DISSERTATION

on

SERUM CREATINE PHOSPHOKINASE AS AN PROGNOSTIC INDICATOR IN ACUTE ORGANOPHOSPHORUS POISONING

Submitted in partial fulfilment of requirements for

M.D. DEGREE BRANCH I

GENERAL MEDICINE

OF

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

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GOVERNMENT THIRUVARUR MEDICAL COLLEGE AND HOSPITAL

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DECLARATION

I solemnly declare that the dissertation titled " **SERUM CREATINE PHOSPHOKINASE AS AN PROGNOSTIC INDICATOR IN A C U T E ORGANOPHOSPHORUS POISONING** " is done by me at Government Thiruvarur medical college and Hospital,Thiruvarur from march 2021 to march 2022 under the guidance and supervision of Dr.M.NATARAJ,MD., Dr.G.KARTHIKEYAN,MD.The dissertation is submitted to TheTamilnaduDr.M.G.R. Medical University towards the partial fulfilment of requirements for the awardof

M.D. Degree (Branch I) in General Medicine.

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CERTIFICATE FROM DEAN

This is to certify that the dissertation titled "**SERUM CREATINE PHOSPHOKINASE AS AN PROGNOSTIC INDICATOR IN ACUTE ORGANOPHOSPHOROUS POISONING**" is a bonafide work done by **Dr. DIVYA. R**, Post graduate student of the Department of General Medicine, Government Thiruvarur Medical College, Thiruvarur, during the academic year 2020-2023. This work has not previously formed the basis for the award of any degree.

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ABBREVIATIONS

OPC-Organophosphorous compounds

Ach-Acetylcholine

Ch-Choline

S.AchE- Serum Acetylcholinesterase

S.CK- serum creatine phosphokinase

POP- Peradeniya Organophosphorus

Poisoning

CNS- Central nervous system

IMS- Intermediate syndrome

P2AM-Pralidoxime

OPIDP-Organophosphate Induced Delayed Polyneuropathy

CONTENTS

INTRODUCTION

INTRODUCTION

- ✓ Organophosphorus compounds are commonly used pesticides andinsecticides. In developing countries like India, suicidal deaths from organophosphorus poisoning is increasing day by day. Incidence of accidental ingestion and exposure through inhalational and contact is also on raising trend. As these poisons are easily available in both rural and urban settings, people can use it harm themselves anytime. Low cost of opc is also an attribution factor. Raising trends of opc poison has become a great threat to both health sector and government. OPC'S are most commonly associated with serious human toxicity accounting for more than 80% of pesticide related hospitalizations.

- ✓ Apart from educating people about the adverse effects of these poisons, it becomes our primary duty to treat the affected patients properly. Early identification of the patients and starting the treatment as early as possible forms the base of successful poison case management. The need of early decontamination has been emphasized by many expertsand articles. This is has to be prioritized along with proper fluid management and antidote administration.

- ✓ Easily available laboratory investigations predicting toxicity could be a boon to the treating doctors in resource limited settings. One such important predictor which has been quoted in various articles is serum creatine phosphokinase.CK is an indicator of muscle fiber necrosis and is proposed to indicate the impending respiratory failure and complications. Hence serial monitoring of CK levels would help us in assessing the outcome of the patients. This would also help us in deciding the rapid and timely interventions necessary to avoid death due to organophosphorus compound poisoning.

AIMS AND OBJECTIVES

AIM AND OBJECTIVES

➢ AIM OF THE STUDY

- To use Creatine kinase as a prognostic indicator in acute organophosphorous poisoning.

➢ OBJECTIVES OF THE STUDY

- To estimate the levels of a creatine phosphokinase levels serially on day of admission and on day 3 of OPC poisoning and to correlate itwith severity

- To correlate the levels of Creatine phosphokinase with complications

REVIEW OF LITERATURE

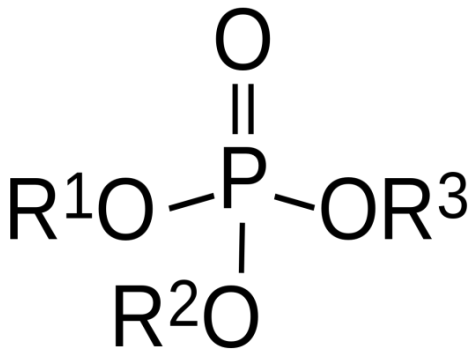
REVIEW OF LITERATURE

HISTORY

In 1800, Lassaigne synthezised OPC while he saw reaction ofalcohol with phosphoric acid. Later in 1854, Tetra Ethyl Pyrophosphate (TEPP) was prepared by Phillipe de Clerment . Years later in 1932, Lange and kreuger, discovered dimethyl diethyl phosphor fluoridates .Based on this, Schrader synthesised 2000 compounds like parathion, tabun, sarin etc. These compounds were used by german military during warfare as a weapon . In 1930, Jamaican jinger palsy incident provoked scientist to know about the mechanism of action of OPC. Oximesin and its effectiveness in OPC poisoning was introduced by DAVIS in 1955 . Many tragedies have happened down the lane becauseof opc poisoning, one such noticeable tragedy happened in INDIA in 2005, when food was contaminated with ethion.

CHEMICAL STRUCTURE

The structure of OPC compound is



OPC are esters of phosphoric acid

The phosphorous atom in centre is attached to either oxygen (P=O) or sulfur (P=S) and three side chains, x group R1 and R2 alkyl, alkoxy, amidomercapten or other groups by a double bond . X group is the principal metabolite, based on which the species is identified. OPC haveunstable structure because of which they disintegrate easily into many radicles, which are usually harmless.

MECHANSISM OF ACTION:

- ACETYLCHOLINE (Ach) is present in both CNS and PNS. It acts as a neurotransmitter in all post synaptic parasympathetic nerve endings.

- It is concentrated in synapses of both sympathetic and parasympathetic ganglia and also in skeletal muscles. Ach is hydrolyzed to acetic acid andcholine by an enzyme ACETYL CHOLINESTARASE (Ach E)

- Two types of Ach E are True acetyl cholinesterase, which is found in tissues anderythrocytes &Pseudocholinesterase which is seen in liver and serum.

- Ach binds with Ach E to form acetylcholine enzyme complex. The enzyme gets acetylated.The acetylated enzyme hydrolysis into acetic acid and choline and freeenzyme is released. This process of acetylation occurs very rapidly.

- OPC compounds cause phosphorylation of acetylcholine enzyme complex in contrast to acetylation which is normally occuring.The phosphorylated enzymewill convert into free enzyme very slowly. There is excess availability of acetylcholine in synapse due to non availability of free enzyme.

