A Dissertation on

"A STUDY TO ASSESS THE UTILITY OF PERADENIYA ORGANOPHOSPHORUS POISONING (POP) SCALE, POISONING SEVERITY SCORE (PSS) AND GLASGOW COMA SCALE (GCS) IN PREDICTING SEVERITY AND TREATMENT OUTCOME IN ACUTE ORGANOPHOSPHORUS POISONING PATIENTS ADMITTED IN A TERTIARY CARE CENTRE "

Submitted in partial fulfilment of requirements for

M.D. DEGREE BRANCH – I

GENERAL MEDICINE

OF

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

INSTITUTE OF INTERNAL MEDICINE

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003

MAY 2023

CERTIFICATE-I

This is to certify that this dissertation entitled **"A STUDY TO ASSESS THE UTILITY OF PERADENIYA ORGANOPHOSPHORUS POISONING (POP) SCALE, POISONING SEVERITY SCORE (PSS) AND GLASGOW COMA SCALE (GCS) IN PREDICTING SEVERITY AND TREATMENT OUTCOME IN ACUTE ORGANOPHOSPHORUS POISONING PATIENTS ADMITTED IN A TERTIARY CARE CENTRE "** submitted by Dr.ANNADURAI S appearing for M.D. Branch I - General Medicine Degree examination in MAY-2023 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfilment of regulations of the TamilNadu Dr. M.G.R. Medical University, Chennai. I forward this to the TamilNadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

Prof. Dr. NALINI KUMARAVELU, M.D.,
Professor of Medicine,
Institute of Internal medicine,
MMC & RGGGH,
Chennai- 600 003.

PROFESSOR
INSTITUTE OF INTERNAL MEDICINE
MMC & RGGGH, CHENNAI-600 003

Prof. Dr. C.HARIHARAN, M.D.,
Professor and Director,
Institute of Internal Medicine,
MMC & RGGGH,
Chennai- 600 003.

DIRECTOR AND PROFESSOR
Institute of Internal Medicine
Madras Medical College,
Govt. General Hospital
Madras-600 003

Prof. Dr. S.RAGUNANTHANAN, M.D.,
Professor of Medicine,
Chief of IMCU and Toxicology,
Institute of Internal medicine,
MMC & RGGGH,
Chennai- 600 003.

Prof. Dr. E. THERANIRAJAN, M.D, DCH, MRCPH (UK), FRCPH (UK)
The Dean,
MMC & RGGGH,
Chennai- 600 003

DEAN
MADRAS MEDICAL COLLEGE
CHENNAI-600 003.

CERTIFICATE OF GUIDE

This is to certify that the dissertation titled, **" A STUDY TO ASSESS THE UTILITY OF PERADENIYA ORGANOPHOSPHORUS POISONING (POP) SCALE, POISONING SEVERITY SCORE (PSS) AND GLASGOW COMA SCALE (GCS) IN PREDICTING SEVERITY AND TREATMENT OUTCOME IN ACUTE ORGANOPHOSPHORUS POISONING PATIENTS ADMITTED IN A TERTIARY CARE CENTRE "** is a bonafide work done by **Dr. ANNADURAI S**, registration number : 200120100505 at Madras Medical College, Chennai in partial fulfilment of the university rules and regulations for award of M.D., Degree in General Medicine (Branch-I) under my guidance and supervision during the academic year 2020-2023.

Prof. Dr. NALINI KUMARAVELU M.D.
Professor of Medicine,
Institute of Internal Medicine,
Madras Medical College,
RGGGH, Chennai – 600 003.

PROFESSOR
INSTITUTE OF INTERNAL MEDICINE
MMC & RGGGH, CHENNAI-600 003

DECLARATION BY THE CANDIDATE

I, **Dr. ANNADURAI S,** Registration number **200120100505** hereby solemnly declare that the dissertation entitled **"A STUDY TO ASSESS THE UTILITY OF PERADENIYA ORGANOPHOSPHORUS POISONING (POP) SCALE, POISONING SEVERITY SCORE (PSS) AND GLASGOW COMA SCALE (GCS) IN PREDICTING SEVERITY AND TREATMENT OUTCOME IN ACUTE ORGANOPHOSPHORUS POISONING PATIENTS ADMITTED IN A TERTIARY CARE CENTRE "** was done by me at the Institute of Internal Medicine, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai during the academic year 2020 - 2023 under the guidance and supervision of **Prof.Dr.NALINI KUMARAVELU M.D., & Prof.Dr.S.RAGUNANTHANAN M.D.,** This dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University, Chennai towards the partial fulfilment of requirement for the award of M.D. Degree in General Medicine (Branch I)

Place: CHENNAI

Date: 14/12/22

Dr.ANNADURAI S
Post Graduate student,
M.D. General Medicine,
Institute of Internal Medicine,
Madras Medical College,
Chennai - 600 003.

ACKNOWLEDGEMENT

I express my heartfelt gratitude to **the Dean, Prof. Dr. E.THERANIRAJAN, M.D., DCH. MRCPH (UK), FRCP (UK).** Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-3 for permitting me to do this study.

I am very grateful to **Prof. Dr.NALINI KUMARAVELU, M.D.,** Professor of Medicine, Institute of Internal Medicine, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-3 who guided and trimmed my work throughout the period of my study and for her constant support.

I am thankful to the Director and Head of the Department, **Prof. Dr. C. HARIHARAN, M.D,** who had approved and supported my work.

I also thank **Prof. Dr.S.RAGUNANTHANAN, M.D,** Professor of Medicine, who provided expert advice for this study.

I am very much thankful for the help rendered by the Registrar **Dr. P.BALAMANIKANDAN, MD** and my Assistants Professor **Dr. SENTHILPRIYAN, M.D., & Dr. KIRUTHIKA,M.D.,** for their constant help and encouragement.

I am very much thankful for the help rendered by my beloved colleagues I thank all my professional colleagues (Dr. VISHNU VARTHAN, Dr. PAVITHRA, Dr. HEMAVARTHINI, Dr. SAKTIVEL,) for their support and valuable criticism.

Above all, I express my heartfelt gratitude to my Parents and Friends for their unwavering love, prayers and encouragement. I would not have reached this far without them.

I am extremely thankful to all the Members of the **INSTITUTE ETHICS COMMITTEE** for giving approval for my study.

I sincerely thank all the patients who have submitted themselves for this study and made it possible.

TABLE OF CONTENTS

LIST OF TABLES

ABBREVIATIONS

OPC : Organophosphorus compounds

IMCU : Intensive Medical Care Unit

COPIND : Chronic organophosphate Induced Neuropsychiatric disorder

OPIND : OPC Induced delayed polyneuropathy

AChE : Acetylcholinesterase

PChE : Plasma pseudocholinesterase

SGPT/ALT : Alanine Amino Transeferse

SGOT/AST : Aspartate Amino Transeferase

ALP : Alkaline Phosphatase

FFP : Fresh Frozen Plasma

HR : Heart Rate

RR : Respiratory Rate

BP : Blood Pressure

IMS : Intermediate

Syndrome CREAT : Creatinine

K + : Potassium

MV : Mechanical Ventilation

WHO : World health organisation TEPP : Tetraethyl pyrophosphate 2-PAM : Pralidoxime

ABSTRACT

Introduction:

Organophosphate compounds are often used for homicidal and suicidal purposes and continues to be an important cause of poisoning in developing countries including India.It accounts for about 80% of pesticide related hospital admissions. In rural areas hospitals are not adequately staffed or equipped to treat very sick patients. ICU and ventilators are in short supply to meet the above burden. In such situations early assessment by clinical markers is very important to categorise the severity and early referral to higher centres so that aggressive treatment can be initiated immediately.

Aims and objectives:

Study aimed to assess the severity of OP poisoning by POP scale, PSS and GCS and to compare POP scale, PSS and GCS in predicting the treatment outcome in OPC poisoning.

Material and Methods:

In our study, 142 patients who satisfied the inclusion and exclusion criteria with Organophosphorus compound poisoning were included.POP score, PSS and GCS were assessed at the time of presentation and were grouped based on the severity. These scores were evaluated individually in predicting the need for ventilator requirement and mortality in OPC poisoning and the scores were compared with each other, so as to assess which score was better in predicting the severity of OPC poisoning.

Results:

Ventilator requirement and mortality was found to be in 37.3% and 8.5% of the patients respectively. Intubation and mortality rates were higher in patients with a severe category of POP score, PSS and GCS with a significant p value of (0.005), than in the mild to moderate category . All three scores correlated well in predicting the requirement of mechanical ventilation and mortality in OPC poisoning cases.

Conclusion:

All the three scoring systems are simple and effective tools that can be assessed based on the clinical examination. For resource limited countries, like India, any of the three scoring systems can be used at the primary health care setting level which helps in making timely decisions regarding need for mechanical ventilation and timely shifting of patients to the ICU care setting for management.

INTRODUCTION

Organophosphorous (OPC) compounds produce significant morbidity and mortality in India. Because India is being an agricultural country, Organophosphorus insecticides are the main agents for crop protection and pest control. Therefore, it is probable to have negative consequences on farmers who unintentionally overexpose themselves when handling these pesticides. They are used as petroleum additives and nerve gases like Sarin,tabun and Soman in chemical warfare[1,2].Due to its low cost and wide availability, it has also turned into a preferred method for self-poisoning. Approximately 300000 individuals per year die from self-poisoning by pesticides worldwide, with rural areas accounting for the majority of these deaths.Mortality is as high as 70% in developing countries like India.[3] In India, insecticide poisoning and hanging are the two most popular ways to end one's life, with males (70.54%) and females (29.47%) committing suicide at higher rates.[4] Insecticides containing organophosphorus (OP) may be one of the most widespread global causes of poisoning-related morbidity and mortality. Due to the accessibility of these substances, it is common in poorer nations like India. In rural areas of India, OP pesticide self-poisoning is a significant clinical issue. In areas where very toxic OP insecticides are available, accidental poisoning is a problem even though it kills significantly fewer individuals.

Organophosphates are structurally related to acetylcholine and bind covalently with the cholinesterase molecule. This causes increased level of Ach at synapses, which overstimulates postsynaptic receptors in the central and peripheral nervous systems.The stimulation of muscarinic and nicotinic ACh receptors in the parasympathetic system, sympathetic ganglia, neuromuscular junction, and within the nervous system results in the clinical symptoms of acute organophosphorus poisoning.[5]

The type of organophosphorus substance involved determines the severity and timing of the clinical symptoms of poisoning.Most common complication that leads to death is respiratory failure.The majority of this issue is concentrated in rural hospitals, which annually treat hundreds of patients who have been poisoned by pesticides. These hospitals frequently lack the staff or resources necessary to handle these seriously ill patients. To handle the burden, there is a shortage of ICU beds and ventilators. To classify the severity in such circumstances and to expeditiously assign patients to a higher level of care where aggressive treatment can start right away, early clinical marker assessment is crucial. Numerous research have been conducted to examine the diagnostic and prognostic usefulness of blood cholinesterase levels in OP poisoning and their association with neurological symptoms.[6,7]

Serum cholinesterase can be utilised as a diagnostic marker, however the majority of research have found that it plays a very small function as a prognosis marker.[8,9] In order to accurately forecast the severity of a condition and the clinical outcome, it is crucial to compare the readily available grading systems.

The aim of the study is to compare POP (Peradeniya Organophosphorus) scale, PSS(Poisoning Severity Score), and GCS(Glasgow Coma Scales) in predicting the severity and treatment outcome in OP Poisoning.

AIMS AND OBJECTIVES

- To assess the correlation of POP scale, PSS and GCS in predicting

Clinical severity,

- Ventilator requirement and
- Treatment outcome in OPC poisoning patients.

REVIEW OF LITERATURE

Organophosphates are a class of chemical compounds made mostly of amides, esters, and thiols that are phosphoric acid derivatives. They make up the largest class of insecticides used in farming, particularly in south India.

The first OPCs were created by mediaeval alchemists. Phillipe de Clermont and Jean Louis Lassaigne, however, were among the early innovators in the methodical investigation of these compounds in the early nineteenth century. Tetraethyl pyrophosphate (TEPP) the first organophosphorus chemical, was created in 1854,[10] but it was not actively used until World War II. Gerhard Schrader, a German chemist, experimented with OPC as insecticides in the 1930s and later as potential warfare agents(Tabun, Sarin, and Soman) [the G series weapons].

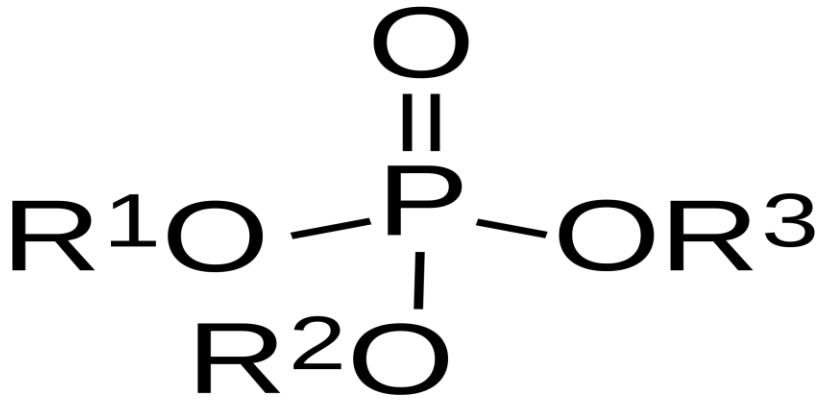
After World War II Organophosphate pesticides were first synthesised in considerable amounts by American companies. The first substance was Parathion.Then followed by other compounds like Malathion, Azinophosmethyl etc.

There are now many distinct OPs with various biological characteristics that are utilised as fungicides, insecticides, nematocides, and insecticides. Organophosphates and carbamates are strong cholinesterase inhibitors that can result in severe cholinergic toxicity when ingested, inhaled, or applied topically. Although they are chemically different, organophosphates and carbamates have comparable clinical toxicity symptoms and need the same treatment after an overdose.

STRUCTURE OF ORGANOPHOSPHATES

Organophosphorus compounds are a varied category of substances, however they all share some similar chemical characteristics. Organophosphorus has two organic side chains (R1 and R2), an extra side chain that serves as the leaving group, and a central phosphorus atom with a double bond to either oxygen (P=O) or sulphur (P=S) (x). The specific leaving group for each OP compound may be a cyanide, thiocyanate, halide, phosphate, phenoxy, thiophenoxy, or carboxylate group. In most common pesticides, the R1 and R2 groups are either two methyl or two ethyl ester groups, which together create the dimethyl or diethyl OPs. These groups are aryl or alkyl groups and are found in most common pesticides.[11]

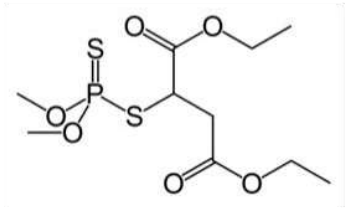
The general chemical structure

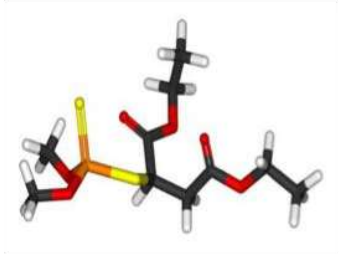


CHEMICAL STRUCTURE OF SOME OF THE

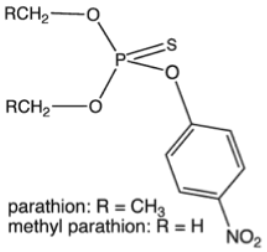
COMMON ORGANOPHOSPHATE POISONS

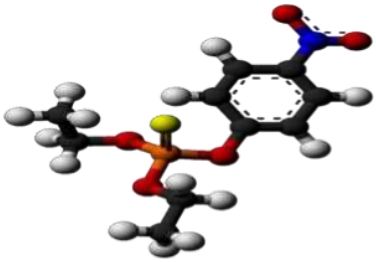
Malathion:



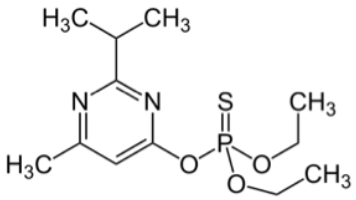


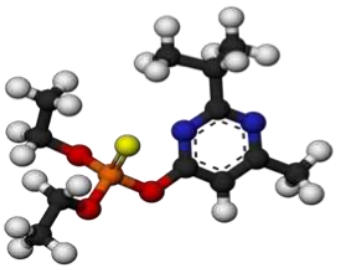
Parathion



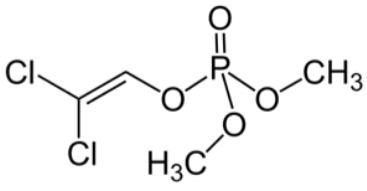


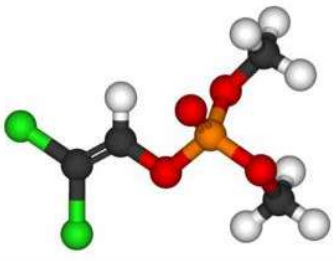
Diazinon

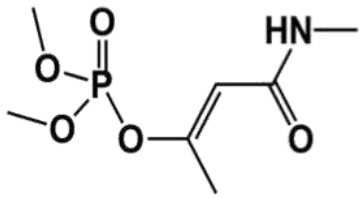
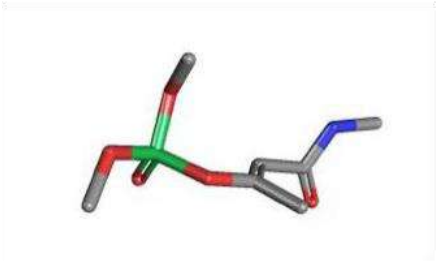
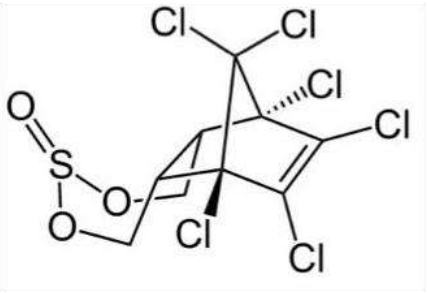




Dichlorvos





Monocrotophos  	Endosulfan  

CLASSIFICATION [12]

1) Based on volatility

- Insecticides made of **low-volatile** substances

  Eg: methyl parathion, chlorpyrifos, and dimethoate.

- **Highly flammable**: used in nerve gas form in chemical warfare

  Sarin, Tabun, VX

  OR

2) Chemical categorization

- **Aryl phosphates**

  HETP, demeton, malathion, and trichlorfon.

- **Alkyl phosphates**

  diazinon, methyl parathion, parathion, and paraoxon

  (OR)

3. Depending on toxicity

a) Highly hazardous or extremely toxic: LD 51-500 mg/kg or 1-50 mg/kg chlorpyrifos, diazinon, dimethoate, fenthion, methyl parathion, monocrotophos, phorate, and quinalphos.

b) Moderately toxic (LD 501–5000 mg/kg).

Slightly toxic (LD: greater than 5000mg/kg) Malathion, Temephos, and Triazophos LD, which is the dosage in mg/kg of body weight that results in 50% of exposed animals dying.

LOW TOXICITY	MODERATE TOXICITY	HIGH TOXICITY
Bromophos	Chlorpyrifos	Tetraethyl pyrophosphate
Eltrimfos	Crotoxyphos	Parathion
Lodofenphos	Cyanophos	Phorate
Malathion	Cythiote	Monochrotophos
Phoxim	Acephate	Chlormephos
Temephos	DEF	Cyanofenpos
Tetrachlorvinphos	Diazinon	Demeton
	Dichlofenthion	Dicrotophos
	Dimethoate	EPN
	Dichlorphos	Fenamiphos
	Ethion	Fonofos
	Formothion	Isofenphos
	Heptenofos	Mephosfolon
	Isoxathion	Mevinphos
	Methyltrithion	Phosphamidon
	Naled	Terbufos
	Oxydemeton-methyl	
	Pyrazophos	
	Quinolphos	
	Sulprofos	
	Triazophos	

MODES OF OPC POISONING

1. **Accidental poisoning.**
2. **Suicidal poisoning.**

ROUTES OF POISONING

1. **INHALATION**: Pesticides applied to plants, animals, household surfaces, or carpets in poorly ventilated spaces may be inhaled through the air.
1. **INGESTION** : of fruits and vegetables that have been pesticide-treated before eating.
2. **CONSUMING** water from containers that have been tainted with poison that has been left on the premises.
3. **ABSORPTION**: Unwashed hands after touching insecticides can absorb.

PHARMACOKINETICS

Compounds containing organophosphorus are absorbed by the skin, digestive system, and lungs. Because cutaneous absorption is influenced by the lipid affinity of the chemicals, it is accelerated by damaged skin, dermatitis, and hotter climatic conditions.[13]

The Organophosphorus Compounds stored in the body fat, kidneys, liver, and salivary glands after absorption. [14]

Since phosphorothioates [P=S] like parathion and bromophos are more lipid soluble than phosphates [P=O] like dichlorvos, they are more likely to be

stored in body fat, which contributes to long-term intoxication and might lead to relapse after a person has undergone clinical recovery.

Because they are often lipophilic in nature, organophosphorus chemicals can pass the blood-brain barrier.[15] The liver's cytochromeP450-mediated mono-oxygenases detoxify these substances.[16]

The cholinesterase enzyme is inhibited by organophosphates. They are phosphoric acid derivatives or carbamic acid esters. They firmly phosphorylate the cholinesterase enzyme, or occasionally irreversibly. As a result, cholinesterase activity will be suppressed, particularly acetyl cholinesterase in synapses and on the membranes of red blood cells as well as butyryl cholinesterase in plasma. It can take from 60 minutes to many weeks for the phosphorus-Cholinesterase link to be broken.[17] The human body contains both a particular Acetylcholinesterase (true cholinesterase) and a non-specific Butyrylcholinesterase (pseudocholinesterase).

The plasma half-life of these drugs varies significantly, from a few minutes to a few hours, depending on the route of administration and the kind of molecule. In the liver, oxidation is the main mode of metabolism.

Due to subcutaneous lipid storage followed by later chronic systemic release following redistribution, "highly fat soluble drugs" like chlorfenthion may cause cholinergic overactivity over a prolonged duration of days to weeks. After what appears to be good management, these chemicals can potentially result in recurring release.

Nearly 90% of OPC and its metabolites are removed by faeces and urine, both of which happen within 48 hours. While some substances may stay in the body for longer periods than others, very few substances are eliminated unchanged in urine and faeces.

MECHANISM OF ACTION

Organophosphates typically produce significant acute toxic effects. The sympathetic and parasympathetic ganglia, neuromuscular junctions, sympathetic fibres to sweat glands, post ganglionic parasympathetic terminal junctions, and nerve terminals of the central nervous system all contain acetyl choline as a neurotransmitter. ACh is released into synapses or neuromuscular junctions after depolarization. This ACh binds to post synaptic receptors, activating the receptors and causing an action potential to propagate.

OPCs are inhibitors of the carboxylic ester hydrolase, which includes chymotrypsin, non-specific proteases, hepatic and plasma carboxyl esterases, BuChE, AChE, and BuChE. The majority of the clinical symptoms of both carbamate and OP poisoning are caused by AChE inhibition. We don't fully understand the clinical implications of other enzymes being inhibited.

