PERADENIYA ORGNOPHOSPHOROUS SCALE (POP) AS A PREDICTOR OF MORTALITY IN ORGANOPHOSPHOROUS POISONING

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Dissertation submitted to

BLDE (Deemed to be University) Vijayapur, Karnataka

In partial fulfillment of the requirements for the degree of

DOCTOROFMEDICINE

IN

GENERAL MEDICNE

Under the guidance of

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DEPARTMENTOFGENERAL MEDICNE

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HOSPITAL & RESEARCH CENTRE, VIJAYAPUR

KARNATAKA

2020

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TITLE OF THE TOPIC

"PERADENIYA ORGANOPHOSPHOROUS SCALE(POP) AS A PREDICTOR OF MORTALITY IN ORGANOPHOSPHOROUS POISOINING"

MD IN GENERAL MEDICINE

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ABSTRACT

INTRODUCTION

Organophosphorous chemical poisoning is a significant cause for emergency admission in the many hospitals in India. OP compounds are employed as insecticides, herbicides, and nerve gases as chemical warfare agents . The likelihood of poisoning has increased due to these substances widespread availability. Indian studies show a mortality rate of 4–30%. The most frequent consequence of OP poisoning that results in mortality is respiratory failure. Survival may be increased with early detection and appropriate ventilator assistance. India has 48,000 ventilators and around 95,000 ICU beds . All OP poisoning victims are not treated in ICUs in the Indian system due to a lack of resources. Therefore, it's crucial to identify at the initial assessment any clinical traits or criteria that could indicate the necessity for ventilator assistance. In the Indian context, little research has been done on the Peradenya OP compound scale . Identifying the early requirement for ventilator support may be a quick and easy process. Patients who score highly on the POP scale have increased morbidity and death, according to a study by Senayeke et al.

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LIST OF ABBREVIATIONS

Ach	Acetylcholine
anti-ChE	Anticholinesterase
DDT	Dichloro diethyl trichloro ethane.
EDRF	Endothelium-derived relaxing factor
x^2	Chi square value
HS	Highly significant
IMS	Intermediate syndrome
IV	Intravenous
No	Number
OP	Organophosphorus
OPC	Organophosphorus compound
OPIDP	Organophosphate induced delayed polyneuropathy
2-PAM	Pralidoxime
PChe	Pseudocholinesterase
POP scale	Peradeniya Organophosphorus poisoning scale
S	Significant.
TEPP	Tetraethyl pyrophosphate
TOCP	Organophosphate triortho cresylphosphate

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INTRODUCTION

Organophosphorus poisoning reached epidemic numbers, with over 3 million cases reported annually in 1990. Poisoning by organophosphorus compounds is largely a problem in underdeveloped countries[1]. The most common medical condition is organophosphorus chemical poisoning. India is experiencing a toxic emergency**.** Organophosphorus compound poisoning is a serious condition. In most hospitals across the country, an essential indication for emergency admission isIndia[2]. Schrader was the first to produce organophosphorus compounds shortly before and during WWII. They were first used as an agricultural pesticide before being considered as possible chemical warfare agents[2]. Organophosphorus Pesticides, herbicides, and chemical warfare agents all contain (OP) chemicals. Nerve gases are a type of nerve gas[2]. Its popularity has grown due to its extensive use and accessibility. The possibility of toxicity from these chemicals Although poisoning is a possibility, Most incidences of suicidality result from occupational exposure or unintentional consumption intent. Because of their widespread availability, OP pesticide poisoning has become a global problem and public health issue that affects many number of people.

India is a tropical nation whose economy is based primarily on agriculture. More than 60% of Indians are farmers. Due to their easy availability and low cost, pesticides are the most frequent dangerous substances to which farmers are exposed, with OPCompound being the most prevalent in addition to accidental intoxication from using these substances as agricultural insecticides[3]. These substances are also used for homicidal and suicidal purposes.

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Pesticide poisoning kills more than 220,000 people per year, according to the World Health Organization. Toxicity and fatality rates in developing nations such as India and Sri Lanka are alarmingly high [4].

Organophosphates cause cholinergic overactivity by irreversibly blocking cholinesterase, leading to increase of acetylcholine at synapses and neuromuscular junctions. Organophosphorus chemicals have been shown to have a direct cardiotoxic effect[1, 4.]

In Indian research, mortality rates range from 4 to 30%. The most common complication of Organophosporous poisoning that leads to death is respiratory failure. Early detection and ventilatory assistance increase the chance of survival. In the Indian system, all Organophosphorous poisoining cases are not treated in ICUs because of lack of resources. As a result, clinical characteristics and criteria for predicting the requirement for ventilatory support must be recognized at the initial assessment.

After OP poisoning, serum cholinesterase levels are easier to measure and are usually low. In the Indian context, the Peradeniya OP poisoning scale has gotten little attention to determine its necessity, it might be a simple and effective technique early on in the course for ventilatory assistance As a result, this research was done to determine the degree of organophosphorus chemical toxicity from both a clinical and an epidemiological standpoint calculating serum choline esterase levels and utilizing Peradeniya scoring.

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OBJECTIVES

To study the role of Paradeniya Organophosphorous Poisoining(POP) Scale in predicting mortality in organophosphorous poisoining

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REVIEW OF LITERATURE

Organophosphorus poisoning

History:

Ten years prior to the discovery of physostigmine [2], Clermont provided the first description of the synthesi of tetraethyl pyrophosphate (TEPP), a highly potent compound of the organophosphorus anticholinesterase (anti-ChE) class. Homestead noted a century later that it was uncommon that the investigator lived long enough to report on the flavour of the substance. Organophosphate triorthocresylphosphate (TOCP) [6] was added to a popular treatment in the 1930s, which caused thousands of individuals in the Caribbean to experience "Jamaican ginger paralysis." The first work on the synthesis of diethyl and dimethyl phosphoflouridates was written by Lange and Krueger in 1932, and it served as the impetus for contemporary research on organophosphorus compounds. After experiencing a persistent choking sensation and blurred vision after inhaling these substances, Schrader decided to test this group of chemicals for insecticidal activity. After synthesising nearly 2000 compounds, Schrader[2] (1952) identified the structural requirements for insecticidal (and, as was later discovered, anti-ChE) action. One of the earliest substances in this series, parathion (a phosphorothioate), later developed into the most widely applied insecticide in this category. Organophosphates have gained a lot of popularity due to their efficiency.

Because of their efficiency as insecticides and lack of environmental accumulation, organophosphates have become quite popular. They break down into harmless radicals within 4 days of treatment due to their fragile chemical nature. They have replaced DDT as the preferred insecticide agent since they do not accumulate in the body or environment like DDT and other organochloride compounds do.

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These chemicals are mostly used as pesticides ,mainly insectides in agriculture. Some compounds are used in human and veterinary practise. Organophosphorus compounds have been used as lubricants, flame retardants also. The invention and deployment of certain of these chemicals as highly effective warfare agents have worldwide implications.

Table 1: Various Sources of organophosphorus pesticides[7]

Domestic • Garden sheds—insecticidal preparations, fertilizers. • Surface and room sprays. • Baits for cockroaches and insects (ex chlorpyrifos) • Shampoos against head lice (for example, malathion) • Pet preparations (ex: pet washes, collars)
Industrial or occupational • Crop protection and livestock dipping • Fumigation
Terrorism or warfare (nerve agents)

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ANATOMY & PHYSIOLOGY OF AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system is the part of the nervous system that controls the body's visceral functions. This aids in the regulation of arterial pressure, gastric motility, secretions, urine bladder control, perspiration, and body temperature, among other things. The autonomic nervous system's ability to change visceral function quickly and intensely is one of its most noticeable features.

The hypothalamus, brain stem, and spinal cord all have autonomic nervous system centers.

Sympathetic**:** Spinal cord -Segments: T1 – L1

Pre - vertebral ganglia - coeliac and hypogastric plexus

Parasympathetic**:** central nervous system – Via III, VIII, IX, X cranial nerves [8]

Spinal cord - S2, S3 and S4 nerves

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NEUROMUSCULAR JUNCTION

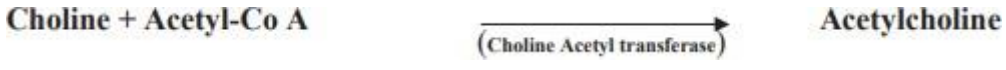
ACETYLCHOLINE:

Acetylcholine (Ach), a neurotransmitter, was created for the first time by BAYER in 1867. HUNT identified it as a powerful pharmacological agent for the first time in 1906**.**

Acetylcholine is produced in the following sites:

a) Autonomic effector sites innervated by postganglionic parasympathetic fibers.
b) Preganglionic autonomic fibers of sympathetic and parasympathetic ganglion cells and adrenal medulla.
c) Motor end-plates on skeletal muscle .
d) Synapses in the central nervous system**.**

The Ach is made from choline and coenzyme A (CoA) in the axoplasm, with the help of the enzyme choline acetyltransferase. Choline, which enters the nerve terminal via a carrier-mediated transport pathway, is derived from the extracellular fluid.



Around 20% of the Ach in the nerve terminal is found loose in the axoplasm, and the remaining 80% is housed in vesicles, each of which holds about 4-5 x 10^5 molecules of Ach.

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There are many Ach reservoirs or pools within the nerve terminal. Nerve impulses can release the Ach's releasable pool (80%), but some cannot (the non-releasable pool or stationary pool). The 7 Ach located inside the vesicles make up the releasable pool, whereas the Ach in the axoplasm make up the non-releasable pool. It is common practise to divide the releasable pool into two categories: instantly available and reserved..

Figure No:1 NEUROCHEMISTRY OF AUTONOMIC PATH[9]

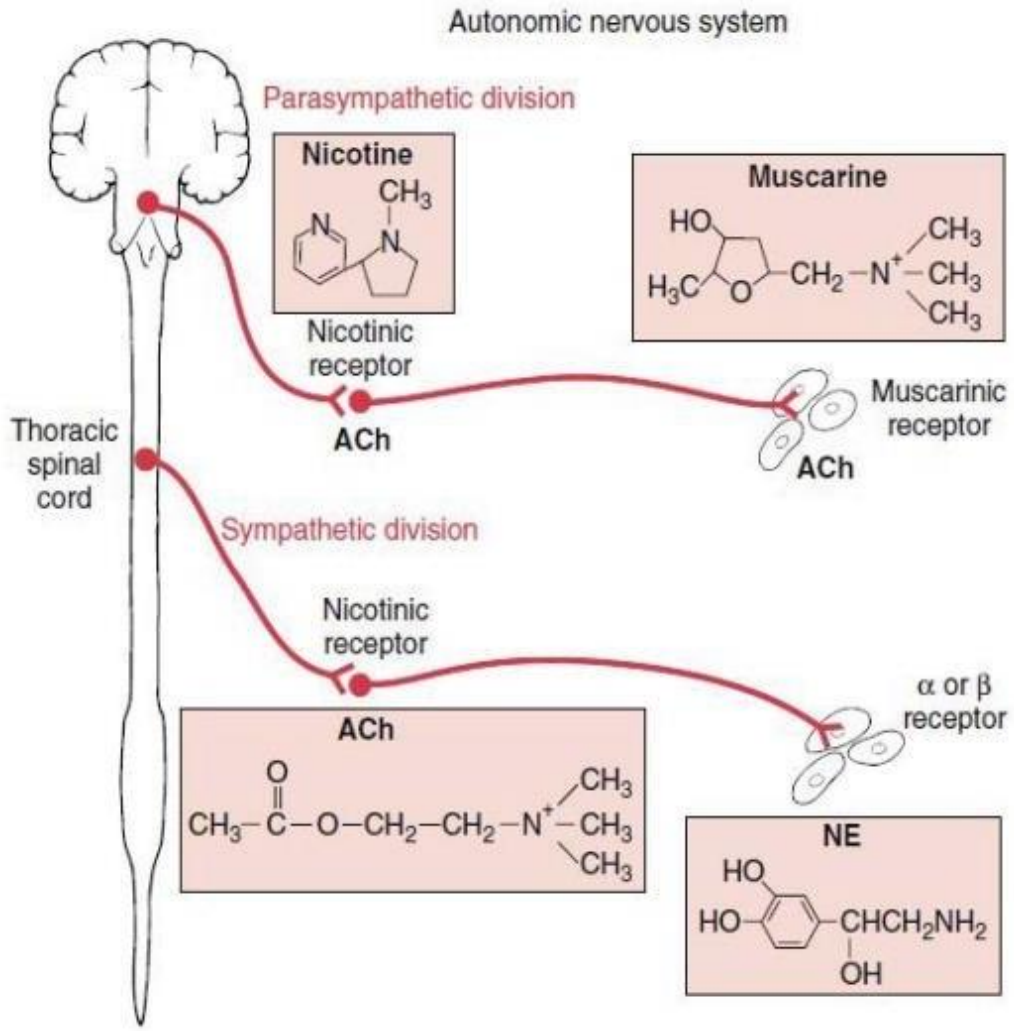

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Figure No2- THE ORGAN SPECIFIC ARRANGEMENT OF ANS[9]

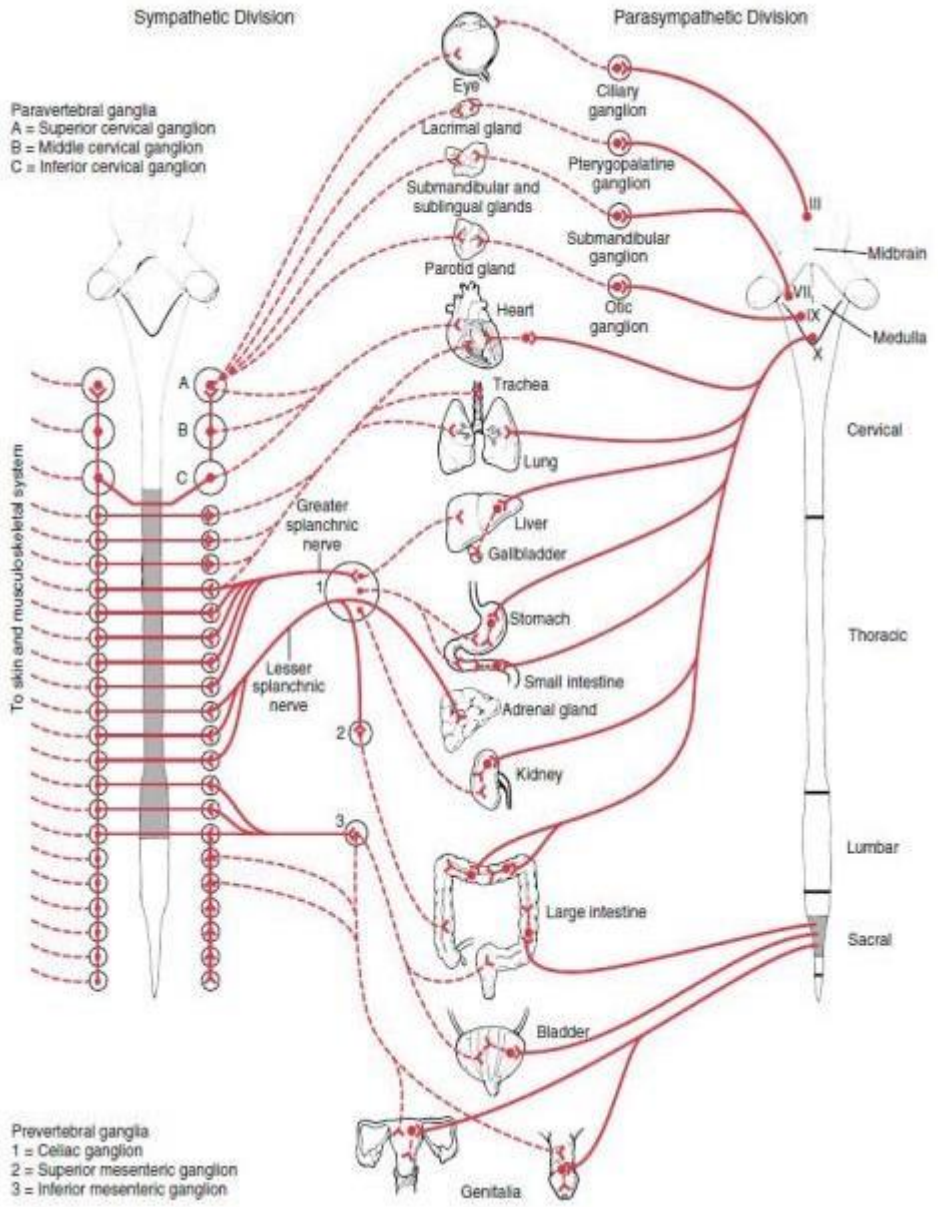

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Table2.EFFECTS OF DIRECT-ACTING CHOLINOCEPTOR STIMULANTS[10]

ORGAN	RESPONSE
Eye	
Sphincter muscle of iris	Contraction (miosis)
Ciliary muscle	Contraction for near vision
Heart	
SA node	Decrease in rate
Atria	Decrease in contractile strength
AV node	Decrease in conduction velocity
Ventricles	Decrease in contractile strength
Blood vessels	
Arteries	Dilation, Constriction
Veins	Dilation, Constriction
Lung	
Bronchial muscle	Contraction
Bronchial glands	Stimulation
Gastrointestinal tract	
Motility	Increases
Sphincters	Relaxation
Secretions	Stimulation
Urinary bladder	
Detrusor	Contraction
Trigone and Sphincter	Relaxation
Glands	
Salivary, lacrimal	Secretion
Nasopharyngeal	Secretion

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Acetylcholine acts via two different types of receptors:

Muscarinic receptors :

A toad faeces toxin called muscarine specifically stimulates muscarinic receptors in the body. Postganglionic neurons of the parasympathetic nervous system and postganglionic cholinergic neurons of the sympathetic nervous system stimulate effector cells.