CLASSIFICATION OF OPC

I) BASED ON CHEMICALSTRUCTURE

A) Alkylphosphates:

1. HETP (Hexaethyl tetraphosphate)
2. TEPP (tetraethyl pyrophosphate)tetron,fosvex

   Dimefox
3. Isopestox
4. Malathion
5. Sulfoteppa
6. Systox,demeton
7. Dipterex

B) Arylphosphate

i. Paroxon

ii. Parathion

iii. EPN-o,

iv. Methylparathiono, o-dimethyl o-pnitrophenylthiophosphate

- BASED ON TOXICITY:

1. **HIGH TOXICITY:**

   MONOCHROTOPHOS, ETHYL PARATHION, CHLORTHIPHOS,DICHLORVAS, METHYL PARATHION

2. **MODERATE TOXICITY:**

   MALATHION, CHLORPYRIFOS, DIAZINON , FENTHION

3. **MILD TOXICITY:**

   PARATHION, DIMETHOATE, PROPHENOPHOS,TRIAZOPHOS

PHARMACOKINETICS

BASED ON:

1) Target organ's distance from contact
2) metabolism- local/ systemic
3) elimination route
4) hydrolyses by esterase

Around majority of compound is eliminated within 48 hours of exposure by urinary and fecal excretion.Cholinergic crisis may occur when unmetabolized OPC are mobilized from fat store – Fenthion,Chlorfenthion

Prolonged absorption from intestine & reabsorption from fat store mayallow the insecticide concentration for up to 48 hrs

➢ MECHANISM OF ACTION:

- OPC inhibit enzyme AChE. The mechanism of inhibition of the enzyme is by reacting with the esteratic site on the acetyl cholinesterase molecule. The bond formed between phosphorus atom and the esteratic site of enzyme is stable and requires hours to weeksto reverse depending on the type of OP compounds.

- Phosphorylated enzyme is inhibited because of occupation of its active site. It is incapable of carrying out its normal function of hydrolyzing acetylcholine. The effect of the OPC poisoning is therefore the result of continuing increased production of acetylcholine at the neuromuscular junction, resulting in depolarisation block.

- This phosphorylated enzyme can undergo spontaneous hydrolysis or dealkylation. Due to spontaneous hydrolysis active enzyme cholinesterase is released and this is called reactivation. The phosphorylated enzyme can also undergo dealkylation. Once this occurs, reactivation is impossible. This process is called ageing.

AGING OF ENZYME

- For 24 to 72 hours, PC — Ach E is irrevocably bound.

- Aging is the process of one of the R groups leaving the phosphate molecule.

- Once ageing has taken place, denovo synthesis of Ach E is necessary to restore thesupply.

- In carbamates, ageing cannot happen.
- In 24 hours, Ach E hydrolyzed on its own.
- Dichlorvos, dicrotophosdicaphon, dimethoate, temephos, crotoxyphos, diethyl OPC low propensity for ageing benefit from P2AM.- parathion, chlorpyriphos, phorate, phosfolan,TEPP, coumaphos, diazinon, ehion, chlorothion, demeton are examples of dimethyl phosphoryl compounds that accelerate AchE ageing

- Without antidotal treatment with an enzyme reactivator, permanent attachment to the acetylcholinesterase enzyme ("ageing") may occur after a variable delay. Dimethylated Organophosphates (OPs) reactivate suppressed AChE more quickly than diethylated OPs.

- The half-life of ageing may be prolonged by oxime antidote therapy;

- Therefore, early oxime administration is likely to be beneficial.

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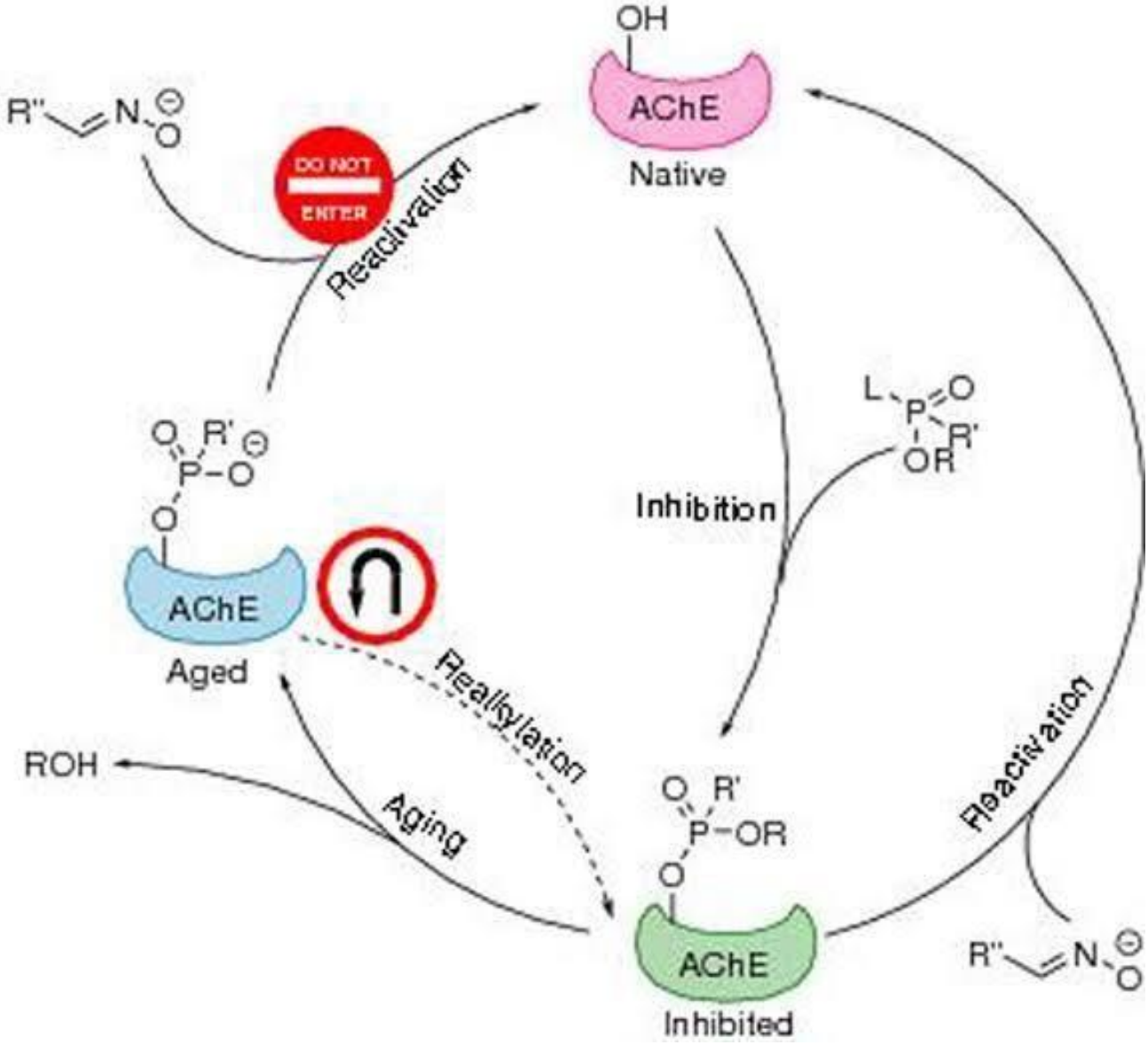

- The rate of poisoning onset and its severity are determined by three separate processes, specifically the phosphorylation of ChE by OPC. Ageing, Reactivation

- Clinical symptoms only appear in cases of acute poisoning after serum cholinesterase activity has been suppressed to a level greater than 50%,and the severity of symptoms is inversely correlated with serum cholinesterase activity inhibition.