The main target of organophosphates is the B-esterase AChE. The physiological function of AChE is to hydrolyze the neurotransmitter acetyl choline,[18] which is released for the propagation of action potentials in the peripheral and central nervous systems, to acetic acid and choline. There are esteratic and aromatic anion sites in the AChE active site. They are solely

connected to the serine moiety of the esteratic site by organophosphate. Presynaptic nerve terminals absorb the choline that AChE produces by hydrolyzing acetyl choline. As a result, ACh builds up at the cholinergic synaptic connections when organophosphate inhibits acetylcholinesterase. Most organs include cholinergic receptors of the nicotinic and muscarinic types, and this overstimulates them.

The esteratic site of the enzyme, which is also the active site, is where organophosphates with a P=O moiety phosphorylate hydroxyl groups. Its ability to affect its physiological substrate is hindered by this. Initially, the Michelis complex forms, which then triggers the phosphorylation of AChE. Both reactions happen extremely quickly, which shows that the enzyme is apt to each OPC. The link between acetyl choline carbonyl atoms at the same site of the enzyme is less stable than the bond between the esteratic site of the AChE and the phosphorous atom.

In contrast to the enzyme-carbon link, which can break in a matter of microseconds, the enzyme-phosphorus connection can break in a matter of hours to days depending on the chemical makeup of the OPs.The velocity of this spontaneous reaction depends on the chemical make-up of the "R" substitute and the hydrolysis of phosphorylated AChE by water, which is a very slow process. However, a class of hydroxylamine derivatives known as oximes is used to treat OP poisoning because it can help AChE dephosphorylation. If the enzyme-inhibitor complex is already aged, the phosphorylated AChE's reactivation might not take place. Ageing [19,20,21] is the loss of one of the "R" groups through non-

enzymatic hydrolysis, whose pace depends on the chemical composition of the alkyl group and typically takes place over a period of 24 to 48 hours. Only newly created enzymes, which could take several days, can replace an aged AChE.

PHYSIOLOGY OF CHOLINERGIC TRANSMISSION:

ACETYLCHOLINE

In all preganglionic autonomic fibres, postganglionic parasympathetic fibres, neuromuscular junction, and several interneuron synapses in the CNS, acetylcholine is the predominate neurotransmitter.

Two classes of Ach receptors

1) Muscarinic

2) Nicotinic

MUSCARINIC RECEPTORS:

These are typically found on autonomic effector cells in the CNS, heart, blood vessels, eye, smooth muscles, sweat glands, and glands of the gastrointestinal, respiratory, and urinary tracts.

Five subtypes of muscarinic receptors.[22]

- **M1** – Autonomic ganglia, gastric glands and CNS
- **M2** – Heart
- **M3** – Visceral smooth muscles, Exocrine glands & Vascular endothelium
- **M4** – CNS
- **M5** – CNS

NICOTINIC RECEPTORS:

They are mostly found in the autonomic ganglia and neuromuscular junction. Based on where they are found, there are two subtypes.

- **$N_{(M)}$ -** Neuromuscular junction
- **$N_{(N)}$** – autonomic ganglia and CNS

ACETYLCHOLINESTERASE(AChE) :

The muscle's basement membrane, the membranes of the motor end plates, and the membranes at the nerve terminals all contain the protein acetyl cholinesterase. The enzyme's individual molecules can bind and break down several acetylcholine molecules.

Three types:

1. **Brain acetylcholinesterse** : seen as tetramer (G4 form) and monomer (G1 form).
2. **RBC acetylcholinesterase**: specific or true acetylcholinesterase found in Red cell, nervous tissue, skeletal muscle.
3. **Plasma acetylcholinesterase**: butryl or Pseudo cholinesterase: found in Plasma, liver, heart, pancreas, brain.

Cholinesterases hydrolyze Ach into acetate and choline to inhibit its effects on the body. The body has two cholinesterases: butryl cholinesterase and acetyl cholinesterase, also known as (pseudo cholinesterase).

Ach activity in the body is inhibited by acetyl cholinesterase, which hydrolyzes acetyl choline. All cholinergic sites, RBCs, and grey matter exhibit it.

Ach is hydrolyzed slowly by "butryl cholinesterase," which also breaks down ingested esters. This can be observed in the liver, gut, white matter, and plasma. These are "more sensitive" to inhibition by organophosphosphates, and after inhibition with these substances, their levels significantly decrease.

ANTICHOLINESTERASES

Anticholinesterases inhibit cholinesterases, thereby protect acetyl choline from hydrolysis. That leads to potentiation of cholinergic effects.

They can be classified into.

1) **Reversible** -- Physostigmine, Edrophonium, rivastigmine, donepezil,Neostigmine,Pyridostigmine,

2) **Irreversible** – organophosphates and few carbamates Anticholinesterases bind and inhibit number of enzymes, but more clinical importance is their action on the esterase.

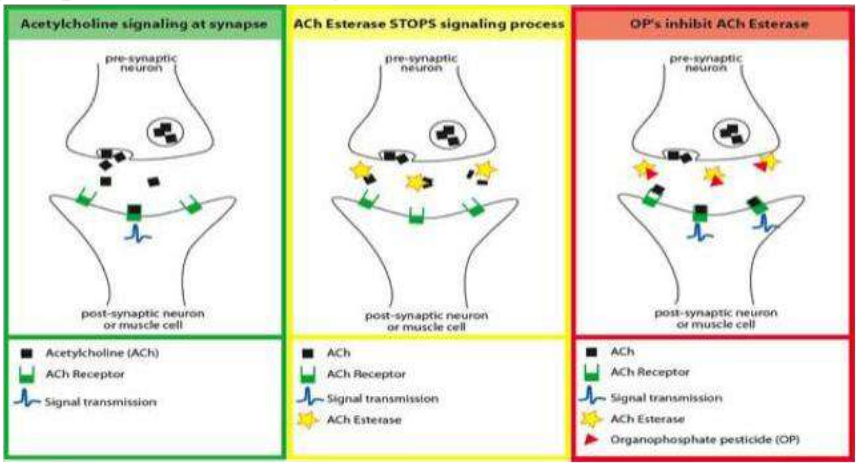
1) INHIBITION OF ACETYLCHOLINESTERASES (AchE):

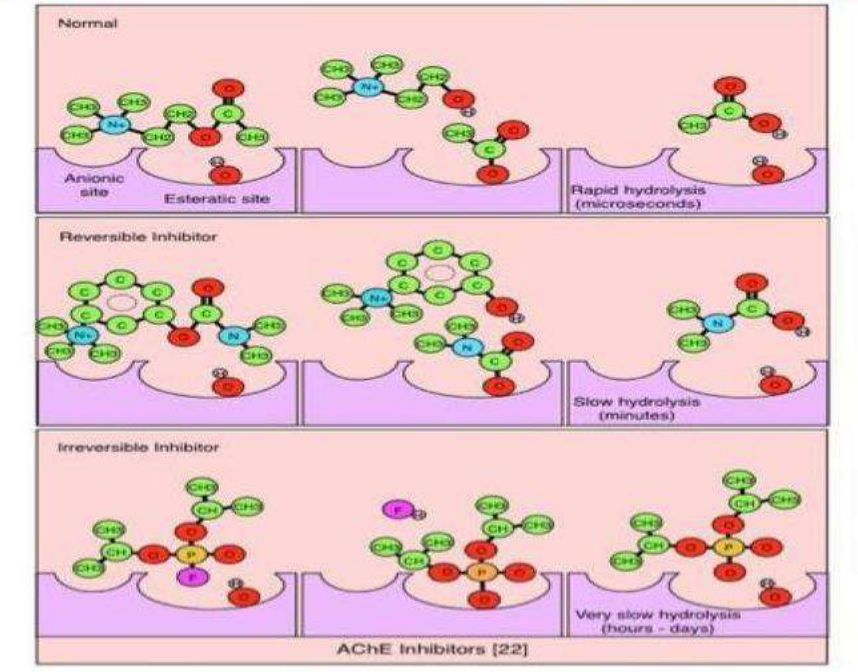
Anionic and esteric sites are present in anti-acetyl cholinesterases. Ach quickly hydrolyzes after binding to the anionic site on acetyl-cholinesterases. The substrate cannot be attached because reversible anti-acetyl cholinesterases mix with acetylcholinesterases at the same anionic site. The OPC are extremely lipid soluble organic derivatives of phosphorus-containing acids. In doing so, they join with "esteric sites of acetylcholinesterase" and phosphorylate it, causing it to overstimulate at first and thereafter block synaptic conduction.

The enzyme will become inactive and unable to continue hydrolyzing Ach after being phosphorylated. Finally, this leads to an endogenous buildup of Ach at the sites of cholinergic transmission, which in turn culminates in unchecked cholinergic overactivity.

This binding cannot be undone, unless early pharmaceutical intervention is made. "Many parameters that affect toxicity differ, including the rate of inactivation (phosphorylation) and reactivation (dephosphorylation)." A few of them include the chemical makeup of the substance, the species' capacity for metabolism, and the extent of tissue distribution. Denovo synthesis of new enzyme, which happens very slowly at a rate of 1% per day, and spontaneous dephosphorylation, which may take even up to 1000 hours, are the two methods for reactivating dormant cholinesterases.

Organophosphate attacks Acetylcholinesterase:





The inactive phosphorylated enzyme doesn't react with water at all or only very slowly. Reactivation happens a million times more quickly if more reactive OH groups, such as oximes, are utilised. The inhibited enzyme "ages," or decreases in responsiveness to reactivating agents with time. One of the "R" groups in the enzyme's active site may separate non-enzymatically, leaving a monoalkyl or monoalkoxyl phosphoryl group behind.

The negatively charged phosphate group of the enzyme cannot be attacked by a nucleophile like hydroxyl or oxime any longer, hence once "ageing" of the enzyme takes place, the inactivation "cannot be reversed." As a result, the enzyme is permanently blocked. Aging happens quickly with chemical warfare chemicals like soman.

2) INHIBITION OF NEUROPATHY TARGET ESTERASES (NTE):

Neuropathy target esterase Inhibition is responsible for the organophosphate induced delayed neuropathy (OPIDN) after its transformation to an aged form.

CLINICAL FEATURES

- **Acute toxicity**
- **Intermediate syndrome**
- **Chronic toxicity**

The rate of AchE inhibition of the organophosphorus agents, the mode of absorption, the enzymatic conversion into the active metabolite, and the lipophilicity of the organophosphorus agent all influence the onset and duration of AChE inhibition.

Oral & respiratory route : clinical features occurs within 3hrs

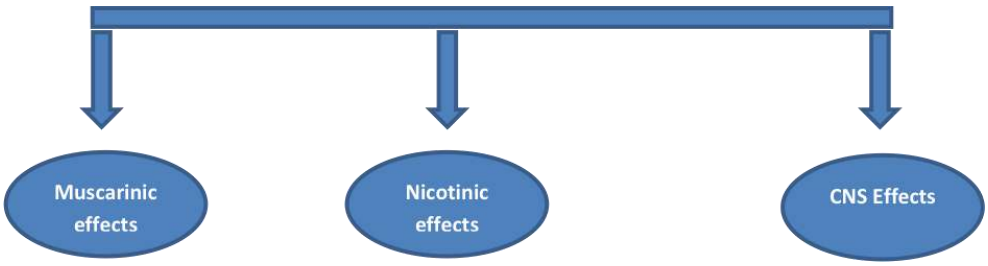
Dermal absorption: can be delayed upto 12 hours

ACUTE TOXICITY

The Symptoms occurs as early as 5 min and even within 15mins of ingestion death can also occurs. Patients will be symptomatic around 8-24 hours mostly .Onset of symptoms mainly depends on the agent, the route of poisoning ,and the degree of exposure.The drugs that require metabolic activation, such as malation or extremely lipid-soluble agents like fenthion and chlorfenthion, can take the longest to show clinical characteristics.

SIGNS AND SYMPTOMS

Mainly based on these receptors activation



MUSCARINIC EFFECTS:

SLUDGE SYMPTOMS

- salivation
- lacrimation
- urination
- defecation
- Gastrointestinal upset
- Emesis

"**DUMBBELS**"

- defecation
- urination
- miosis
- bronchorrhea
- bronchospasm
- emesis
- lacrimation
- salivation.

Miosis is the muscarinic finding that is most frequently observed among the ones mentioned above. The most significant symptom of muscarinic toxicity is bronchorrhea, which frequently looks like pulmonary edema.

Cardiovascular system:	Hypotension,Bradycardia [23]
Respiratory system:	Rhinorrhea, bronchospasm, Bronchorrhea, cough, severe respiratory distress
Gastrointestinal :	Hypersalivation, vomiting, Abdominal pain, diarrhoea
Genitourinary :	Incontinence
Ocular :	Miosis, blurring of vision
Glands :	increased sweating & lacrimation

NICOTINIC EFFECTS:

Nicotine selectively activates the nicotinic receptors, and either tubocurarine or hexamethonium blocks this activity. The pentameric structure of these receptors resembles a rosette.

N_M and N_N are the two subtypes of nicotinic receptors (Which is previously known as N_1 and N_2)

Fasciculations of the muscles [23]

cramps

generalised weakness

flaccid paralysis (of the skeletal muscles)

tachycardia and transient hypotension.

Other symptoms include:

vertigo

drowsiness

Lethargy

mental confusion

headaches

respiratory centre depression

convulsions, and coma

INTERMEDIATE SYNDROME:

It is defined as Delayed onset of muscle weakness without fasciculations or cholinergic characteristics, which might appear 24-96 hours following acute organic phosphorus compound poisoning,[25] This type of paralysis, also known as type II paralysis [26], typically lasts 4 to 18 days.

The majority of individuals with intermediate syndrome first exhibit characteristic cholinergic signs and symptoms, which improve over the course of one to two days with atropine and other treatments.

Due to persistent cholinesterase inhibition and necrosis of the muscles, it develops one to four days after poisoning. Intermediate syndrome is more frequently associated with the drugs chlorpyrifos, dimethoate, monocrotophos, parathion, sumithion, fenthion, ethyl parathion, methyl parathion, diazinion, and malathion. Acute respiratory paresis, neck flexor weakness, and motor cranial nerve palsies are the main symptoms of muscular weakness and paralysis. The inability to elevate the neck or sit up, ophthalmoparesis, delayed eye movements, facial weakness, swallowing difficulty , limb weakness (mainly proximal), areflexia, respiratory paralysis, and death are some examples of paralytic symptoms.

MECHANISMS

The release of organophosphates from adipose tissue, which act on nicotinic receptors, may be the cause of late paralysis. An AChR malfunction caused by prolonged depolarization. At the neuromuscular junction, there is a

downregulation of nicotinic AChR, or a reduction in the density of active AChRs.

Myopathy induced by OPC is contentious. It was once hypothesised as the cause of IMS, but it is now suggested that it is caused by cholinergic overactivity and shares the same origin as IMS. In rat experiments, oximes acts as a cholinesterase reactivator and prevents muscle necrosis right after poisoning.

CHRONIC TOXICITY:

- **Chronic organophosphate induced neuropsychiatric disorder (COPIND)**
- **OPC Induced delayed polyneuropathy(OPIND)**
- **Parkinsonism**

CHRONIC ORGANOPHOSPHATE INDUCED NEUROPSYCHIATRIC DISORDER (COPIND): [27]

After extended exposure to OP chemicals, a syndrome known as chronic organophosphate (OP)-induced neuropsychiatric illness occurs

Clinical characteristics

- Confusion [28]
- Lethargy
- Anxiety [29]
- emotionally unstable
- Depression
- Irritability

- Schizophrenia
- Choreoathetosis
- EEG Changes
- Cogwheel rigidity
- Dystonic reactions

OPC INDUCED DELAYED POLYNEUROPATHY(OPIND) [30]

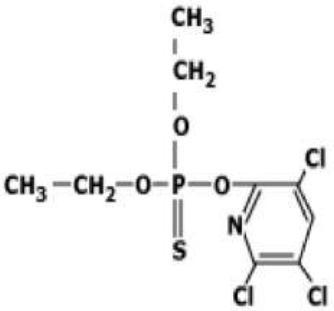
OPIND is caused by the enzyme neuropathy target esterase (NTE) being inhibited.

Clinical features: [31,32,33,34]

- Demyelination of long nerves:–motor dysfunction,distal limb weakness,muscle cramps
- Sensory dysfunction:paresthesias that may be chronic or recurrent.

Agents associated with organophosphorous induced delayed neuropathy

Chlorpyrifos
Leptophos
Malathion
Merphos
Mipafox
Trichlorfon
Triorthocresylphosphate (TOCP)



Chlorpyrifos

PARKINSONISM:

As a result of long term toxicity caused by OPC chemicals that resemble MPTP (Methyl phenyl tetra hydropyridine), Parkinsonism can develop.

DIFFERENCE BETWEEN INTERMEDIATE SYNDROME AND DELAYED POLYNEUROPATHY

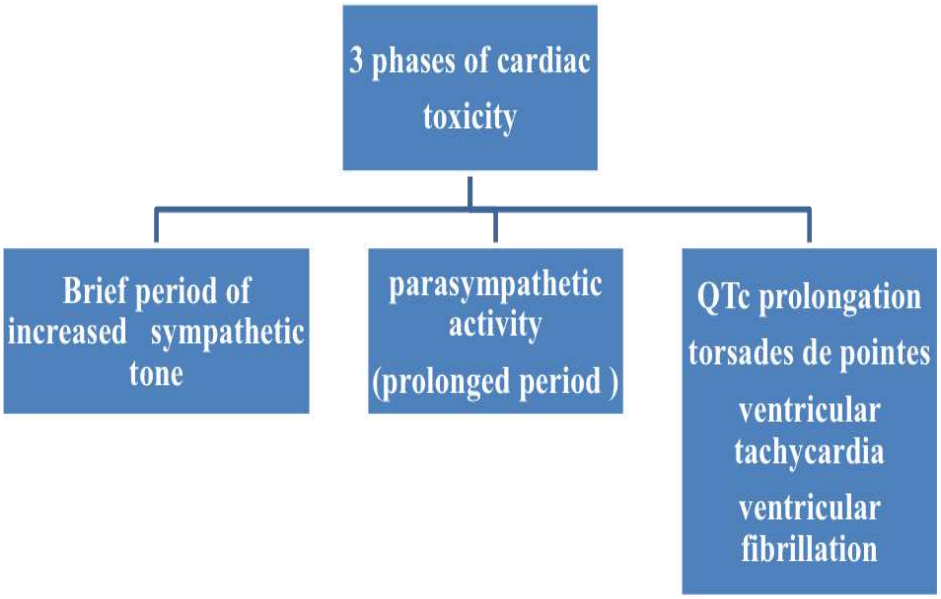
	Intermediate syndrome	Delayed Polyneuropathy
Latent period	1-4 days	2-3 weeks
Site of weakness	Proximal	Distal
Limb muscle	Involved	Not involved
Neck muscle	Involved	Not involved
Cranial Nerve	Yes	No
Respiratory muscle	Involved	Not involved
Electromyogram	Tetanic fade	Denervation

Additional effects

- Patients with severe OPC poisoning experienced acute kidney injury [35] that required renal replacement treatment.
- Acute pancreatitis is caused by ductular hypertension and high cholinergic activation.
- Hepatitis

CARDIOVASCULAR DISORDER:

Ludomisky et al described cardiac toxicity in OPs poisoning [36]



DIAGNOSIS OF ORGANOPHOSPHORUS POISONING:

- History of poisoning
- The smell of poison
- Muscarinic,nicotinic,CNS features [37,38]
- Improvement in symptoms and signs after using pralidoxime and atropine.
- Lab tests : reduced levels of plasma pseudocholinesterase (PChE) and RBC acetylcholinesterase (AChE) [39]

PChE	AChE
Sensitive indicator	Specific indicator
Decreased in other conditions also eg:genetic deficiency, Medical illness	OPC poisoning level will be reduced
Recovery within weeks after exposure	Recovery can take for several months

OPC poisoning is diagnosed clinically, and as OPC poisoning is linked to a high mortality rate, it is crucial to make the diagnosis as soon as the patient is admitted so that treatment can start.

Based on symptoms, indicators, and cholinesterase levels, patients are classified as having mild, moderate, or severe poisoning for management purposes in order to reduce complications and enhance prognosis.

Identification of the compound at the time of admission is crucial for identifying patients who are at risk of respiratory failure.

For instance, monocrotophos poisoning causes respiratory paralysis to appear within a short period of time after intake. [40]

Miosis and muscle fasciculations are the poisoning symptoms that are most helpful in making a diagnosis.

SEVERITY ASSESSMENT

(BASED ON SYMPTOMS):

MILD	MODERATE	SEVERE
Patient walks &talks	cannot walk	Patient unconscious
Headache, vomiting, abdominal pain sweating, salivation, rhinorrhoea	Fasciculations Restlessness Miosis Soft voice	No pupillary reflex Flaccid paralysis Fasciculations Increased bronchial Secretions Crepitations/ Wheeze Convulsions Respiratory failure
AChE :1.6 to 4u/l	0.8-2 u/l	<0.8 u/l

PERADENIYA ORGANOPHOSPHORUS POISONING (POP) SCALE

The Peradeniya Organophosphorous Poisoning (POP) Scale is a scoring system developed in 1993 by N. Senanayake, H. J. de Silva, and L. Karalliedde [41]. It is based on the common clinical characteristics of organophosphorus poisoning, which are evaluated on a three-point scale ranging from 0 to 2

- **0-3 score - mild poisoning**
- **4 to 7 - moderate poisoning**
- **8 to11 - severe poisoning**

Pupil Size	>2mm	0
	<2mm	1
	Pinpoint	2
Respiratory rate	<20	0
	>20	1
	20 with central cyanosis	2
Heart rate	>60	0
	41-60	1
	<40	2
Fasiculation	None	0
	Present generalized or continuous	1
	Present Generalized and continuous	2
Level of consciousness	Conscious and rationale	0
	Impaired response to verbal commands	1
	No response to verbal commands	2
Seizure	Absent	0
	present	1

POISONING SEVERITY SCORE

The PSS is a classification scheme for cases of poisoning in adults and children. This scheme should be used for the classification of acute poisonings regardless of the type and number of agents involved.

Table 2. Poisoning severity score (11)

	None	Minor	Moderate	Severe	Fatal
Organ	0	1	2	3	4
	No symptoms or signs	Mild transient, and spontaneously resolving symptoms	Pronounced or prolonged signs or symptoms	Severe or life-threatening	Death
Cardiovascular system		Isolated extrasystoles	Bradycardia (HR 40-50 in adults) Tachycardia (HR 140-180 in adults) Chest pain Conductance, disturbance Hypertension Hypotension	Bradycardia (HR < 40 for adults) Tachycardia (HR > 180 for adults) Cardiac arrest	
Respiratory system		Irritation, coughing, breathlessness, mild dyspnea, mild bronchospasm	Prolonged coughing, bronchospasm, dyspnea, stridor, hypoxemia requiring extra oxygen	Manifest respiratory insufficiency airway obstruction, pulmonary edema, ARDS, pneumonitis	
		Chest X-ray: abnormal with minor or no symptoms	Chest X-ray: abnormal with moderate symptoms	Chest X-ray: abnormal with severe symptoms	
Nervous system		Vertigo, tinnitus, ataxia	Unconsciousness with appropriate response to pain	Deep coma unresponsive to pain	
		Mild extrapyramidal symptoms Paresthesia	Confusion, agitation, hallucinations,delirium Infrequent, generalized, or local seizures	Extreme agitation Generalized seizures, status epilepticus	
GI tract		Vomiting, diarrhea, pain	Pronounced or prolonged vomiting, diarrhea, pain ileus	Massive hemorrhage, perforation	
			Dysphagia	Severe dysphagia	
Metabolic imbalance		Mild acid-base disturbances Mild electrolyte and fluid disturbances	More pronounced acid-base disturbances More pronounced electrolyte and fluid disturbances	Severe acid-base disturbances Severe electrolyte and fluid disturbances	
		Mild hypoglycemia	More pronounced hypoglycemia	Severe hypoglycemia	
Liver		Minimal rise in serum enzymes	Rise in serum enzymes no diagnostic biochemical or clinical evidence of liver dysfunction	Rise in serum enzymes biochemical or clinical evidence of liver dysfunction	
Kidney		Minimal proteinuria/hematuria	Massive proteinuria/hematuria Renal dysfunction	Renal failure.	
Muscular system		Mild pain, tenderness	Pain, rigidity, cramping, fasciculations	Intense pain, extreme rigidity, extensive cramping,fasciculations	
			Rhabdomyolysis	Rhabdomyolysis with complications	
Local effects on skin		Irritation, 1st degree burns	2nd degree burns in 10%–50% of body surface or 3rd degree burns in <2% of body	2nd degree burns in >50% of body surface or 3rd degree burns	
Local effects on eye		Irritation, redness, lacrimation, mild palpebral edema	Intense irritation, corneal abrasion Minor (punctate) corneal ulcers	Corneal ulcers (other than punctate), perforation	

ARDS, Acute Respiratory Distress Syndrome.

