlessness	Excessive salivation	Altered sensorium	Sweating	Diarrhoea	Seizures	Miosis/ pupil size(POP Score)	Bradycardia	Fasciculation (POP Score)	Tachynoea	Neck muscle weakness	Cyanosis	Pulse rate (/Minute)	Systolic (mm Hg)	Diastolic (mm Hg)	Temperature (^{0}F)	Respiratory rate (/Minute)	Mental status (POP score)	Respiratory system	Cardiovascular system	Per abdomen	GCS Score	POP Score	1	2	3	Complications	Various Complications	Mechanical ventilation	Outcome
86	806412	44	F	H	C	ORAL	U	6	25	su	A	A	A	A	A	A	A	1	P	1	A	A	A	35	126	70	98.2	18	0	N	SB	N	14	4	2337	2511	2737	A	A	n	S
87	806989	42	F	L	MC	ORAL	U	4	15	su	A	A	A	A	A	A	A	A	A	A	A	A	A	65	132	80	98.6	20	0	N	N	N	13	0	4823	4834	5550	A	A	n	S
88	807361	19	M	s	m	ORAL	20	8	30	su	P	A	A	A	A	A	A	1	P	A	A	A	A	55	104	66	98.6	20	0	N	SB	N	11	2	990	1108	1239	A	A	n	S
89	807705	23	M	L	C	ORAL	50	5	42	su	P	A	P	A	A	A	A	1	P	1	P	P	A	52	100	70	98.6	30	0	Tc	SB	N	10	4	776	542	368	P	HT	Y	S
90	807944	28	M	L	Pr	ORAL	100	4	68	su	P	P	P	P	P	A	A	2	P	2	P	P	P	46	110	76	99	38	2	Tc	SB	N	7	8	458	377	153	P	HT	Y	E
91	808253	42	M	f	MC	ORAL	U	4	30	su	P	A	A	A	A	A	A	1	A	A	A	A	A	106	134	84	98.2	18	0	N	T	N	11	1	1775	992	-	A	A	n	S
92	808899	52	F	H	m	ORAL	10	6	20	su	A	A	A	A	A	A	A	A	A	A	A	A	A	70	120	70	98.2	19	0	N	N	N	10	0	1890	1950	2100	A	A	n	S
93	809124	41	M	f	U	ORAL	U	4	6	su	A	A	A	A	A	A	A	A	A	A	A	A	A	65	134	84	98	20	0	N	N	N	14	0	3933	4008	4199	A	A	n	S
94	809477	20	F	s	MC	ORAL	50	5	40	su	P	P	P	A	A	A	A	1	P	1	P	P	A	56	122	68	98.6	32	0	Tc	SB	N	9	4	2670	1880	1540	P	ARF	n	S
95	809490	35	M	L	C	ORAL	U	7	62	su	P	P	P	P	P	A	A	2	P	1	P	P	A	42	130	76	98.4	34	2	Tc	SB	N	7	6	556	-	-	P	Psy	Y	E
96	810202	38	M	L	Pr	ORAL	U	8	52	su	P	P	P	P	A	A	A	2	P	1	P	P	A	45	126	80	98.6	30	1	Tc	SB	N	7	6	1004	975	776	P	RF	Y	S
97	810565	64	F	f	MC	ORAL	20	4	22	su	P	A	A	A	A	A	A	1	A	A	A	A	A	72	140	84	98.2	20	0	N	N	N	13	1	2261	2556	2672	A	A	n	S
98	810788	21	M	L	MC	ORAL	10	6	12	su	A	A	A	A	A	A	A	A	P	A	A	A	A	68	100	66	98.2	20	0	N	N	N	14	0	4311	4689	4930	A	A	n	S
99	811232	55	M	L	C	ORAL	U	6	30	su	P	A	A	A	A	A	A	1	P	A	A	A	A	56	120	76	98.6	18	0	N	SB	N	11	2	2544	2667	2890	A	A	n	S
100	811453	24	M	L	Fn	ORAL	10	7	15	su	P	A	A	A	A	A	A	A	P	A	A	A	A	58	104	70	98.6	19	0	N	SB	N	12	1	3056	3099	3112	A	A	n	S

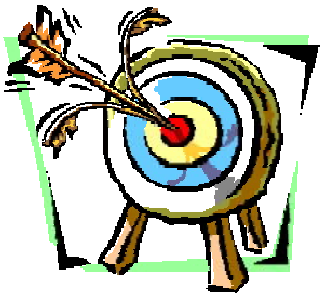
ANNEXURE-IV

KEY TO MASTER CHART

^{0}F	-	Degree Fahrenheit
A	-	Absent
ARF	-	Acute renal failure
ARDS	-	Acute respiratory distress syndrome
BP	-	Blood pressure
C	-	Chlorpyrifos
CA	-	Cardiac arrhythmia
CNS	-	Central nervous system
CV	-	Convulsions
DM	-	Dimethoate
DV	-	Dichlorvos
DZ	-	Diazinon
E	-	Expired
ECG	-	Electrocardiogram
F	-	Female
f	-	Farmer
Fn	-	Fenthion
FC	-	Fenchlorophos
H	-	Housewife
HT	-	Hypotension
L	-	Labourer
m	-	Malathion

M	-	Male
MC	-	Monocrotophos
mL	-	Millilitre
mm Hg	-	Millimeters of mercury
MP	-	Methylparathion
n	-	No
N	-	Normal
P	-	Present
POP	-	Peradenya Organophosphorus Poisoning
Pr	-	Parathion
Psy	-	Psychosis
RF	-	Respiratory failure
s	-	Student
su	-	Suicidal
S	-	Survived
SB	-	Sinus bradycardia
T	-	Tachycardia
Tc	-	Tachypnoea
U	-	Unknown
U/L	-	units per liter
Y	-	Yes

Introduction



Objectives

Review of Literature

Methodology

Results

Discussion

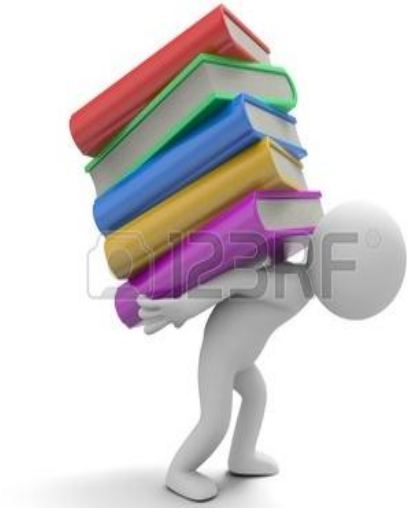
Conclusion

Summary

Bibliography

Annexure-I

Annexure-II



Annexure-III

Annexure-IV