- Cholinesterase in mild poisoning is 20–50%. Cholinesterase levels drop to 10–20% in the case of moderate poisoning.

- The cholinesterase level drops to less than 10% in cases of severe poisoning.

➢ CLINICAL FEATURES

- The agent, quantity and route of entry, determines the clinical manifestations of OP poisoning. Consumption and inhalation produce faster symptom development than cutaneous exposure. The effects of consumptionemerge in compounds within 30-90 minutes, at most 24 hours.
- They require metabolic bioactivation and are very lipophilic.

Muscarinic receptors	Nicotinic receptors	Central receptors
Cardiovascular	**Cardiovascular**	**General effects**
Bradycardia	Tachycardia	Anxiety
Hypotension	Hypertension	Restlessness
Respiratory	**Musculoskeletal**	Ataxia
Rhinorrhoea	Weakness	Convulsions
Bronchorrhoea	Fasciculations	Insomnia
Bronchospasm	Cramps	Dysarthria
Cough	Paralysis	Tremors
Gastrointestinal		Coma
Nausea/vomiting		Absent reflexes
Increased salivation		Respiratory depression
Abdominal cramps		Circulatory collapse
Diarrhoea		
Faecal incontinence		
Genitourinary		
Urinary continence		
Eyes		
Blurred vision		
Increased lacrimation		

➢ LOCAL EFFECTS:

- Systemic symptoms begin to develop later after the commencement of GI symptoms. Usually have respiratory consequences when inhaled. Symptoms following ocular exposure typically start in the eyes.

➢ SYSTEMIC OUTCOMES:

- There are three distinct clinical phases that can be seen:First cholinergic stage.
- The middle-range syndrome (IMS)
- Delayed Polyneuropathy Induced by Organophosphate.
- Chronic Neuropsychiatric Disorders caused by Organophosphates.
- The Ach buildup at the cholinergic synapses causes the cholinergic phase, which can be divided into
- Muscarinic (all postganglionic nerve endings)
- Nicotinic acid (Autonomic ganglia and skeletal muscle end plate)Symptoms of the CNS (synapses in CNS)

The Intermediate Syndrome (IMS)

- This type II paralysis first described by Wadia et al. In 1974 and later christened as –Intermediate syndrome(IMS)‖ by Senanayake, Karalliedde L. The syndrome is of Acute onset, seen within 24-96 hrs(1-4days) after poisoing, affecting conscious patients without fasciculations or other cholinergic manifestations. The cardinal features of this syndrome is muscle weakness affecting predominantly proximal limb muscles and neck flexors. The muscles innervated by motor cranial nerves III,VII and X are affected in different combinations. These patients were conscious and showed marked anxiety, sweating, dyspneic and restlessness caused by progressive hypoxia.

- The neck muscle weakness is a constant feature. Patients will have difficulty to raise head above the pillows. Weakness of shoulder abduction and hip flexion could be noted. However normal strength in the distal muscle gives a false impression that the limbs are spared. Tendon reflexes are diminished or in most patients with no sensory impairment. Complete recovery occurs within 4-18 days if adequate ventilator support is given. But altered function at neuromuscular junction may persist upto 2yrs after its occurrence

- The syndrome carries great mortality if not recognized in time and treated. The agents commonly responsible are fenthion, monochrotophos and Dimethoate. Respiratory insufficiency develops over 6 hrs approximately. Initially patient uses accessory muscles of ventilation. There is increase in ventilator rate, sweating, restlessness and later cyanosis if not recognized patient soon becomes unconscious and death follows. A consensus from literature search appears that IMS may result from inadequate therapy with oximes.
- IMS is likely to result from post synaptic neuromuscular dysfunction. The symptom complex begins at a time when the cholinesterase function is very low and the OP compounds is still detectable in the body. As blood levels of OPC's fall and OPC's tissue redistribution occurs the motor end plates may be rechallenged by the cholinesterase inhibitor in the presence of inadequate circulatory oximes.

➢ Organophosphate Induced Delayed Polyneuropathy (OPIDP)

- It is a distal motor axonopathy develops following a latent period of 2-4 weeks after the cholinergic crisis. The main clinical features are distal muscle weakness especially of feet and hand. The weakness is preceded by limb pain and paresthesia. Wasting of distal muscles of particularly small muscle of the hand and those of anterior and peroneal compartments of the leg is a inevitable consequence. In some patients pyramidal tract signs appear after a few weeks or few months. Recovery is variable. The phosphorylation of an enzyme neuropathy target esterase in nervous tissue is considered to be responsible for the polyneuropathy. Several out breaks of OPIDP have occurred in various countries where the poison was traced in most instances to be accidental contamination or adulteration of cooking oils with mineral oils. In 1930's more than 50000 US citizens became paralyzed after drinking Jamica ginger contaminated with TOCP.

➢ Chronic Organophosphate Induced Neuropsychiatric Disorder (COPIND)

- These are the delayed complications due to acute exposure to high dose of OPC.
- The manifestations are depression, anxiety, memory disturbances, dystonic reactions, cog-wheel rigidity, schizophrenia. It is due to the sequelae of convulsions, respiratory failure, cardiac arrythmias and anoxia.

➢ CLINICAL SEVERITY SCORING

Table 1: Peradeniya organophosphorus poisoning (POP) scale

Parameter	Criteria	Score
Pupil size	≥2 mm	0
	<2 mm	1
	pinpoint	2
Respiratory rate	<20/min	0
	≥20/min	1
	≥20/min with central cyanosis	2
Heart rate	>60/min	0
	41-60/min	1
	<40/min	2
Fasciculation	None	0
	Present, generalized/ continuous	1
	Both generalized and continuous	2
Level of consciousness	Conscious and rationale	0
	Impaired response to verbal command	1
	No response to verbal command	2
Seizures	Absent	0
	present	1
0-3: mild poisoning, 4-7: moderate poisoning, 8-11: severe poisoning		

➢ TREATMENT:

- **DECONTAMINATION:**

Patient must be decontaminated immediately, removing the contaminated clothes, washing the skin with soap and water is mandatory Gastric lavage: performed using largest possible oro-gastric tubes with 50-100ml of fluid/lavage, preferably within 1 hrof ingestion protect airway in patients with impaired consciousness.