Severity Grades [42]

- **NONE (0):** No symptoms or signs related to poisoning
- **MINOR (1):** Mild, transient and spontaneously resolving symptoms
- **MODERATE (2):** Pronounced or prolonged symptoms
- **SEVERE (3):**Severe or life-threatening symptoms
- **FATAL (4):** Death

MANAGEMENT

UNKNOWN POISONING/SUSPECTED OPC POISONING:

Uncertain whether the patient ingested OPC toxicity then ATROPINE TEST can be done by 0.6-1 mg of IV atropine are administered results in pulse rate increasing to 25 beats per minute and skin flushing if Patient has minimal or no toxicity.

INITIAL MANAGEMENT [43]

Poisoning by organophosphorus is a medical emergency

If patients have respiratory distress, tracheal intubation if needed should be performed after performing early resuscitation with oxygen supply.

The patient needed artificial ventilation due to their massive poison consumption, increased secretions, depressed mental state, and hypoventilation.

IV line should be fastened

Inotrope support should be administered if the patient has hypotension in addition to fluid resuscitation using crystalloids.

Urine catheter should be introduced before the patient has urinary retention from the antidote.

DECONTAMINATION

- The patient should thoroughly wash their skin and hair with soap and water because their clothes and hair would have been contaminated by pesticides.
- Remove any contaminated clothing(Poison may be absorbed through the skin)
- Use either water or saline to irrigate the exposed eyes.
- To prevent unintentional poisoning exposure, the healthcare worker should wear an apron, gloves, and a mask.

GASTRIC LAVAGE:

Ryles tube should be placed for stomach lavage once the patient has been stabilised if the patient arrives within 1–2 hours of consuming the poison.

ACTIVATED CHARCOAL

Activated charcoal is effective in preventing the further absorption of poison in the GIT.

The dose is 1g/kg (maximum dose 50g) and should be given after stomach wash if patients present within 2 hours.

ANTIDOTE THERAPY:

1. ATROPINE:

Atropine suppresses cholinergic activation by competing with acetylcholine at muscarinic receptors.[44]

Initially dose of 1.2 mg -3mg iv bolus is given.

The **indication** for atropine are

a. miosis,

b. excessive sweating

c. bradycardia

d. hypotension

e. reduced air entry due to bronchorrhoea & bronchospasm

Check the aforementioned 5 markers for improvement 5 minutes after atropine bolus.

If there is no improvement, atropine dosage should be doubled and should be done so repeatedly until an adequate response is seen.

The **target end point** are

a. Clear chest on auscultation

b. Heart rate>80/min

c. Axilla –dry

d. Systolic BP- >90mmHg

e. Pupils no longer pinpoint

After achieving this goal, make plans for an atropine infusion. Atropine was infused at a rate of 10–20% of the whole initial dose every hour for maintenance.

The patient is observed by keeping an atropine chart, documenting the patient's temperature, pulse rate, blood pressure, secretions, and pupils first every hour and later every four hours.

Most of the time, atropine dosage should be weaned off over the course of two to five days after poisoning.

Monitor for **atropine toxicity** like

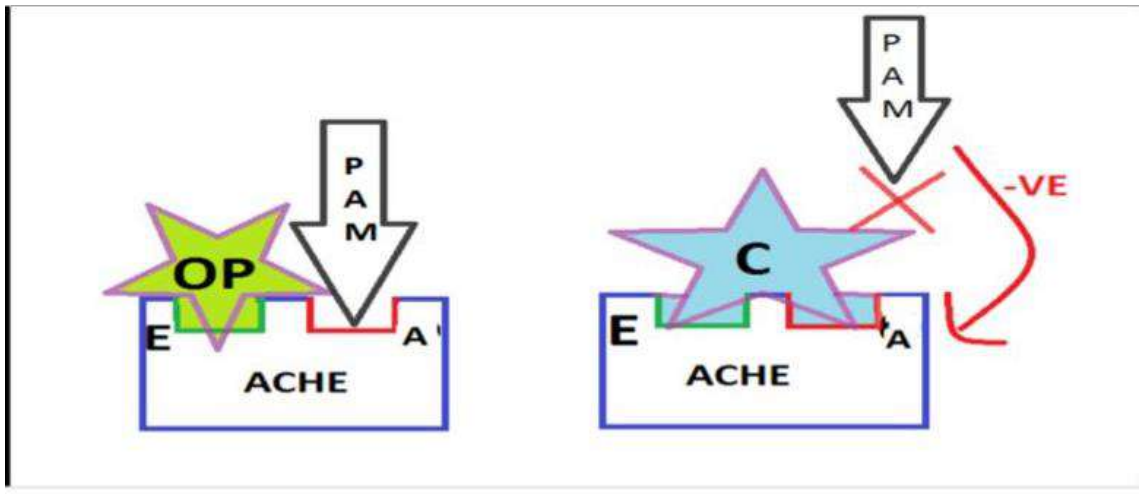
- Absent bowel sounds
- Fever
- Confusion
- Sometimes life threatening arrhythmias and Rhabdomyolysis.

Stop the atropine infusion for 60 minutes if patients show signs of atropine toxicity, then continue at a lesser dose when the symptoms reverse.

2.PRALIDOXIME

Pralidoxime (2-PAM) and other medications in its class, such as obidoxime, reactivate cholinesterase.[45,46]

MECHANISM OF ACTION:



These medications work by blocking the muscarinic and nicotinic effects of OPC toxins.

Organophosphates impede the action of the acetylcholinesterse enzyme by attaching to its hydroxyl component (esteric site).

The pralidoxime displaces the phosphate by attaching to the opposite site (unblocked, anionic site).

Patients with carbamate toxicity are not administered the oximes. The only condition for which this medication is utilised is organophosphate poisoning.

DOSE: ACCORDING TO WHO [47]

Bolus dose : **30 mg/kg 2PAM over 30 min**

Maintenance dose :**continuous infusion of 8mg/kg/hour**

The duration of treatment is till atropine is required (2-5days)

Pralidoxime use longer the 24 hours is indicated only in case of unaged OPCs that are redistributed from fat.

Due to the slower rate of ageing of diethyl compounds than that of dimethyl groups, oximes are more beneficial in these compounds.

OBIDOXIME [48]

loading dose 250mg

Followed by Infusion at 0.5mg/kg/hour

GLYCOPYRROLATE

It decreases secretions in opc poisoning with fewer neurological side effects such as delirium and agitation.

It may also be utilised in conjunction with atropine when a patient exhibits excessive secretions, undue tachycardia, or neurological signs of over-atropinization.

DOSE: The initial infusion dose of 7.5 mg of glycopyrrolate in 200ml of saline is started, and the dose is then increased to achieve the desired effects of a dry mucous membrane. [48]

If more is needed, it can also be administered 0.2 mg IM stat, followed by a repeat dosage every six hours.

DIPHENHYDRAMINE:

It is Centrally acting anticholinergic drug

If atropine is not available- can be used.

BENZODIAZEPINES [49]

Patients who have consumed organophosphorus poisoning typically experience agitated delirium. The cause is multifaceted and includes the pesticide itself, atropine toxicity, hypoxia, alcohol consumed along with the poison, and medical issues. Some patients require medication even when prevention or treatment of underlying causes constitutes the cornerstone of treatments. Diazepam therapy is beneficial for people who are agitated suddenly. Diazepam is the first-line treatment for seizures, but they are rare in well-oxygenated patients who have pesticide poisoning. Organophosphorus nerve

agents seem to cause seizures more frequently (such as soman and tabun). Diazepam may lessen neurological damage and shield against respiratory failure and mortality, according to animal studies, but there aren't many human trials. **VARIOUS OTHER TREATMENTS**

Only a few mechanisms are used by current treatments. Numerous novel medicines have been investigated, however the findings were conflicting. Future research may, however, turn out multiple accessible medicines operating at various locations that could supplement current therapy.

Magnesium sulphate inhibits ligand-gated calcium channels, reducing acetylcholine release from pre-synaptic terminals and enhancing the function of neuromuscular junctions by reducing NMDA receptor activation-mediated CNS overstimulation [50]. Magnesium sulphate was found to reduce mortality in a trial including people who had been exposed to organophosphorus insecticides. However, due to the small sample size, non-randomized assignment (every fourth patient received the intervention), and the publication's limited description of the technique and dosage of the magnesium sulphate utilised, these findings should be regarded with caution.

Clonidine, an alpha2-adrenergic receptor agonist, also lowers acetylcholine release and synthesis from presynaptic terminals.[51] Clonidine medication is beneficial for animals, particularly when combined with atropine, but effects on humans are unknown.

In Brazil and Iran, **sodium bicarbonate** [52,53] is sometimes used instead of oximes to treat organophosphorus poisoning. However, a Cochrane review found that there is currently insufficient evidence to determine whether sodium bicarbonate should be used in humans who have been poisoned with organophosphorus. Blood pH increases (up to 745–755) have been reported to improve outcomes in dogs through an unknown mechanism.

Sodium bicarbonate intravenous infusion was more helpful in nerve agent overdose.[53]

Fresh frozen plasma has been used in bioscavenger therapy under the theory that the BuChE enzyme may sequester free toxins in blood, removing them from circulation.[54] However, there is not enough evidence at this time to confidently endorse this bioscavenger therapy.

Early implementation of enteral feeding improves outcomes by preventing enterohepatic circulation in the critically unwell. Patients with OPC may need prolonged ventilation as result of IMS development, and these patients benefit most from early enteral feeding.

Organophosphorus removal from the blood may enhance the effectiveness of other treatments.

However, a recent non-randomised controlled trial in China revealed a benefit of **hemofiltration** following poisoning with dichlorvos, which has poor solubility in fat and hence should have a very small volume of distribution. The functions of haemodialysis and hemofiltration are still unclear. Organophosphorus poisoning therapies are now the subject of a systematic review, but randomised controlled trials are required to create effective, evidence-based therapy recommendations.

DRUGS FOR FUTURE:

These medications have been shown to be potent, highly selective, and reversible acetylcholinesterase inhibitors.[55,56]

- **Huperzine A**
- **ZT-1**

MECHANICAL VENTILATION:

Early diagnosis of respiratory failure leads to intubation and mechanical ventilation, a life-saving treatment for OP toxicity.

INDICATIONS

I.	Respiratory Gas Tensions	
	i	Direct Indices
		Arterial Oxygen Tension < 50 mm Hg on room air
		Arterial Co_2 Tension > 50 mm Hg in the absence of metabolic alkalosis
	ii	Derived Indices
		P a o_2/ Fio_2 < 250 mm of Hg
		PA-aOo_2 (Pulmonary arterial-alveolar O2 gradient) > 350 mm of Hg
		Vd/Vt> 0.6
II.	Clinical - Respiratory Rate (RR)> 35 breaths/min	
III.	Mechanical Indices	
	Tidal Volume 5 ml/kg	
	Vital capacity < 15 ml/kg	
	Maximum inspiratory force <- 25 cm of H_2O	

COMPLICATIONS

It can occur upto 43% of cases

Death can often occur within 24 hours in untreated cases or after variable periods of up to 10 days in those who reach a hospital and are in patients given optimal management.

Early deaths are mostly related to

1) CNS depression

2) Seizures

3) Ventricular arrhythmias

4) Respiratory failure due to

• Excessive bronchial secretions

• Bronchospasms

• Pulmonary oedema

• Paralysis of respiratory muscles or

• Apnoea associated with depression of the medullary respiratory centre

Late mortality is caused by

Respiratory failure – associated with Infection Pneumonia Septicaemia

Complications related to protracted period of mechanical ventilation and intensive care management.

Late unexpected ventricular arrhythmias, respiratory failure,sudden collapse and death may occur.

The most frequent consequence of acute OP poisoning is respiratory failure.

OTHER COMPLICATIONS :

Liver function abnormalities, blood dyscrasias, coagulopathy, pancreatitis and ulcerative stomatitis.

MORTALITY

Respiratory failure is the most frequent cause of death in acute organophosphorus poisoning. Cardiac arrest was reported as the cause of death in 10% of patients by Singh et al. According to Namba, mortality occurs within 24 hours in untreated cases and takes up to 10 days in treated instances.

The average time for full recovery is ten days. The poison used, the length of exposure, and the atropinization of all poisons all affect the mortality rate. The mortality rate in Indian studies ranged from 4 to 38%. The fastest hydrolization of the carboxyester group into compounds with negligible or no anticholinesterase action gives malathion the lowest toxicity. The highest fatality rate is in fenthion.

MATERIALS AND METHODS

STUDY DESIGN:

Prospective Observational Study

STUDY PERIOD:

6 Months (May to November)

STUDY AREA:

Madras Medical College and Rajiv Gandhi Government General Hospital

STUDY POPULATION:

142 patients diagnosed as organophophorus poisoning in Toxicology Intensive Medical Care Unit in Madras Medical College and Rajiv Gandhi Government General Hospital

SAMPLE SIZE:

142

ETHICAL CLEARANCE :

Obtained

CONSENT:

Informed consent obtained from all patients for clinical examination and for investigations. Patient confidentiality was maintained.

INCLUSION CRITERIA :

All patients >18 years of age admitted in Toxicology ward with known or suspected organophosphate poisoning, identified as

- History of consumption of an OPC insecticide
- Classical clinical features of OPC Poisoning– miosis, hypersalivation, fasciculations, characteristic odor of stomach wash.
- Patient giving consent to the study

EXCLUSION CRITERIA:

- Patients <18 years of age
- Patients not willing to participate in the study
- Patients who have consumed other poisons with the OPC
- Referral patients treated elsewhere

METHODOLOGY:

- The prospective observational study is proposed to be conducted after obtaining informed consent from the patients admitted in toxicology unit, IIM, MMC & RGGGH
- After satisfying the inclusion and exclusion Criteria patients will be included in the study.
- All patients will be evaluated with detailed history and thorough clinical examination at the time of admission
- POP score, PSS and GCS will be assessed at the time of presentation and grouped based on the severity.

- Routine and specific lab investigations like Serum cholinesterase,ECG,chest X ray, ABG will be done at the earliest.
- These scores are individually evaluated in predicting the ventilator requirement and mortality in OP compound poisoning and the scores are compared with each other, as to asses which score is better in predicting the severity of OP poisoning

STATISTICAL ANALYSIS

Descriptive analysis like frequency and percentage were calculated. Inferential statistics like chi square test was applied for categorical data. P value was set significant at 0.05.

OBSERVATION AND RESULTS

Table 1: DISTRIBUTION ACCORDING TO AGE (N=142)

Age	N	%
≤20	21	14.8%
21-30	52	36.6%
31-40	37	26.1%
41-50	20	14.1%
>50	12	8.5%
Total	142	100%

In our study, the total number of participants were 142.

In our study majority of the patients are in between the age group 21-30 years. (36.6%)

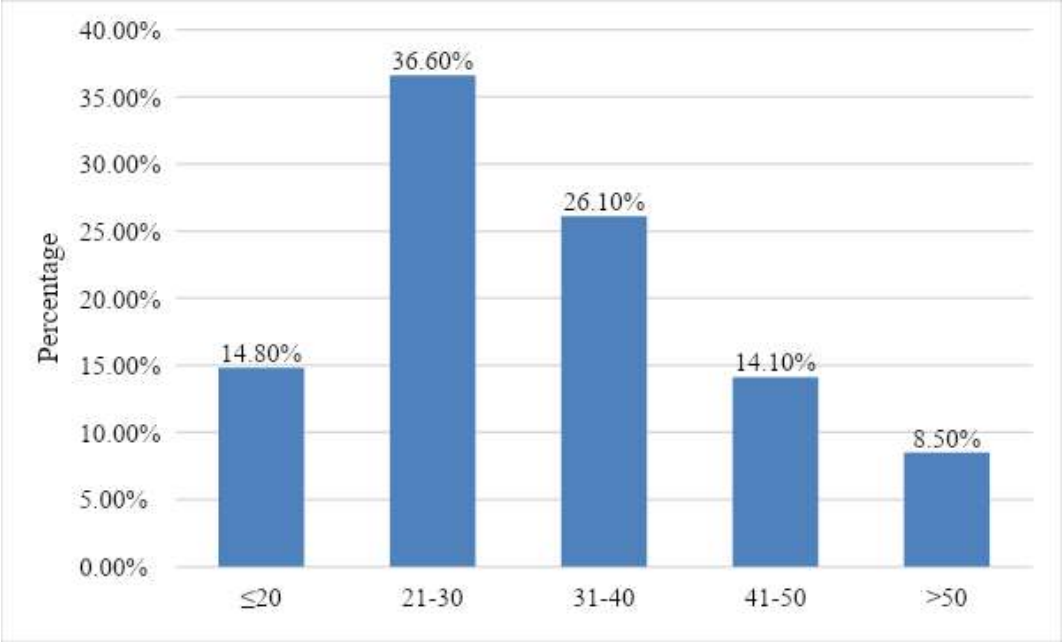


Table 2:DISTRIBUTION ACCORDING TO GENDER (N=142)

Gender	N	%
Female	62	43.7%
Male	80	56.3%
Total	142	100%

In our study out of total participants of 142 patients, 80 of them are males (56.3%), and 62 of them are females (43.7%)

In our study the majority of patients are males.

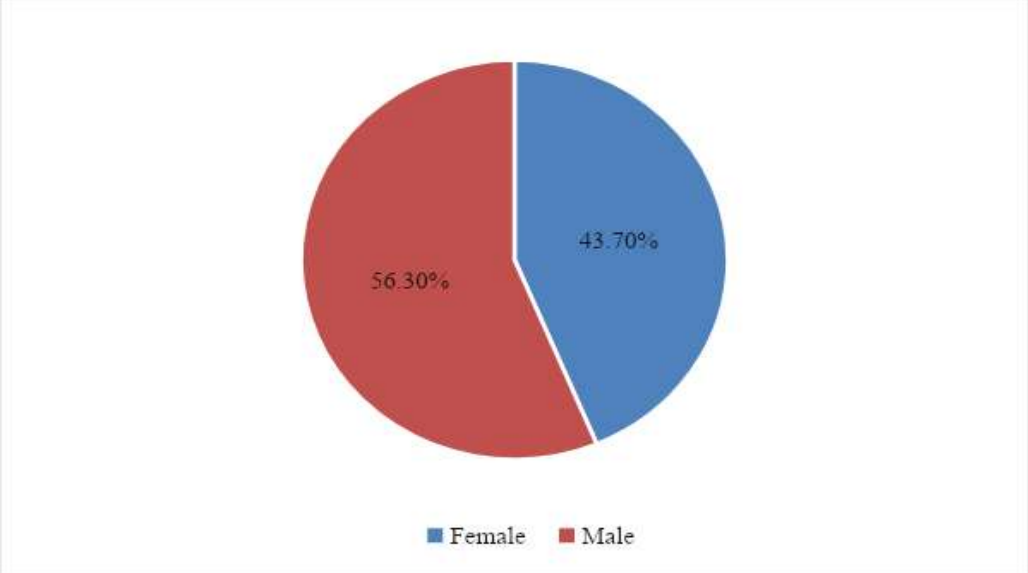
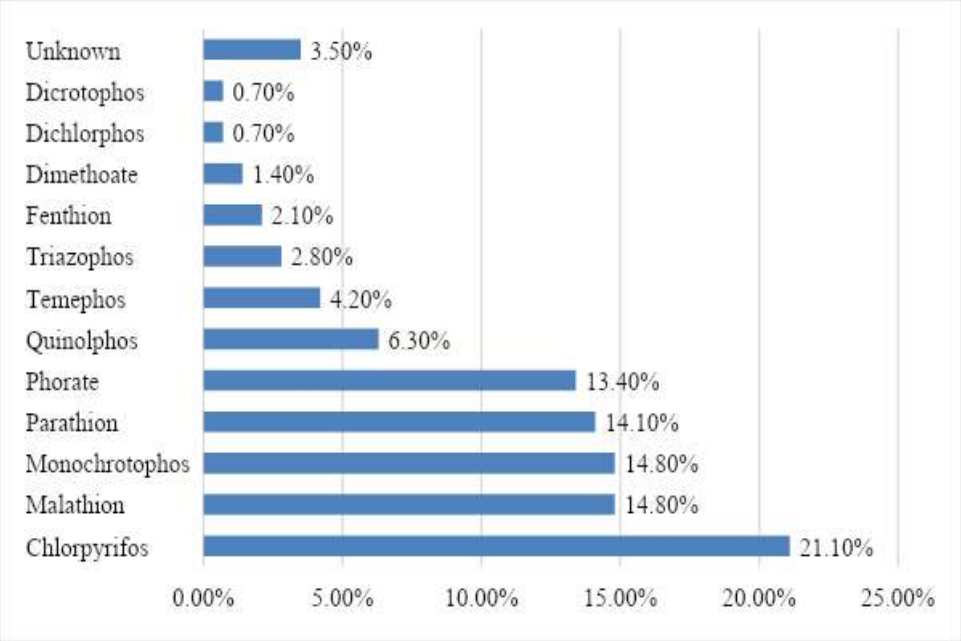


Table 3: DISTRIBUTION ACCORDING TO OPC COMPOUNDS (N=142)

OPC compounds	N	%
Chlorpyrifos	30	21.1%
Malathion	21	14.8%
Monochrotophos	21	14.8%
Parathion	20	14.1%
Phorate	19	13.4%
Quinolphos	9	6.3%
Temephos	6	4.2%
Triazophos	4	2.8%
Fenthion	3	2.1%
Dimethoate	2	1.4%
Dichlorphos	1	0.7%
Dicrotophos	1	0.7%
Unknown	5	3.5%
Total	142	100%



In our study the most common OPC compound is Chlorpyrifos (21.10%),(N=30), followed by Malathion and Monochrotophos (14.8%) ,(n=21)each.