Nicotinic receptors :

Nicotinic receptors are activated by nicotine in pre- and postganglionic neurons of the sympathetic and parasympathetic nervous systems, as well as in the membranes of skeletal muscle fibres at the neuromuscular junction.

Metabolism of Acetyl choline:

The junctional acetylcholinesterase is the enzyme that breaks down Ach in the synaptic cleft. The muscle's basement membrane and possibly the motor end plate and nerve membrane terminals are related to the protein acetyl cholinesterase. A number of molecules can be bound and hydrolyzed by a single enzyme molecule. molecules of acetylcholine When a nerve impulse is released, at least 10 active enzyme sites are thought to be available for each molecule of Ach. Each Ach molecule only interacts with the substrate once thanks to this architecture. It then proceeds to quickly hydrolyze (in less than 1msec).

TYPES OF CHOLINESTERASES:

Two forms of cholinesterase are present in vertebrates which break down acetyl [12]choline.

Serum cholinesterase: (Pseudo cholinesterase- or Butyryl Cholinesterase)

It is found in the intestinal mucosa, liver, pancreas, and plasma. The main organ is the liver. Variations are brought on by liver disease, chronic

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inflammation, malnutrition, usage of morphine, codeine, succinylcholine, and hypersensitivity reactions**.**

RBC cholinesterase: (True, Specific Cholinesterase)

It is found in erythrocytes, grey matter, lungs, spleen, and erythrocytes. Acetylcholine is rendered inactive by interacting with the anionic and esteratic sites on the RBC cholinesterase enzyme. It falls in those with pernicious anaemia and after using anti-malarial drugs. RBC cholinesterase can be measured more accurately than serum cholinesterase. [53]

Anionic site:- Its negative charge makes it attractive to the quaternary nitrogen ion + of acetylcholine (N).

Esteratic site: - It draws the carboxyl group of the acetylcholine molecule, which acetylates the enzyme's esteratic site and causes choline to split.

The acetyl group and esteratic site are immediately removed when mixed with water, yielding acetic acid. This liberates the esteratic site of the enzyme, enabling further acetyl choline inactivation.

PHARMACOLOGY: [2,5,13]

Organophosphorus insecticides are a chemical family with a wide range of structures. They are mainly phosphoric or phosphonic acid ester, amide, or thiol derivatives, with the general formula being:

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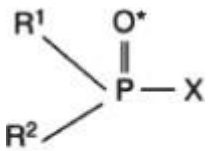

*or S

The alkyl or aryl groups that R1 and R2 can be allow for a variety of replacements. Any of a broad variety of substituted or branched aliphatic, aromatic, or heterocyclic groups linked to phosphorus via a liability bond, typically -O- or -S-, can serve as the "leaving group," or group X. The double bond atom in phosphorothioate and phosphate is either O or S. Since it is inherently more stable, the P=S form is often used in the synthesis of insecticides. This form can subsequently be converted in vivo into the biologically active form, oxon.

Because phosphates are more volatile and directly poisonous, converting phosphothioates to phosphates can be harmful. The relative tightness of the connection to acetylcholinesterase, the duration necessary for hydrolysis, potency, and the onset of symptoms are all influenced by the numerous attached groups to the various sites of phosphorus or sulphur moiety.

The majority of organophosphate pesticides are readily absorbed across all modes of administration: cutaneous, respiratory, gastrointestinal, and conjunctival. Organophosphate is classed as a direct cholinesterase inhibitor or an indirect cholinesterase inhibitor that must be converted to an active metabolite[13,14]. Plasma half life varies from a few minutes to a few hours depending on the substance and the method of delivery. after a single injection. Metabolism is mostly accomplished through oxidation, esterase hydrolysis, and the transfer of a portion of the molecule to glutathione.

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The majority of organophosphorus chemicals are eliminated in the urine almost exclusively as hydrolysis products. Within 48 hours, 80-90 percent of the bacteria are gone. In the urine, only a small percentage of organophosphorus chemicals and their active forms (oxons) are unaltered. Some compounds (fenthion, fenithrothion) have been shown to last longer in the body.

Pharmacokinetics:

The skin, mucous membranes, conjunctiva, gastrointestinal system, and lungs absorb most organophosphorus chemicals quickly and effectively. In the liver, cytochrome P450-mediated monooxygenases detoxify these substances.

However, several metabolites formed during the conversion of parathion, diazinon, and malathion to oxons are more dangerous than the original compounds.

Table 3: Classification of Insecticides[12]:

OrganoChlorine Compounds	**OrganoPhosphorus Compounds**	**Carbamates**
Methoxychlor DDT	Chlorthion	Carbaryl
HCH (BHC) Lindane	Diazinon	Pyrolan
Chlordene Hepatochlor	Dioxathion	Dimetilan
Dieldrin	Dimethoate	Propoxur
Aldrin	EPN	Synthetic Pyrethroids
	Malathion (OMS-1)	
	Fenthion (OMS – 2)	
	Methylparathion	
	Parathion	
	Ronnel Trichlorfos	
	Dichlorvos, Chlorpyrifos	

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Table 4- FREQUENTLY ENCOUNTERED ORGANO PHOSPHORUS POISONINGS IN HOSPITAL EMERGENCIES:[12,16]

GENERIC NAME	TRADE NAME
Monocrotophos	Nuvacron, Huvacron
Endosulphan	Endrin, Jayasulphan
Fenthion	Baytex, Lebaycid, Fenthiosul
Fenitrothion	Tik-20, Folithion, Agrothion, Vikathion
Malathion	Finit, vegfru, Malatox, Agromal, Cythion
Methyl Parathion	Kathion, Maladan, Malazene, Sulmathion
Metapar	Licel
Phorate	Agrotex, Parahit, Metacid, Folidol-M
Oxydemeton	Dragnet, Phoratox
Methyl Quinalphos	Knock Out, Metasystox

Clinical manifestations: Three groups can be made out of the cholinesterase poisoning symptoms[4,13,17,18].

1. Muscarinic: Postganglionic parasympathetic manifestations

2. Nicotinic: Autonomic ganglionic and somaticomotor (NMJ) effects

3.Central: Central nervouso system effects

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Table 5 – Muscarinic manifestations:

Bronchial tree	Tightness in chest, wheezing, Dyspnoea increased bronchial secretion, cough, pulmonary edema, cyanosis
Gastro intestinal system	Nausea, vomiting, abdominal tightness, cramps, diarrhea, tenesmus, fecal incontinence
Sweat glands	Increased sweating
Salivary glands	Increased salivation
Lacrimal glands	Increased secretion
Cardiovascular system	Bradycardia, hypotension
Pupils	Miosis
Ciliary body	Blurring of vision
Bladder	Frequency, urinary Incontinence

Table 6 – Nicotinic manifestations:

Striated muscle	Muscle twitching, Fasciculations, cramps, weakness, respiratory muscle weakness
Sympathetic ganglion	Pallor, tachycardia, hypertension.

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Central nervous system manifestations:

Excessive dreaming, insomnia, headache, tremor, depression, drowsiness, confusion, slurred speech, generalised weakness, coma with absent reflexes, type I paralysis, Cheyne' Stokes respiration, convulsions, depression of respiratory and circulatory centres with dyspnoea, cyanosis, and drop in blood pressure .[5,19]

Acute cholinergic crisis: An increase in secretions (such as bronchorrhea, salivation, sweating, and tears), bronchoconstriction (such as chest tightness and wheezing), vomiting, and an increase in gastrointestinal motility are all brought on by the buildup of acetylcholine at muscarinic sites (abdominal tightness and cramps). Organophosphorus substances cause diagnostic miosis and vision blur in the eye.

Increased refractory period and conduction time in the sinoatrial and atrioventricular nodes, decreased effective refractory period of atrial muscle fibers, and perhaps coronary blood flow restriction is the main cardiac effects[13].

Muscle fasciculations, cramping, weakness, and diaphragmatic paralysis are all symptoms of nicotine poisoning, which can lead to paralysis, areflexia, and respiratory failure. Hypertension, tachycardia, papillary dilatation, and pallor are all autonomic consequences. Nicotinic actions on the electrophysiological system may cause a prolonged depolarization block.

Restlessness, emotional lability, headache, tremor, drowsiness, disorientation, slurred speech, ataxia, widespread weakness, respiratory depression, delirium, convulsions, coma, and death are all central nervous system consequences.

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During this early cholinergic phase, impacts on the heart (bradycardia and other arrhythmias), breathing (central or peripheral respiratory failure), and brain (depression of important centres) are likely to result in death. Normally, the cholinergic phase lasts between 24 and 48 hours.

Intermediate syndrome (Wadia type II syndrome)[5,13,17,20,21]:

Some individuals acquired muscle paralysis after recovering from cholinergic crisis but before the predicted onset of delayed polyneuropathy, a condition known as "Intermediate syndrome" (IMS). The condition usually appears within 24-96 hours following ingestion. The patient is normally awake.

There are no fasciculations or other cholinergic symptoms. Acute respiratory paralysis, bulbar musculature weakness, nuchal weakness, proximal limb weakness, and depressed or normal tendon reflexes are seen in the patient.

The most usually impacted muscles are the external ocular muscles. Muscle weakness is the most common symptom of this disease, which mostly affects the neck flexors and proximal limb muscles. The flaw is symmetrical on both sides.

As a result, minor irregularities may be ignored. Over the course of around 6 hours, respiratory insufficiency develops. The patient may become unconscious and die if left ignored. Recovery can take 2-4 times as long as development, ranging from 4 to 18 days.

When cholinesterase activity is too low and organophosphate chemicals are found in the body, the symptom complex develops. Treatment of the acute cholinergic crisis with atropine and oximes may have been successful. In the

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presence of insufficient circulating oxime, the motor endplate may be rechallenged with the cholinesterase inhibitor as the organophosphate blood level lowers and tissue redistribution occurs.

There may be substantial stereochemistry associated with the effect of oximes on the neuromuscular junction because IMS appears to develop preferentially with specific organophosphorus chemicals like fenthion (fat-soluble and sustained toxicity).

According to Senanayake and Karelliede, A conformational shift in the acetylcholine receptor, which transforms the depolarization neuromuscular block into a non depolarization block, which is characterised by a fade on tetanic stimulation, may be the origin of IMS. Three IMS patients underwent electromyographic testing, and the results showed no post-tetanic facilitation, no fade with tetanic stimulation, and no fade upon low-frequency stimulation, pointing to a postsynaptic abnormality.

However, these findings have not been replicated, and subclinical fading has been identified utilising repeated nerve stimulation with a train of ten stimuli at various frequencies, however these results have not been independently verified. Wadia et al. found only a decrease in the amplitude of the compound muscle action potential at 30Hz and no drop at 3Hz or 10Hz in patients with IMS. However, experiments on neuromuscular transmission using 3 Hz recurrent nerve stimulation show a decline in response. Wide variations in results have been seen as a result, including decreases at low stimulation frequencies of 1 to 3 Hz, normal series at 10, 20, or 50 Hz, or decreases at intermediate frequencies of 10 to 20 Hz, normal findings at both low and higher frequencies[21].

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Organophosphate induced delayed polyneuropathy (OPIDP):[5,13,18,20,22]

It usually happens after 2-3 weeks of exposure (after cholinergic crisis).The distal weakness of the feet and hands are the primary symptoms. Initially the patient complains of weakness in both lower extremities.. Glove and stocking paraesthesia, leg cramping, calf soreness, limb atrophy, foot drop, wrist drop, and claw hand are all unavoidable outcomes. After a few weeks or months, pyramidal signs may arise. Ataxia, positive Romberg sign, high steppage gait, and ankle jerk loss are also possible symptoms. Bilateral symmetrical flaccid paralysis can develop in some instances. Over the following few months, sensory issues disappear, but paralysis endures. More severe cases have symptoms that persist for years, whereas milder cases may heal in 15 months to several years.

The axonal neuropathy symptoms seen in the electrophysiological studies using OPDIP organophosphate include slowed motor and sensory transmission as well as a reduction in the number of neurons in the axon.. Evidence of distal muscle partial denervation. Neuropathologic OPIDP studies show that big distal neurons are usually the first to be impacted. Myelin degeneration occurs first, followed by axonal degeneration. A target that is attached to the membrane OPIDP[6,7]'s main target is esterase, often known as neuropathy target esterase.The axonal membrane is coupled to certain organophosphorus chemicals. the active site of an enzyme The structure of the target protein is changed first. The enzyme then goes through one of two processes: if the binding compound is a phosphate, phosphonate, or phosphoraminate, the enzyme rapidly 'ages,' and the leaving group likely permanently binds to an adjacent macromolecule site that has yet to be identified; if the binding compound is a phosphinate, sulfonate, or carbamate, the enzyme cannot 'age,' and the neuropathy target esterase is protected.

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According to certain theories, the enzyme implicated in neuropathy target esterase is linked to calmodulin kinase II. When calmodulin kinase II is phosphorylated, it becomes active, causing an increase in axoplasmic calcium concentration, cytoskeletal protein disruption, and eventually axonal degeneration[13]

Table 7: Comparison of intermediate syndrome and delayed polyneuropathy

Variable	Intermediate syndrome	Delayed polyneuropathy
Time of onset after Poisoning	1-4days	2-3 weeks
Sites of weakness Limb muscles Neck muscles Cranial nerves Respiratory muscles	Proximal + + + +	Distal - - - -
Electromyogram	Titanic fade	Denervation
Recovery, from time of Onset	4-18days	6-12months
OPs commonlyinvolved	Fenthion Dimethoate Monocrotophos	Methamidophos Trichlorfon Leptophos

Behavioral effects:[5,17,18]

The behavioral changes have been documented following acute or chronic organophosphorus poisoning. Impaired memory and vigilance, diminished information processing and psychomotor speaking, linguistic irregularities, depression, anxiety, and irritability are the most often reported neuropsychological consequences. It takes months for these symptoms to go away. The amount of acetylcholinesterase during the early stages of intoxication was positively correlated with EEG, which tends toward quicker frequency and

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greater voltages. Brain SPECT has shown perfusion abnormalities, especially in the parietal lobe.