Administer activated charcoal: dose initial 60-100 gms, followed by1.25ms to 0.5gms/kg every 1-4 hrs.

➢ TREATMENT:

i) **Anticholinergics -** atropine /glycopyrrolate.

ii) AchE reactivation:

- **Anticholinergic medication:** Atropine

  - It is a tertiary amine, a competitive antagonist of acetylcholine at muscuranic post synaptic membrane and in the CNS. In symptomaticpoisoned adults , Inject 1.8-3 mg (3-5 ml) of atropine, bolus. Check whether targets are achieved.
  - Aim for heart rate >80 beats per minute, SBP > 80 mm Hg, and a clear chest

(atropine won't dry focal areas of aspiration). Double the atropine dose everyfive minutes if you have not achieved these targets.

- Review patient every 5 min. Once these parameter are improving,repeat last same or smaller dose of atropine.
- If improvement in these parameters is persistent and satisfactory after 5 min, now you can plan for atropine infusion.Calculate total dose of atropine required for rapid atropinisation.
- Start hourly atropine infusion at 20% of total dose of atropine required for atropinisation. Most patients do not need >3-5 mg (5-9 ml) per hour of atropine infusion.
- Once your patient is stabilised, use the Targets checklist to lower the infusion rate by 20% every four hours.
- In patients who are intubated, unconscious, have an in situ oropharyngeal airway, and have intermediate syndrome, do not use oral secretions to guide therapy.
- To adjust the atropine dose, ignore perspiration.
- Stable Patients who have a clear chest but a heart rate that is slightly belowtarget do not require more atropine.
- Once the patient is stable, the most crucial indicator for adjusting the atropinedosage is bronchorrhea.
- Absence of bowel noises, fever, and confusion are signs of atropine poisoning.

- If the patient has experienced an atropine toxicity, stop the atropine infusionfor 60 minutes.
- Once the patient's temperature has dropped and he or she has calmed down,re-start the infusion at 80% of the original pace.
- PRALIDOXIME is used only to treat people who have ingested organophosphorus.

  - ✓ Bolus dose: 30 mg/kg PAM over 30 minutes. Adults-2g
    Obidoxime @ 4mg/kg over 20min
  - ✓ Maintenance dose: continuous infusion of 8 mg/kg per hour. Adults-500mg/hr
  - ✓ Obidoxime- 0.5mg/kg/hr infusion

PAM must be administered intravenously. For both bolus and maintenance, administer slowly. Vomiting, hypertension, cardiac arrhythmia, or a cardiac arrest can all resultfrom a rapid infusion.

The class of Organophosphorus compounds affects how effective pralidoxime is. The drug P2AM is ineffective in Profenofos.

PAM is effective for up to 12 hours when used with dimethyl organophosphorus substance. And for up to 5 days in the diethyl organophosphorus compound.

- Due to the short-lived and spontaneous reversibility of the cholinesterase inhibition incarbamate intoxication, pralidoxime is generally not advised.

- However, pralidoxime may be used empirically if the precise agent is not known and thepatient suffers considerable toxicity.

- **OTHER DRUGS:**

- Diazepam is used when the patients are agitated and who develop seizures. Diazepam appears to counteract some aspects of CNSderived symptoms which are not affected by atropine.
- Diazepam 10 mg slow IV push, repeated as necessary. Up to 30 mgdiazepam per 24 hours can be given.

- Protective effects of clonidine or likely to involve multiple effects including blockade of acetylcholine release and post synaptic muscarinic receptors, Transient inhibition of acetylcholinesterase, Inhibits the release of acetylcholine from central and peripheral cholinergic neurons.

- **MANAGEMENT OF INTERMEDIATE SYNDROME:**

- ✓ After exposure, it often manifests 12 to 96 hours later. Pharyngeal weakness and actiontremors are early indicators of the intermediate syndrome (difficulty in deglutition or pooling of secretions in pharynx). Later, the patient experiences respiratory muscle paralysis, deep tendon jerk loss, cranial neuropathies, and the inability to flex the neck.Patients with tremors and pharyngeal weakness are more likely to develop the full intermediate condition that necessitates intubation and ventilation. Later, the patient experiences respiratory muscle paralysis, deep tendon jerk loss, cranial neuropathies, and the inability to flex the neck. Total symptomatic treatment for intermediate syndrome.
- ✓ PAM should useserumm while offering sufficient ventilator support. Because the patientcould experience breathing problems, they should stay in the hospital for up to 5 days.

✓ **MANAGEMENT OF DELAYED POLYNEUROPATHY:**

- No specific drugs are available. Physiotherapy could help to an certain extent

➢ MORTALITY:

- In India and other underdeveloped nations, the mortality rate ranges from 4 to38%. The poison employed, the amount, the length of time following exposure, and the atropinization of the poisons all affect mortality.
- The fastest hydrolysis of the carboxy ester group into compounds with negligible or no anticholinesterase action gives malathion the lowest toxicity.The highest fatality rate is in phenthion.
- Death due to Respiratory failure may be because of increased secretions, Bronchospasms, respiratory muscle paralysis, respiratory center depression
- Other causes of early death includes seizures, arrythmias
- Death in late stages is due to secondary infections , sepsis, complications related to mechanical ventilators, ventricular arrhythmias.

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- **SERUM CREATINE PHOSPHOKINASE IN OPC POISONING:**

- In an experimental investigation performed on rats with an OP, the serum CPK activity increased but the tissues' ChE activity decreased. In poisoning instances and in patients who died as a result of poisoning, the CPK activity was markedly increased. Myonecrosis, or the destruction of muscle membrane, manifests as a significant rise in serum creatine phosphokinase. The mitochondria are where the first alterations occur; they enlarge and then exhibit lysis of the central cristae.

- Although the structures of various AChEIs differ, the myopathic alterations they cause are the similar, indicating that they share a common mechanism. It is not the direct action of these inhibitors on muscle that causes this process; rather, it is an excess of Ach and its interactions within AChRs. Muscle hyperactivity, such as fasciculations, is the common denominator.