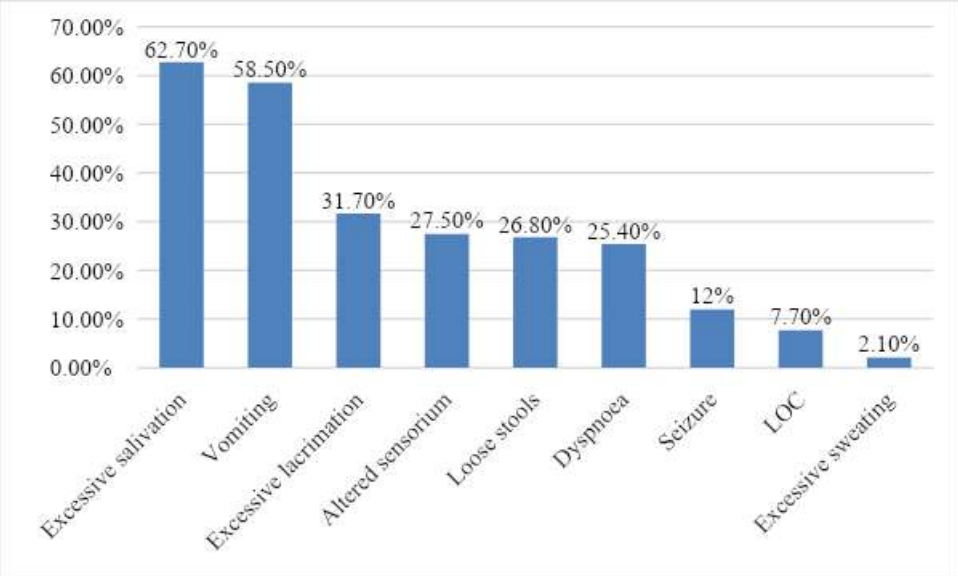
Table 4: DISTRIBUTION ACCORDING TO OCCUPATION (N=142)

Occupation	N	%
Farmer	64	45.1%
Student	52	36.6%
House wife	19	13.4%
IT staff	3	2.1%
Bank manager	1	0.7%
Driver	1	0.7%
Tailor	1	0.7%
Teacher	1	0.7%
Total	142	100%

In our study farmers consumed OPC poison more commonly (45.1%) (n=64),followed by students (36.6%) (n=52).

Table 5: DISTRIBUTION ACCORDING TO COMPLAINTS (N=142)

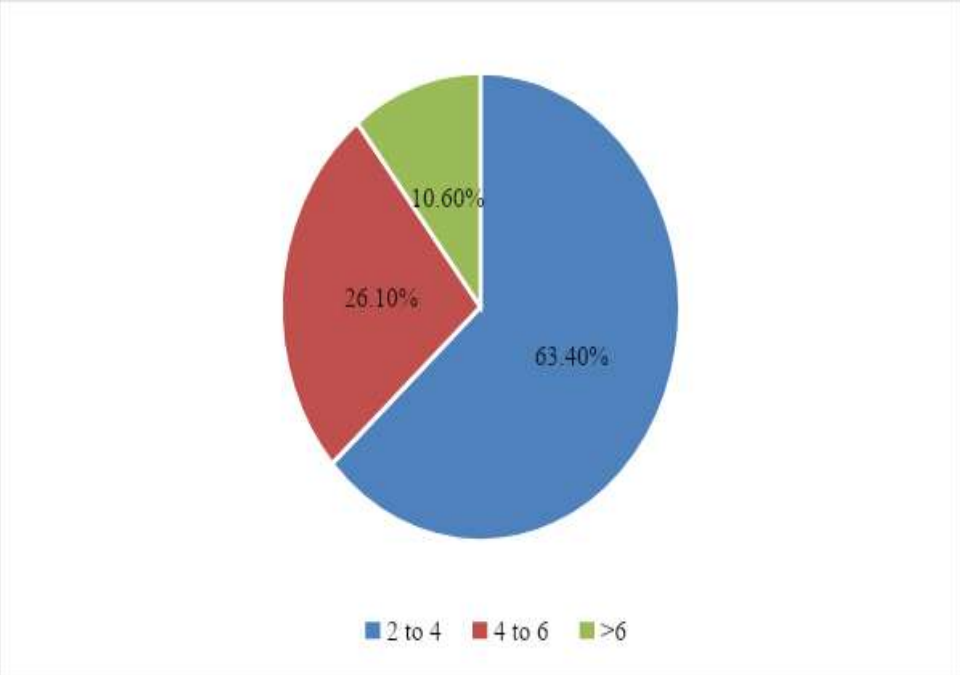
Complaints	N	%
Excessive salivation	89	62.7%
Vomiting	83	58.5%
Excessive lacrimation	45	31.7%
Altered sensorium	39	27.5%
Loose stools	38	26.8%
Dyspnoea	36	25.4%
Seizure	17	12%
LOC	11	7.7%
Excessive sweating	3	2.1%



In our study most common complaints are Excessive salivation (62.7%) (n=89),followed by vomiting(58.5%) (n=83),Excessive lacrimation (31.7%)(n=45),altered sensorium(27.5%)(n=39),loose stools(26.8%)(n=38).

Table 6: DISTRIBUTION ACCORDING TO LAG TIME FOR ADMISSION AFTER CONSUMING OPC POISON (N=142)

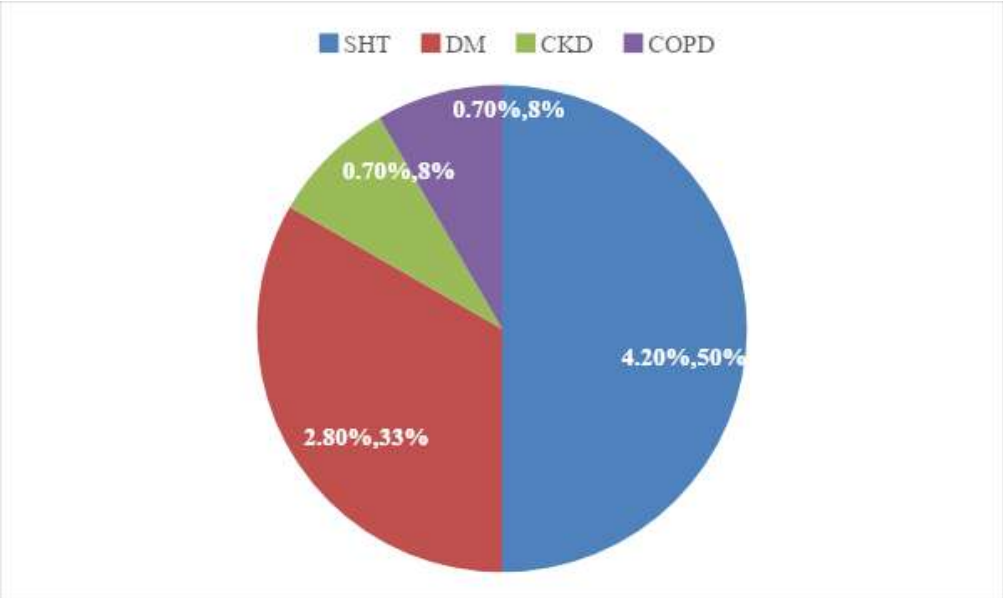
Lag time for admission after opc poisoning (hrs)	N	%
2-4	90	63.4%
4-6	37	26.1%
>6	15	10.6%
Total	142	100%



In our study (63.4%)(n=90) of the patients admitted in hospital within 2-4 hours of OPC poisoning,26.1% (n=37) within 4-6 hours and 15 patients (10.6%) admitted after 6 hours.

Table 7: DISTRIBUTION ACCORDING TO COMORBIDS (N=142)

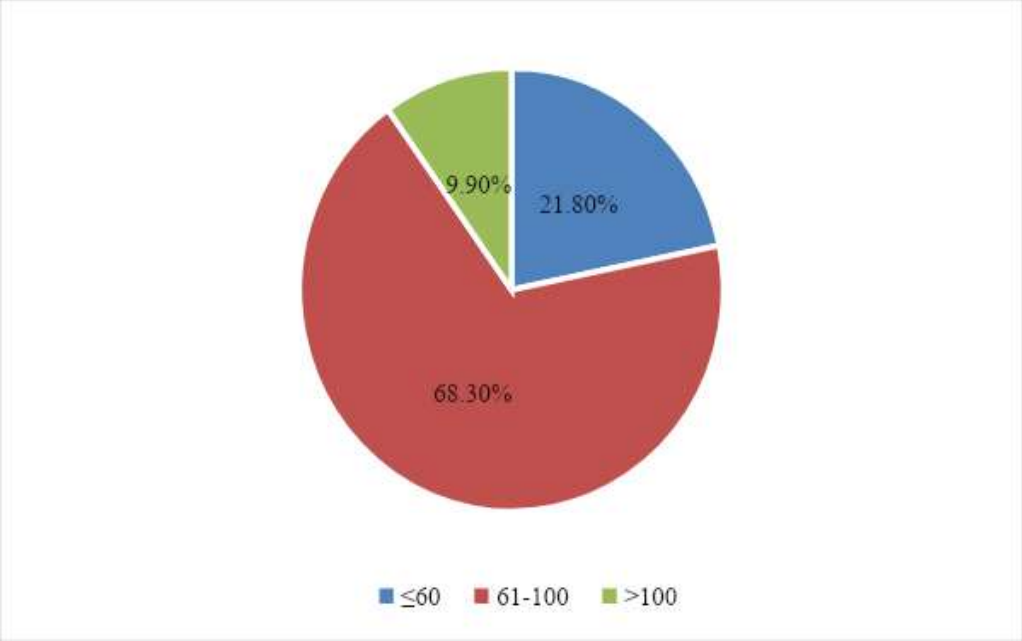
Past history	N	%
SHT	6	4.2%
DM	4	2.8%
CKD	1	0.7%
COPD	1	0.7%



In our study 6 patients (4.2%) had SHT, 4 patients (2.8%) had DM, 1 patient(0.7%) had CKD and 1 patient (0.7%) had COPD.

Table 8: DISTRIBUTION ACCORDING TO HEART RATE (N=142)

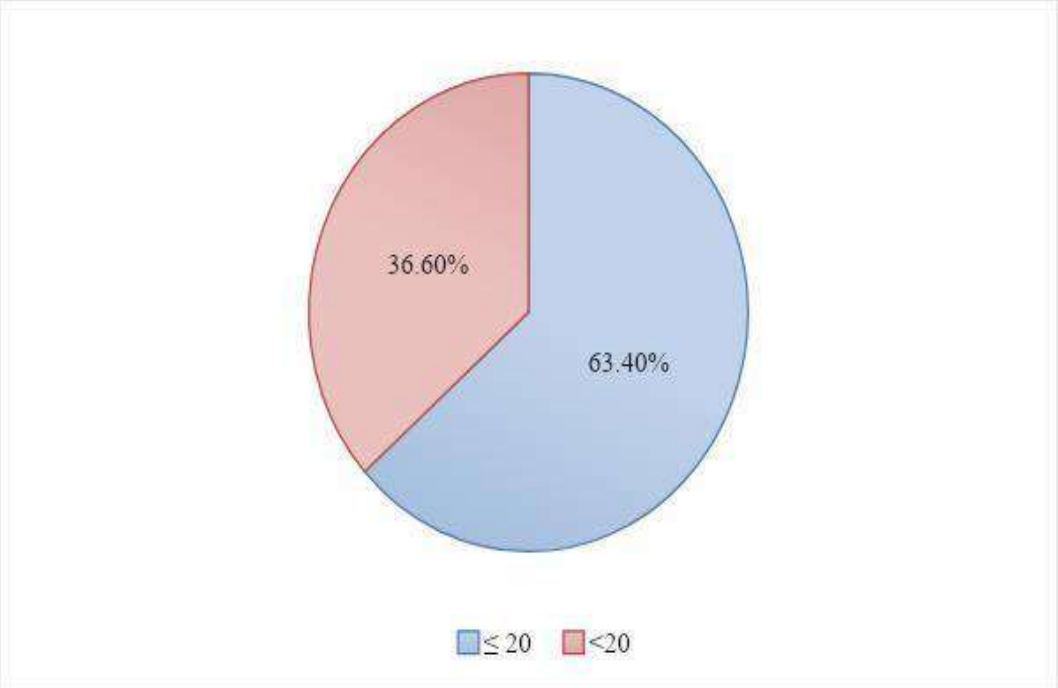
Pulse Rate (per min)	N	%
≤60	31	21.8%
61-100	97	68.3%
>100	14	9.9%
Total	142	100%



In our study 31 patients (21.8%) had Bradycardia,97 patients (68.3%) had normal heart rate and 14 patients (9.9%) had Tachycardia.

Table 9: DISTRIBUTION ACCORDING TO RESPIRATORY RATE (N=142)

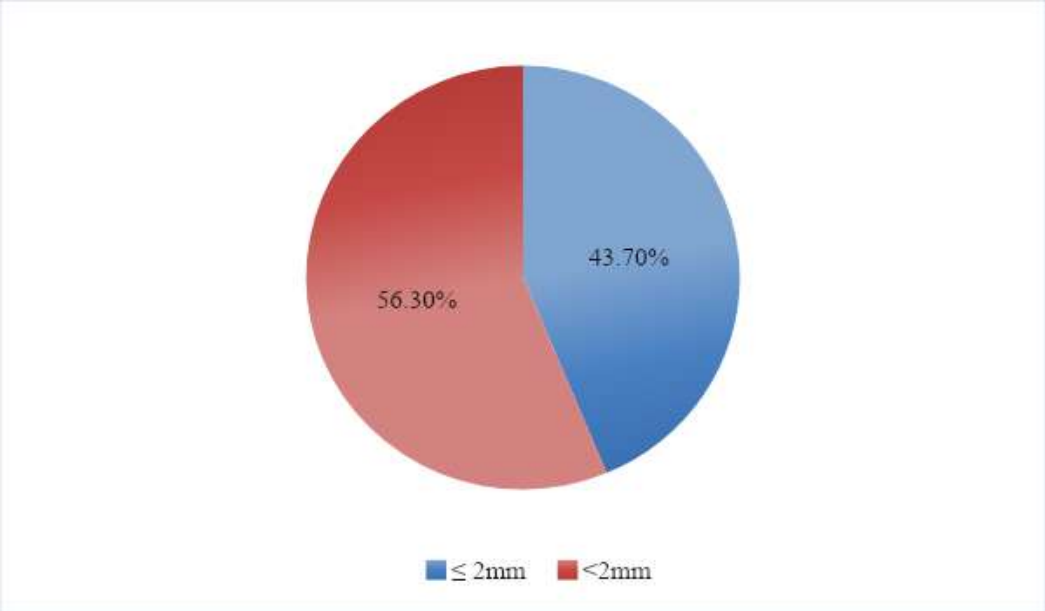
Respiratory rate (per min)	N	%
≤ 20	90	63.4%
>20	52	36.6%
Total	142	100%



In our study 52 patients (36.6%) had tachypnoea.

Table 10: DISTRIBUTION ACCORDING TO PUPIL SIZE (N=142)

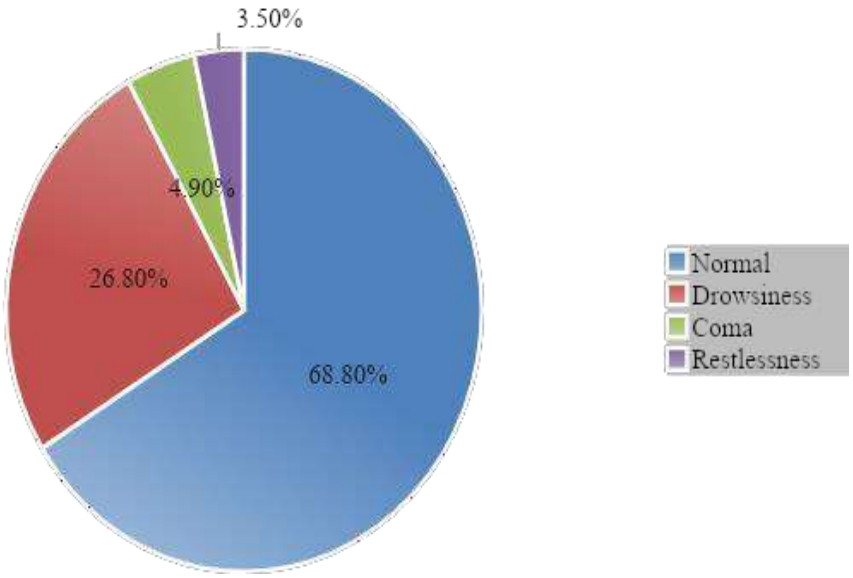
PUPIL(mm)	N	%
≤ 2mm	62	43.7%
>2mm	80	56.3%
Total	142	100%



In our study 62 patients (43.7%) had pin point and constricted pupils.

Table 11: DISTRIBUTION ACCORDING TO SENSORIUM (N=142)

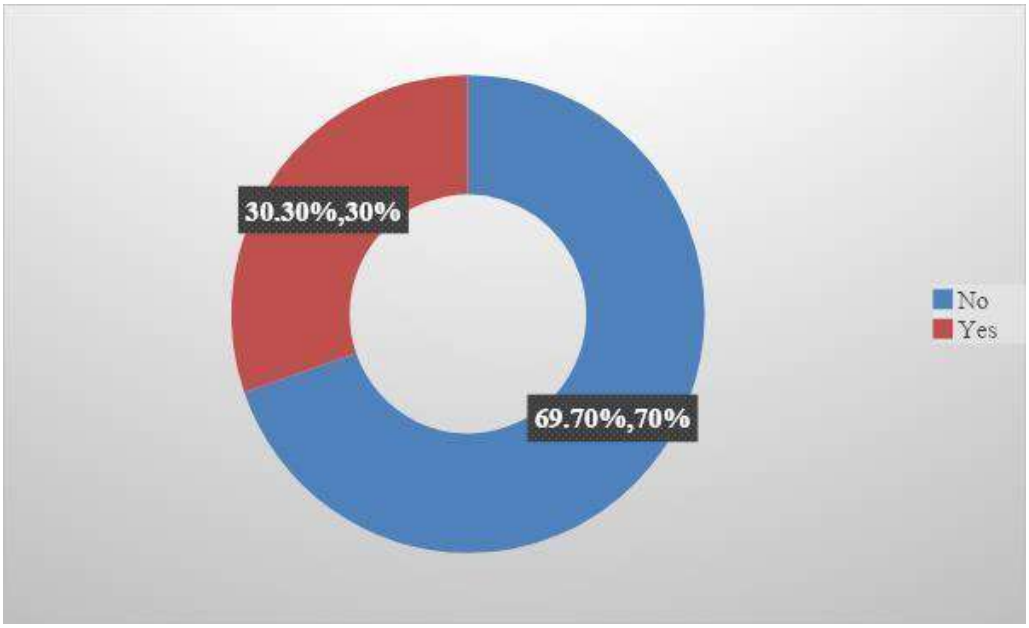
Sensorium	N	%
Normal	92	68.8%
Drowsiness	38	26.8%
Coma	7	4.9%
Restlessness	5	3.5%
Total	142	100%



In our study 38 patients (26.8%) presented with drowsiness, 7 patients (4.9%) in coma, 5 patients (3.5%) with restlessness and others (n=92)(68.8%) are with normal sensorium.

Table 12: DISTRIBUTION ACCORDING TO FASCICULATION (N=142)

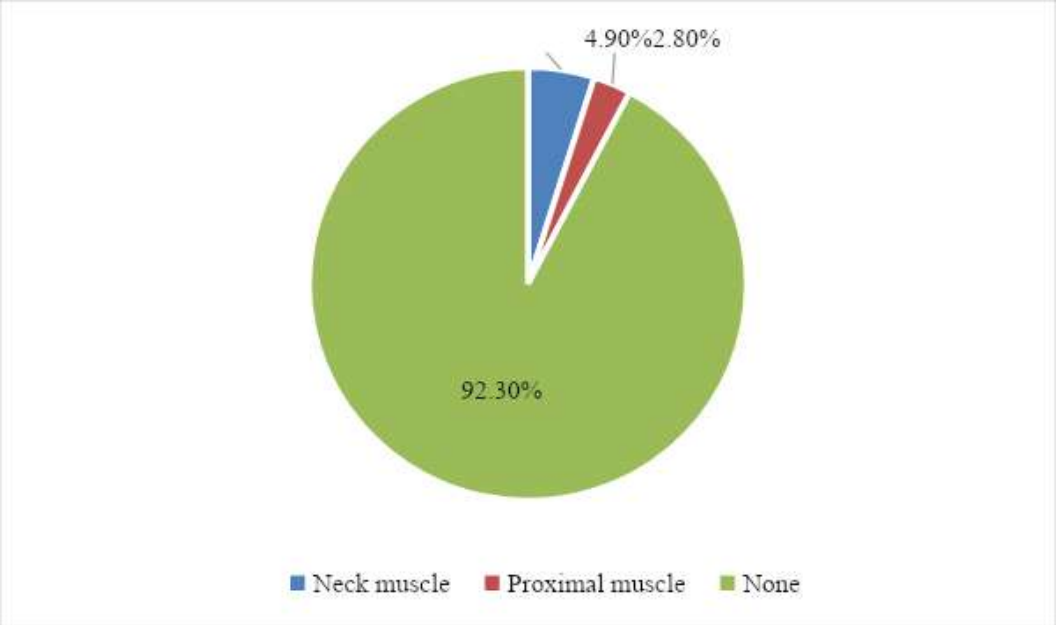
Fasciculation	N	%
No	99	69.7%
Yes	43	30.3%
Total	142	100%



In our study on examination 43 patients (30.3%) had fasciculations.

Table 13: DISTRIBUTION ACCORDING TO MUSCLE WEAKNESS (N=142)

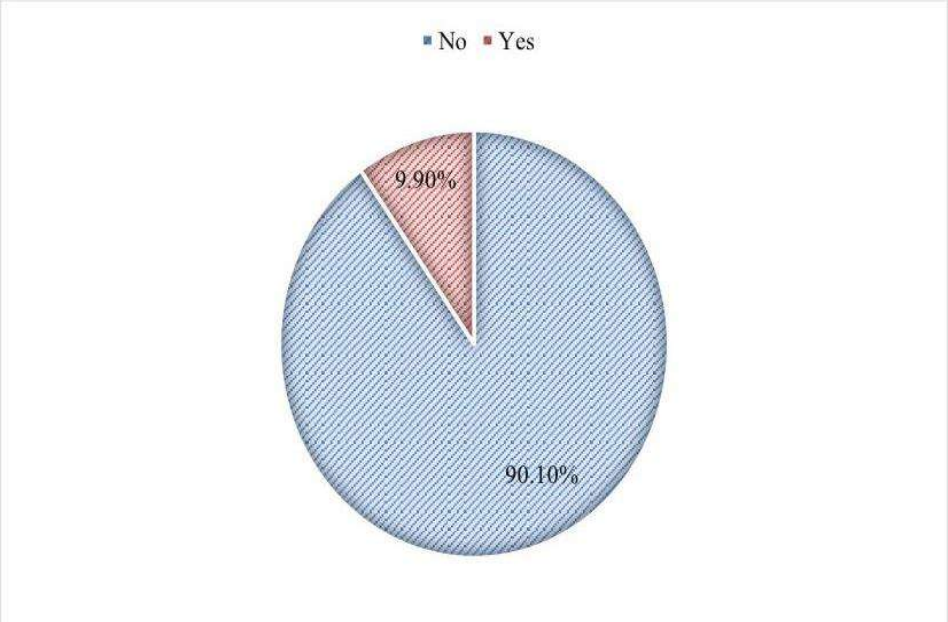
Weakness	N	%
Neck muscle	7	4.9%
Proximal muscle	4	2.8%
None	131	92.3%
Total	142	100%



In our study 7 patients(4.9%) had neck muscle weakness and 4 patients (2.8%) had proximal muscle weakness.