Extra pyramidal manifestations:[17,18]

Organophosphorus exposure can cause these symptoms 4 to 40 days later. Symptoms in individuals who survive may go away on their own in 1-4 weeks. Dystonia, rest tremor, cog-wheel rigidity, and choreoathetosis were the most common features. These characteristics are frequently bilateral and asymmetrical. This behaviour has been linked to the inhibition of acetyl cholinesterase in the human extrapyramidal system, which is abundant in cholinergic neurons and anticholinesterase.

Grading of severity of organophosphorus poisoning:

In acute organophosphorus poisoning, there are various techniques for grading severity. Among the initial categories, one put forth by Namba et al.[19] (1971) was never validated in the present or the past. Senanayake N.[23] (1993) recently proposed the Paradeniya Organophosphorus Poisoning (POP) scale for evaluating the severity, which is based on five cardinal manifestations of organophosphorus poisoning as stated in the table below (Table 8)

Peradeniya organophosphorus poisoning (POP) scale grading.

Score	Grade
<4	Mild
4-7	Moderate
>7	Severe

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Table 8: Peradeniya organophosphorus poisoning (POP) scale

Sl. No.	Parameter	Score
1	**Miosis** Pupil size >2mm Pupil size <2mm Pupils pin point	 0 1 2
2	**Fasciculations** None Present butnot generalized or continuous Generalized and continuous with central cyanosis	 0 1 2
3	**Respiration** Respiratory rate ≤20/min Respiratory rate >20/min Respiratory rate >20/min with central cyanosis	 0 1 2
4	**Bradycardia** Pulse rate >60/min Pulse rate 41-60/min Pulse rate ≤40/min	 0 1 2
5	**Level of consciousness** Conscious and rational Impaired, responds to verbal commands Impaired, no response to verbal commands (if convulsion present add 1)	 0 1 2
	TOTAL	11

cardinal clinical manifestations are used in the POP scale, and each one is assigned a severity score that is added to the others to determine the severity on a range from 1 to 11.

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To determine the severity of organophosphorus poisoning, Bardin et al.4,[23] (1990) changed their grading system. This grading takes into account previous exposure to or consumption of organophosphorus, suicidal attempts, clinical symptoms, and uses examinations (decreased PaO2 and abnormal chest roentgenogram) to make an early determination of respiratory failure.

Revised grading for organophosphorus poisoning:[4,25]

Grading	Criteria
Mild poisoning	History of intake/ exposure **Mild signs:** - Normal consciousness - Secretions 1+ - Fasciculations 1+
Severe poisoning	**Severe signs:** - Altered consciousness -Secretions 3+ - Fasciculations 3+
Life threatening poisoning	Suicide attempt - Stupor - PaO2 <75mm Hg (<10mm Hg) - Abnormal chest x ray

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(1+ indicates mild secretions, few fasciculations; 3+ indicates copious secretions, generalized fasciculations. At least two criteria are required for grade, if fewer use previous grades).

Grade	**Cholinesterase activity**
Normal	> 50%
Mild	20-50%
Moderate	10-20%
Severe	<10%

DIAGNOSIS OF ORGANO-PHOSPHORUS POISONING:[24]

1. History of exposure to organophosphorus compounds within the last 24 hours
2. Symptoms and Signs of poisoning
3. pralidoxime and atropine showing effects
4. Inhibition of cholinesterase activity in the blood.

MANAGEMENT OF ACUTE ORGANOPHOSPHOROUS POISONING[12,27]:

1 . Supportive measures:

Oral suction of secretions

Maintenance of circulation

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Establishment of respiration

2. Prevention of absorption :

Decontamination

Emesis

Adsorbant

Cathartics

Bowel wash

3. Specific chemotherapy:

Atropine

Oximes

Treatment of complications

Prevention of further absorption:

Gastric lavage[27]: Though it is most effective within 30 minutes of intake, it is sometimes recommended at the time of admission, after the required steps to protect the airway have been taken. It's done with a big bore tube filled with 3-5 litres of water or saline and 150-200 mL aliquots per wash.

Activated charcoal[5,17,29]: Administer one dose of activated charcoal 1-2g/kg without a cathartic.

Antidotal therapy (Atropine and Oximes)[5,13,17,26,30]:

Atropine: Atropine functions as a physiological antidote, effectively blocking the effects of muscarinic receptors (bronchorrhea, salivation, bradycardia

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etc).Initial dose is 2-4 milligrams given intravenously. Repeated every 5-10 minutes till finished.The symptoms of muscarinic toxicity are reduced[13,17]. Anticholinergic therapy's conclusionThe tracheobronchial tree secretions are cleared and the tracheobronchial tree is dried.almost all secretions The early response to atropine is the pupillary response, however it is not the same as the atropine response. The therapeutic goal Atropine is not contraindicated in the presence of tachycardia.(A heart rate of more than 140 beats per minute should be avoided.)Atropine infusions (0.02-0.08 mg/kg/h) have resulted in considerable weight loss as compared to traditional intermittent treatment in some centres of therapy.[5,17,26]

Atropine also passes the blood-brain barrier, preventing acetylcholine buildup in the central nervous system.

Atropine overdose can cause symptoms like dry mouth, dry, flushed, heated skin, dilated pupils, excitation, delirium, palpitation, hypotension, cardiovascular collapse, convulsions, and coma. Most of these signs and symptoms resemble acute organophosphorus poisoning. The proper dose should be modified after this condition is determined.

Glycopyrrolate:[4,6,25]

If there is no sign of central toxicity, glycopyrrolate (0.5 mg/kg), a quartenary ammonium compound, can be used instead of atropine. For the treatment of organophosphorus poisoning, it has a number of theoretical advantages over atropine. It cannot have any central effects because it cannot cross the blood-brain barrier. Additionally, there is less tachycardia and better control of bronchial discharge, which results in a decrease in respiratory infections.

Oximes (Cholinesterase reactivators):

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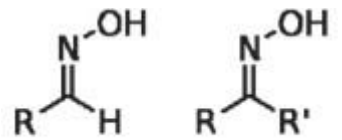

aldoxime ketoxime

Pralidoxime(2-Hydroxy iminomethyl- 1 –pyridinium chloride, 2-PAM; pridine 2 aldoxime methyl chloride)[2,13]

Three actions are attributed to pralidoxime[17].

1. Reactivation of cholinesterase by cleavage of phosphorylated active sites

2. A direct reaction and detoxification of unbounded organophosphorus molecules

3. Endogenous anticholinergic effect at normal doses

Pralidoxime competes with the organophosphorus molecule's phosphate moiety in order to inhibit the acetyl cholinesterase enzyme. This activity occurs only after the enzyme has been poisoned and inhibited, following which the enzyme "Ages" and becomes irreversibly inactive.

Pralidoxime has positive effects for a very long period after exposure, however it is most effective when administered early in the infection. The oximes have the best chance of working if administered within 6 hours of the poisoning. Cholinesterase reactivators must be administered within 24 to 48 hours of organophosphate exposure in order to be effective, according to early research[13].

The kidneys are responsible for pralidoxime elimination, and after 12 hours, 80% of the dose is recovered unchanged in the urine. The half-life of plasma is 75 minutes. Adults should have 100-150 millilitres of 0.9 percent

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alcohol with 1-2 grammes of pralidoxime chloride in it. 30 minutes of intravenous sodium chloride administration [13, 17, 30]

This dosage may vary.If muscle weakness and fasciculations do not improve after an hour, repeat the procedure. This dosage is repeated every 6-12 hours for a total of 24 to 48 hours.. As a result, a continuous infusion for severe poisoning, a dose of 500mg/hour has been recommended.[13,17,31]

Before very high plasma levels (400 mg/mL) are attained, 2-PAM's negative effects in humans have either been absent or minimal. The rate of elevation may cause dizziness, impaired vision, and an increase in diastolic blood pressure. Sudden cardiac and respiratory arrest have resulted with rapid IV infusion.[5,13]

Other oximes: Sugar oximes (a chemical mixture of glucose and 2-PAM derivatives) appear to be a promising way to improve CNS penetration.Obidoxime (Toxogonin) is more effective than 2-PAM since it has two active sites per molecule. The H series of oximes, including HI-6 and HJ-6 (named after Hagedorn), were developed to counteract nerve agents used in chemical warfare.They have direct central and peripheral anticholinergic actions in addition to reactivating cholinesterase[13].

STUDIES ON SEVERITY OF OP POISONINGAND NEED FOR VENTILATORY SUPPORT.

Proudfoot.A,(1982) classified the severity of Organophosphorus poisoning based n PChe levels as: mild [20-50% o normal (2160-5280 IU)], moderate [10-20% of normal (1080-2160iu)], and severe [less than 10% of normal (540-1080 IU)]. The normal range varies from 5400 - 13200 IU.Molly J Coye et.al., (1987) followed up three groups of agricultural workers exposed to

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Organophosphorus pesticides , in the absence of baseline values for cholinesterases (red cell and serum cholinesterase). They found that both red cell and serum cholinesterase values were decreased during the follow up period. They also confirmed that in the absence of baseline values, interpretation of single post exposure value is not useful because of the wide range of normal cholinesterase activity and advised sequential estimations to be better indicators.

In 1990, Thomas Chang-Yao Tsao et al. found that over the course of five years, roughly 40% of patients with carbamate or organophosphorus poisoning experienced respiratory failure, and half of them passed away as a result.

In order to ascertain whether serum cholinesterase levels at the time of admission following acute organophosphorus poisoning have any predictive value with regard to severity, treatment, APACHE scoring, and the need for ventilation, Semir Nouira et al[35]. (1994) estimated serum cholinesterase levels. According to the assessments mentioned above, they did not discover a connection between serum cholinesterase levels and organophosphorus poisoning.

The early identification of those patients requiring ventilatory support was studied in 1994 by Goswamy et al[36], in King Edward VII memorial hospitalBombay, India. They concluded that unconsciousness, miosis, fasciculations and low plasma choline esterase levels were of greatest predictive values.

Goel A et al[37] (1998) evaluated 103 consecutive patients of OP poisoning at JIPMER, Pondicherry with special reference to need for ventilatory support of which 34.95% required mechanical ventilation. Patients who had a Glasgow coma scale below 6 required ventilator assistance in 75% of cases.. [64].70% of

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pts with pupil size less than or equal to 1 mm, 100% of pts with convulsions, 92.86% of pts with fasciculation score greater than 7 required mechanical ventilation. 66.67% of patients with severe poisoning required ventilatory support.

A.Ramani et.al.[38] (1998) studied 25 cases of organophosphorus poisoning and found a good prognostic value for serum cholinesterase from samples drawn at regular intervals.

Kundu et al[39] (2001) conducted a study of 108 hospitalized patients to determine the factors that would predict death in patients who had eaten OP, Burdwan Medical College Hospital looked at factors such length of time before hospitalisation, amount of poison consumed, respiratory paralysis at the time of admission, and amount of PAM needed.

Dua A et al[40], (2001) studied 50 patients admitted to Post Graduate Institute of Medical sciences, Rohtak and estimated serum cholinesterase with respect to clinical profile. The results obtained revealed that serum cholinesterase levels were depressed in poisoning patients but the enzyme level did not correspond with the severity of poisoning. Lee p, Tai d.y[41] (2001)studied clinical features and evaluated the APACHE II scoring system as an alternative index for measuring severity of Organophosphorus poisoning along with estimation of serum cholinesterase levels as a predictor for weaning from mechanical ventilation. Threshold values for weaning were found to be 2900 IU / L and 7500 IU / L.

Aygun D et al[42].,(2002) found that serum cholinesterase level estimations are useful in diagnosis of Organophosphorus poisonings in acute phase but shows no relation to severity of poisoning.

Weissmann-Brenner et al,[43] (2002) in a multi hospital survey of OP compound poisoning at Israel defense forces medical corps (1979 to 1997) described

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characteristics, clinical course and outcome of patients admitted to emergency rooms with diagnosis of OP poisoning. Of the 97 patients studied, 53 presented with mild intoxication, 22 with moderate and 13 had severeintoxication, 5 of whom died. There was a direct correlation between inhibition of butryl cholinesterase and severity of intoxication. Butryl cholinesterase inhibition was higher than 86% in 37% of moderately affected patients, between 51-86% in 50% of severe cases. The number and intensity of clinical manifestations especially miosis, sweating, hyper salivation increased as the severity of intoxication worsened.

Eddleston M (2008) et al[44], assessed usefulness of butyryl cholinesterase activity on admission for predicting severity in acute OP poisoning and found that plasma butyryl cholinesterase activity on admission can provide useful information and can be used to predict death when insecticide ingested is known.

50 patients were evaluated in a study by Reihman S. et al.[45] (2008) that was done at Bir Hospital in Nepal between August 2004 and September 2005. According to the Peradeniya OP poisoning (POP) scale, patients were categorised as having mild, moderate, or severe poisoning. Only 4% of patients had severe poisoning, compared to 26% of patients with mild poisoning. 14% of all patients passed away. Serum cholinesterase levels and poisoning severity were directly associated (p 0.001). The POP scale and serum cholinesterase upon presentation both seemed to be helpful in determining the severity of poisoning, especially when considering the larger doses of atropine, extended hospital stays, and requirement for mechanical ventilation.

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MATERIALS AND METHODS

7.1. SOURCE OF DATA

Patients admitted in the medicine ICU/WARDS OF BLDEUS Shri BM Patil medical college and research Centre, Vijayapura and who fulfill the inclusion criteria.

Patients attending the medicine OPD/ executive health check-up schemes that fulfill the inclusion criteria

Period of study:

The study will be conducted during the period of OCTOBER 2020 to JANUARY 2022.

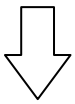
7.2 METHOD OF COLLECTION OF DATA:

The data is collected according to proforma in terms of details history, clinical examination and necessary investigations of the patients who fulfill the inclusion criteria

METHODOLOGY

Patient on admission will be assessed according to Paradeniya Organophosphorous scale

Patient will be given a total score by adding the individual score allotted to each parameter as per Paradeniya organophosphorous scale and will be allotted to mild moderate and severe groups as per the total score of the patient



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Patient will be followed till the final outcome(discharge/death) in each group

Paradeniya organophosphorous scale

Parameter	Criteria	Scor
Pupil size	≥2 mm	0
	<2 mm	1
	Pinpoint	2
Respiratory rate	<20/min	0
	≥20/min	1
	≥20/min with central cyanosis	2
Heart rate	>60/min	0
	41 to 60/min	1
	<40/min	2
Fasciculation	None	0
	Present, generalized/continuous	1
	Both generalized and continuous	2
Level of consciousness	Conscious and rationale	0
	Impaired response to verbal commands	1
	No response to verbal commands	2
Seizures	Absent	0
	Present	1

Scale:

Mild :0-3

Moderate :4-7

Severe :8-11

Type of study: Cross Sectional Study

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Assuming that 58% of the subjects in the population have the factor of interest, the study would require a sample size of: 94 for estimating the expected proportion with 10% absolute precision and 95% confidence.