- Serum total CK activity significantly increases as soon as 30 minutes of carbofuran injection and continues to rise for 3 hours, causing fasciculations and myopathy.
- Examining the serum and diaphragm under the influence of acute carbofuran poisoningrevealed numerous distinctive changes in CK isoenzymes. Within 30 minutes, theisoezyme CK-MM type was enhanced >2 fold in the diaphragm and remained noticeably higher than control at 24 hours. Following the return of regular muscular action, the amount of CK that was leaking decreased.
- As cpk could be a marker of respiratory failure, it could be used as an prognosticindicator in opc poisoning

MATERIALS AND METHODS

MATERIALS AND METHODS

- **Study centre:** Department of General Medicine GTMCH
- **Duration of study: 1 year** (From March 2021 to march 2022)
- **Study design** **:** Observational study
- **Sample Size** **:** 100patients
- **Analysis Plan:** SPSS, Epi info

- **Inclusion criteria**

1. Age more than 18 years of both sexes
2. Patients willing to give informed consent
3. Patients who have consumed organophosphorus compound in the past 24hours

- **Exclusion Criteria**

1. Age less than 18 years
2. Non complying patients who do not give consent to participate in the Study.
3. Patients with co-existing illness like Coronary artery disease, Diabetes mellitus, Hypertension, Pulmonary diseases and other chronic illness.
4. Patients who had trauma, intramuscular injections in the recent past.
5. Patients on prior medicines like statins, fibrates, anti-platelets, anti-coagulants and steroids.
6. Patients who have consumed alcohol with poison.

❖ **Methodology:**

- After obtaining the informed consent details of history and clinical examination were recorded. Peradeniya OP poisoning scale was applied to all study subjects and the severity of OP poisoning was graded as mild, moderate, severe. In all study subjects blood was collected on admission, day 3, for estimation of serum creatine phosphokinase. Other routine investigations weredone.

❖ **Statistic analysis:**

- Continuous variables are represented in mean, median, mode and standard deviation.
- Categorical variables are represented in frequencies and percentages.
- When a Categorical Variable is associated with a categorical variable,the variables are represented in both by tables and bar diagrams. For test of significance, chi-square test is used. Fisher's exact test is used when more than 20% of the cell values have expected cell value less than5.
- When a continuous variable is associated with continuous variable,correlation tests are used.
- When the paired samples variables such as variable at admission, day one and at discharge is associated with the categorical variables such as outcome,clinical severity, and then repeated measures ANOVA is used.
- P-value less than 0.05 is considered statistically significant.

RESULTS

➢ OBSERVATION AND RESULTS

RESULTS

1) Population characteristics:

A) Age:

Table 1: Age Distribution

Age (In years)	Cases	
	No	%
11-20	**4**	**4**
21-30	**27**	**27**
31-40 years	35	35
41-50 years	16	16
51-60	11	11
61-70	6	6
71-80	1	1
Total	100	100.0
Range	18-73 years	
Mean	37.7 years	
S.D	13 years	