Table 14: DISTRIBUTION ACCORDING TO RESPIRATORY MUSCLE WEAKNESS (N=142)

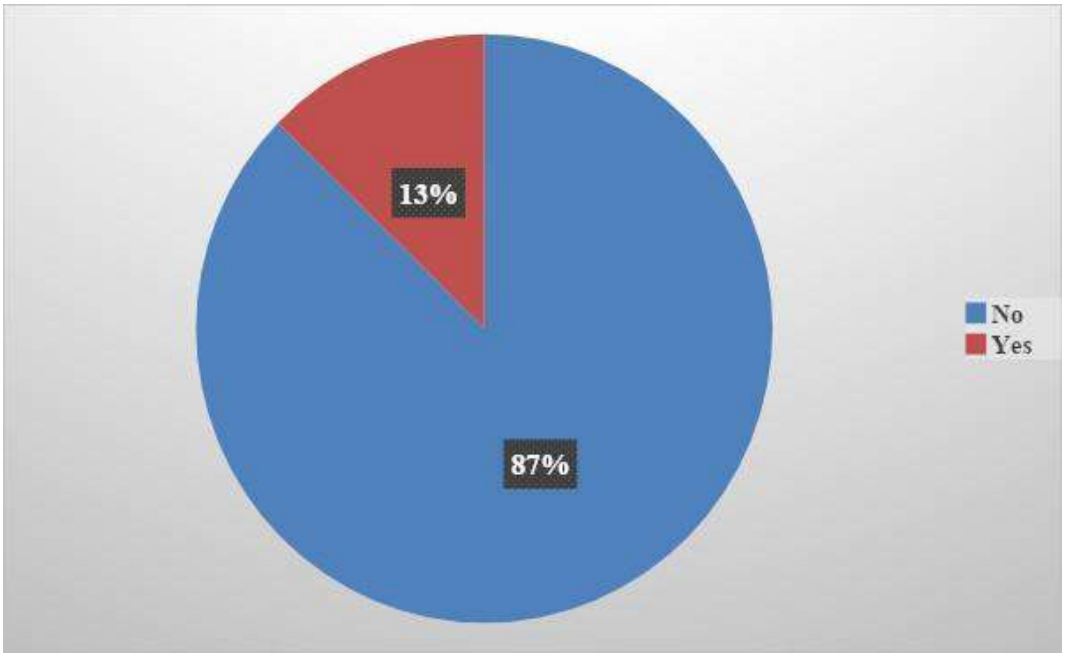
Respiratory muscle weakness	N	%
No	128	90.1%
Yes	14	9.9%
Total	142	100%



In our study 14 patients (9.9%) presented with respiratory muscle weakness.

Table 15: DISTRIBUTION ACCORDING TO SEIZURE (N=142)

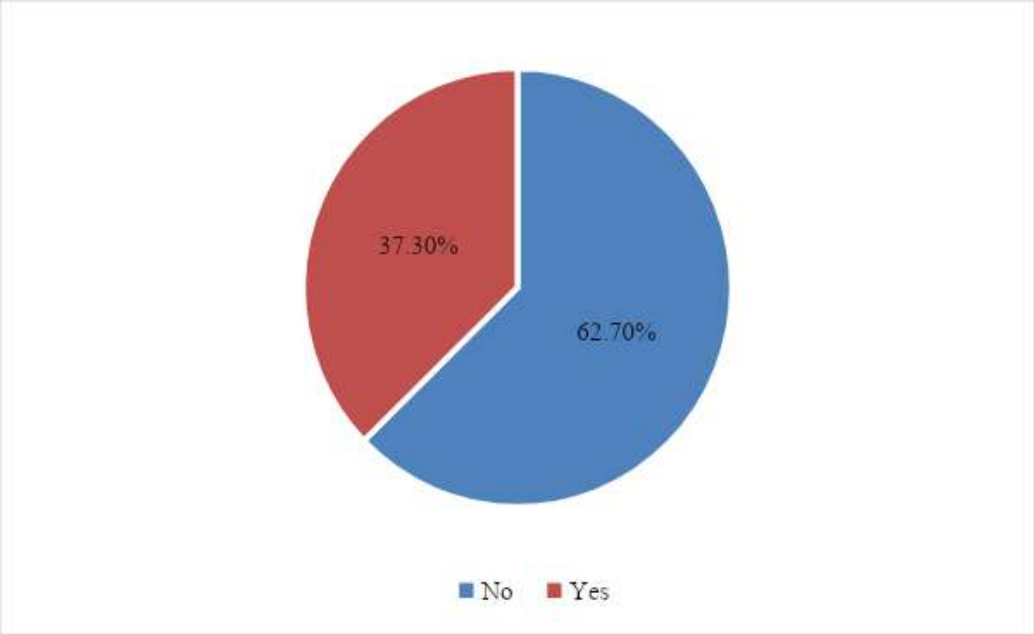
Seizure	N	%
No	124	87.3%
Yes	18	12.7%
Total	142	100%



In our study 18 patients (12.7%) had a seizure episode at the time of admission.

Table 16: DISTRIBUTION ACCORDING TO MECHANICAL VENTILATION REQUIREMENT (N=142)

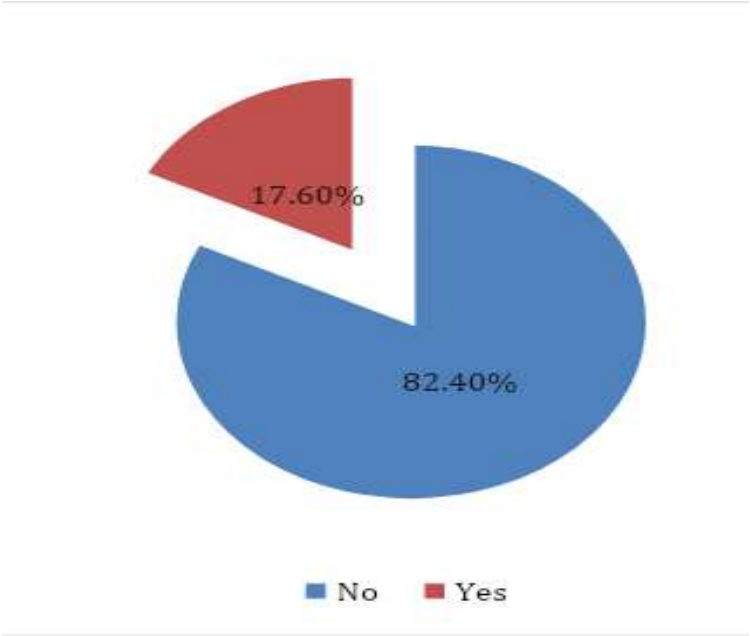
Mechanical ventilation required	N	%
No	89	62.7%
Yes	53	37.3%
Total	142	100%



In our study 53 patients (37.3%) required mechanical ventilation.

Table 17: DISTRIBUTION ACCORDING TO IMS (N=142)

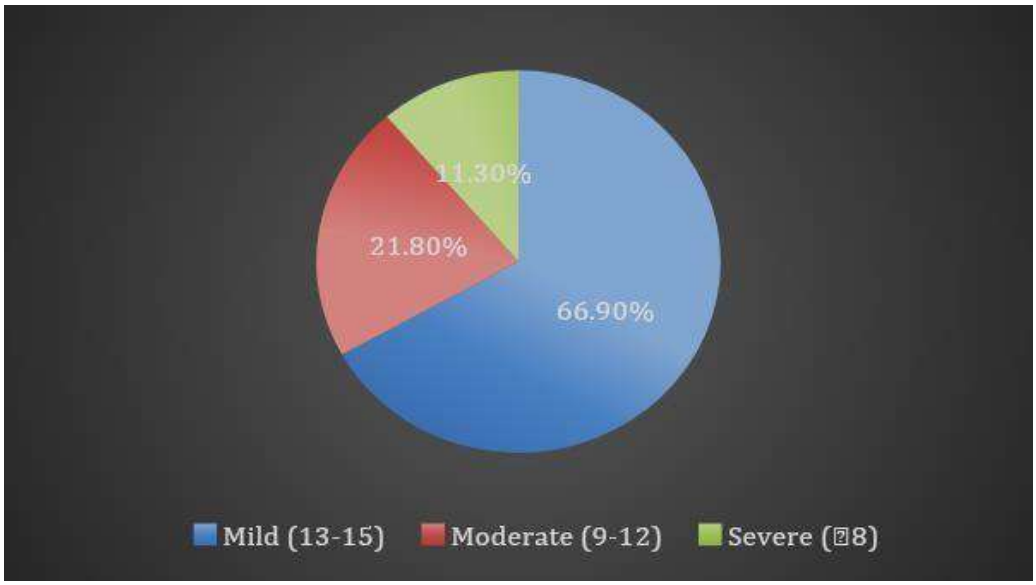
IMS	N	%
No	117	82.4%
Yes	25	17.6%
Total	142	100%



In our study 25 patients (17.6%) developed intermediate syndrome.

Table 18: DISTRIBUTION ACCORDING TO GCS (N=142)

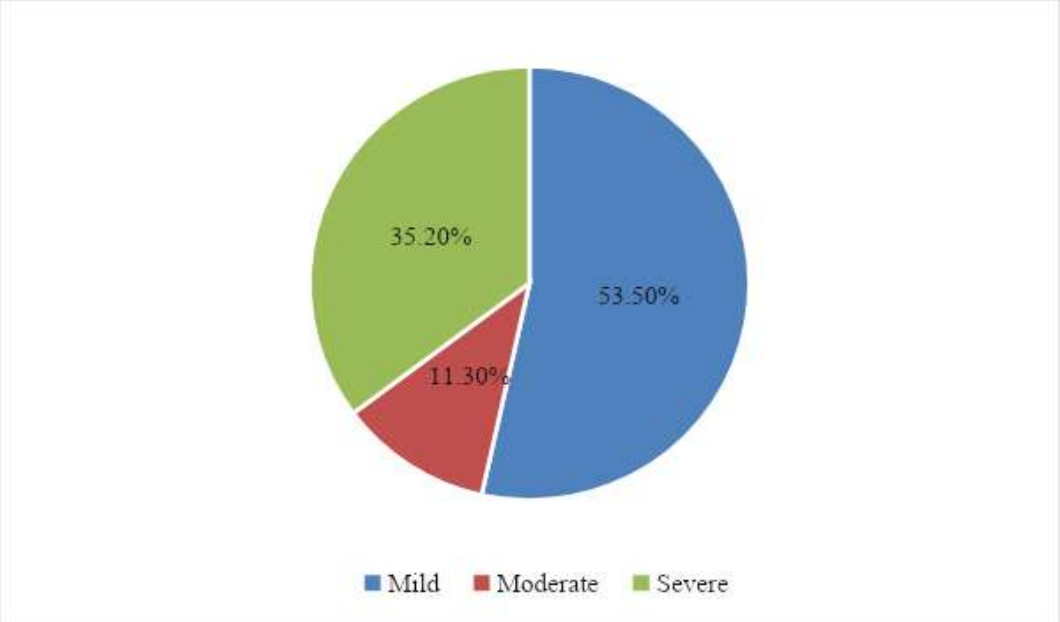
GCS	Frequency	Percentage
Mild (13-15)	95	66.9%
Moderate (9-12)	31	21.8%
Severe (≤8)	16	11.3%
Total	142	100%



In our study,on admission, 95 patients (66.9%) were categorised as mild, 31 patients(21.8%) in moderate and 16 patients (11.3%) in severe according to GCS.

Table 19: DISTRIBUTION ACCORDING TO S.CHOLINESTERASE (N=142)

Serum Cholinesterase(U/L)	N	%
Mild	76	53.5%
Moderate	16	11.3%
Severe	50	35.2%
Total	142	100%



In our study 76 patients (53.5%) came under mild, 16 patients (11.3%) under moderate and 50 patients (35.2%) under severe category according to serum cholinesterase level.

Table 20: DISTRIBUTION ACCORDING TO POP SCALE (N=142)

POP	Frequency	Percentage
Mild (1-3)	97	68.3%
Moderate (4-7)	33	22.5%
Severe (>7)	13	9.2%
Total	142	100%

In our study 97 patients (68.3%) categorised under mild,33 patients (22.5%) under moderate and 13 patients (9.2%) under severe category according to POP SCALE.

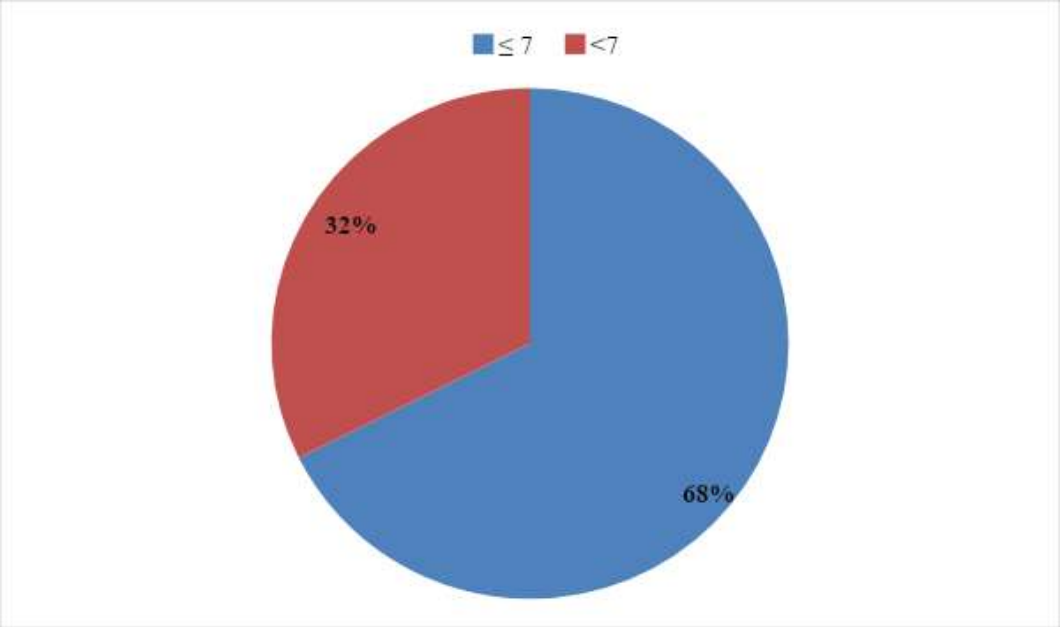
Table 21: DISTRIBUTION ACCORDING TO POISONING SEVERITY SCORE (N=142)

Poisoning Severity Score	Frequency	Percentage
None (0)	21	14.8%
Mild (1)	65	45.8%
Moderate (2)	37	26.1%
Severe (3)	19	13.4%
Total	142	100%

In our study 65 patients (45.8%) categorised under mild, 37 patients (26.1%) under moderate and 19 patients (13.4%) under severe category according to poisoning severity score.

Table 22: DISTRIBUTION ACCORDING TO IMCU STAY (N=142)

IMCU STAY (Days)	N	%
≤ 7	96	67.6%
>7	46	32.4%
Total	142	100%



In our study, treatment of 96 patients (67.6%) in IMCU were less than 7 days and 46 patients (32.4%) stayed more than 7 days.

Table 23: DISTRIBUTION ACCORDING TO OUTCOME (N=142)

Outcome	Frequency	Percentage
Recovered	130	91.5%
Expired	12	8.5%
Total	142	100%

In our study 12 patients (8.5%) expired and others 130 (91.5%) recovered.

DESCRIPTIVE STATISTICS TO COMPARE THE OUTCOME WITH VARIOUS PARAMETERS BY CHI-SQUARE TEST

Table 24: COMPARISON OF MECHANICAL VENTILATION REQUIRED WITH GCS

Mechanical ventilation required	GCS			Total
	Mild (13-15)	Moderate (9-12)	Severe (≤8)	
No	84 (94.4%)	5 (5.6%)	0 (0%)	89
Yes	11 (20.8%)	26 (49.1%)	16 (30.2%)	53
Total	95 (66.9%)	31 (21.8%)	16 (11.3%)	142
Chi Square: 82.496, P value: <0.001 (Significant)				

In our study, out of 95 patients in the mild category of GCS 11 patients (11.5%) required mechanical ventilation, out of 31 patients in moderate category 26 (83.8%) required mechanical ventilation and all 16 patients(100%) in the severe category required mechanical ventilation.

Table 25: COMPARISON OF MECHANICAL VENTILATION REQUIRED WITH S.ACETYLCHOLINESTERASE

Mechanical ventilation required	AchE			Total
	Mild (>4500)	Moderate (4500-2500)	Severe (<2500)	
No	74	10	19	103
Yes	2(2.6%)	6(37.5%)	31(62%)	39
Total	76	16	50	142
P value: <0.001 (Significant)				

In our study, mechanical ventilation was required in 2 (2.6%) out of 76 patients in the mild category, 6(37.5%) out of 16 patients in the moderate category, and 31(62%)patients out of 50 in the severe category according to the S.AchE levels.

Table 26: COMPARISON OF MECHANICAL VENTILATION REQUIRED WITH POP SCALE

Mechanical ventilation required	POP			Total
	Mild (1-3)	Moderate (4-7)	Severe (>7)	
No	85 (95.5%)	4 (4.5%)	0 (0%)	89
Yes	12 (22.6%)	28 (52.8%)	13 (24.5%)	53
Total	97 (68.3%)	32 (22.5%)	13 (9.2%)	142
Chi Square: 82.09, P value: <0.001 (Significant)				

In our study,mechanical ventilation was required in 12 (12.3%) out of 97 patients in the mild category,28(87.5%) out of 32 patients in the moderate category, and in all 13 patients (100%) in the severe category according to the POP scale.

Table 27: COMPARISON OF MECHANICAL VENTILATION REQUIRED WITH POISONING SEVERITY SCORE

Mechanical ventilation required	Poisoning Severity Score				Total
	None (0)	Mild (1)	Moderate (2)	Severe (3)	
No	21 (23.6%)	61 (68.5%)	7 (7.9%)	0 (0%)	89
Yes	0 (0%)	4 (7.5%)	30 (56.6%)	19 (35.8%)	53
Total	21 (14.8%)	65 (45.8%)	37 (26.1%)	19 (13.4%)	142
Chi Square: 101.69, P value: <0.001 (Significant)					

In our study, mechanical ventilation was required in 4 (6.1%) out of 65 patients in the mild category, 30 (81%) out of 37 patients in the moderate category and all the 19(100%) patients in the severe category according to the poisoning severity score.

Table 28: COMPARISON OF LAG TIME FOR ADMISSION AFTER OPC POISONING WITH OUTCOME

Lag time for admission after opc poisoning (Hrs)	Outcome		Total
	Recovered	Expired	
2-4	88 (67.7%)	2 (16.7%)	90 (63.4%)
4-6	32 (24.6%)	5 (41.7%)	37 (26.1%)
>6	10 (7.7%)	5 (41.7%)	15 (10.6%)
Total	130 (100%)	12 (100%)	142 (100%)
Chi Square: 17.743, P value: <0.001 (Significant)			

In our study most of the patients 5 (33.3%) out of 15, expired are admitted after 6 hours of OPC poison consumption followed by 5(13.5%) out of 37 patients expired are admitted 4-6 hours after OPC poisoning and 2 (2.2%) out of 90 patients expired within 4 hours of OPC poisoning.

Table 29: COMPARISON OF MECHANICAL VENTILATION REQUIREMENT WITH OUTCOME (N=142)

Mechanical ventilation required	Outcome		Total
	Recovered	Expired	
No	89 (100%)	0 (0%)	89
Yes	41 (77.4%)	12(22.6%)	53
Total	130 (100%)	12 (100%)	142
Chi Square: 22.011, P value: <0.001 (Significant)			

In our study out of 53 patients who required mechanical ventilation 12 patients (22.6%) expired and 41 patients (77.4%) recovered.

Table 30: COMPARISON OF S.ACETYLCHOLINESTERASE LEVEL WITH OUTCOME

OUTCOME	AchE			Total
	Mild (>4500)	Moderate (4500-2500)	Severe (<2500)	
RECOVERED	76	14	40	130
EXPIRED	0	2(12.5%)	10(20%)	12
Total	76	16	50	142
P value: <0.001 (Significant)				

In our study,out of 76 patients categorised under mild category none(0%) expired,out of 16 patients under moderate category 2(12.5%) patients expired and out of 50 patients under severe category 10(20%) expired according to S.AchE levels.

Table 31: COMPARISON OF GCS WITH OUTCOME

GCS	Outcome		Total
	Recovered	Expired	
Mild (13-15)	94 (72.3%)	1 (8.3%)	95 (66.9%)
Moderate (9-12)	31 (23.8%)	0 (0%)	31 (21.8%)
Severe (≤8)	5 (3.8%)	11 (91.7%)	16 (11.3%)
Total	130 (100%)	12 (100%)	142 (100%)
Chi Square: 84.779, P value: <0.001 (Significant)			

In our study, out of 95 patients categorised under mild category 1 patient (1.05%) expired,out of 31 patients under moderate category,none(0%) expired and out of 16 patients under severe category 11(68%) patients expired according to GCS.

Table 32: COMPARISON OF POP SCALE WITH OUTCOME

POP	POP		Total
	Recovered	Expired	
Mild (1-3)	97 (74.6%)	0 (0%)	97 (68.3%)
Moderate (4-7)	26 (20%)	6 (50%)	32 (22.5%)
Severe (>7)	7 (5.4%)	6 (50%)	13 (9.2%)
Total	130 (100%)	12 (100%)	142 (100%)
Chi Square: 37.228, P value: <0.001 (Significant)			

In our study, out of 97 patients categorised under mild category none(0%) expired,out of 32 patients under moderate category 6(18.7%) expired and out of 13 under severe category 6 (46.1%) expired according to the POP scale.

Table 33: COMPARISON OF POISONING SEVERITY SCORE WITH OUTCOME (N=142)

Poisoning Severity Score	Outcome		Total
	Recovered	Expired	
None	21 (16.2%)	0 (0%)	21 (14.8%)
Mild	97 (50%)	0 (0%)	65 (45.8%)
Moderate	26 (27.7%)	6 (8.3%)	37 (26.1%)
Severe	7 (6.2%)	6 (91.7%)	19 (13.4%)
Total	130 (100%)	12 (100%)	142 (100%)
Chi Square: 69.55, P value: <0.001 (Significant)			

In our study,out of 65 patients categorised under mild category none(0%) expired,out of 37 patients under moderate category 6(16.2%) expired and out of 19 patients under severe category 6 (31.5%) expired according to poisoning severity score.

Table 34: COMPARISON OF IMCU STAY WITH OUTCOME

IMCU	Outcome		Total
	Recovered	Expired	
≤ 7	96 (73.8%)	0 (0%)	96 (67.6%)
>7	34 (26.2%)	12 (100%)	46 (32.4%)
Total	130 (100%)	12 (100%)	142 (100%)
Chi Square: 27.355, P value: <0.001 (Significant)			

In our study,out of 96 patients with less than 7 days of IMCU stay, they had better outcome (100%) recovered and none(0%) expired. Out of 46 patients with more than 7 days of IMCU stay 12 patients (26%) expired and 34 (74%) recovered.

DISCUSSION

Acute Organophosphorus poisoning is one of the most common poisoning in the developing world and poses significant health problems and also a potential cause of human mortality. And majority of the cases occur in the Asian countries due to lack of regulation and easy availability of poisoning. According to WHO Acute OPC Poisoning is the most common poisoning and it causes 3 million human deaths worldwide each year.

OPC poisoning is highly preventable and completely treatable if reported early and treated properly. The most important step in the management is the assessment of the patient for the need for ventilatory support. Hence early recognition and transfer of the patient to the ICU holds the key for the better prognosis. Since most of these poisonings occur in the rural areas, where the basic infrastructure is very poor, clinical scales play an important role in predicting the severity of the situation.

Acute OPC poisoning is one of the most common poisoning encountered in Madras Medical College and Rajiv Gandhi Government General Hospital.
In our study, 142 patients who satisfied the inclusion and exclusion criteria with Organophosphorus poisoning were included.