Sample size calculation

With anticipated Proportion of Respiratory failure among OP Poisoning patients 58%[(ref)] the study would require a sample size of 94 patients with 95**%** level of confidence and 10% absolute precision**.**

Formula used

$$n = \frac{z^2\ p*q}{d^2}$$

Where Z= Z statistic at α level of significance

d^2= Absolute error

P= Proportion rate

q= 100-p

Statistical analysis

The collected data will be loaded into a Microsoft Excel sheet, and statistical analysis will be carried out using the social sciences statistical software (Verson 20). Results will be shown as counts, percentages, graphs, and Mean (Median) SD. The independent t test will be used to compare continuous variables that are normally distributed. Mann Whitney U test will be used for variables that are not normally distributed. The Chi square test will be used to compare categorical variables. It will be deemed statistically significant if p0.05. Every statistical test will run in two-tailed mode.

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INCLUSION CRITERIA

1 . Non alcoholic

2 . Patients whose age between 15- 70

3 . Both sexes are included.

4 . Patients who have a history of exposure to organophosphorous chemicals within the preceding day and have the typical clinical symptoms of organophosphorous compound poisoning

EXCLUSION CRITERIA

1. Patients with a history of serious systemic illness
2. Patients who consumed poison other than organophosphorous compound
3. Patients who were treated treatment with atropine before admission
4. Patients who has Alcohol consumption along with poison

Investigations or interventions required in this study are routine standard procedures. There is no animal experiment involved in this study. Investigations include:

1. Complete Blood Count
2. Absolute Eosinophil Count
3. Erythrocyte sedimentation rate
4. Complete Urine Examination
5. Random blood sugar
6. Serum cholinesterase
7. Renal Function Test
8. Liver Function Tests
9. Toxicology Report from Gastric aspirate
10. Electrocardiogram
11. Chest X-Ray

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OBSERVATION AND RESULTS

Age (years)	Frequency	Percent
> 70	3	3.2
10-20	18	18.9
20-30	47	49.5
30-40	16	16.8
40-50	4	4.2
50-60	6	6.3
60-70	1	1.1
Total	95	100.0

Table 9- : Age distribution in the study

In the study majority of subjects were in the age group 20-30 years (49.5%), 18.9 % were in the age group 10-20 years and 16.8% are in the age group 30-40 years

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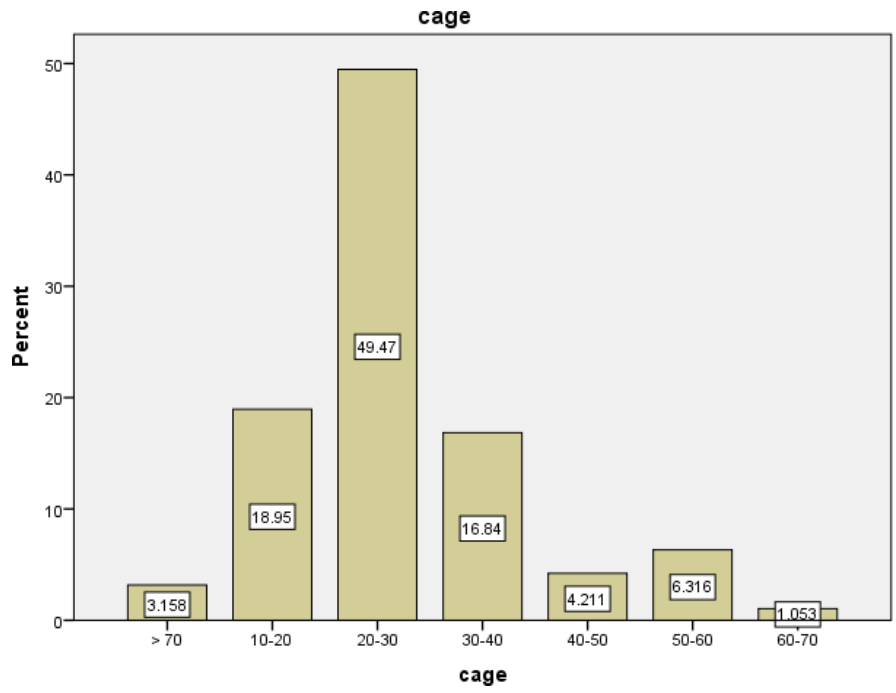

Figure 3 : Bar diagram showing age distribution in the study

In the study majority of subjects were in the age group 20-30 years (49.5%), 18.9 % were in the age group 10-20 years and 16.8% are in the age group 30-40 years

Parameter	Frequency	Percent
Female	32	33.7
Male	63	66.3
Total	95	100.0

Table 10: sex distribution in the study

In this study 66.3 % were males(majority) and 33.7 % were females

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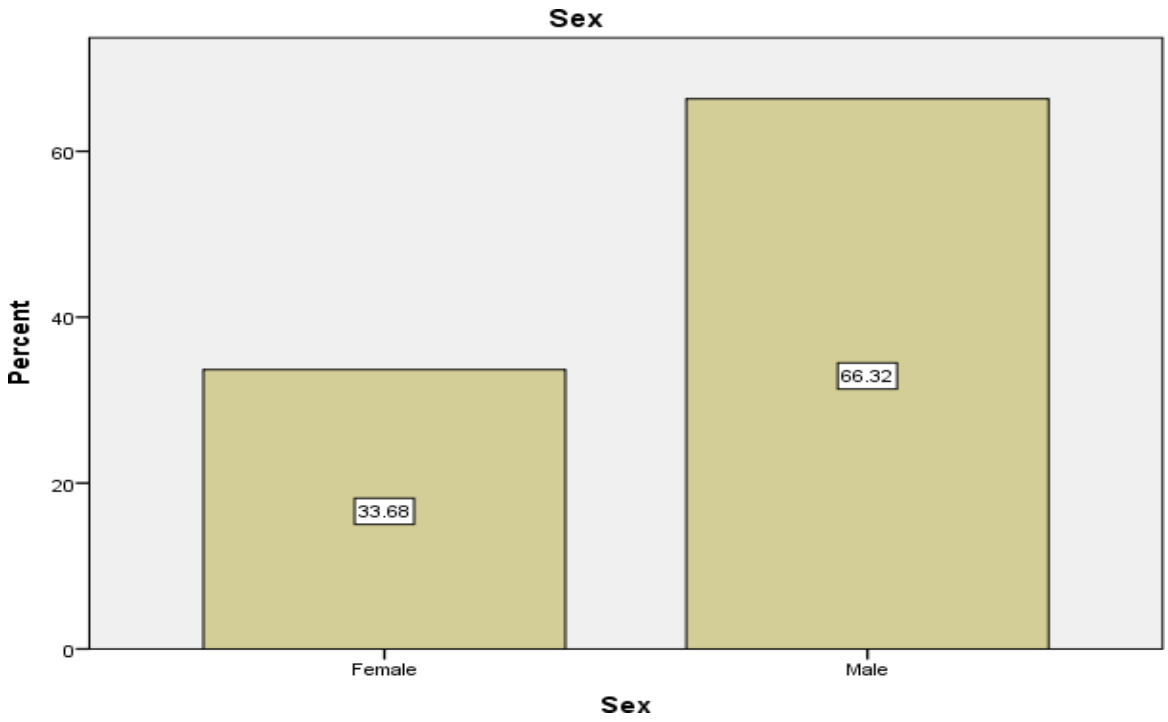

Figure 4: Bar diagram showing sex distribution in the study

In this study 66.3 % were males(majority) and 33.7 % were females

Occupation	Frequency	Percent
barber	2	2.1
driver	11	11.6
farmer	23	24.2
housewife	14	14.7
laborer	2	2.1
painter	4	4.2
Student	39	41.1
Total	95	100.0

Table 11: Table showing occupation of subjects

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In this study majority of the subjects (41.1%) are students.24.2% are farmers,11.6% are drivers

Outcome	Frequency	Percent
Alive	82	86.3
Died	13	13.7
Total	95	100.0

Table 12: Table showing outcome among subjects

In this study 86.3% of the subjects are alive and 13% of the subjects are dead

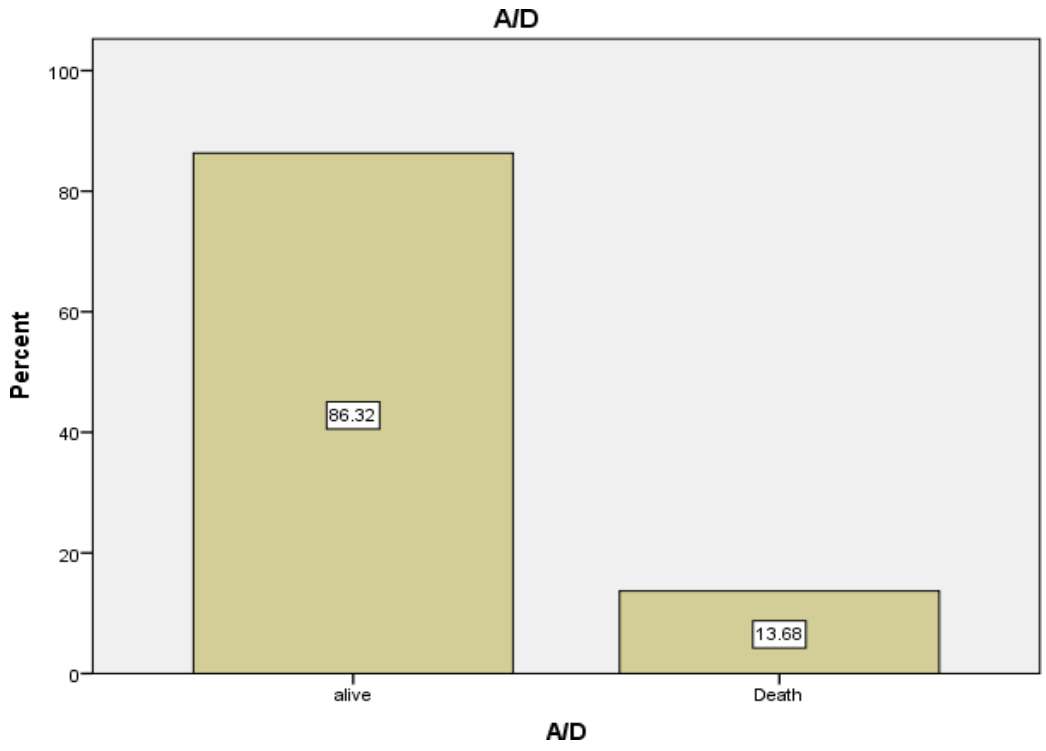

Figure 5: Bar diagram showing outcome of patients

In this study mortality was 13.68% . 86.3% of patients survived

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Route	Frequency	Percent
Oral	94	98.9
skin	1	1.1
Total	95	100.0

Table 13: Table showing route of intake

In this study majority of the subjects took OP compound Orally (94%) and the next common route is via exposure through skin (1%)

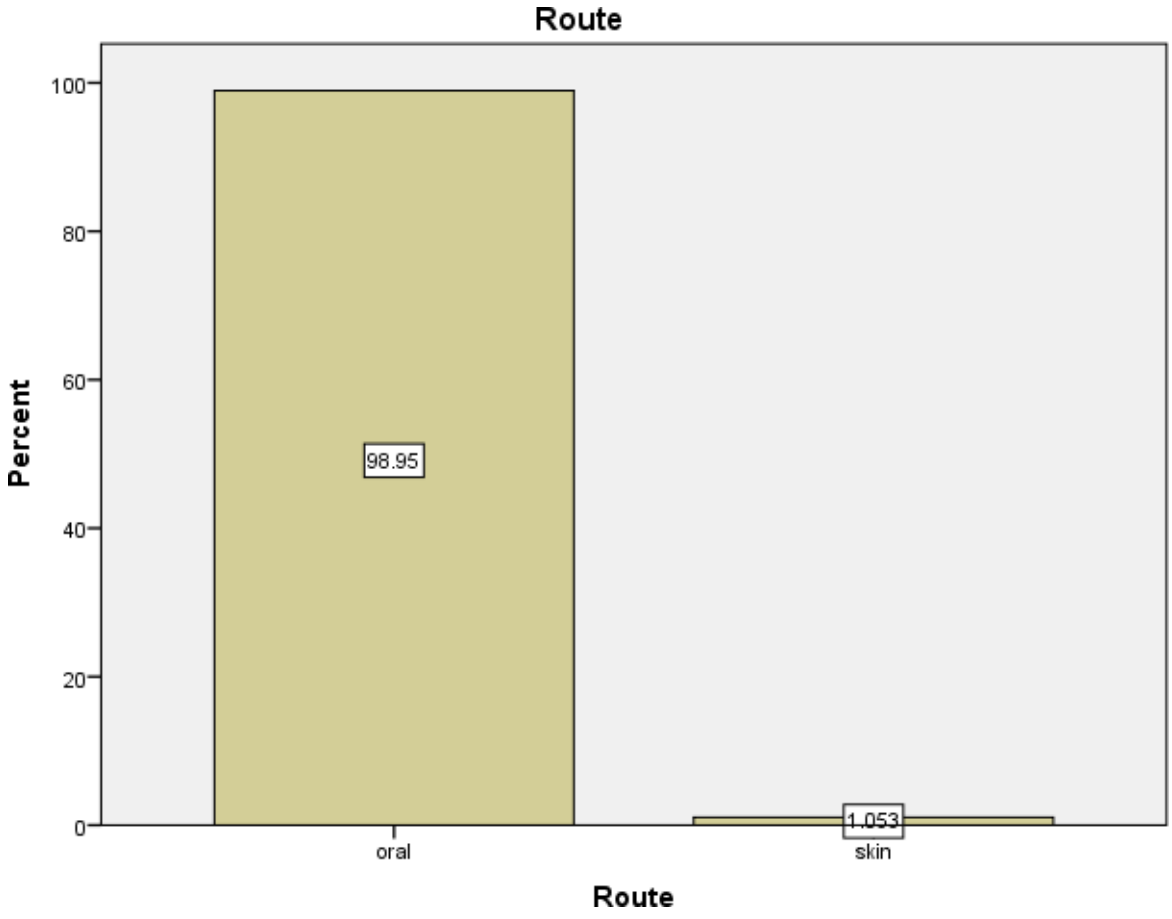

Figure 6: Graph showing route of intake

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In this study 98.95% of patients consumed the poison orally

Marital status

Marital status	Frequency	Percent
Married	48	50.5
Unmarried	47	49.5
Total	95	100.0

Table 14:Table showing marital status

In this study,50.5% of the subject are married and 49.5% of the subjects are unmarried