Fig 1: Bar Diagram showing Age Distribution

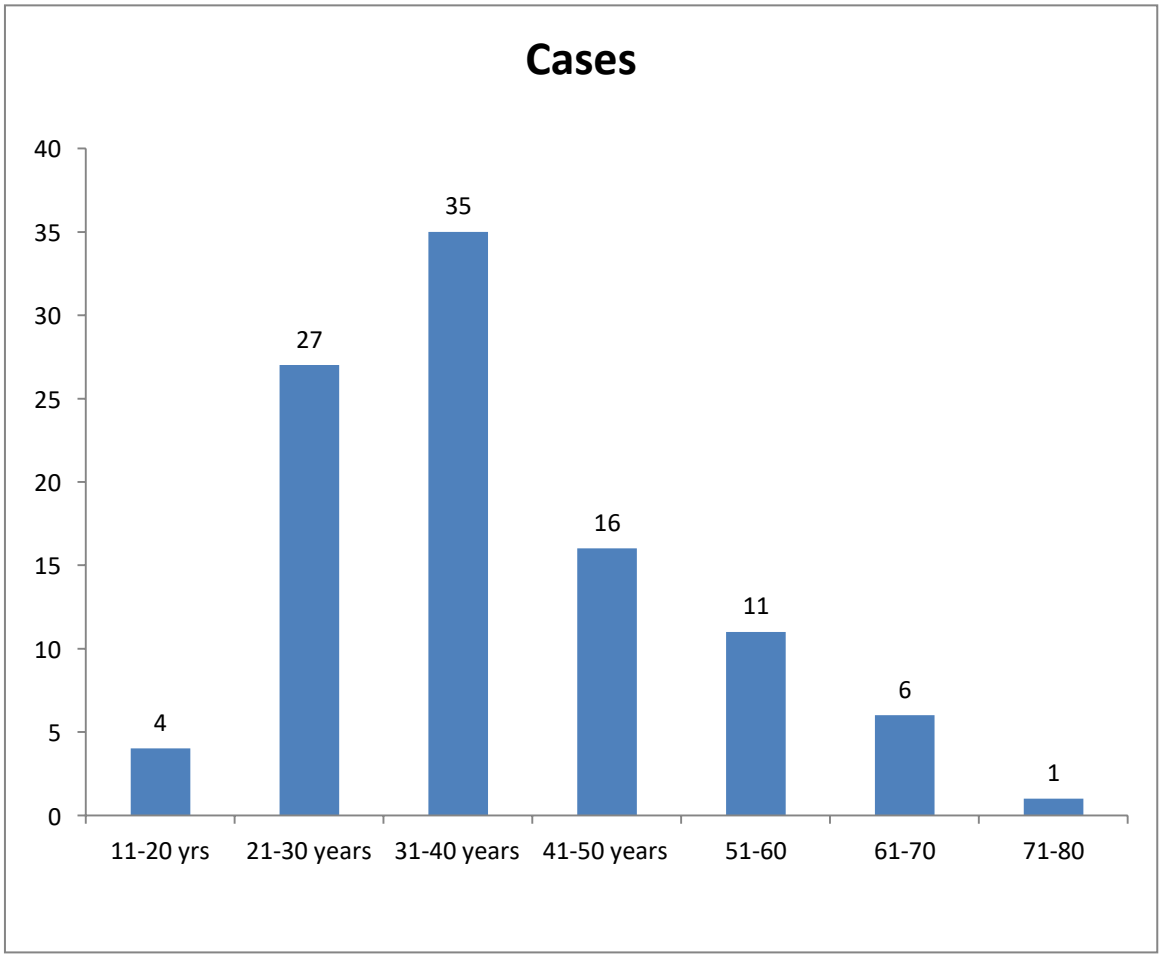

Table 2: Distribution of Gender

	Cases	
	No	**%**
Male	34	34
Female	66	66
Total	100	100.0

Fig 2: Pie Diagram showing Distribution of Gender

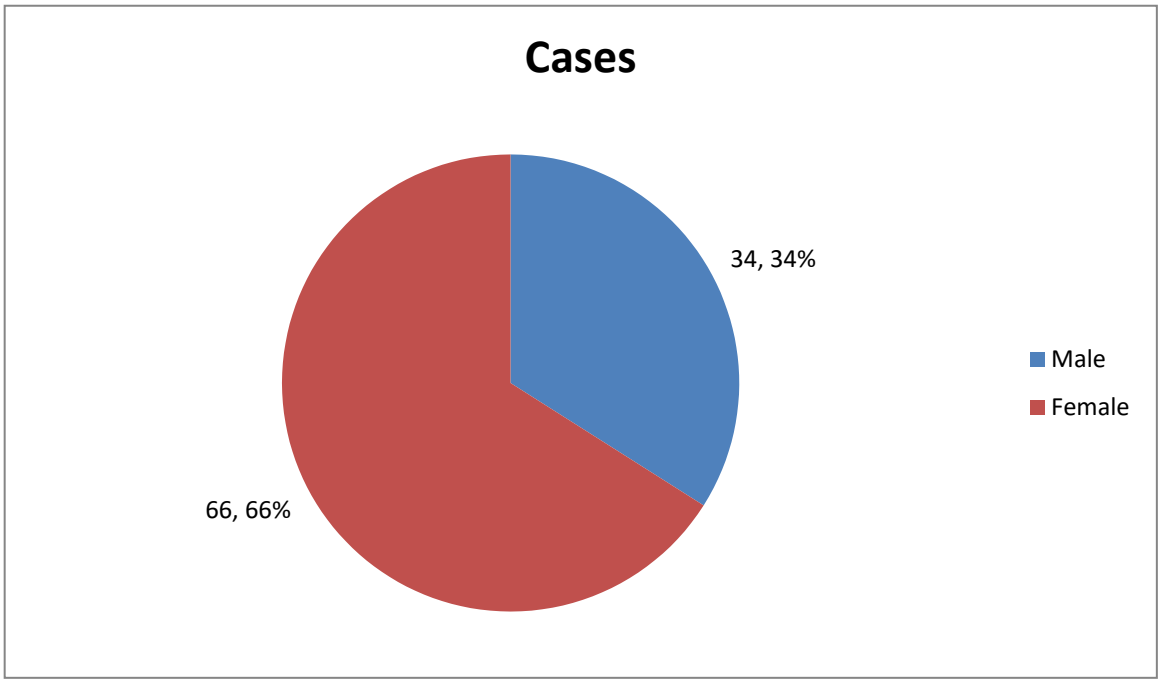

Table 3: Distribution of Mode of Ingestion

	Cases	
	No	**%**
Oral	98	98
Aural	2	2
Total	100	100.0

Fig 3: Pie Diagram showing Distribution of Mode of Ingestion

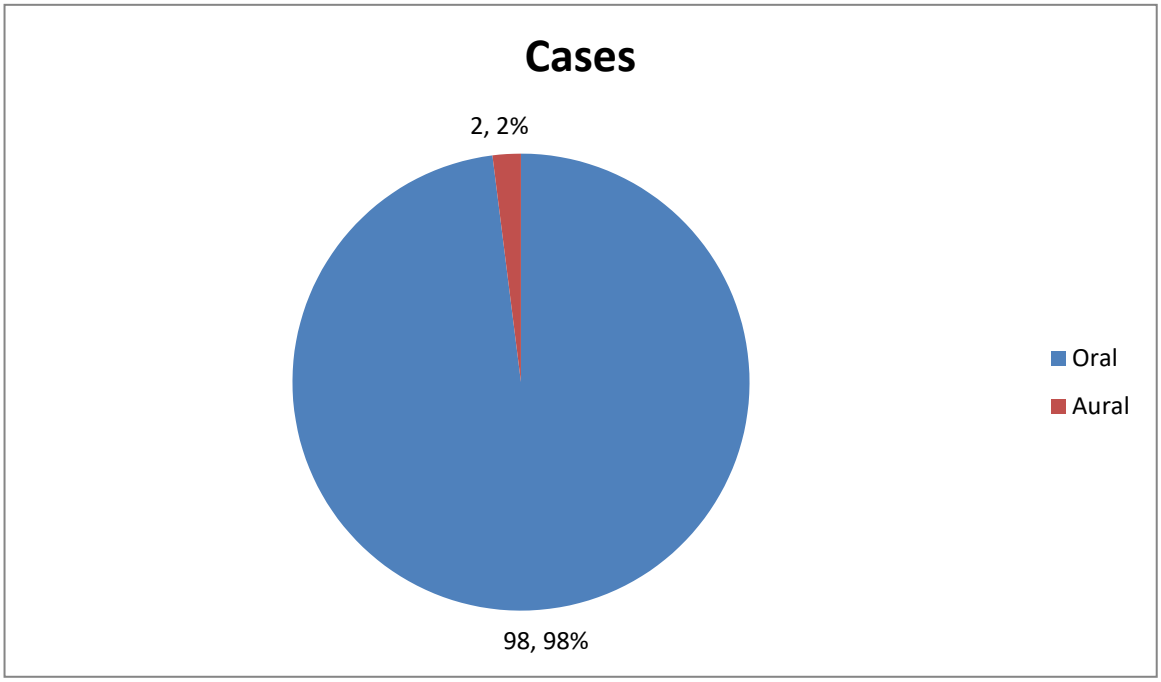

Table 4: Distribution of Quantity of Intake

Quantity (ml)	Cases	
	No	%
<25	40	40
25-50	27	27
50-75	7	7
75-100	26	26
Total	65	100.0