In this study, 142 patients with a definite history of OPC poisoning presenting to a tertiary care hospital, were assessed using the internationally validated scoring systems like the POP scale, PSS, and GCS to predict the severity of OPC poisoning and treatment outcome. In the present study the ventilator requirement and mortality was found to be in 37.3% and 8.5% of

patients respectively. This was correlated with a study conducted by J V Peter et al [57], where 65.7% of patients required mechanical ventilation and had a mortality of 13.1%.Incidence is higher (36.6%) in the age group of 21-30 years followed by 26.1% in the age group of 31-40 years.

In our study out of total participants of 142 patients, 80 of them are males (56.3%) , and 62 of them are females (43.7%) .The majority of patients are males showing that the incidence of poisoning is more in males.This Correlates with the findings of the previously published studies. However , one study done by M.Vishwanathan et al [58] showed that the majority of patients were females(66%).

In our study the most common OPC compound is Chlorpyrifos (21.10%),(N=30), followed by Malathion and Monocrotophos (14.8%) ,(n=21)each. So the type of compound predicts the prognosis of OPC Poisoning.In our study farmers consumed OPC poison more commonly (45.1%) (n=64),followed by students (36.6%) (n=52). The most common complaints are Excessive salivation (62.7%) (n=89),followed by vomiting(58.5%) (n=83),Excessive lacrimation (31.7%)(n=45),altered sensorium(27.5%)(n=39),loose stools(26.8%)(n=38).

In our study (63.4%)(n=90) of the patients admitted in hospital within 2-4 hours of OPC poisoning,26.1% (n=37) within 4-6 hours and 15 patients (10.6%) admitted after 6 hours. In terms of comorbids 6 patients (4.2%) had SHT, 4 patients (2.8%) had DM,1 patient(0.7%) had CKD and 1 patient (0.7%) had COPD.

The most frequent nicotinic features are 31 patients (21.8%) had Bradycardia,97 patients (68.3%) had normal heart rate and 14 patients (9.9%) had Tachycardia.Robert et al [59] study found ,19% of the patients had bradycardia, and another study conducted by Semir Nouria [60] , 17 % had bradycardia.In our study 52 patients (36.6%) had tachypnoea.The most marked muscarinic feature in our study was 62 patients (43.7%) had pin point and constricted pupils..In our study 38 patients (26.8%) presented with drowsiness, 7 patients (4.9%) in coma, 5 patients (3.5%) with restlessness and others (n=92)(68.8%) were with normal sensorium,43 patients (30.3%) had fasciculations.

In our study 7 patients(4.9%) had neck muscle weakness and 4 patients (2.8%) had proximal muscle weakness and 14 patients (9.9%) presented with respiratory muscle weakness.In our study 18 patients (12.7%) had a seizure episode at the time of admission.In our study 25 patients (17.6%) developed intermediate syndrome.In our study 53 patients (37.3%) required mechanical ventilation. Statistically this is significant with p value of < 0.0001.Hence Ventilator requirement is an important prognostic indicator in Organophosphorus poisoning.

In our study,on admission, 95 patients (66.9%) were categorised as mild, 31 patients(21.8%) in moderate and 16 patients (11.3%) in severe according to GCS.

In our study 76 patients (53.5%) came under mild, 16 patients (11.3%) under moderate and 50 patients (35.2%) under severe category according to serum cholinesterase level.In our study 97 patients (68.3%) categorised under

mild,33 patients (22.5%) under moderate and 13 patients (9.2%) under severe category according to POP SCALE. In our study 65 patients (45.8%) categorised under mild, 37 patients (26.1%) under moderate and 19 patients (13.4%) under severe category according to poisoning severity score.In our study, treatment of 96 patients (67.6%) in IMCU were less than 7 days and 46 patients (32.4%) stayed more than 7 days.Totally 12 patients (8.5%) expired and others 130 (91.5%) recovered.

In our study, out of 95 patients in the mild category of GCS 11 patients (11.5%) required mechanical ventilation,out of 31 patients in moderate category 26 (83.8%) required mechanical ventilation and all 16 patients(100%) in the severe category required mechanical ventilation.Mechanical ventilation was required in 2 (2.6%) out of 76 patients in the mild category, 6(37.5%) out of 16 patients in the moderate category, and 31(62%)patients out of 50 in the severe category according to the S.AchE levels.Mechanical ventilation was required in 12 (12.3%) out of 97 patients in the mild category,28(87.5%) out of 32 patients in the moderate category, and in all 13 patients (100%) in the severe category according to the POP scale.Mechanical ventilation was required in 4 (6.1%) out of 65 patients in the mild category, 30 (81%) out of 37 patients in the moderate category and all the 19(100%) patients in the severe category according to the poisoning severity score.Requirement of ventilator support is high when the patient on admission has severe toxicity clinically by using peradynia scoring scale(POP),GCS and PSS which has high statistical significance,so that can be

used as a prognostic indicator in OPC Poisoning.This was well correlated with the study conducted by Raveendra, et al.[61]

In our study most of the patients 5 (33.3%) out of 15, expired are admitted after 6 hours of OPC poison consumption followed by 5(13.5%) out of 37 patients expired are admitted 4-6 hours after OPC poisoning and 2 (2.2%) out of 90 patients expired within 4 hours of OPC poisoning.Mortality were higher among the patients who got admitted after 6 hours.This was well correlated with the study conducted by Raveendra, et al.[61] In our study out of 53 patients who required mechanical ventilation 12 patients (22.6%) expired and 41 patients (77.4%) recovered.

In our study,out of 76 patients categorised under mild category none(0%) expired,out of 16 patients under moderate category 2(12.5%) patients expired and out of 50 patients under severe category 10(20%) expired according to S.AchE levels.Out of 95 patients categorised under mild category 1 patient (1.05%) expired,out of 31 patients under moderate category,none(0%) expired and out of 16 patients under severe category 11(68%) patients expired according to GCS.Out of 97 patients categorised under mild category none(0%) expired,out of 32 patients under moderate category 6(18.7%) expired and out of 13 under severe category 6 (46.1%) expired according to the POP scale.Out of 65 patients categorised under mild category none(0%) expired,out of 37 patients under moderate category 6(16.2%) expired and out of 19 patients under severe category 6 (31.5%) expired according to poisoning severity score.These are all well correlated with the study conducted by Raveendra, et al.[61]

In our study,out of 96 patients with less than 7 days of IMCU stay, they had better outcome (100%) recovered and none(0%) expired. Out of 46 patients with more than 7 days of IMCU stay 12 patients (26%) expired and 34 (74%) recovered.

Intubation rates were significantly higher in patients with moderate and severe grades whereas mortality rates were higher in patients with severe grades of POP score, PSS and GCS with a significant p value of 0.001.

All the three scoring systems were well correlated in predicting the need for intubation and mortality in OPC poisoning cases. These are simple and effective tools and can be assessed based on the clinical examination.

The Glasgow Coma Scale (GCS) and the International Program on Chemical Safety Poison Severity Scale (IPCS PSS) were both found to be equally accurate at predicting the outcome of patients poisoned by OP pesticides, according to a multicenter cohort study conducted in Sri Lanka by J O J Davies et al, [62].

To determine the effectiveness of the PSS and GCS scoring systems in predicting the severity and clinical consequences of OP poisoning, S Chandrashekar et al.[63] conducted an observational clinical study at the department of medicine Kurnool Medical College, Andhra Pradesh, India, on 100 patients I predicting severity and outcomes of OPC poisoning , GCS and PSS are both useful tools.

A prospective cross-sectional study was carried out by Shashank Tripathi [64] at the tertiary health centre in Nagpur between October 2011 and September 2013. In order to correlate the predictive value of GCS, POP, and serum acetylcholinesterase levels in acute OP poisoning, the study was conducted. 17.5% of patients had fatality, and patients with severe grades of POP and GCS scores had greater mortality rates than other patients.

CONCLUSION

One of the most frequent poisonings in this region of the world is caused by OPC compounds. The majority of these are either accidental or suicidal and come from rural backgrounds. The majority of deaths result from failure to report to medical facilities on time or from inadequate infrastructure at nearby hospitals (primary health care centres). As a result, the majority of these patients receive ICU support from tertiary care centres. Much time is wasted, and our study revealed the same. Different grading systems are frequently applied during first medical care to help identify the severity and later assist in early referral to a tertiary care institution for ICU support.

Additionally, because biochemical testing is not readily available at all of the centres, these clinical grading systems must be used to determine the severity of OPC poisoning. Furthermore, these scoring systems are straightforward, efficient tools that a primary care physician can use in a primary health centre for early assessment and prompt referral for a better treatment outcome.

LIMITATIONS

- Small sample size.Hence results cannot be extrapolated to general population
- Serial estimation of the biochemical parameters during the course of hospital stay was not done.

BIBLIOGRAPHY

1. Zhao X, Wu C, Wang Y, Cang T, Chen L, Yu R, et al. Assessment of toxicity risk of insecticides used in rice ecosystem on Trichogramma japonicum, an egg parasitoid of rice lepidopterans. J Econ Entomol. Feb 2012;105(1):92-101.

2. Chen SW, Gao YY, Zhou NN, Liu J, Huang WT, Hui L, et al. Carbamates of 4'-demethyl-4-deoxypodophyllotoxin: synthesis, cytotoxicity and cell cycle effects. Bioorg Med Chem Lett. Dec 15 2011;21(24):7355-8.

3. Trupati H Trivedi et al , API TEXT BOOK OF MEDICINE, 11 nth Edition, Chapter 4 , Organophosphorus compound poisoning, page 2263-2266.

4. Deaths A. Suicides in India 2015, National Crime Records Bureau, Ministry of Home Affairs; 2015 pg-255

5. WHO in collaboration with UNEP, 1990. Public Health Impact of Pesticides used in Agriculture. Updated June 2007, WHO, Geneva.

6. Saravanapavananthan T. Serum pseudocholinesterase estimation in the management of organophosphate poisoning cases and the effect of PAM on regenerating it. Singapore medical journal. 1987;28:166.

7. Aygun D, Doganay Z, Altintop L, Guven H, Onar M, Deniz T, Sunter T. Serum acetylcholinesterase and prognosis of acute organophosphate poisoning. Journal of Toxicology: Clinical Toxicology. 2002;40:903-10.

8. Persson HE, Sjöberg GK, Haines JA, de Garbino JP. Poisoning severity score. Grading of acute poisoning. Journal of Toxicology: Clinical Toxicology. 1998;36:205-13.

9. Dreisbach RH. Cholinesterase inhibitor pesticides. In : Hand Book of poisoning 11th ed. Longe medical publications. California. 1983: 106-14.

10. Robert S. Hoffman, Lewis S. Nelson, Mary Ann Howland, Neal A. Lewin, Neal E. Flomenbaum, Lewis R. Goldfrank et al, Goldfrank's MANUAL OF TOXICOLOGIC EMERGENCIES, Chapter 109, Insecticides: Organic phosphorus compounds and carbamates, page 837.

11. Greaves, Alana K.; Letcher, Robert J.; Chen, Da; McGoldrick, Daryl J.; Gauthier, Lewis T.; Backus, Sean M. (2016-10-01). "Retrospective analysis of organophosphate flame retardants in herring gull eggs and relation to the aquatic food web in the Laurentian Great Lakes of North America".

12. Paranthaman P, Raja P,Madhavan V,Arun prabu P. MANUAL OF TOXICOLOGY –AN INDIAN PERSPECTIVE, Chapter 3, Organophosphorus Poisoning, Page 26-49.

13. Kumar S, Fareedullah M, Sudhakar Y, Venkateswarlu B, Kumar E. Current review on organophosphorus poisoning. Arch Appl Sci Res. 2010;2(4):199-215.

14. Marrs TC. Organophosphate poisoning. Pharmacology & therapeutics. 1993;58(1):51-66.

15. Bajgar J. Organophosphates Nerve Agent Poisoning: Mechanism of Action, ⁄ Diagnosis, Prophylaxis, and Treatment. Advances in clinical chemistry. 2004;38:151-216.

16. Fest C, Schmidt K-J. The chemistry of organophosphorus pesticides: Springer Science & Business Media; 2012.

17. Paranthaman P, Raja P,Madhavan V,Arun prabu P. MANUAL OF TOXICOLOGY –AN INDIAN PERSPECTIVE, Chapter 3, Organophosphorus Poisoning, Page 26-49.

18. Watson WA, Livtovitz TL, Rodgers GC Jr, et al .2002 annual report of the American Association of poison control centres Toxic Exposure Suveillance System. Am J Emerg Med 2003;21:353.

19. Maroni M. Review of toxicological properties and biotransformation of organophosphorous esters. In : WHO Manual Of Analytical maethods. Cremona : WHO collaboration center for occupational health 1985 ; 3-39

20. Karalliedde L, Senanayake N : Organophosporous insecticide poisoning. British Journal of anaeasthesia 1989; 63; 736-750.

21. Davies DR, Green AL. The kinetics of reactivation by oximes of cholinesterase inhibition by Organophosphorous compounds. Biochemical Journal 1956; 63: 529-535.

22. KD TRIPATHI, Essentials of MEDICAL PHARMACOLOGY, Seventh Edition, Chapter 7b, Cholinergic System and Drugs Page 99-112.

23. Karalliedde L, Senanayake N. Acute organophosphorous insecticide poisoning in Sri Lanka. Forensic Science International 1988; 36: 97-100.

24. Xie, W., Stribley, J.A., Chatonnet, A., et al. Postnatal development delay and supersensitivity to organophosphate in gene-targeted mice lacking acetylcholinesterase.J. Pharmacol. Exp.Ther.,2000,293:892–902.

25. Senanayake N, Karalliedde L. Neurotoxic effects of organophosphorus insecticides. An intermediate syndrome. The New England journal of medicine. 1987;316(13):761-3.

26. Sidell FR. Clinical effects of organophosphorus cholinesterase inhibitors. Journal of applied toxicology: JAT. 1994;14(2):111-3.

27. Moretto A, Lotti M. Poisoning by organophosphorus insecticides and sensory neuropathy. Journal of neurology, neurosurgery, and psychiatry. 1998; 64(4):463-8.

28. Sevim S, Aktekin M, Dogu O, Ozturk H, Ertas M. Late onset polyneuropathy due to organophosphate (DDVP) intoxication. The Canadian journal of neurological sciences Le journal canadien des sciences neurologiques. 2003;30(1):75-8.

29. Yang PY, Tsao TC, Lin JL, Lyu RK, Chiang PC. Carbofuran-induced delayed neuropathy. Journal of toxicology Clinical toxicology. 2000;38(1):43-6.

30. Dickoff DJ, Gerber O, Turovsky Z. Delayed neurotoxicity after ingestion of carbamate pesticide. Neurology. 1987;37(7):1229-31.

31. Glynn P. Neuropathy target esterase. The Biochemical journal. 1999;344 Pt 3:625-31.

32. Aygun D, Onar MK, Altintop BL. The clinical and electrophysiological features of a delayed polyneuropathy developing subsequently after acute organophosphate poisoning and it's correlation with the serum acetylcholinesterase. Electromyography and clinical neurophysiology. 2003;43(7):421-7.

33. Abou-Donia MB. Organophosphorus ester-induced chronic neurotoxicity. Archives of environmental health. 2003;58(8):484-97.

34. Johnson MK. Organophosphorus esters causing delayed neurotoxic effects: mechanism of action and structure activity studies. Archives of toxicology. 1975;34(4):259-88.

35. Kumar S, Fareedullah M, Sudhakar Y, Venkateswarlu B, Kumar E. Current review on organophosphorus poisoning. Arch Appl Sci Res. 2010;2(4):199-215..

36. Ludomirsky A, Klein H, Sarelli P, Becker B, Hoffman S, Taitelman U, et al. Q-T prolongation and polymorphous (torsades de pointes) ventricular arrhythmias associated with organophosphorus insecticide poisoning. Am J Cardiol 1982; 49:1654-8

37. Taturi J, Roberts J. Organophosphorous poisoning. Annals of emergency medicine 1987; 16: 193-202.

38. Singh S, Sharma N. Neurological syndromes following organophospate poisoning. Neurology India 2000 ; 48 (4) : 308-13.

39. Baloch ,G.H., A.H.Khan ,C.Madhudas, B.R.Devrajani, S.Z.A.Shah, T.Dos and S.A.Shah, 2011.Outcome of acute organophosphorus Poisoning at Liaquat university hospital Hyderabad.World Applied Sci.J., 13 : 266-268.

40. Balali-Mood , M. and M.Shariat,1998. Treatment of organophosphate poisoning.Experience of nerve agents and acute pesticide poisoning on the effect of oximes. J. Physiol.-Paris,92: 375-378.

41. Senanyake N, Sanmuganathan PS. Extrapyramidal manifestations complicating Organ phosphorous insecticide poisoning. Human and experimental Toxicology 1995; 14: 600-604.

42. Persson H, Sjöberg G, Haines J, Pronczuk de Garbino J. Poisoning Severity Score: Grading of acute poisoning. J Toxicology - Clinical Toxicology (1998) 36:205-13.

43. V.Palaniyappen .Current Concepts in the Management of Organophosphorus Compound Poisoning .http://apiindia.org/medicine-update-2013/chap 95

44. Afzali ,S.,2002.The comparison of atropine & atropine + oxime in organophosphate poisoning with muscarinic signs.Sci. J.Hamadan Univ. Med.Sci.Health Serv., 9: 37 -40.

45. Narang U, Narang P, Gupta O. Organophosphorus Poisoning: A social calamity .J Mahatma Gandhi Inst Med Sci 2015;20: 46-51

46. Banerjee, I., S.K.Tripathi and A.S.Roy , 2011. A study on comparative evaluation of add- on pralidoxime therapy over atropine in the management of organophosphorus poisoning in a tertiary care hospital. JK Sci ., 13: 65-69.

47. Buckley NA ,Eddleston M, Li Y ,et al. Oximes for acute organophosphate pesticide poisoning .Cochrane Database System Rev 2011; CD005085.

48. Johnson MK, Jacobsen D, Meredith TJ, Eyer P, Heath AJ, Ligtenstein DA, et al. Evaluation of antidotes for poisoning by organophosphorus pesticides. Emergency Medicine. 2000;12(1):22-37.

49. Taylor P. Anticholinesterase agents In: Gilman AG, Goodman LS, Rall TW, Murad F eds. The pharmacological basis of therapeutics. New York: Mac Millan 1985; 110-129.

50. Johnson DD, Wibcox CW. Studies on the mechanism of protective and antidote actions of diazepam in Organophosphorous poisoning. European journal of pharmacology 1975; 34: 127-132.

51. Liu WF. A symptomatological assessment of organophosphate induced lethality in mice: comparison of atropine and clonidine protection. Toxicol Lett 1991; 56:19-32.

52. Wong A, Sandron CA, MagalhaesAS,Rocha LC. Comparative efficacy of pralidoxime Vs sodium bicarbonate in rats and humans severely poisoned with OP pesticides. J ToxicolClinToxicol 2000; 38:554-5.

53. Balali-Mood M, Ayati MH, Ali-Akbarian H. Effects of high doses of sodium bicarbonate in acute organophosphate pesticide poisoning. ClinToxicol 2005; 43:571-4.

54. Stanworth SJ, Brunskill SJ, Hyde CJ, McClelland DB, Murphy MF. Is fresh frozen plasma clinically effective? A systematic review of randomized clinical trials. Br J Haematol 2004; 126:139-52.

55. Virendra K Goyal, K K Pareek, Medicine update 2019, Volume 29 ,Chapter 222, Organophosphorus Poisoning : Current Management Guidelines. Page 1107-1110.

56. Reddy N, Murty O. The Essentials of forensic medicine and Toxicology .26th Edition, 2007.

57. Peter JV, Thomas L, Graham PL, Moran JL, Abhilash KP, Jasmine S, Iyyadurai R. Performance of clinical scoring systems in acute organophosphate poisoning. Clinical toxicology. 2013;51:850-4.

58. M.Vishwanathan, K srinivasan. Poisoning by bug poison. A preliminary study. Journal of Indian Medical Association 1962, vol.39; No. 7: 345-349

59. Robert J, Zwiener, Charles M Ginsburg. Organophosphorous and carbamate poisoning in infants and children. Pediatrics 1988; 81:121-126.

60. Nouira S, Abroug F, Elatrous S, Boujdaria R, Bouchoucha S. Prognostic value of serum cholinesterase in organophosphate poisoning. CHEST Journal. 1994;106(6):1811-4.

61. Raveendra, et al. Utility of POP Scale, PSS and GCS in Predicting Severity And treatment outcome in acute organophosphorous poisoning. International Journal of Contemporary Medical Research 2020;7(2):B20-B24.

62. Davies JO, Eddleston M, Buckley NA. Predicting outcome in acute organophosphorus poisoning with a poison severity score or the Glasgow coma scale. QJM: An International Journal of Medicine. 2008;101:371-9.

63. Chandrasekhar S, Rahim MA, Quraishi SM, Theja CR, Kiran KS. An observational clinical study of assessing the utility of PSS (Poison Severity Score) and GCS (Glasgow Coma Scale) scoring systems in predicting severity and clinical outcomes in op poisoning. J Evidence Based Med Healthcare. 2017;4:2325-32.

64. Tripathi S. Prognostic value of glasgow coma scale, poisoning severity score and serum acetylcholinesterase levels in organophosphorus poisoning. Journal of Evolution of Medical and Dental Sciences. 2014;3:3415- 22.

PROFORMA

A STUDY TO ASSESS THE UTILITY OF PERADENIYA ORGANOPHOSPHORUS POISONING (POP) SCALE, POISONING SEVERITY SCORE (PSS) AND GLASGOW COMA SCALE (GCS) IN PREDICTING SEVERITY AND TREATMENT OUTCOME IN ACUTE ORGANOPHOSPHORUS POISONING PATIENTS ADMITTED IN A TERTIARY CARE CENTRE

Name: IP No:

Date of admission:

Age/ Sex:
Occupation:
Address:
Final Diagnosis:

History of presenting complaints:

- Time and date of consumption of organophosphorus compound poison:
- Compound Name :
- Time elapsed after consuming the compound:
- Amount of compound consumed:
- Difficulty in breathing:
- Excessive sweating:
- Excessive salivation:
- Vomiting /Loose stools:
- Excessive lacrimation:
- Seizures:
- Loss of consciousness:
- Consumed alcohol:

Past history:

- Any comorbids
- Drug intake. Yes/no

Personal history:

- Alcoholic. : Yes /No
- Smoker :Yes /No

- General Physical examination:
- Appearance
- Built: well/mod/poor
- Nourishment:
- Wasting
- Pallor
- Cyanosis
- Clubbing
- Pedal edema
- Lymphadenopathy
- Icterus

Vitals:

- Blood pressure:
- Pulse:
- R.R :
- Temperature:
- Breath smell:
- Tongue:
- Skin:

Systemic examination:

C.N.S :

- Sensorium :Normal/Restlessness/Drowsyness/Coma
- GCS : VEM=
- Pupils:
- Fasciculations:
- Weakness : proximal muscle/neck muscle
- Respiratory muscle : Single breath count
  Accessory muscles

P.A :

R.S :

C.V.S:

Peradeniya organonphosphorus poisoning (POP) scale

SL NO	PARAMETER		SCORE	PATIENT SCORE
1	MIOSIS	Pupil size >2mm	0	
		Pupil size ≤2mm	1	
		Pupils pin point	2	
2	FASCICULATIONS	None	0	
		Present but not general-ized or continuous	1	
		Generalized and continu-ous	2	
3	RESPIRATION	Respiratory rate ≤20/min	0	
		Respiratory rate >20/min	1	
		Respiratory rate >20/min with central cyanosis	2	
4	BRADYCARDIA	Pulse rate >60/min	0	
		Pulse rate 41-60/min	1	
		Pulse rate ≤40/min	2	
5	LEVEL OF CON-SCIOUSNESS	Conscious and rational	0	
		Impaired, responds to verbal commands	1	
		Impaired, no response to verbal commands	2	
6	CONVULSION	present	1	
	Total Score		11	

INVESTIGATIONS:

COMPLETE BLOOD COUNT:

TOTAL COUNT	
DIFFERENTIAL COUNT	
HEMOGLOBIN	
PLATELET	

RLE:

RANDOM BLOOD SUGAR	
UREA	
CREATININE	
TOTAL BILIRUBIN	
DIRECT BILIRUBIN	
SGOT	
SGPT	
Na/K	
CPK	
LDH	
URIC ACID	

SERUM CHOLINESTERASE LEVELS:

ARTERIAL BLOOD GAS ANALYSIS:

pH	
pO_2	
pCO_2	
HCO_3	

URINE ROUTINE:

ALBUMIN:

SUGAR:

RBCs:

PUS CELLS:

ECG FINDINGS:

CHEST XRAY FINDINGS:

POISONING SEVERITY SCORE:

Management:

- Body wash:
- Stomach wash:
- Atropine:
- P2AM:
- Others:
- Intubation hours after consumption of OPC Poisoning
- Ventilation:
- Those who developed intermediate syndrome:
- IMCU STAYS :
- OUTCOME : RECOVERED/EXPIRED

PATIENT CONSENT FORM

நோயாளி ஒப்புதல் படிவம்

ஆய்வு விவரம் : **A STUDY TO ASSESS THE UTILITY OF PERADENIYA ORGANOPHOSPHORUS POISONING (POP) SCALE, POISONING SEVERITY SCORE (PSS) AND GLASGOW COMA SCALE (GCS) IN PREDICTING SEVERITY AND TREATMENT OUTCOME IN ACUTE ORGANOPHOSPHORUS POISONING PATIENTS ADMITTED IN A TERTIARY CARE CENTRE**

ஆய்வு மையம்: ராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை.