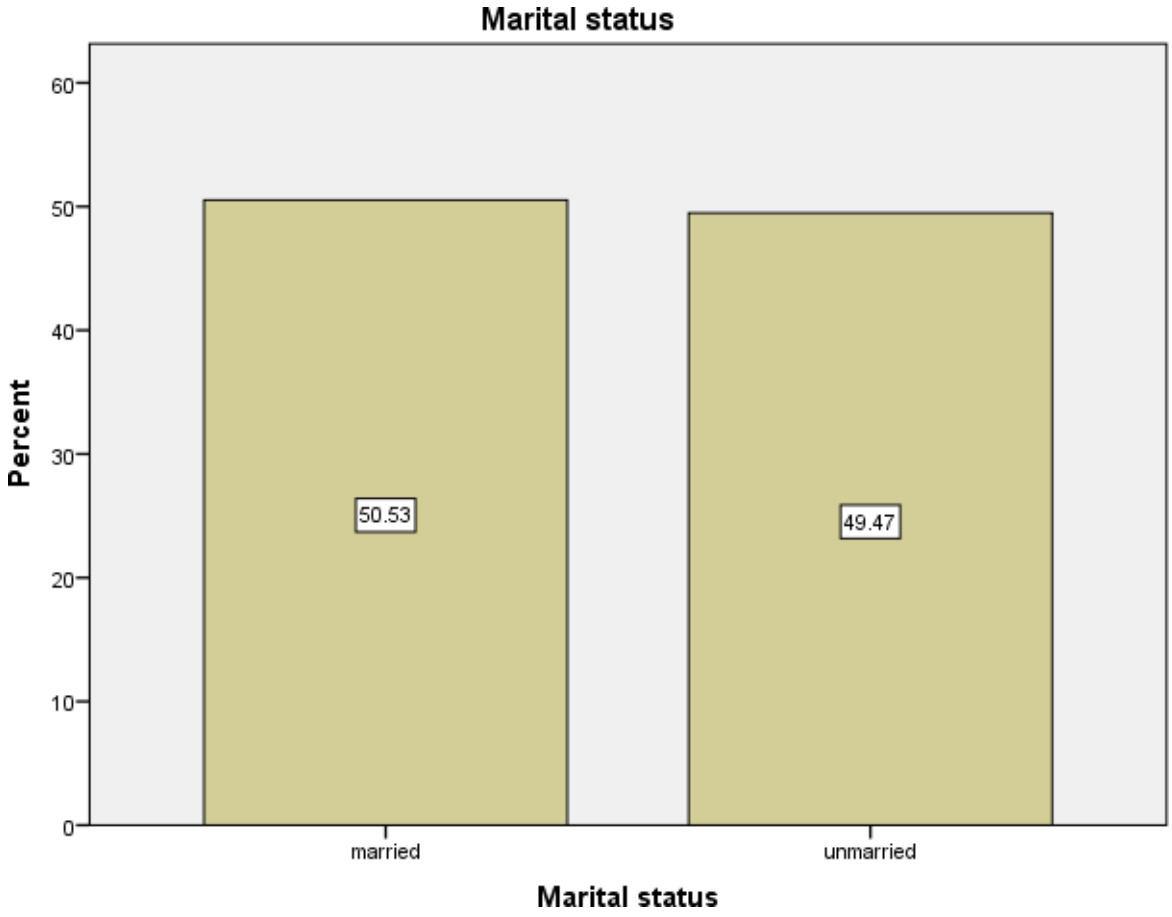

Figure 7:Bar chart showing marital status

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In our study 50.53% were married and 49.47% patients were unmarried

		Frequency	Percent
Valid	Accidental	10	10.5
	Suicidal	85	89.5
	Total	95	100.0

Table 15: Showing intention of poisoning

In this study majority of the subjects have the intention to commit suicide while taking the poison (89.5%) and the remaining 10.5 % have accidental exposure to OP poison

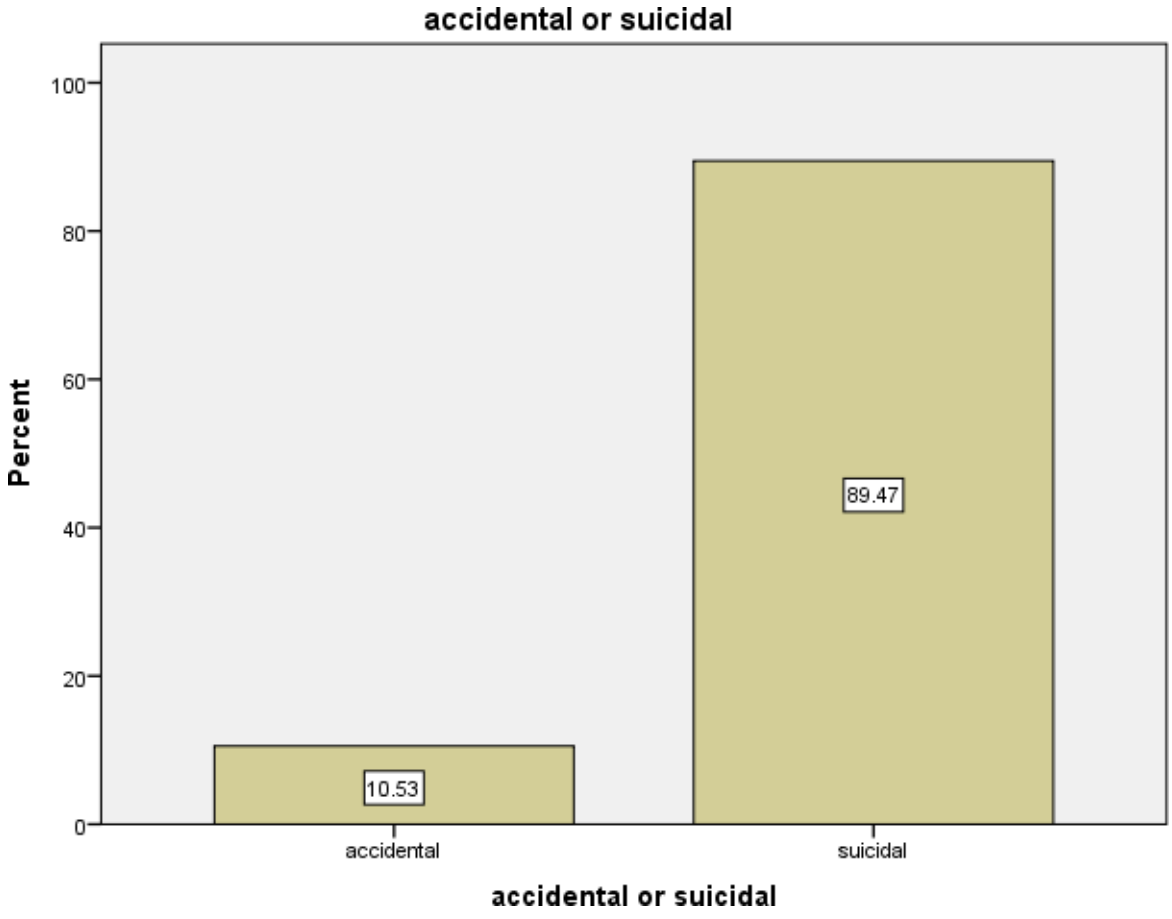

Figure 8: Bar diagram showing mode of poisoning

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In this study 89.47% subjects consumed poison with an intention to commit suicide

Parameter	Frequency	Percent
mild	64	67.4
moderate	27	28.4
severe	4	4.2
Total	95	100.0

Table 16: Table showing the severity of poisoning as per POP scale

In our study Majority of the subjects (67.4%) fall in mild categeory of POP score ,28.4% fall in Moderate categeory,4% fall in Severe categeory

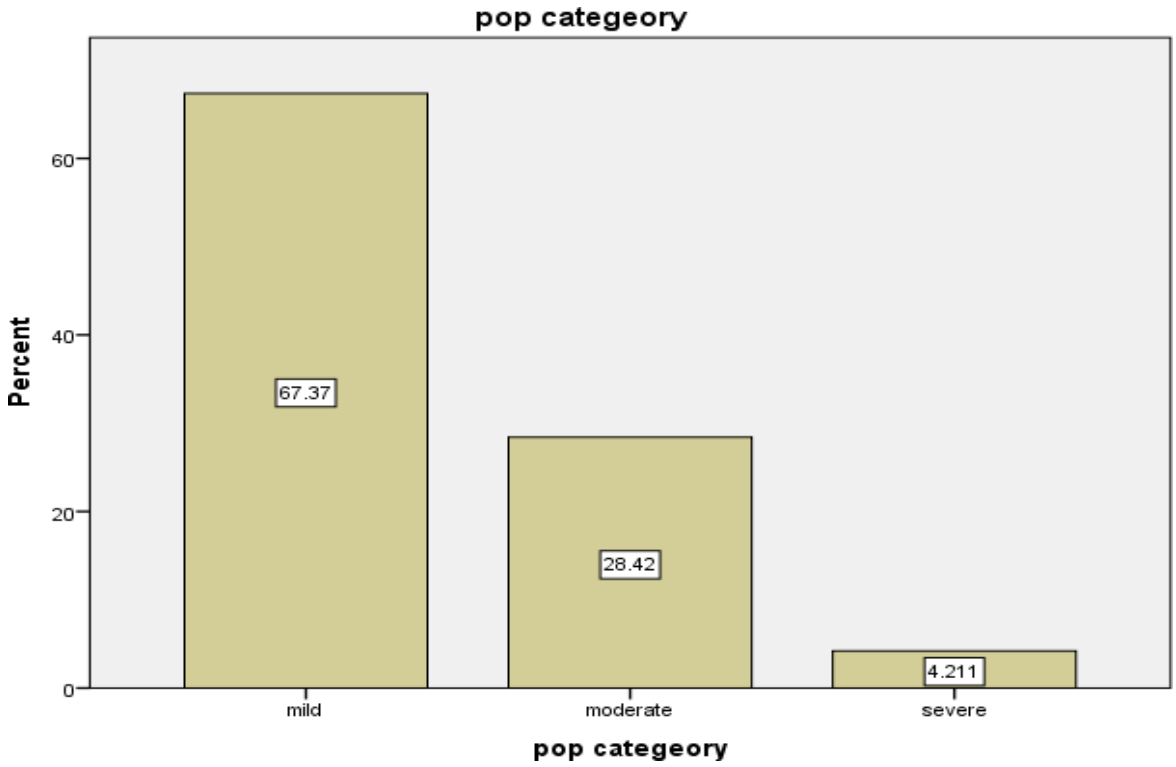

Figure 9: **Bar chart showing severity of poisoning according to POP scale**

67.37% of patients in the study belonged to mild grade of poisoning. 4.2% had severe poisoning.

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			Ventilator		Total
			Intubated	Not intubated	
pop categeory	Mild	Count	1	63	64
		% within pop categeory	1.6%	98.4%	100.0%
	moderate	Count	15	12	27
		% within pop categeory	55.6%	44.4%	100.0%
	severe	Count	4	0	4
		% within pop categeory	100.0%	0.0%	100.0%

Table 17: Table showing severity of POP scale and requirement of ventilator support

In this study 1.6 % of the subjects in Mild categeory were intubated in comparison with 55.6 % in the moderate categeory and 100 % in severe categeory

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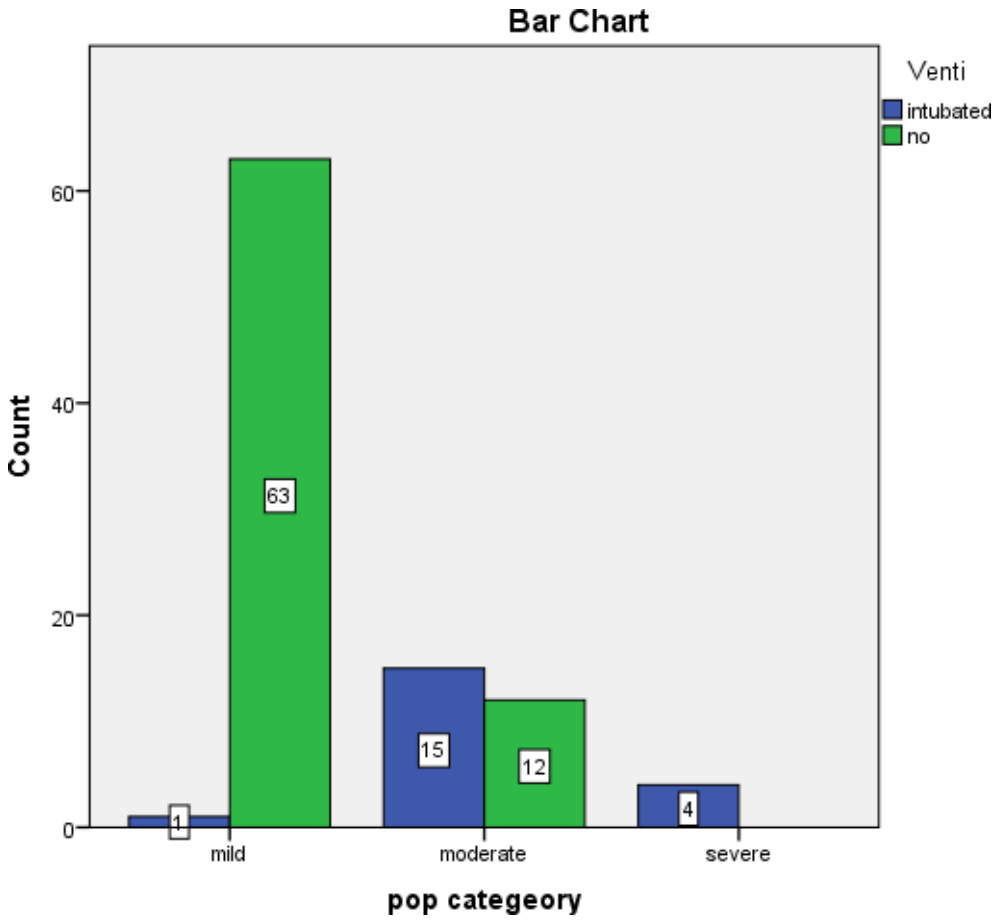

Figure 10: Bar showing severity of POP scale and requirement of ventilator

In this study only 1.6% of patients with mild grade of poisoning according to POP scale required ventilatory support, where has 99.4% did not required ventilatory support. Most of patients with moderate (55.6%) and severe poisoning (100%) according POP scale required ventilatory support. The statistical significance of this was very strong.

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			ALIVE OR DEAD		Total
			alive	Death	
pop categeory	Mild	Count	63	1	64
		% within pop categeory	98.4%	1.6%	100.0%
	moderate	Count	18	9	27
		% within pop categeory	66.7%	33.3%	100.0%
	severe	Count	1	3	4
		% within pop categeory	25.0%	75.0%	100.0%

Table 18 :Table showing correlation between POP categeory and outcome among subjects

In this study the subjects falling in the severe categeory of POP score has a mortality of 75%.For mild categeeory it is 1.6% and moderate categeory it is 33.3%.

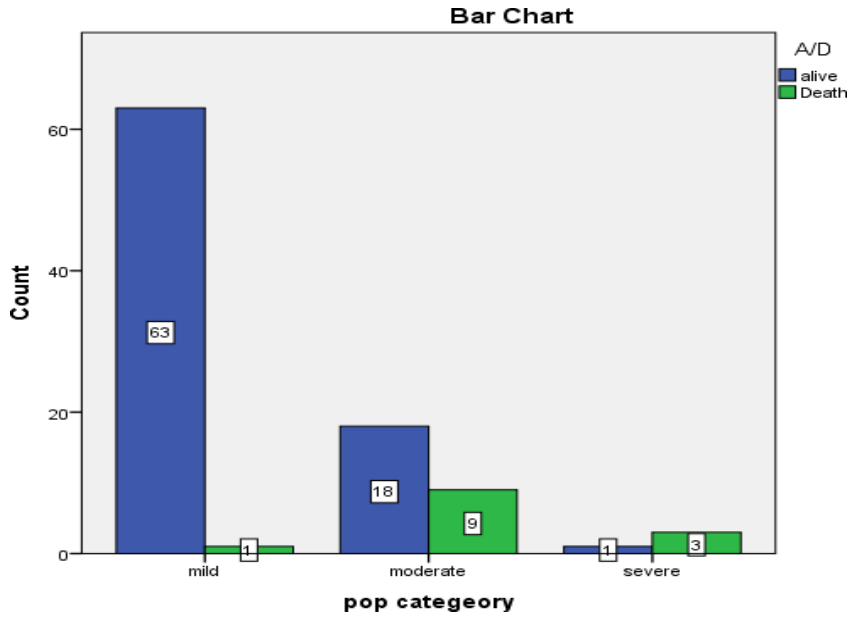

Figure 11 : Figure shows the correlation between POP categeory and outcome among the subjects

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In this study the subjects falling in the severe categeory of POP score has a mortality of 75%.For mild categeeory it is 1.6% and moderate categeory it is 33.3%.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
total score(11)	.208	95	.000	.871	95	.000
Cholinesterase	.123	95	.001	.919	95	.000

a. Lilliefors Significance Correction

Nonparametric Correlations

Correlations

			Cholinesterase	total score(11)
Spearman's rho	Cholinesterase	Correlation Coefficient	1.000	-.619**
		p-value	.	.000
		N	95	95
	total score(11)	Correlation Coefficient	-.619**	1.000
		p-value	.000	.
		N	95	95

**. Correlation is significant at the 0.01 level (2-tailed).

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DISCUSSION

Organophosphate (OP) compounds are utilised in both home and industrial settings. These are cholinesterase inhibitors with the potential to cause severe cholinergic poisoning .