Fig 4: Pie Diagram showing Distribution of amount of intake

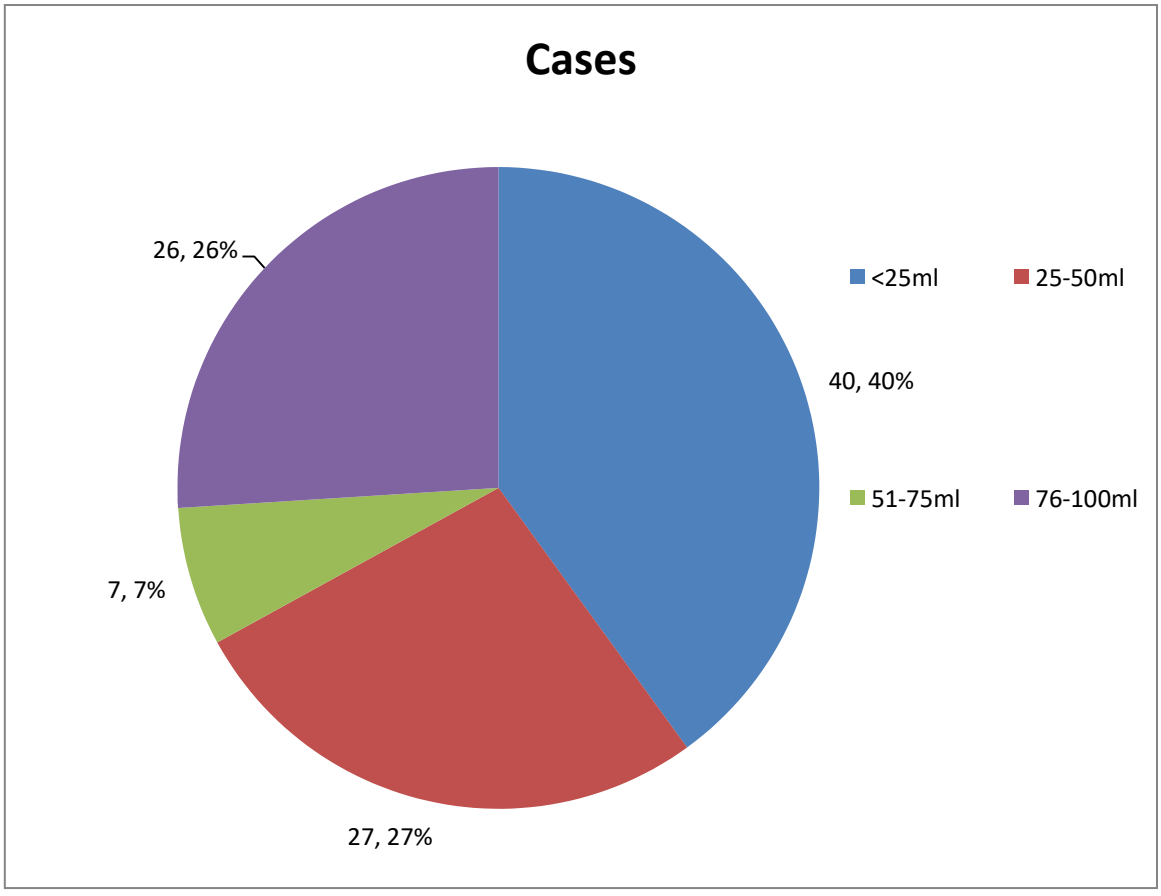

Table 3: Distribution of Composition of poison

	Cases	
	No	**%**
Chlorpyrifos	42	42
Dichlorvas	2	2
Dimethoate	16	16
Monochrotophos	25	25
Parathion	11	11
Phorate	1	1
Profenofos	3	3
Total	100	100.0

Fig 4: Bar Diagram showing Distribution of composition

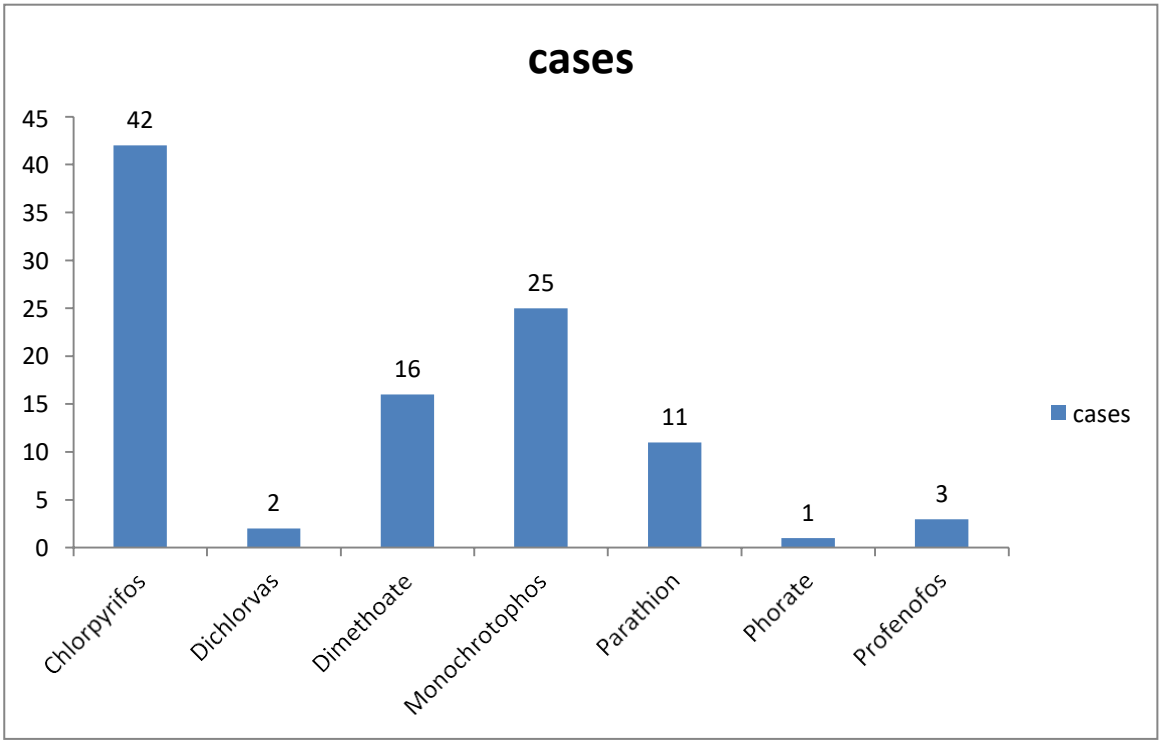

Table 5: Distribution of Time of Presentation

	Cases	
	No	%
<1hr	12	12
1-2hr	25	25
2-3hr	32	32
3-4hr	16	16
4-5hr	3	3
>5hr	12	12
Total	100	100.0