நோயாளியின் பெயர்:

நோயாளியின் வயது:

அடையாள எண் :

நோயாளி இந்த பெட்டிகளை (√) செய்யலாம்:-

- மேற்கண்ட ஆய்விற்கான நடைமுறையின் நோக்கத்தை நான் புரிந்து கொண்டேன் என்பதை உறுதிப்படுத்துகிறேன். கேள்வி கேட்க எனக்கு வாய்ப்பு உள்ளது, எனது முழு திருப்திக்கும் எனது எல்லா கேள்விகளுக்கும் சந்தேகங்களுக்கும் பதில் அளிக்கப்பட்டுள்ளது.
- ஆய்வில் நான் பங்கேற்பது தன்னார்வமானது என்பதையும், எனது சட்ட உரிமைகள் பாதிக்கப்படாமல், காரணமின்றி எந்த நேரத்திலும் திரும்பப் பெற எனக்கு சுதந்திரம் உள்ளது என்பதையும் நான் புரிந்துகொள்கிறேன்.
- மருத்துவ ஆய்வின் ஸ்பான்சர், ஸ்பான்சர் சார்பாக பணிபுரியும் மற்றவர்கள், நெறிமுறைக் குழு மற்றும் ஒழுங்குமுறை அதிகாரிகள் எனது சுகாதார பதிவுகளைப் பார்க்க எனது அனுமதி தேவையில்லை என்பதை நான் புரிந்துகொள்கிறேன்,

தற்போதைய ஆய்வு மற்றும் மேற்கொண்டுள்ள எந்தவொரு ஆராய்ச்சியையும் பொறுத்தவரை இது தொடர்பாக, நான் ஆய்வில் இருந்து விலகினாலும் இந்த அணுகலை ஒப்புக்கொள்கிறேன். எவ்வாறாயினும், சட்டத்தின் கீழ் தேவைப்படாவிட்டால், மூன்றாம் தரப்பினருக்கு வெளியிடப்பட்ட அல்லது வெளியிடப்பட்ட எந்தவொரு தகவலிலும் எனது அடையாளம் வெளிப்படுத்தப்படாது என்பதை நான் புரிந்துகொள்கிறேன். இந்த ஆய்வில் எழும் எந்தவொரு தரவு அல்லது முடிவுகளின் பயன்பாட்டை கட்டுப்படுத்த வேண்டாம் என்று நான் ஒப்புக்கொள்கிறேன்.

- மேற்கண்ட ஆய்வில் பங்கேற்கவும், ஆய்வின் போது கொடுக்கப்பட்ட அறிவுறுத்தல்களுக்கு இணங்கவும், ஆய்வுக் குழுவுடன் உண்மையுடன் ஒத்துழைக்கவும், எனது உடல்நலம் அல்லது நல்வாழ்வில் ஏதேனும் சரிவு ஏற்பட்டால் அல்லது எதிர்பாராத அல்லது ஏதேனும் ஏற்பட்டால் உடனடியாக ஆய்வு ஊழியர்களுக்கு அறிவிக்கவும் ஒப்புக்கொள்கிறேன். அசாதாரண அறிகுறிகள்.
- இந்த ஆய்வில் பங்கேற்க நான் இதன்மூலம் ஒப்புக்கொள்கிறேன்.
- தேவைக்கேற்ப விரிவான மருத்துவ பரிசோதனை மற்றும் இரத்த விசாரணைகளை மேற்கொள்ள நான் இதன்மூலம் அனுமதி அளிக்கிறேன்.

புலனாய்வாளரின் கையொப்பம் பங்கேற்பாளரின் கையொப்பம் / கட்டைவிரல் எண்ணம்

நோயாளியின் பெயர் மற்றும் முகவரி

MASTER CHART

S.NO	Name	Age	Sex	Occupation	Compound	Lag time for admission after OPC POISONING(Hrs)	Amount consumed(ml)	GCS(3-15)	Pulse rate(per min)	Respiratory rate(per min)	SBP(mmhg)	DBP(mmhg)	SpO2(%)	Breath smell	Sensorium	Pupils(mm)	Fasciculations	Seizure	Creatinine(mg/dl)	TB(mg/dl)	SGOT(mg/dl)	SGPT(mg/dl)	Na+(mEq/L)	K+(mEq/L)	CPK(IU/L)	LDH(IU/L)	AchE(U/L)	POP scale(0-11)	POISONING SEVERITY SCORE(0-4)	Atropine requirement(amp)	Intubation hours after consumption of	Mechanical Ventilation	IMS	IMCU stay(Days)	Outcome
1	Murali	22	Male	Student	Phorate	3	20	15	76	18	110	70	99	Normal	Normal	3	No	No	0.9	1.0	25	17	143	3.6	36	96	6335	0	1	2	Nil	No	No	2	Recovered
2	Sudha	18	Female	Student	Chlorpyrifos	2	15	15	80	18	100	60	98	Normal	Normal	3	No	No	1.0	0.9	16	17	137	3.9	42	86	9325	0	1	2	Nil	No	No	1	Recovered
3	Muniyammal	46	Female	Housewife	Chlorpyriphos	6	130	11	65	22	90	60	96	Kerosene	Drowsiness	Pinpoint	No	No	1	1.2	43	39	136	4.1	56	106	3150	3	2	8	Nil	No	No	6	Recovered
4	Malar	27	Female	Housewife	Triazophos	3	25	15	82	16	100	70	98	Normal	Normal	3	No	No	0.8	1.0	36	24	139	3.7	26	78	8060	0	0	2	Nil	No	No	1	Recovered
5	Sundar	28	Male	IT staff	Chlorpyrifos	4	120	10	56	26	100	60	96	Kerosene	Drowsiness	1	No	No	1.1	1.3	46	47	132	3.5	13	75	4570	4	2	24	4	Yes	No	8	Recovered
6	Susila	38	Female	Housewife	Monochrotophos	8	150	7	62	26	90	50	94	Kerosene	Drowsiness	Pinpoint	Yes	Yes	1.5	1.2	65	56	136	3.6	64	105	2300	8	3	48	8	Yes	No	10	Expired
7	Bala	13	Male	Student	Phorate	2	15	15	96	16	100	70	99	Normal	Normal	3	No	No	0.8	1	32	28	140	3.7	16	28	11300	0	0	1	Nil	No	No	1	Recovered
8	Mohan	40	Male	Tailor	Monochrotophos	7	140	7	56	28	90	50	90	Kerosene	Drowsiness	Pinpoint	Yes	Yes	1.7	1.6	52	49	136	3.3	59	104	4269	6	3	52	8	Yes	Yes	9	Expired
9	Valli	26	Female	Student	Parathion	5	80	13	58	22	90	60	97	Kerosene	Drowsiness	1	No	No	0.6	0.9	40	36	138	4.2	42	110	3650	4	2	32	6	Yes	No	8	Expired
10	Palani	52	Male	Farmer	Chlorpyrifos	4	180	7	63	28	90	50	99	Kerosene	Drowsiness	Pinpoint	Yes	Yes	0.6	0.7	36	27	134	3.5	56	86	2370	5	2	46	4	Yes	No	10	Recovered
11	Muniraj	36	Male	Farmer	Parathion	7	70	15	88	18	110	80	98	Normal	Normal	3	No	No	0.7	1	39	27	139	3.7	26	103	8360	0	1	6	Nil	No	No	2	Recovered
12	Suganya	24	Female	Student	Phorate	6	30	15	92	16	100	70	96	Normal	Normal	4	No	No	0.6	1.1	40	36	142	4.1	39	89	7800	0	1	4	Nil	No	No	2	Recovered
13	Murugan	45	Male	Plumber	Chlorpyrifos	4	150	10	62	22	90	60	99	Kerosene	Drowsiness	Pinpoint	Yes	No	1.3	1.6	52	47	134	3.9	35	92	4150	4	2	44	5	Yes	No	12	Recovered
14	Munusamy	56	Male	Farmer	Chlorpyrifos	8	50	15	96	18	130	90	98	Kerosene	Normal	3	No	No	2.1	1.0	23	17	140	4.7	36	113	9776	0	1	6	Nil	No	No	3	Recovered
15	Kalavathy	18	Female	Student	Phorate	3	15	15	86	18	100	80	99	Normal	Normal	4	No	No	0.6	0.9	23	22	138	3.5	25	117	11236	0	0	2	Nil	No	No	1	Recovered
16	Mastan	24	Male	Student	Phorate	6	20	15	87	18	100	80	99	Normal	Normal	4	No	No	0.8	1	34	31	137	3.6	24	122	4780	0	0	2	Nil	No	No	1	Recovered
17	Mani	27	Male	IT staff	Monochrotophos	3	160	7	56	28	80	50	90	Kerosene	Drowsiness	Pinpoint	Yes	Yes	1.6	2.0	56	51	136	3.4	49	112	2487	8	3	54	5	Yes	Yes	14	Recovered
18	Kavitha	26	Female	Student	Fenthion	8	120	15	58	18	100	80	99	Kerosene	Normal	3	No	No	0.7	1.2	23	21	145	3.9	34	132	2659	1	3	26	12	Yes	No	11	Recovered
19	Malliga	36	Female	Housewife	Chlorpyriphos	4	60	15	78	16	100	70	98	Normal	Normal	2	No	No	0.6	0.9	24	22	137	3.6	43	121	4780	1	1	6	Nil	No	No	3	Recovered
20	Malathi	40	Female	Teacher	Parathion	6	130	13	62	24	90	70	99	Kerosene	Drowsiness	1	No	No	2.0	1.7	54	49	133	3.4	56	155	2300	3	2	56	12	Yes	No	14	Recovered
21	Karthik	31	Male	Driver	Fenthion	8	100	15	62	16	100	70	99	Kerosene	Normal	2	No	No	0.5	1.0	21	17	142	4.5	23	102	23100	1	2	10	12	Yes	No	14	Recovered
22	Alibaba	40	Male	Salesman	Chlorpyrifos	7	90	15	76	18	110	80	98	Normal	Normal	2	No	No	1.3	1.1	21	17	136	4.3	25	123	2173	1	2	26	Nil	No	No	4	Recovered
23	Sudhakar	17	Male	Student	Parathion	4	15	15	82	18	110	80	97	Normal	Normal	4	No	No	0.9	1.2	25	28	136	3.7	26	124	2610	0	1	4	Nil	No	No	1	Recovered
24	Madura	29	Female	Housewife	Monochrotophos	7	125	15	58	22	100	60	96	Kerosene	Normal	2	No	No	0.8	0.6	34	30	136	4.6	53	116	2063	3	2	48	9	Yes	No	8	Recovered
25	Nandhakumar	54	Male	Farmer	Parathion	6	180	11	106	26	90	60	91	Kerosene	Drowsiness	Pinpoint	Yes	No	1.1	1.2	52	49	134	4.1	48	132	2163	5	2	56	8	Yes	Yes	14	Recovered
26	Malarvizhi	21	Female	Student	Phorate	5	30	15	96	16	100	70	96	Normal	Normal	4	No	No	0.9	0.9	31	30	150	3.6	13	96	6890	0	1	6	Nil	No	No	2	Recovered
27	Rudra	36	Female	Housewife	Chlorpyrifos	5	175	8	110	28	110	60	98	Kerosene	Drowsiness	Pinpoint	Yes	No	1.4	1.2	56	48	135	4.1	86	136	1960	5	3	60	12	Yes	Yes	15	Expired
28	Manikkam	18	Male	Student	Parathion	5	150	10	58	24	100	60	99	Kerosene	Restlessness	1	No	No	1.0	1.7	32	36	140	4	68	150	2050	4	2	48	6	Yes	No	7	Recovered
29	Sangavi	28	Female	Housewife	Monochrotophos	7	120	5	117	30	90	60	90	Kerosene	Drowsiness	Pinpoint	Yes	Yes	0.6	1	25	35	135	4.9	36	96	1600	7	3	56	8	Yes	Yes	15	Recovered
30	Arjunan	50	Male	Farmer	Chlorpyrifos	6	80	15	78	18	120	70	98	Normal	Normal	3	No	No	0.7	0.5	45	21	145	3.7	35	136	2450	0	1	2	Nil	No	No	2	Recovered
31	Malini	32	Female	Housewife	Parathion	5	30	15	78	16	100	80	96	Normal	Normal	3	No	No	0.9	0.7	31	26	142	4.1	37	143	4860	0	1	4	Nil	No	No	2	Recovered
32	Sunil	37	Male	Farmer	Malathion	2	20	15	87	16	100	70	96	Normal	Normal	4	No	No	0.9	0.7	21	17	135	4.1	26	98	5688	0	0	2	Nil	No	No	1	Recovered
33	Arokiyasamy	48	Male	Farmer	Dicrotophos	8	150	9	58	28	90	60	96	Kerosene	Drowsiness	1	No	No	1.1	1.2	42	41	143	3.6	26	133	1859	5	2	44	9	Yes	No	10	Recovered
34	Surya	25	Male	Student	Dimethoate	4	50	15	96	18	120	80	97	Normal	Normal	3	No	No	0.7	0.6	16	21	143	3.5	23	146	7800	0	1	4	Nil	No	No	2	Recovered
35	Rani	38	Female	Housewife	Phorate	6	150	10	54	26	100	60	96	Kerosene	Drowsiness	Pinpoint	Yes	No	0.8	0.9	46	43	139	3.7	36	97	2350	4	2	54	7	Yes	No	8	Recovered
36	Preethi	22	Female	Student	Triazophos	2	20	15	78	16	110	80	98	Normal	Normal	3	No	No	0.5	0.6	34	36	141	3.9	26	117	5200	0	0	2	Nil	No	No	1	Recovered
37	Sulochana	32	Female	Housewife	Chlorpyrifos	7	130	3	42	32	80	50	90	Kerosene	Coma	Pinpoint	Yes	Yes	1.6	1.2	56	51	132	3.5	56	132	1600	7	3	58	8	Yes	Yes	16	Expired
38	Sabari	16	Male	Student	Malathion	4	10	15	96	16	100	70	98	Normal	Normal	4	No	No	0.6	0.9	21	19	136	3.9	23	140	11356	0	0	2	Nil	No	No	1	Recovered
39	Ramesh	19	Male	Student	Parathion	3	10	15	68	16	120	70	99	Normal	Normal	3	No	No	1.0	1.1	31	26	142	3.6	32	153	4963	0	0	2	Nil	No	No	1	Recovered
40	Ashwini	26	Female	Housewife	Phorate	4	30	15	78	16	110	80	99	Normal	Normal	3	No	No	0.6	0.9	31	19	139	3.7	56	186	5180	0	1	4	Nil	No	No	2	Recovered
41	Soniya	46	Female	Housewife	Monochrotophos	6	130	9	62	32	90	60	96	Kerosene	Drowsiness	1	No	No	1.3	1.1	56	51	142	3.9	56	196	2100	3	3	56	6	Yes	No	10	Recovered
42	Saravanan	45	Male	Farmer	Chlorpyrifos	2	25	15	101	16	130	80	96	Normal	Normal	4	No	No	1.1	0.6	32	19	142	3.9	32	165	5960	0	1	2	Nil	No	No	2	Recovered
43	Nallusamy	56	Male	Farmer	Malathion	3	50	15	96	16	140	80	96	Normal	Normal	4	No	No	0.5	0.6	36	35	141	4.3	65	175	4450	0	1	2	Nil	No	No	2	Recovered
44	Ashif ali	19	Male	Student	Phorate	2	10	15	86	18	100	80	99	Normal	Normal	4	No	No	1.1	0.7	21	17	139	3.6	23	65	4566	0	0	2	Nil	No	No	1	Recovered
45	Pooja shree	22	Female	Student	Malathion	2	25	15	73	18	100	80	99	Normal	Normal	3	No	No	1.1	0.9	40	41	145	3.9	37	153	9800	0	1	2	Nil	No	No	1	Recovered
46	Poongodi	34	Female	Housewife	Monochrotophos	6	Not known	12	65	26	100	70	99	Kerosene	Drowsiness	1	No	No	0.9	1.1	32	19	135	3.7	65	137	2350	3	2	48	6	Yes	No	7	Recovered
47	Ilayaraja	30	Male	Driver	Malathion	3	20	15	86	16	90	60	98	Normal	Normal	3	No	No	0.9	0.6	35	36	137	3.6	24	98	13060	0	1	2	Nil	No	No	1	Recovered
48	Pushpa	16	Female	Student	Phorate	2	30	15	86	16	100	70	96	Normal	Normal	3	No	No	1.0	0.9	32	31	143	3.6	27	183	6870	0	1	4	Nil	No	No	2	Recovered