Exposure at work, exposure through an accident, or intentional consumption can all result in organophosphate poisoning. The diagnosis is typically based on a history of exposure and includes telltale symptoms of cholinergic excess, but it can be challenging if the patient has been exposed unintentionally or disoriented. Utilizing the Peradeniya Organophosphorus Poisoning (POP) scale, the severity of the poison is assessed.This study was conducted to evaluate the role of Paradeniya organophosphorous scale as a predictor of mortality in OP poisoining .

According to our analysis, a majority of participants (50%) are in the 20–30 age range. The average patient age was under 40 in 81% of cases. The investigations of Reihman et al.[45], Goel et al.[37], Doshi et al.[46], and Noiura et al.[35] are contrasted with this.

According to this survey, men (63% of cases) outnumbered women (32% of cases). In this study, the male to female ratio is 1.9:1. The gender distribution described by Shankar et al.[47] (1.48:1), A Goel et al.[37] (2.5:1), and Gupta et al.[48] accords to this.

In our study, there were 48 married patients (50.5%), and there were 49.5% single patients. In this study, there are 1.02 married people for every unmarried person. This can be explained by the fact that marriages take place sooner in India.

In our study, majority of the subjects were students (41.1%), then farmers who constituted about 24.2% of cases,which is followed by housewives of 14.7%. This is in variance to findings by Reihman et al[45] where farmers

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constituted 14% and housewives' 28%. A Goel et al[37] have reported in their study that 43% of patients were unemployed.

In our analysis, 89.5 percent of the cases involved poison consumption with a suicidal intent. The fact that OP chemicals are typically readily available as pesticides and that they are freely accessible at pesticide stores may be the cause of their frequent use in suicidal attempts. In contrast, Reihman et al[45], Noiura et al[35] (90%), Goel et al[37] (96.1%), and Gupta et al[48] (91%), reported values.

In our study majority (94%) took the poison via oral route where as the remaining 1.1% has accidental exposure of the poison via skin

In our study 86.3% of the patients who took the poison are alive where as 13.7% of the patients died which is in comparison with Das.B.W[51] et al(13.3%), Arup kumar kundu et al[39] (13.3%), Noiura et al[35] (10%), Reihman et al[45] (14%).

In our study majority of the subjects fell in Mild categeory of POP score (67.4%),28.4% in the moderate categeory and 4% fell in severe categeory

In our study only 1.6% patients in mild grade of poisoining as per POP score required ventilatory support where as 99.4% did not require ventilatory support .Majority of the patients in Moderate categeory of POP score (55.6%) required ventilatory support where as 100% of the patients in severe categeory of POP score required ventilatory support. Reihman et al[45] found out a significant association between POP score and PChe levels, POP score and hospital stay, total dose of atropine required, mechanical ventilation. The higher the score of POP scale, higher was the suppression of PChe levels. A Goel et al[37] compared individual components of POP scale and concluded that they can be used in predicting the need for ventilatory support.

In our study subjects falling in the severe categeory of POP score has a mortality of 75%,33.3% for moderate categeory and 1.6% for mild categeory

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In our study the mortality among subject in Mild POP categeory is 1.5%,in Moderate categeory of POP score it is 33.3% and in severe categeory of POP score it is 75%.

So it can be said that, POP score can be a better predictor to categorize patients who might not require ventilator support and who has a less chance of death. However as mentioned earlier, studies with a large sample size and a heterogeneous population have to conducted to confirm the results

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CONCLUSION

An increase in POP score in OP poisoining is associated with increase in mortality,Hence POP score can be useful to estimate the mortality in OP poisoining patients

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SUMMARY

This study was conducted with a sample size of 94 patients who were referred to our hospital for organophosphorous poisoining from January 2012 to June 2022.Majority of the patients were younger than 30 years with male preponderance.Majority of them were students.Majority of the participants were students.Majority of the participants(50.5%) are married.89.5 % of the participants took OP compound with an intention to commit suicide.At the end of the study 86,3% of the participants were alive.Majority of them took the poison with an intention to commit suicide.98.9% of the partipants took the poison via oral route and the rest has exposure via skin.67.4% of the partipants were falling in Mild categeory of POP Score.In this study 100% of the patients who fell in severe categeory of POP score required ventilatory support where as 55.6% of those in Moderate categeory required ventilatory support and 1.6% of those in Mild categeory required ventilatory support.In this study the mortality rate of subjects falling in severe categeory was 75%,for moderate categeory it is 33.3% and 1.6% for Mild categeory.In our study as the POP score increases and as patients falls in severe categeory of POP score the mortality increased and so the need for ventilatory support . Hence on the initial assessment of patient of OPpoisoining POP score can be used to categerise patients into mild,moderate and severe categeories.Patients falling in Severe POP score categeory can be referred to higher centres with ventilator support to reduce mortality where as those falling in mild to moderate POP scores categeory can be managed in the Primary health care setting ensuring optimal utilisation of scarce resources especially in developing country like India.

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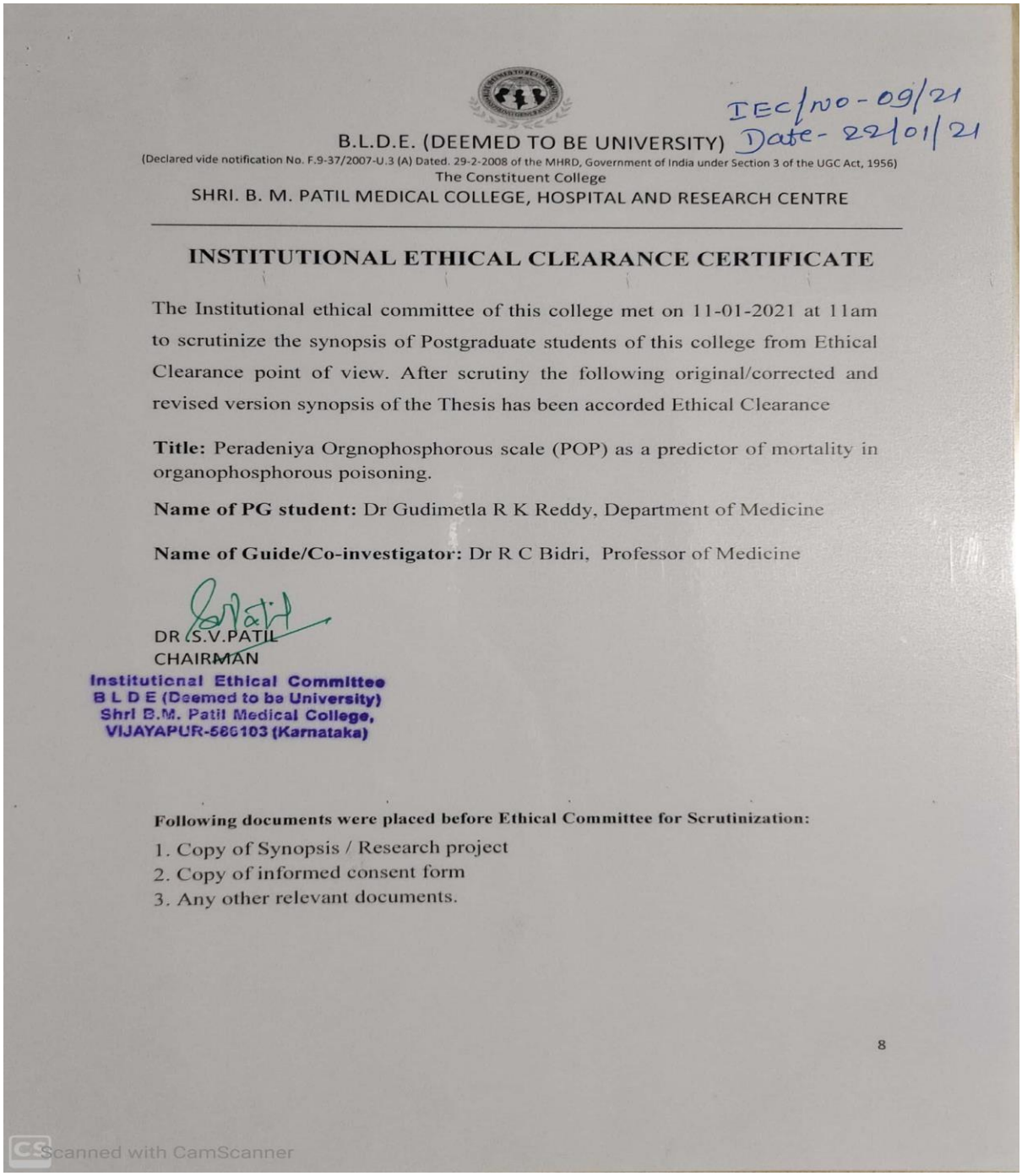
Annexure I

Ethical Clearance Certificate



B.L.D.E. (DEEMED TO BE UNIVERSITY) IEC/NO-09/21 Date- 22/01/21

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: Peradeniya Orgnophosphorous scale (POP) as a predictor of mortality in organophosphorous poisoning.

Name of PG student: Dr Gudimetla R K Reddy, Department of Medicine

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

DR S.V.PATIL
CHAIRMAN
Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

8

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Annexure II

SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE,VIJAYAPUR-586 103

RESEARCH INFORMED CONSENT FORM

TITLE OF THE PROJECT : PERADENIYA ORGANOPHOSPHOROUS SCALE(POP) AS A PREDICTOR OF MORTALITY IN ORGANOPHOSPHOROUS POISOINING

PG GUIDE : DR. R. C. BIDRI

PG STUDENT : DR. GUDIMETLA.R.K.REDDY

PURPOSE OF RESEARCH:-

BENEFITS:-

I understand that my participation in this study will help the investigator to diagnose the disease better and will help in the management of the disease.

PROCEDURE:-

I understand that relevant history will be taken and I will undergo detailed clinical examination after which necessary investigations will be done and accordingly treatment will be given.

RISK AND DISCOMFORTS:-

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I understand there is no risk involved and I will experience no pain during the procedures performed.

CONFIDENTIALITY:-

I understand that medical information produced by this study will become a part of my hospital records and will be subjected to the confidentiality and privacy regulation of the said hospital. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file.

If the data are used for publication in the medical literature or for teaching purposes no names will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand I may see the photographs, videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:-

I understand that I may ask more questions about the study at any time Concerned. The researcher is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:-

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in this study at any time without prejudice. I also understand that the researcher may terminate my participation in this study at any time after he has explained the reasons for

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doing so and has helped arrange for my continued care by my own physician, if this is appropriate.

INJURY STATEMENT:-

I understand that in the unlikely event of injury to me resulting directly from my participation in this study and if such injury were reported promptly, then medical treatment will be available to me, but no further compensation will be provided. I understand that by my agreement for my participation in this study, I am not waiving any of my legal rights.

I have explained to (patient's / relevant guardian's name) the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Investigator / P. G. Guide Date

I confirm that …….(Name of the PG guide / chief researcher) has explained to me the research, the study procedures that I undergo and the possible risks and discomforts as well as benefits that I may experience. I have read and I understand this consent form. Therefore, I agree to give my consent for my participation as a subject in this research project.

_____ _____

Participant / guardian Date

_____ _____

Witness to signature Date

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ANNEXURE III

OPC POISOINING CASE PROFORMA

PROFORMA

Name IP number

Age: Sex

Address : Occupation :

Date of Admission : Date of discharge:

Chief Complaints :

History of present illness:

Past history:

Personal History:

Physical Examination:

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On Examination :

VITALS:

Temperature:

Pulse:

Respiratory rate:

Blood pressure:

GENERAL CONDITION:

Pallor:	Yes/ No
Icterus:	Yes/ No
Cyanosis:	Yes/No
Clubbing:	Yes/No
Lymphadenopathy:	Yes/No
Edema:	Yes/No
Pt condition on discharge	Improved/Worsened/same/Expired

SYSTEMIC EXAMINATION:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

CENTRAL NERVOUS SYSTEM:

PER ABDOMEN EXAMINATION:

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INVESTIGATIONS:

COMPLETE BLOOD COUNT –

Total count	CELLS/CMM
Neutrophils	%
Lymphocytes	%
Eosinophils	%
Basophils	%
Monocytes	%
Haemoglobin	GM/Dl
Platelet count	LAKHS/CMM

ESR	MM/HR

AEC	CELLS/CMM

URINE COMPLETE:

ALBUMIN	MG/Dl
SUGAR	MG/Dl
RBC	PER MICRO LT
EPITHELIAL CELLS	PER HPF

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PUS CELLS	PER MICRO LT

RBS	MG/Dl

BLOOD UREA	MG/Dl
SERUM CREATININE	MG/Dl
SERUM SODIUM	MEQ/Lt
SERUM POTASSIUM	MEQ/Lt

LIVER FUNCTION TEST :

TOTAL BILURUBIN	MG/Dl
CONJUGATED BILURUBIN	MG/Dl
UNCONJUGATED BILURUBIN	MG/Dl
SGOT	UNITS/Lt
SGPT	UNITS/Lt
ALBUMIN	MG/Dl
ALP	UNITS/Lt

SERUM CHOLINESTERASE	IU/Lt

ECG :

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POP Score:

Parameter	Value	Score
Pupil Size		
Respiratory Rate		
Heart rate		
Fasciculations		
Level of Consciousness		
Seizures		

Total score and categeory:

Intention:Accidental/Suicidal

Provisional Diagnosis:

Outcome:Alive/Dead

Intubated/Not intubated

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ANNEXURE IV

MASTER CHART

S.NO	Case no	Ip no	Name	Age	Sex	Occupation	Marital status	Intention	DOA	DOD	Place	Route	Amount(	Compound	Pulse(bpm	BP	RR(cpm)	Temp(	Fasciculation	Pupil size	Level of consciousness	Seizures	total score(11)	pop categeory
1	1	28635	Netra ravindra	20	F	Student	unmarried	suicidal	21/01/202	26/01/202	rural	oral	20	unknown	64	90/60	18	3	present		Impaired to verbal command	absent	2	mild
2	2	30512	mahananda dareppa	15	F	student	unmarried	suicidal	23/01/202	28/01/202	rural	oral	unknown	monocrotophos	66	100/60	22	3	generalised		Impaired to verbal command	absent	5	moderate
3	3	40473	bhirappa siddappa	22	M	driver	unmarried	suicidal	31/01/202	02-04-2022	rural	oral	unknown	dichlorovas	58	100/60	18	3	continuous		conscious and rationale	absent		moderate
4	4	47209	lakkappa pujari	75	M	farmer	married	suicidal	02-05-2022	02-09-2022	rural	oral	30	monocrotophos	58	130/88	18	3	present		conscious and rationale	absent	1	mild
5	5	37016	heena nganiyar	17	F	Student	unmarried	accidenta	29/01/202	30/01/202	rural	oral	10	chlorpyrifos	70	100/70	16	3	none		conscious and rationale	absent	0	mild
6	6	55298	basavaraj hanumanth	26	M	driver	married	suicidal	12-02-2022	17-02-2022	urban	oral	unknown	unknown	62	96/70	22	3	generalised		conscious and rationale	absent	3	mild
7	7	70901	roopa gangappa	22	F	student	unmarried	suicidal	24-02-2022	26-02-2022	urban	oral	unknown	phorate	60	100/60	21	3	none		conscious and rationale	absent	2	mild
8	8	67338	pretam	16	M	student	unmarried	suicidal	22-02-2022	23-02-2022	urban	oral	30	propenofos	60	90/70	18	3	none		conscious and rationale	absent	0	mild
9	9	58479	girish bheemanna	22	M	barber	unmarried	suicidal	14-02-2022	16-02-2022	rural	oral	10	dimethoate	62	94/68	22	3	continuous		conscious and rationale	absent	4	moderate
10	10	53594	rekha shanker	19	F	student	unmarried	suicidal	10-02-2022	19-02-2022	rural	oral	15	monocrotophos	58	90/70	22	3	continuous	pinpoint	conscious and rationale	absent	5	moderate
11	11	56466	siddappa pujari	30	M	farmer	married	suicidal	12-02-2022	14-02-2022	rural	oral	unknown	unknown	66	100/70	21	3	none		conscious and rationale	absent	1	mild
12	12	73901	danamma shekappa	18	F	housewife	unmarried	suicidal	27-02-2022	05-03-2022	rural	oral	40	chlorpyrifos	58	110/80	23	3	generalised		conscious and rationale	absent	4	moderate
13	13	78682	yalaling lankapagol	35	M	driver	unmarried	suicidal	04-03-2022	05-03-2022	rural	oral	20	propenofos	64	140/90	18	3	none		conscious and rationale	absent	0	mild
14	14	78462	siddappa gopal	36	M	farmer	unmarried	suicidal	03-03-2022	10-03-2022	rural	oral	40	unknown	58	110/70	21	3	generalised		conscious and rationale	absent	4	moderate
15	15	110121	janaverasiddha pujari	23	M	student	married	suicidal	31-03-2022	31-03-2022	rural	oral	100	monocrotophos	58	100/60	24	3	continuous	pinpoint	Impaired to verbal command	absent	7	moderate
16	16	80144	soumya umesh	31	F	housewife	married	suicidal	04-03-2022	10-03-2022	rural	oral	50	monocrotophos	66	110/80	22	3	generalised		Impaired to verbal command	absent	3	mild
17	17	110111	shanker hebbal	24	M	barber	unmarried	suicidal	30-03-2022	08-04-2022	rural	oral	45	unknown	58	110/70	21	3	none		Impaired to verbal command	absent	4	moderate
18	18	108534	gurulingappa padagalgi	55	M	farmer	married	suicidal	29-03-2022	05-04-2022	rural	oral	40	unknown	66	100/80	22	3	none		conscious and rationale	absent	2	mild
19	19	97021	peerappa siddappa loni	26	M	farmer	married	accidenta	18-03-2022	19-03-2022	rural	oral	100	propenofos	42	130/80	26	3	generalised	pinpoint	Impaired to verbal command	present	7	moderate
20	20	97100	basamma tarachand	45	F	housewife	married	accidenta	18-03-2022	26-03-2022	rural	oral	unknown	dimethoate	46	130/70	22	3	continuous		conscious and rationale	absent	4	moderate
21	21	110064	akshara sunil hipargi	19	F	Student	unmarried	accidenta	30-03-202	05-04-202	rural	oral	10	unknown	61	144/80	21	3	generalised		Impaired to verbal command	absent	2	mild
22	22	81267	yaseen bisanal	30	M	farmer	married	accidenta	05-03-202	08-03-202	rural	oral	20	propenofos	62	140/80	21	3	generalised		Impaired to verbal command	absent	3	mild
23	23	134966	umesh muddanur	28	M	laborer	married	accidenta	21-04-202	26-04-202	rural	oral	unknown	unknown	62	150/70	22	3	generalised		Impaired to verbal command	absent	4	moderate
24	24	127602	ravikanth bechar	18	M	Student	unmarried	suicidal	14-04-202	24-04-202	urban	oral	20	monocrotophos	58	128/80	26	3	continuous	pinpoint	No response to verbal commands	present	8	severe
25	25	98317	akshara saiwalli	14	F	Student	unmarried	suicidal	19-03-202	22-03-202	rural	oral	unknown	monocrotophos	58	130/72	24	3	generalised	pinpoint	Impaired to verbal command	present	7	moderate
26	26	99436	danamma ramanna	18	F	housewife	unmarried	suicidal	21-03-202	24-03-202	rural	oral	unknown	unknown	66	136/80	22	3	none		conscious and rationale	absent	1	mild
27	27	117033	mallikarjun byalal	21	M	Student	unmarried	suicidal	06-04-202	12/04/200	urban	oral	unknown	monocrotophos	62	140/78	22	3	generalised		conscious and rationale	absent	3	mild
28	28	131545	kiranbabu	28	M	painter	married	suicidal	18-04-202	28-04-202	urban	oral	unknown	chlorpyrifos	58	110/70	22	3	none		conscious and rationale	absent	3	mild
29	29	117726	irranna r dolli	33	M	farmer	married	suicidal	06-04-202	09-04-202	rural	oral	20	unknown	62	136/70	22	3	generalised		conscious and rationale	absent	1	mild
30	30	125218	umesh hadimani	27	M	driver	married	suicidal	12-04-202	18-04-202	urban	oral	unknown	unknown	58	110/70	18	3	generalised		conscious and rationale	absent	3	mild
31	31	256792	manoj tejappa pujari	24	M	Student	unmarried	suicidal	17-11-202	18-11-202	rural	oral	20	monocrotophos	66	100/70	18	3	generalised		conscious and rationale	absent	1	mild
32	32	303693	aishwarya hosamani	15	F	Student	unmarried	suicidal	20-12-202	23-12-202	rural	oral	unknown	propenofos	56	110/80	20	3	none		conscious and rationale	absent	1	mild
33	33	297846	mahantesh bamanhalli	50	M	farmer	married	suicidal	15-12-202	16-12-202	rural	oral	unknown	chlorpyrifos	56	110/80	22	3	generalised	pinpoint	Impaired to verbal command	present	7	moderate
34	34	163625	mallapa gangappa bisnl	24	M	laborer	unmarried	suicidal	14-05-202	17-05-202	rural	oral	unknown	unknown	62	110/70	22	3	generalised		conscious and rationale	absent	3	mild
35	35	177408	vikash tamanna	20	M	Student	unmarried	suicidal	25-05-202	27-05-202	urban	oral	20	monocrotophos	62	100/70	22	3	none		conscious and rationale	absent	1	mild
36	36	155075	bhagyawathi layappa	16	F	Student	unmarried	suicidal	08-05-202	11-05-202	urban	oral	unknown	monocrotophos	58	110/70	18	3	none		conscious and rationale	absent	1	mild
37	37	153048	bagavva rangappa	55	F	housewife	married	suicidal	06-05-202	09-05-202	rural	oral	unknown	unknown	62	140/80	22	3	generalised		Impaired to verbal command	absent	2	mild
38	38	127612	Rrenuka shivalingappa	27	F	housewife	married	suicidal	15-04-202	05-05-202	rural	oral	unknown	propenofos	50	112/80	24	3	continuous	pinpoint	Impaired to verbal command	present	7	moderate
39	39	135686	santosh shina	21	F	Student	unmarried	suicidal	22-04-202	26-04-202	rural	oral	unknown	monocrotophos	62	110/70	16	3	continuous		conscious and rationale	absent	1	mild
40	40	159615	subbaraiainapur	75	M	farmer	married	accidenta	11-05-202	13-05-202	rural	skin	unknown	monocrotophos	58	134/80	18	3	generalised		conscious and rationale	absent	2	mild
41	41	253374	prajwal gangnalli	17	M	Student	unmarried	accidenta	23/04/202	25/04/202	rural	oral	unknown	propenofos	62	110/70	21	36	none		conscious and rationale	absent	1	mild
42	42	163337	ameenappa sabu	91	M	farmer	married	accidenta	28/03/202	30/03/202	rural	oral	unknown	monocrotophos	55	100/70	26	3	continuous	pinpoint	Impaired to verbal command	present	7	moderate
43	43	58671	mahananda mmatapati	21	F	Student	unmarried	accidenta	16/05/2021	19/05/2021	rural	oral	unknown	monocrotophos	66	110/70	22	3	none		conscious and rationale	absent	1	mild
44	44	126297	pallavi suresh kengalgti	16	M	Student	unmarried	suicidal	29/07/202	08-01-2021	rural	oral	unknown	monocrotophos	66	130/80	21	36	generalised		conscious and rationale	absent	2	mild
45	45	78682	yalling h lankappagol	35	M	farmer	married	suicidal	02-04-202	03-05-202	rural	oral	unknown	propenofos	64	110/70	18	37	none		conscious and rationale	absent	0	mild
46	46	166277	vaishali yallappa katnalli	18	F	Student	unmarried	suicidal	29/03/202	04-04-202	urban	oral	unknown	chlorpyrifos	62	120/70	22	36	generalised		conscious and rationale	absent	2	mild
47	47	19667	basavaraj bhimappa	24	M	Student	unmarried	suicidal	13/04/2021	17/04/202	rural	oral	100	unknown	62	90/70	21	36	none		conscious and rationale	absent	1	mild
48	48	15725	siddu amogi handiganur	25	M	painter	unmarried	suicidal	04-10-202	18/04/202	rural	oral	100	unknown	56	100/70	24	36	generalised	pinpoint	Impaired to verbal command	present	7	moderate
49	49	80838	veenashri b hiremath	21	F	Student	unmarried	suicidal	16/06/202	17/06/202	rural	oral	unknown	monocrotophos	56	90/70	26	36	generalised	pinpoint	No response to verbal command	present	8	severe
50	50	80884	appasaheb siddappa	27	M	driver	married	suicidal	16/06/2021	18/06/202	rural	oral	unknown	unknown	58	90/70	26	36	generalised		Impaired to verbal command	present	2	mild
51	51	106692	altaf miyasab yalgar	21	M	Student	unmarried	suicidal	07-12-202	18/07/202	rural	oral	unknown	unknown	58	90/70	23	36	none		conscious and rationale	present	3	mild
52	52	112251	kaveri paramanand mali	22	F	housewife	unmarried	suicidal	14/7/2021	17/07/202	rural	oral	unknown	unknown	68	100/80	22	36	none		conscious and rationale	absent	1	mild
53	53	122284	ammangouda laxman	40	M	farmer	married	suicidal	26/07/2021	29/07/202	rural	oral	100	monocrotophos	62	110/70	24	36	generalised		conscious and rationale	absent	2	mild
54	54	141924	ravigodihal	20	M	Student	unmarried	suicidal	08-12-202	18/08/202	urban	oral	50	chlorpyrifos	62	120/70	22	36	generalised		conscious and rationale	absent	3	mild
55	55	165727	basavaraj shinde	55	M	farmer	married	suicidal	09-02-202	09-06-202	rural	oral	100	unknown	64	110/80	21	36	none		Impaired to verbal command	absent	2	mild
56	56	168053	dundawwa sidaraya	48	F	housewife	married	suicidal	09-04-202	09-07-202	rural	oral	100	unknown	66	110/88	18	36	generalised		conscious and rationale	absent	1	mild
57	57	175770	madiwalappa telagani	35	M	farmer	married	suicidal	09-12-202	27/09/202	rural	oral	unknown	unknown	56	90/60	26	36	continuous	pinpoint	Impaired to verbal command	present	7	moderate
58	58	176836	mangala hyalawar	17	F	Student	unmarried	suicidal	13/09/202	15/09/202	rural	oral	unknown	monocrotophos	66	100/70	24	36	continuous		conscious and rationale	absent	2	mild
59	59	191392	somaninga hugar	25	M	Student	unmarried	suicidal	25/09/202	28/09/202	rural	oral	50	propenofos	58	110/70	18	36	generalised		conscious and rationale	absent	2	mild
60	60	207015	veeresh chandrayya	34	M	driver	married	suicidal	10-09-202	15/09/202	rural	oral	unknown	unknown	56	100/60	26	36	continuous	pinpoint	Impaired to verbal command	present	7	moderate
61	61	214728	shankar somasul	25	M	Student	unmarried	suicidal	14/10/202	21/10/202	rural	oral	50	unknown	66	110/80	22	36	continuous		conscious and rationale	absent	3	mild
62	62	226894	shivanandn pawar	28	M	driver	married	suicidal	23/10/202	29/10/202	urban	oral	100	unknown	58	110/70	24	36	generalised		conscious and rationale	absent	4	moderate
63	63	229423	dharamaraj biradar	20	M	Student	unmarried	suicidal	26/10/202	27/10/202	urban	oral	unknown	unknown	64	110/80	18	36	none		conscious and rationale	absent	0	mild
64	64	230847	nagarajm adaraki	36	M	farmer	married	suicidal	27/10/202	31/10/202	rural	oral	unknown	unknown	62	110/80	22	36	generalised		conscious and rationale	absent	3	mild
65	65	240267	parashuram mareppa	35	M	farmer	married	suicidal	11-01-202	11-02-202	rural	oral	unknown	unknown	56	90/70	26	36	generalised	pinpoint	Impaired to verbal command	present	7	moderate
66	66	245464	santram ramkumbar	60	M	farmer	married	suicidal	11-07-202	13/11/202	rural	oral	100	monocrotophos	66	140/80	24	36	none		conscious and rationale	absent	2	mild
67	67	259645	savitri r bolegaon	19	F	Student	unmarried	suicidal	20/11/202	24/11/202	urban	oral	100	unknown	68	110/70	26	36	generalised		conscious and rationale	absent	2	mild
68	68	259688	santosh todalabagi	32	M	painter	married	suicidal	19/11/202	21/11/202	urban	oral	unknown	unknown	56	100/70	18	36	none		conscious and rationale	absent	1	mild
69	69	266727	jagadish namadev	52	M	farmer	married	suicidal	24/11/202	27/11/202	rural	oral	unknown	unknown	58	130/70	20	36	none		conscious and rationale	absent	2	mild
70	70	282396	shankarling teli	28	M	Student	married	suicidal	12-05-202	05-12-202	urban	oral	100	monocrotophos	68	110/70	22	36	none		conscious and rationale	absent	2	mild
71	71	289908	sadashiv madarkhandi	30	M	driver	married	suicidal	12-10-202	12-11-202	rural	oral	unknown	unknown	66	110/80	18	36	none		conscious and rationale	absent	0	mild
72	72	293122	sharanamma hoonalli	28	F	housewife	married	suicidal	12-12-202	17/12/202	rural	oral	unknown	unknown	60	100/60	25	36	generalised		conscious and rationale	absent	3	mild
73	73	294374	vishal ramesh naik	21	M	Student	unmarried	suicidal	13/12/202	01-05-202	urban	oral	unknown	unknown	58	90/60	26	36	continuous	pinpoint	no response to verbal command	present	8	severe
74	74	314535	mohan somanath chavn	28	M	driver	married	suicidal	29/12/202	29/12/202	urban	oral	unknown	unknown	60	90/60	24	36	continuous	pinpoint	no response to verbal command	present	7	moderate
75	75	28355	shreedevi hiremath	26	F	housewife	married	suicidal	21/12/202	22/12/202	rural	oral	unknown	unknown	58	100/80	19	36	none		conscious and rationale	absent	1	mild
76	76	164172	bharati b teli	22	F	housewife	married	suicidal	17/05/202	18/05/202	rural	oral	unknown	unknown	58	110/80	22	36	none		conscious and rationale	absent	2	mild
77	77	58479	girish bhimanna kabbin	22	M	Student	unmarried	suicidal	14/02/202	16/02/202	rural	oral	unknown	chlorpyrifos	54	100/70	16	36	generalised		conscious and rationale	absent	2	mild
78	78	55331	jayashree vittal	22	F	Student	unmarried	suicidal	13/01/202	15/01/202	urban	oral	100	chlorpyrifos	70	90/70	18	36	generalised		conscious and rationale	absent	1	mild
79	79	76288	basappa bmoole	29	M	farmer	married	suicidal	06-11-202	18/06/202	rural	oral	unknown	unknown	58	110/80	22	36	none		conscious and rationale	absent	2	mild
80	80	78100	amarvishnu more	24	M	farmer	married	suicidal	16/06/202	17/06/202	rural	oral	unknown	unknown	56	110/80	28	36	generalised	pinpoint	Impaired to verbal command	present	7	moderate
81	81	81703	giddaya matapati	35	M	farmer	married	suicidal	24/06/202	25/06/202	rural	oral	100	unknown	42	110/80	26	36	generalised	pinpoint	no response to verbal command	present	8	severe
82	82	317216	girish kolli	25	M	Student	unmarried	suicidal	30-12-202	15/1/2022	urban	oral	50	unknown	44	110/80	24	36	generalised	pinpoint	impairedtoverbal command	present	7	moderate
83	83	211848	muttanna aurasang	31	M	driver	married	suicidal	10-12-202	13/10/202	rural	oral	unknown	unknown	78	110/70	19	36	none		conscious and rationale	absent	0	mild
84	84	130679	santosh p kulkarani	40	M	farmer	married	suicidal	08-02-202	08-07-202	rural	oral	unknown	unknown	68	130/80	22	36	none		conscious and rationale	absent	2	mild
85	85	168520	hanamangouda g nadhli	56	M	farmer	married	suicidal	18/05/2022	26/05/202	rural	oral	100	chlorpyrifos	58	140/80	22	36	generalised		conscious and rationale	absent	4	moderate
86	86	166980	jyoti prabhuling biradar	24	M	Student	unmarried	suicidal	17/05/2022	20/05/202	urban	oral	50	monocrotophos	66	100/70	22	37	generalised	pinpoint	conscious and rationale	absent	4	moderate
87	87	293150	anjum gudusab kolur	28	F	housewife	married	suicidal	14/12/2021	16/12/202	rural	oral	unknown	unknown	78	90/70	18	36	generalised		conscious and rationale	absent	1	mild
88	88	151766	satish g sajjan	22	M	Student	unmarried	suicidal	05-05-202	05-07-202	urban	oral	100	propenofos	66	100/70	20	36	generalised		conscious and rationale	absent	2	mild
89	89	146288	kanchana holagi	14	F	Student	unmarried	suicidal	30/04/2022	05-06-202	urban	oral	unknown	unknown	66	90/60	24	36	generalised		conscious and rationale	absent	3	mild
90	90	132706	ningaraj hugar	22	M	Student	unmarried	suicidal	20/04/2022	26/04/202	rural	oral	100	monocrotophos	66	100/60	22	37	continuous		no response to verbal command	present	6	moderate
91	91	127612	renuka sonagar	27	F	housewife	married	suicidal	15/04/2022	05-05-202	rural	oral	unknown	chlorpyrifos	44	100/60	28	37	continuous	pinpoint	Impaired to verbal command	present	7	moderate
92	92	105797	raju denu rathod	28	M	painter	married	suicidal	26/03/2022	28/03/202	urban	oral	unknown	isahion	66	110/70	22	36	continuous		conscious and rationale	absent	2	mild
93	93	99436	danamma jambagi	18	F	Student	unmarried	suicidal	21/03/2022	24/03/202	rural	oral	unknown	unknown	82	100/70	18	36	none		conscious and rationale	absent	2	mild
94	94	99898	gangamma walikar	25	F	housewife	married	suicidal	21/03/2022	24/03/202	rural	oral	unknown	propenofos	78	110/80	22	36	none		conscious and rationale	absent	1	mild
95	95	97109	sangangouda s patil	30	M	driver	married	suicidal	18/03/2022	25/03/202	urban	oral	unknown	chlorpyrifos	60	120/70	25	36	generalised		conscious and rationale	absent	3	mild