S.NO	Name	Age	Sex	Occupation	Compound	Lag time for admission after OPC POISONING(Hrs)	Amount consumed(ml)	GCS(3-15)	Pulse rate(per min)	Respiratory rate(per min)	SBP(mmhg)	DBP(mmhg)	SpO2(%)	Breath smell	Sensorium	Pupils(mm)	Fasciculations	Seizure	Creatinine(mg/dl)	TB(mg/dl)	SGOT(mg/dl)	SGPT(mg/dl)	Na+(mEq/L)	K+(mEq/L)	CPK(IU/L)	LDH(IU/L)	AchE(U/L)	POP scale(0-11)	POISONING SEVERITY SCORE(0-4)	Atropine requirement(amp)	Intubation hours after consumption of	Mechanical Ventilation	IMS	IMCU stay(Days)	Outcome
49	Saroja	40	Female	Tailor	Parathion	6	Not known	5	112	34	90	60	90	Kerosene	Coma	Pinpoint	Yes	No	0.6	0.9	53	51	134	3.3	46	96	1930	5	3	60	6	Yes	Yes	9	Expired
50	Pandiyan	31	Male	Farmer	Malathion	4	50	15	96	16	100	60	96	Normal	Normal	4	No	No	0.7	0.6	39	36	135	3.5	36	89	10632	0	1	2	Nil	No	No	1	Recovered
51	Sarathy	32	Male	Carpenter	Temephos	4	Not known	15	92	18	110	60	96	Normal	Normal	4	No	No	1	0.7	32	36	142	3.9	36	156	5869	0	0	2	Nil	No	No	1	Recovered
52	Senthil kumar	24	Male	Student	Dimethoate	4	70	15	58	20	100	80	96	Kerosene	Normal	1	No	No	0.6	0.8	21	23	136	3.6	56	185	4688	2	1	12	Nil	No	No	3	Recovered
53	Kumaresan	45	Male	Farmer	Monochrotophos	6	Not known	11	56	26	100	60	94	Kerosene	Drowsiness	Pinpoint	Yes	No	0.5	1.1	42	21	142	3.8	25	198	2405	5	2	48	8	Yes	Yes	14	Recovered
54	Pavithra	22	Female	Student	Temephos	2	25	15	92	16	120	70	98	Normal	Normal	4	No	No	0.6	0.9	31	32	138	3.8	25	136	6800	0	0	2	Nil	No	No	1	Recovered
55	Deva	56	Male	Farmer	Phorate	6	160	3	48	36	80	50	86	Kerosene	Coma	Pinpoint	Yes	Yes	0.6	0.9	65	53	136	3.9	56	198	2150	8	3	64	6	Yes	Yes	10	Expired
56	Ashok	19	Male	Student	Temephos	3	45	15	90	14	100	60	98	Normal	Normal	3	No	No	1	0.5	32	25	135	3.6	23	65	7800	0	0	2	Nil	No	No	1	Recovered
57	Adithyan	34	Male	Hotel manager	Dichlorphos	3	50	15	68	16	110	60	98	Normal	Normal	3	No	No	0.4	0.6	36	31	142	3.6	35	165	6320	0	1	8	Nil	No	No	2	Recovered
58	Suresh	43	Male	Farmer	Parathion	4	Not known	5	46	32	90	60	98	Kerosene	Drowsiness	Pinpoint	Yes	Yes	0.9	1.1	36	29	136	3.7	65	136	1750	8	3	62	4	Yes	No	8	Recovered
59	Vijay	21	Male	Student	Malathion	2	40	15	96	16	100	70	96	Normal	Normal	3	No	No	1.1	0.6	39	35	136	3.8	26	94	4680	0	0	2	Nil	No	No	1	Recovered
60	Santhosh	38	Male	IT staff	Monochrotophos	5	125	10	56	26	90	60	99	Kerosene	Drowsiness	Pinpoint	Yes	No	0.8	0.6	37	31	143	3.8	13	45	2416	5	2	36	5	Yes	No	8	Recovered
61	Murali	17	Male	Student	Malathion	2	15	15	92	18	100	80	99	Normal	Normal	3	No	No	1.1	0.6	21	23	142	3.6	32	25	6530	0	0	2	Nil	No	No	1	Recovered
62	Gowtham	25	Male	Student	Temephos	3	30	15	86	18	110	60	99	Normal	Normal	3	No	No	1	1.1	32	30	140	4	31	86	2416	0	1	2	Nil	No	No	2	Recovered
63	Jagan	23	Male	Student	Monochrotophos	5	100	10	42	32	90	60	98	Kerosene	Drowsiness	2	Yes	No	0.6	0.6	31	30	140	3.6	53	124	2563	4	2	24	5	Yes	No	9	Recovered
64	Palani	56	Male	Farmer	Chlorpyrifos	6	150	9	63	24	90	60	99	Kerosene	Restlessness	Pinpoint	Yes	No	0.8	0.8	31	24	136	4.1	31	26	1680	4	3	44	5	Yes	Yes	10	Recovered
65	Abinaya	16	Female	Student	Malathion	3	20	15	86	16	90	60	99	Normal	Normal	4	No	No	0.8	0.7	36	39	138	3.9	56	89	4682	0	1	4	Nil	No	No	2	Recovered
66	Ponnusamy	54	Male	Farmer	Chlorpyrifos	4	100	9	63	26	100	60	98	Kerosene	Drowsiness	2	Yes	No	0.6	0.9	25	21	134	3.6	36	94	3960	4	2	24	Nil	No	No	7	Recovered
67	Vennila	36	Female	Housewife	Parathion	3	50	15	92	16	100	70	96	Normal	Normal	3	No	No	0.6	0.9	21	18	139	3.7	34	155	4100	0	1	6	Nil	No	No	2	Recovered
68	Sugash	21	Male	Student	Chlorpyrifos	6	150	10	58	32	100	60	99	Kerosene	Drowsiness	Pinpoint	Yes	No	0.6	0.7	32	31	141	4.3	36	163	3180	6	2	48	7	Yes	No	8	Recovered
69	Vishnu	28	Male	Student	Malathion	2	15	15	102	18	110	80	96	Normal	Normal	4	No	No	0.6	0.9	36	31	136	3.7	39	149	5680	0	1	4	Nil	No	No	1	Recovered
70	Meenatchi	37	Female	Bank manager	Parathion	7	Not known	9	48	28	90	60	96	Kerosene	Drowsiness	1	Yes	No	1	0.6	32	30	143	4.3	26	121	1960	8	2	44	7	Yes	Yes	12	Recovered
71	Mageshwari	40	Female	Farmer	Monochrotophos	8	Not known	3	42	34	80	50	91	Kerosene	Coma	Pinpoint	Yes	Yes	0.6	0.6	36	41	145	4.5	32	163	1650	8	3	64	8	Yes	Yes	14	Expired
72	Manogaram	56	Female	Housewife	Malathion	5	30	15	96	18	100	70	98	Normal	Normal	3	No	No	0.8	1.1	31	20	142	3.6	34	99	6700	0	1	4	Nil	No	No	2	Recovered
73	Sakthivel	24	Male	Student	Malathion	3	50	15	72	18	100	70	99	Normal	Normal	4	No	No	0.6	0.9	21	23	145	3.9	23	96	6945	0	1	4	Nil	No	No	2	Recovered
74	Ravi	27	Male	Student	Phorate	3	30	15	78	16	100	60	99	Normal	Normal	3	No	No	0.7	0.9	21	17	136	3.9	31	93	6398	0	1	4	Nil	No	No	1	Recovered
75	Monisha	32	Male	IT staff	Monochrotophos	3	50	15	86	16	100	70	99	Normal	Normal	4	No	No	1.1	0.6	31	21	145	3.6	21	163	7230	0	1	4	Nil	No	No	1	Recovered
76	Vatsala	46	Female	Housewife	Phorate	7	125	9	48	22	90	60	96	Kerosene	Drowsiness	Pinpoint	Yes	Yes	0.6	0.7	31	25	136	3.6	32	114	1750	8	2	44	8	Yes	No	10	Recovered
77	Sujatha	31	Female	Housewife	Chlorpyrifos	4	Not known	15	92	16	100	80	99	Normal	Normal	3	No	No	0.5	0.6	12	25	139	3.8	36	89	2486	0	1	4	Nil	No	No	2	Recovered
78	Manisha	23	Female	Student	Parathion	4	20	15	92	18	120	70	99	Normal	Normal	4	No	No	0.9	0.8	32	31	136	4.1	21	107	5630	0	1	4	Nil	No	No	2	Recovered
79	Jaisankar	19	Male	Student	Malathion	2	15	15	101	16	110	80	99	Normal	Normal	3	No	No	0.6	0.9	0.4	31	142	3.8	36	155	5680	0	1	4	Nil	No	No	1	Recovered
80	Manimegalai	22	Female	Student	Parathion	2	40	15	86	16	120	70	99	Normal	Normal	4	No	No	21	0.9	27	18	136	4.1	36	125	7860	0	1	2	Nil	No	No	1	Recovered
81	Kalai	31	Male	Farmer	Monochrotophos	2	30	15	78	16	100	70	96	Normal	Normal	4	No	No	1.1	1.2	24	23	142	3.9	32	136	10632	0	1	6	Nil	No	No	2	Recovered
82	Akash	39	Male	Farmer	Chlorpyrifos	5	150	10	62	26	100	70	99	Kerosene	Drowsiness	Pinpoint	Yes	Yes	0.6	0.9	31	21	146	3.9	56	173	1460	6	3	40	6	Yes	Yes	7	Recovered
83	Prakash	37	Male	Farmer	Malathion	2	30	15	92	16	110	70	99	Normal	Normal	4	No	No	1.0	0.9	13	12	136	4.1	36	65	7900	0	1	2	Nil	No	No	2	Recovered
84	Moorthy	27	Male	Student	Monochrotophos	4	20	15	78	16	100	70	99	Normal	Normal	4	No	No	0.9	0.7	31	24	142	3.6	26	155	11693	0	1	4	Nil	No	No	2	Recovered
85	Ilayaraja	36	Male	Farmer	Fenthion	6	125	12	66	18	100	70	96	Kerosene	Normal	2	No	No	0.6	1.0	32	24	142	3.8	26	162	2100	2	2	20	12	Yes	No	9	Recovered
86	Prabha	21	Female	Student	Temephos	2	15	15	96	16	110	70	98	Normal	Normal	4	No	No	0.9	0.6	35	42	134	3.7	35	96	10634	0	1	4	Nil	No	No	1	Recovered
87	Arun	21	Male	Student	Quinolphos	2	25	15	90	14	100	80	96	Normal	Normal	3	No	No	0.9	0.5	31	30	135	4.1	36	97	8963	0	1	2	Nil	No	No	1	Recovered
88	Varun	42	Male	Farmer	Unknown	5	Unknown	9	56	22	90	60	98	Kerosene	Drowsiness	Pinpoint	Yes	No	0.4	0.6	31	27	136	4.3	25	163	1960	6	2	36	6	No	No	6	Recovered
89	Murugan	48	Male	Farmer	Phorate	4	150	15	96	18	100	70	96	Kerosene	Normal	2	No	No	0.6	1.0	21	17	139	3.6	32	165	2160	2	1	8	72	Yes	Yes	10	Recovered
90	Sankar	19	Male	Student	Quinolphos	4	20	15	98	20	100	60	96	Normal	Normal	3	No	No	1	1	39	35	136	4.3	34	189	3648	2	1	4	Nil	No	No	2	Recovered
91	Geetha	39	Female	Housewife	Chlorpyrifos	6	120	9	63	26	100	60	96	Kerosene	Drowsiness	2	Yes	No	1.2	1.3	56	52	145	3.9	66	198	1963	3	2	30	Nil	No	No	8	Recovered
92	Shalini	22	Female	Student	Chlorpyrifos	3	30	15	96	18	100	70	98	Normal	Normal	3	No	No	0.6	0.9	31	19	136	3.9	34	163	8796	0	1	4	Nil	No	No	2	Recovered
93	Varuna	31	Female	Housewife	Monochrotophos	4	40	15	89	16	100	80	96	Normal	Normal	3	No	No	0.7	0.9	20	18	142	3.7	35	196	4568	0	1	4	Nil	No	No	2	Recovered
94	Shanthi	40	Female	Farmer	Parathion	5	125	15	68	20	100	60	99	Kerosene	Normal	2	No	No	0.9	1	16	9	136	4.6	31	185	1961	2	1	26	56	Yes	Yes	12	Recovered
95	Kalpana	21	Female	Student	Malathion	3	60	15	92	16	110	70	98	Normal	Normal	3	No	No	1.1	0.7	32	27	143	4.8	23	46	3645	0	1	4	Nil	No	No	1	Recovered

S.NO	Name	Age	Sex	Occupation	Compound	Lag time for admission after OPC POISONING(Hrs)	Amount consumed(ml)	GCS(3-15)	Pulse rate(per min)	Respiratory rate(per min)	SBP(mmhg)	DBP(mmhg)	SpO2(%)	Breath smell	Sensorium	Pupils(mm)	Fasciculations	Seizure	Creatinine(mg/dl)	TB(mg/dl)	SGOT(mg/dl)	SGPT(mg/dl)	Na+(mEq/L)	K+(mEq/L)	CPK(IU/L)	LDH(IU/L)	AchE(U/L)	POP scale(0-11)	POISONING SEVERITY SCORE(0-4)	Atropine requirement(amp)	Intubation hours after consumption of	Mechanical Ventilation	IMS	IMCU stay(Days)	Outcome
96	Valli	18	Female	Student	Phorate	4	25	15	86	18	100	70	98	Normal	Normal	4	No	No	0.6	0.9	21	17	132	3.9	21	45	6546	0	0	2	Nil	No	No	1	Recovered
97	Seetha	22	Female	Student	Unknown	5	100	13	56	22	100	70	99	Kerosene	Drowsiness	Pinpoint	Yes	Yes	0.4	0.9	31	36	136	3.9	34	89	1546	8	2	36	6	Yes	Yes	10	Recovered
98	Vasantha	24	Female	Student	Chlorpyrifos	4	20	15	78	16	120	80	96	Normal	Normal	3	No	No	1	0.6	36	32	140	4.2	32	145	4563	0	1	2	Nil	No	No	1	Recovered
99	Karpagam	39	Female	Housewife	Parathion	5	150	10	102	22	90	60	98	Kerosene	Drowsiness	1	Yes	No	0.9	1	36	17	136	4.9	54	189	2163	4	3	40	7	Yes	Yes	10	Recovered
100	Sushmitha	20	Female	Student	Malathion	2	25	15	101	18	100	70	98	Normal	Normal	4	No	No	0.5	1	32	37	145	3.8	12	36	5643	0	0	1	Nil	No	No	1	Recovered
101	Parimala	37	Female	Housewife	Triazophos	2	15	15	69	16	100	60	96	Normal	Normal	3	No	No	0.9	1	37	31	143	4.9	31	49	6484	0	0	1	Nil	No	No	1	Recovered
102	Prema	39	Female	Farmer	Unknown	4	150	8	60	28	90	60	98	Kerosene	Drowsiness	Pinpoint	Yes	Yes	0.6	1.1	14	12	146	4.3	36	98	2163	7	2	36	7	Yes	Yes	10	Recovered
103	Karmegam	19	Male	Student	Unknown	6	125	5	42	36	80	50	94	Kerosene	Coma	Pinpoint	Yes	Yes	0.6	1.6	56	51	149	4.9	36	196	1896	8	3	64	6	Yes	No	14	Expired
104	Sakthi	48	Male	Farmer	Monochrotophos	5	130	15	68	22	100	70	98	Kerosene	Restlessness	Pinpoint	Yes	No	1.1	0.8	38	27	138	3.5	26	78	1546	4	2	28	7	Yes	Yes	9	Recovered
105	Swathi	23	Female	Student	Chlorpyrifos	3	50	15	68	18	110	80	99	Kerosene	Normal	2	No	No	0.8	1	31	30	135	4.1	36	89	3645	1	1	4	Nil	No	No	4	Recovered
106	Kavitha	26	Female	Student	Malathion	4	25	15	76	20	110	70	99	Normal	Normal	4	No	No	1.1	0.6	21	17	143	4.1	36	98	5431	1	1	4	Nil	No	No	2	Recovered
107	Sathya	56	Male	Farmer	Unknown	6	Not known	11	106	22	110	80	99	Kerosene	Restlessness	Pinpoint	Yes	No	0.6	0.7	36	31	142	4.1	32	89	1894	5	2	28	Nil	No	No	6	Recovered
108	Deepika	21	Female	Student	Temephos	2	50	15	78	18	100	70	99	Normal	Normal	3	No	No	0.5	0.7	37	30	135	3.8	32	89	6581	0	1	2	Nil	No	No	2	Recovered
109	Lalitha	38	Female	House wife	Chlorpyrifos	4	120	11	86	22	90	60	99	Kerosene	Drowsiness	Pinpoint	Yes	No	0.9	0.6	21	19	139	3.9	31	156	2348	5	2	36	8	Yes	No	7	Recovered
110	Marichamy	28	Male	Farmer	Quinolphos	4	30	15	92	18	100	70	99	Normal	Normal	3	No	No	0.6	0.6	34	31	139	3.8	32	167	10643	0	1	6	Nil	No	No	2	Recovered
111	Monika	21	Female	Student	Monochrotophos	2	20	15	101	18	100	70	98	Normal	Normal	3	No	No	0.6	0.5	34	42	139	3.5	31	122	10643	0	1	4	Nil	No	No	2	Recovered
112	Sathish	24	Male	Student	Malathion	3	10	15	86	18	100	70	99	Normal	Normal	4	No	No	3.8	1.0	31	27	142	3.6	35	188	154613	0	1	2	Nil	No	No	1	Recovered
113	Kiruthika	31	Female	Housewife	Parathion	2	15	15	74	18	100	80	99	Normal	Normal	3	No	No	0.6	0.8	21	19	144	3.4	21	186	12453	0	1	2	Nil	No	No	2	Recovered
114	Magesh	22	Male	Student	Quinolphos	3	15	15	102	18	110	80	99	Normal	Normal	4	No	No	0.4	1	34	31	136	3.6	13	56	7839	0	1	2	Nil	No	No	1	Recovered
115	Madeshwari	52	Female	Farmer	Chlorpyrifos	6	120	11	56	24	90	60	99	Kerosene	Drowsiness	Pinpoint	Yes	No	1	1	32	21	142	3.6	23	155	1596	6	2	32	8	Yes	Yes	13	Recovered
116	Raja	47	Male	Farmer	Monochrotophos	5	125	12	68	24	110	80	99	Kerosene	Normal	Pinpoint	Yes	No	1.1	0.8	36	32	139	3.8	21	163	2194	4	2	20	7	Yes	No	6	Recovered
117	Kalaivani	27	Female	Student	Quinolphos	3	20	15	78	16	100	60	99	Normal	Normal	4	No	No	1	0.9	34	30	142	3.6	27	168	10643	0	1	4	Nil	No	No	2	Recovered
118	Jagan	19	Male	Student	Quinolphos	2	30	15	68	18	100	60	99	Normal	Normal	4	No	No	1	0.6	31	24	142	3.6	37	108	116434	0	1	2	Nil	No	No	1	Recovered
119	Tamilselvan	39	Male	Farmer	Chlorpyrifos	5	110	10	58	24	100	60	99	Kerosene	Drowsiness	Pinpoint	Yes	No	1.1	0.9	32	36	142	3.9	32	199	2169	5	2	36	6	Yes	No	10	Recovered
120	Kiran	21	Male	Student	Malathion	2	15	15	68	16	90	60	98	Normal	Normal	3	No	No	0.7	0.9	34	36	143	4.3	39	166	9843	0	1	4	Nil	No	No	2	Recovered
121	Moorthy	22	Male	Student	Malathion	4	10	15	62	16	100	70	99	Normal	Normal	3	No	No	1.0	0.9	36	31	143	4.4	31	156	14563	0	1	2	Nil	No	No	2	Recovered
122	Arjun	27	Male	Student	Malathion	2	15	15	78	16	110	60	99	Normal	Normal	3	No	No	0.9	1	36	31	142	3.6	31	163	9631	0	0	2	Nil	No	No	1	Recovered
123	Madhu	21	Female	Student	Chlorpyrifos	2	30	15	78	14	90	60	99	Normal	Normal	3	No	No	0.6	0.9	26	21	143	4.5	24	196	7564	0	1	2	Nil	No	No	1	Recovered
124	Pavithra	47	Female	Farmer	Phorate	5	125	10	46	24	100	70	93	Kerosene	Drowsiness	Pinpoint	Yes	Yes	0.9	1	32	16	136	3.7	26	163	1643	8	2	44	6	Yes	Yes	12	Recovered
125	Prathap	25	Male	Farmer	Chl	4	Not known	13	65	22	100	80	99	Kerosene	Drowsiness	2	Yes	No	0.9	0.6	21	27	145	4.6	32	184	2167	4	2	8	Nil	No	No	3	Recovered
126	Saradha	21	Female	Student	Quinolphos	2	20	15	96	18	100	70	98	Normal	Normal	3	No	No	1.1	1	43	29	146	3.9	12	65	4563	0	1	2	Nil	No	No	1	Recovered
127	Murugan	46	Male	Farmer	Monochrotophos	4	150	9	58	26	90	60	99	Kerosene	Drowsiness	Pinpoint	Yes	No	1	1.1	28	26	141	4.1	30	169	1639	8	2	36	5	Yes	No	9	Recovered
128	Saravanan	36	Male	Farmer	Quinolphos	4	25	15	76	16	100	70	99	Normal	Normal	3	No	No	0.9	0.7	31	25	143	4.6	32	169	7531	0	1	2	Nil	No	No	1	Recovered
129	Poongodi	45	Female	Farmer	Parathion	4	130	5	40	36	80	50	89	Kerosene	Coma	Pinpoint	Yes	Yes	1.3	1.2	52	51	146	3.8	36	193	1637	8	3	64	4	Yes	Yes	13	Expired
130	Anand	14	Male	Student	Quinolphos	2	15	15	69	16	100	70	99	Normal	Normal	3	No	No	0.6	0.5	21	23	136	3.5	32	133	8219	0	0	2	Nil	No	No	1	Recovered
131	Suganya	24	Female	Student	Chlorpyrifos	4	100	15	65	18	100	70	99	Kerosene	Normal	2	No	No	0.9	0.5	21	19	146	4.2	23	177	3496	1	1	4	36	Yes	Yes	7	Recovered
132	Prabu	46	Male	Farmer	Parathion	4	30	15	96	16	100	80	96	Normal	Normal	3	No	No	0.5	1	32	39	136	3.8	21	163	5631	0	1	2	Nil	No	No	1	Recovered
133	Mani	56	Male	Farmer	Chlorpyrifos	5	90	12	65	22	90	60	99	Kerosene	Restlessness	1	Yes	No	0.9	0.6	32	12	136	3.6	32	166	2163	5	2	16	14	Yes	No	8	Recovered
134	Palani	23	Male	Student	Phorate	2	20	15	86	16	100	70	99	Normal	Normal	3	No	No	0.6	1.1	27	25	136	3.8	12	164	6531	0	1	2	Nil	No	No	1	Recovered
135	Periyasamy	71	Male	Farmer	Triazophos	2	20	15	112	20	140	80	99	Normal	Normal	4	No	No	1.1	1	32	31	145	4.5	36	196	5691	0	1	4	Nil	No	No	1	Recovered
136	Saravanan	24	Male	Student	Monochrotophos	3	120	15	88	16	100	70	99	Kerosene	Normal	1	No	No	0.6	0.8	32	36	136	3.5	23	164	3951	1	1	4	42	Yes	Yes	6	Recovered
137	Pushpa	49	Female	Farmer	Chlorpyrifos	3	100	11	80	22	90	60	99	Kerosene	Drowsiness	Pinpoint	Yes	No	0.6	0.8	29	24	137	3.9	31	183	4563	1	2	18	18	Yes	Yes	7	Recovered
138	Karnan	27	Male	Student	Phorate	3	15	15	86	16	100	70	99	Normal	Normal	3	No	No	0.5	0.8	14	17	140	4	69	196	8631	0	1	2	Nil	No	No	1	Recovered
139	Ramesh	46	Male	Farmer	Chlorpyrifos	6	120	3	42	32	80	50	90	Kerosene	Coma	Pinpoint	Yes	Yes	1.3	1.2	56	51	139	4.1	23	185	1834	8	3	64	6	Yes	No	14	Expired
140	Arjun	19	Male	Student	Parathion	4	10	15	101	14	100	70	99	Normal	Normal	3	No	No	1	1	32	31	136	4.1	23	166	8791	0	0	1	Nil	No	No	1	Recovered
141	Arun	41	Male	Farmer	Monochrotophos	4	125	5	44	32	90	60	92	Kerosene	Drowsiness	Pinpoint	Yes	Yes	0.7	0.8	21	19	136	3.5	23	156	1063	7	3	40	4	Yes	No	12	Expired
142	Roja	20	Female	Student	Phorate	2	10	15	98	16	100	60	99	Normal	Normal	3	No	No	0.6	0.6	21	17	136	3.5	12	96	11326	0	0	1	Nil	No	No	1	Recovered

KEY TO MASTER CHART

OPC: Organophosphorous compound

GCS: Glasgow Coma Scale

SBP: Systolic Blood Pressure

DBP: Diastolic Blood Pressure

SPO2: Oxygen saturation

TB: Total Bilirubin

SGOT: Aspartate transaminase

SGPT: Alanine transaminase

Na+: Sodium

K+: potassium

CPK: Creatine phosphokinase

LDH: Lactate dehydrogenase

AchE: Acetylcholinesterase

POP: Peradeniya Organophosphorous scale

IMS: Intermediate syndrome

IMCU: Intensive Medical Care Unit

ETHICAL COMMITTEE APPROVAL

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg. No(CDSCO).ECR/270/Inst./TN/2013/RR-20
EC Reg. No(DHR).EC/NEW/INST/2021/1618
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr. ANNADURAI S,
MD Internal Medicine Post Graduate student,
Institute of Internal Medicine,
Madras Medical College,
Chennai-600 003.

Dear Dr. ANNADURAI S,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY TO ASSESS THE UTILITY OF PERADENIYA ORGANOPHOSPHORUS POISONING (POP) SCALE, POISONING SEVERITY SCORE (PSS) AND GLASGOW COMA SCALE (GCS) IN PREDICTING SEVERITY AND TREATMENT OUTCOME IN ACUTE ORGANOPHOSPHORUS POISONING PATIENTS ADMITTED IN A TERTIARY CARE CENTRE"- NO.27052022.** The following members of Ethics Committee were present in the meeting held on **18.05.2022** conducted at Madras Medical College, Chennai 3.

1. Prof.P.V.Jayashankar,MS Orth.,D.Orth.,M.Ch Orth (Liverpool) :Chairperson
2. Prof.N.Gopalakrishnan,MD.,DM., FRCP, Director, Inst.of Nephrology,MMC,Ch. : Member Secretary
3. Prof. K.M.Sudha, Prof. Inst. of Pharmacology,MMC,Ch-3 : Member
4. Prof. Alagarsamy Jamila ,MD, Vice Principal, Stanley Medical College, Chennai : Member
5. Prof.Meena Suresh, MD.,DGO.,Prof.of Obst & Gynaec, IOG,Chennai : Member
6. Prof.S.Lakshmi, Prof. of Paediatrics ICH Chennai :Member
7. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
8. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
9. Thiru K.Ranjith, Ch- 91 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003.

ANTI-PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled "**A STUDY TO ASSESS THE UTILITY OF PERADENIYA ORGANOPHOSPHORUS POISONING (POP) SCALE, POISONING SEVERITY SCORE (PSS) AND GLASGOW COMA SCALE (GCS) IN PREDICTING SEVERITY AND TREATMENT OUTCOME IN ACUTE ORGANOPHOSPHORUS POISONING PATIENTS ADMITTED IN A TERTIARY CARE CENTRE** " of the candidate **Dr. ANNADURAI S** with registration Number **200120100505** for the award of M.D in the branch of General Medicine. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and the result shows **16%** percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal

PROFESSOR
INSTITUTE OF INTERNAL MEDICINE
MMC & RGGGH, CHENNAI-600 003

ANTI PLAGIARISM RECEIPT

Ouriginal by Turnitin

Document Information

Analyzed document	OPC POISONING THESIS.docx (D150291022)
Submitted	11/20/2022 6:46:00 PM
Submitted by	ANNADURAI S
Submitter email	duraiannammc@gmail.com
Similarity	16%
Analysis address	duraiannammc.tnmg@analysis.urkund.com

Sources included in the report

Entire Document

Hit and source - focused comparison, Side by Side

Submitted text As student entered the text in the submitted document.

Matching text As the text appears in the source.