S.NO	A/D	Venti	PDC	Smell of OP	Smoker	Alcoholic	DM	HTN	Past hist	CVS	RS	PA	CNS	No.of da	Sweat	Neck weak	Cholinesterase	hb	TC	PLT	RBS	CREAT	Na	K	srbil	SGOT	SGPT	ALB	ALP
1	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	yes	29	11.	11.6	2	9	0	14	4	0.		1	4	9
2	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou		no	no	16	1	16.1	2.2	9	0	14	4	0.		1	4	6
3	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	850	1	12.0	1	10	0	14	3	0.		2	4	9
4	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	500	17.	14.2	2.4	10	1	14	4	1.		2	4	8
5	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	61	12	12.3	2.8	11	0	14	4	1.		1	3	7
6	alive	no	op+	present	yes	no	no	no	nil	s1s2+	crepts+	soft	consciou		yes	yes	690	13	7.5	1.5	8	0	14	3	1.		5	3	8
7	alive	no	op+	present	no	yes	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	yes	68	13	1	36	10	0	14	4	1.		2	4	8
8	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	83	14	7.	2.5	10	0	14	4	0.		1	4	14
9	alive	no	op+	present	yes	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	57	15	8.	3.0	11	0	14		0.		1	3	7
10	alive	no	op+	no	no	no	no	no	nil	s1s2+	nvbs+	soft	drowsy	1	yes	yes	15	11	8	2.8	11	0	14	3	0.		1	3	7
11	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	nvbs+	soft	drowsy		no	no	83	1	10.8	4.1	12	0	14	4	0.		1	3	7
12	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou		no	no	60	12	9.	2.5	9	0	14	4	1.		1	3	10
13	alive	no	op+	present	yes	yes	no	yes	nil	s1s2+	nvbs+	soft	consciou		yes	yes	49	14.	6.	2	9	0	14	5	0.		1	3	7
14	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	yes	16	13	12.6	1.5	10	0	13	3	1.		2		11
15	Death	intubated	op+	present	yes	no	no	no	yes	s1s2+	crepts+	soft	drowsy	death	yes	yes	17	14.	8.	3	10	0	16	4	0.		1	3	9
16	alive	no	op+	present	no	no	yes	no	nil	s1s2+	nvbs+	soft	consciou		yes	yes	36	1	10.1	2.3	15	0	14	3	0.		2	3	6
17	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	drowsy	1	yes	yes	16	12	8	1.6	10	0	14	4	1.		2	4	7
18	alive	no	op+	present	yes	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	27	9	5.5	2.5	9		14	3	1.		2	3	9
19	Death	intubated	op+	present	yes	no	no	no	yes	s1s2+	crepts+	soft	drowsy	death	yes	yes	17	10.	15.6	1.8	15	1	14	4	1.		30	3	15
20	alive	no	op+	present	no	no	no	yes	nil	s1s2+	nvbs+	soft	consciou		no	no	76	13	9	1.9	11	0	14	3			2	4	16
21	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	63	14.	8.5	3.7	10	0	14	4	0.		1	3	9
22	alive	no	op+	present	yes	yes	no	yes	nil	s1s2+	nvbs+	soft	consciou		yes	yes	55	15.	7.	2.4	9	0	14	3	0.		1	4	9
23	alive	no	op+	present	yes	no	no	yes	nil	s1s2+	crepts+	soft	drowsy		yes	yes	36	12	2.	8	9	0	13	2	1.		5	4	9
24	alive	intubated	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	drowsy	1	yes	yes	14	15.	7.	1	9	0	13	3	1.		4	4	8
25	Death	intubated	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	drowsy	death	yes	yes	17	9.	36.9	4.7	8	1	15	3	5.	1	11	3	9
26	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	59	12	7.	2.5	11	0	14	4	0.		1	4	11
27	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	89	1	8	1.7	8	0	14		0.		2	4	8
28	alive	no	op+	present	yes	no	no	no	nil	s1s2+	nvbs+	soft	consciou	1	yes	yes	2	15.	15.1	1.4	8	0	14	3	1.		1	5	7
29	alive	no	op+	present	yes	no	no	yes	nil	s1s2+	nvbs+	soft	drowsy		no	no	67	15.	5	1.6	8		14	3	0.		2	33	6
30	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	53	16.	16.5	3.2	9		13	3	0.		3	4	8
31	alive	no	op+	present	yes	no	no	no	nil	s1s2+	nvbs+	soft	drowsy		no	no	78	10.	12.0	5.7	11	0	14	4	1.		3	4	11
32	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	59	12	12	2.0	9	0	14	4	1.		3	3	7
33	Death	intubated	op+	present	yes	yes	yes	no	yes	s1s2+	crepts+	soft	drowsy	death	yes	yes	15	1	20.2	2.1	18	0	13		1.		1		6
34	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	59	13	7.5	2.1	11	0	13	4			1	3	10
35	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	45	15	12	4.1	8	0	14	4	1.		1	4	8
36	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	yes	52	14	15	1.4	9	0	13	3	0.		2	4	6
37	alive	no	op+	present	yes	no	no	yes	nil	s1s2+	nvbs+	soft	drowsy		no	no	40	14	15.0	4	10	0	13	4			2	4	9
38	alive	intubated	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou	2	yes	yes	2	11	27.2	1.7	10	0	13	3	2.		5	4	10
39	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	yes	56	11	12	1	10	0	14	3	3.		6	3	11
40	alive	no	op+	present	yes	yes	no	yes	nil	s1s2+	crepts+	soft	drowsy		no	no	56	13	10.2	1.7	8	0	14	3	0.		1	3	6
41	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou		no	no	52	10	8.	2	9	0	14	4			6	3	11
42	Death	intubated	op+	present	yes	yes	yes	no	nil	s1s2+	crepts+	soft	drowsy	death	no	no	11	10	17.1	1.6	11	1	14		0.		1	2	5
43	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	yes	76	11	7.	2.6	8	0	14	4	0.		1	4	6
44	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	31	12	16.1	2.9	8	0	14	4	0.		1	4	7
45	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou			no	47	14	6.	2	10	0	14	5	0.		1	3	7
46	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	drowsy		yes	no	42	13	13.	2.6	10	0	14	4	0.		1	3	10
47	alive	no	op+	present	yes	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	45	13	9.	2.4	9	0	14	3	0.		1	3	6
48	Death	intubated	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	drowsy	death	yes	yes	1	12	12	4	10	0	14	3	0.		8		11
49	Death	intubated	op+	present	yes	yes	no	no	nil	s1s2+	crepts+	soft	drowsy	death	yes	yes	30	1	16.	3.2	8	0	14				2		7
50	Death	intubated	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	drowsy	death	yes	yes	5	14	8.	2	11	0	14	3	0.	1	6	3	5
51	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	drowsy		yes	yes	5	12	8	2	11	0	13	3	0.	1	5		11
52	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou		yes	yes	35	13	17.0	2.6	10	0	13	4	0.	1	5		11
53	alive	no	op+	present	yes	yes	yes	no	nil	s1s2+	crepts+	soft	consciou		yes	yes	65	12	13	1	20	0	14		1.		2		6
54	alive	no	op+	present	yes	no	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	yes	15	13	13	1	11	0	13	3	1.		2		6
55	alive	no	op+	present	yes	yes	no	yes	nil	s1s2+	crepts+	soft	consciou		yes	yes	8	1	13	1	11	0	14	4	1.		2		6
56	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou		yes	yes	28	11	10.	2.5	10	0	14	3	1.		4		7
57	alive	intubated	op+	present	yes	yes	no	no	nil	s1s2+	crepts+	soft	drowsy	1	yes	yes	5	12	5.	2.9	11	0	13	3	1.		3	2	6
58	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	62	11	5	2	10	0	13	4	1.		3		6
59	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	20	13	1	2	10	0	14	3	0.		3	4	9
60	alive	intubated	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	drowsy	death	yes	yes	1	14	14.9	3.5	10	0	14	4	1.		5	4	6
61	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou		yes	no	42	1	1	3	10	0	13	3	1.		5		6
62	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou		yes	yes	30	13	9.	2.8	11	0	13	3	1.		5	3	6
63	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	yes	64	11	9.	2.7	10	0	13	4	1.		5	3	6
64	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	crepts+	soft	drowsy		no	no	30	15	13.	3	10	0	14	3	1.		5	3	6
65	Death	intubated	op+	present	yes	yes	no	no	nil	s1s2+	crepts+	soft	drowsy	death	yes	yes	8	14	7.	3.0	10	0	14	3			6	2	8
66	alive	no	op+	present	yes	yes	no	yes	nil	s1s2+	crepts+	soft	consciou		yes	yes	50	1	13.	2.4	11	0	14	3	1.		4	3	11
67	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	30	1	14	2.4	11	0	13	3	0.		3	4	16
68	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	58	14	10.	1.9	10	0	14		2.		1	3	3
69	alive	no	op+	present	yes	yes	no	yes	nil	s1s2+	nvbs+	soft	consciou		no	no	56	11	13.	3.0	11		14	4			1	3	4
70	alive	no	op+	present	yes	no	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	yes	37	14.	12	2	9	0	14	3	1.		1	3	4
71	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	crepts+	soft	consciou		yes	yes	80	16	12.	2.6	7		14	3	2.		9	3	7
72	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	10	11		2	10	0	14	3	2.		7	3	7
73	Death	intubated	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	drowsy	death	no	no	1	8	10.	1	11	0	16	3	2.		21	2	15
74	Death	intubated	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	drowsy	death	no	no	50	15	7.	2.5	10	0	14	4	1.		6	4	13
75	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs	soft	consciou		no	no	64	9	7.	2.9	11	0	13	4	1.			3	5
76	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	51	9	13.	3.2	11	0	13	4	1.		6		5
77	alive	no	op+	present	yes	no	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	no	57	15	8.	3.0	9	0	14		0.		1	3	7
78	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	27	12	6.	2.3	12	0	13	3	0.		2	3	4
79	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	81	12	13.	2	11	0	14	4	0.		4		7
80	Death	intubated	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	drowsy	death	no	no	5	12	22.	3	11	5	13	7	3.	3	33	2	8
81	Death	intubated	op+	present	yes	yes	no	no	nil	s1s2+	crepts+	soft	drowsy	death	yes	yes	4	14	10.	1	11	0	13	3		3	11	2	7
82	alive	intubated	op+	present	yes	yes	no	no	nil	s1s2+	crepts+	soft	drowsy	1	yes	yes	10	16	9.	3.8	11	0	14	3	1.		2	2	6
83	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	79	16	15.	3.7	10	0	14	4	0.		5	4	14
84	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	crepts+	soft	consciou		no	no	67	17.	8.	1.6	9	0	14	3	1.		4	4	10
85	alive	no	op+	present	yes	yes	no	yes	nil	s1s2+	crepts+	soft	drowsy		yes	yes	2	16	8.	3	9	0	14	3	1.		4	3	8
86	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou		no	no	51	10	7	2.0	8	0	13		0.		1	4	9
87	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	no	32	11	9.	2.1	9	0	14	3			5	3	8
88	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	61	13	5.	1.9	10	0	13	4			3	3	7
89	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	yes	63	11	7	2.0	9	0	13	3	0.		2	4	8
90	alive	intubated	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		yes	yes	1	15	12.9	2.6	9	0	13	3	0.		1	4	9
91	alive	intubated	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	drowsy	2	yes	yes	2	11	8.	3.9	14	0	14	2	0.		2	2	8
92	alive	no	op+	present	no	no	no	no	nil	s1s2+	crepts+	soft	consciou		no	yes	62	13	10.	3.5	9	1	14	4	0.		2		7
93	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	71	12	7.	0.2	9	0	14	4	0.		1	3	8
94	alive	no	op+	present	no	no	no	no	nil	s1s2+	nvbs+	soft	consciou		no	no	59	12	9.	2.5	8	0	15	4	0.		1	3	12
95	alive	no	op+	present	yes	yes	no	no	nil	s1s2+	crepts+	soft	consciou		yes	no	55	15	7.	1	10	0	14	3	1.		1	3	5