Fig 5: Pie Diagram showing Distribution of Time of Presentation

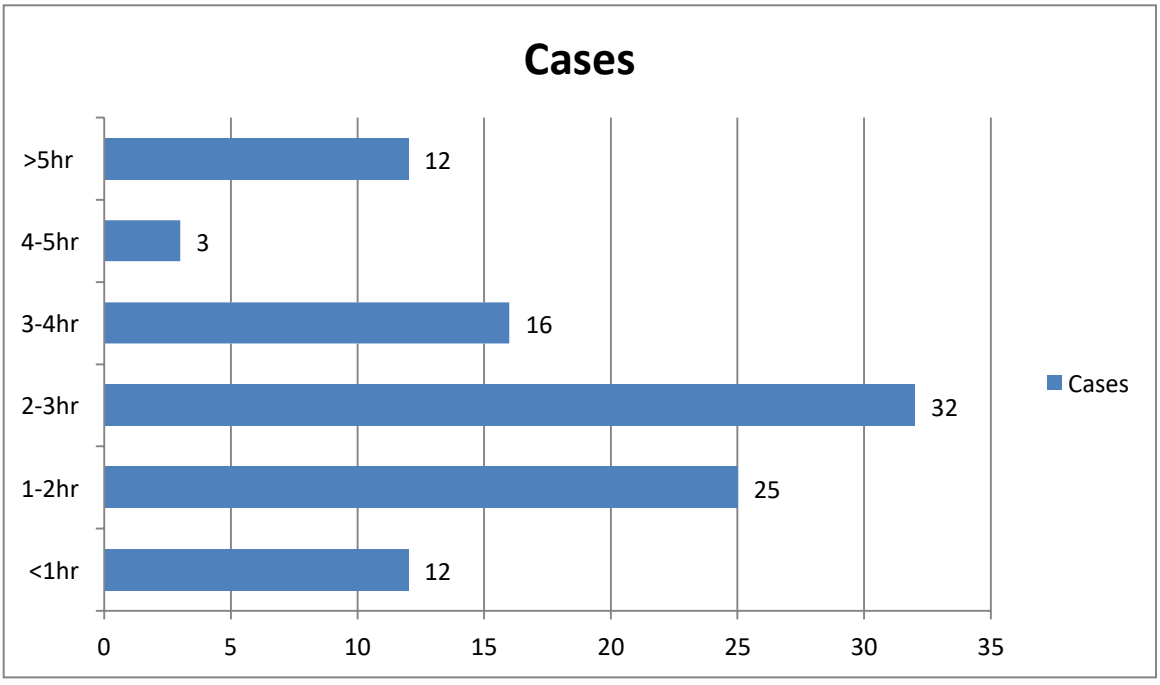

Table 6: Distribution of POP score

	Cases	
	No	**%**
Mild	49	49
Moderate	24	24
Severe	27	27
Total	100	100.0

Fig 6: Pie Diagram showing Distribution of POP score

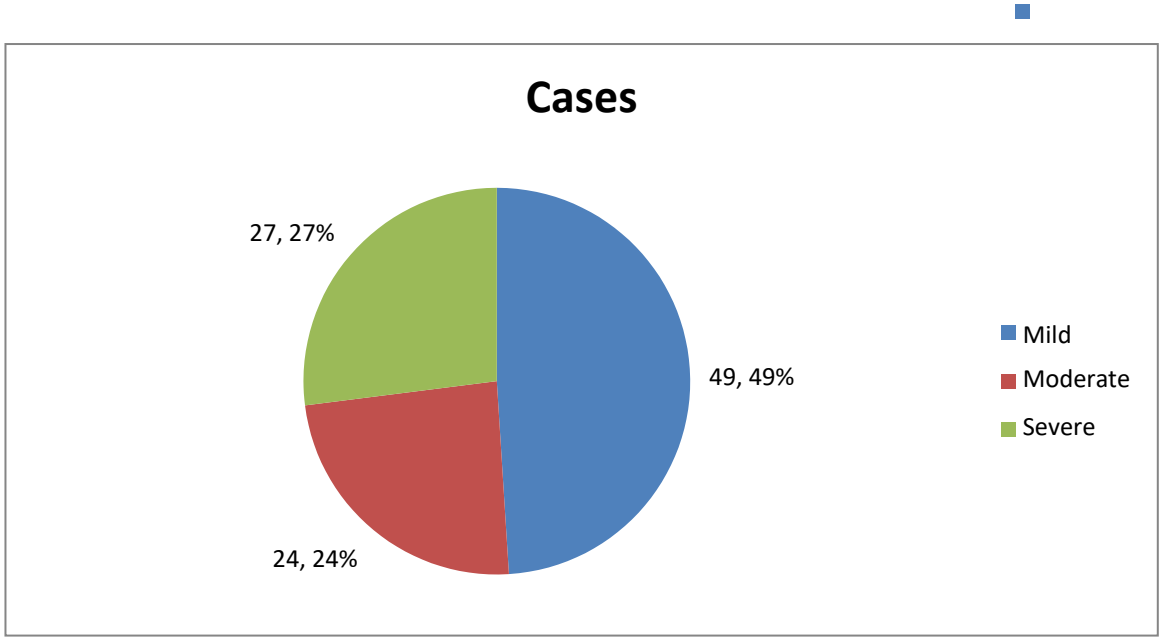

Table 7: Distribution of Patients on Respiratory Failure on Mechanical Ventilation

	Cases	
	No	**%**
Yes	30	
No	70	
Total	100	100.0

Fig 7: Pie Diagram showing Distribution of Patients on Mechanical ventilation

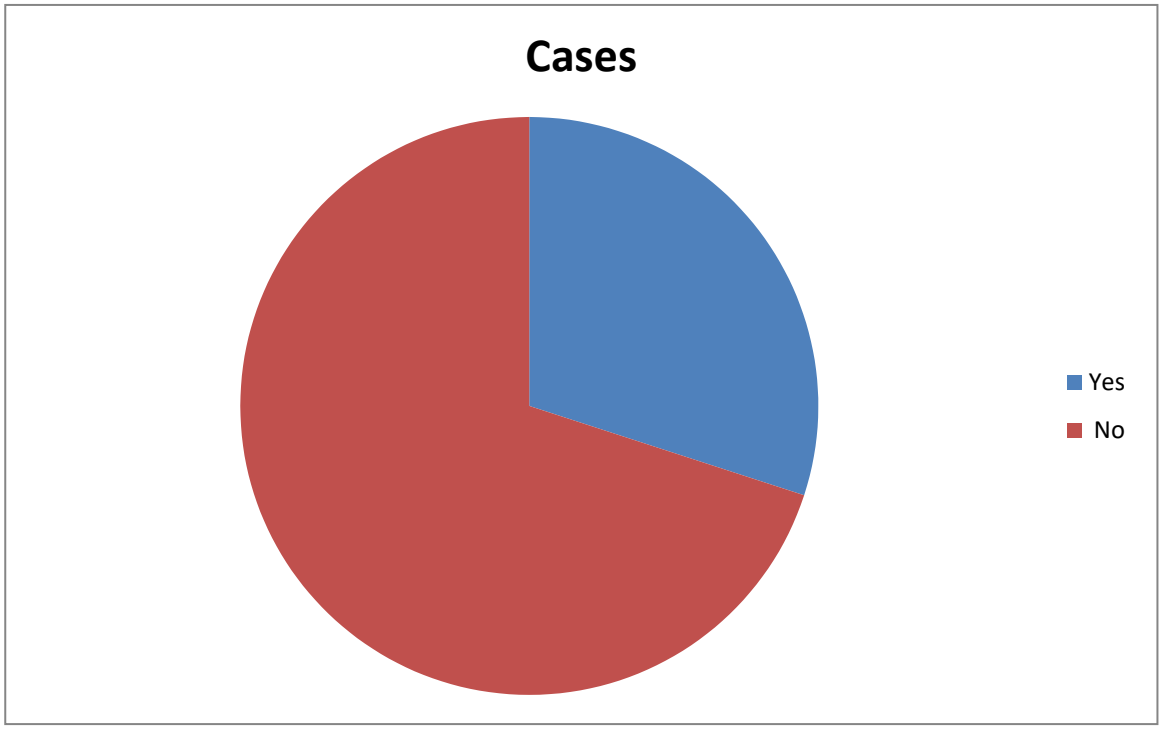

Table 8: Distribution of Patients with Deranged LFT

	Cases	
	No	**%**
Yes	40	
No	60	
Total	100	100.0

Fig 8: Pie Diagram showing Distribution of Patients with deranged LFT

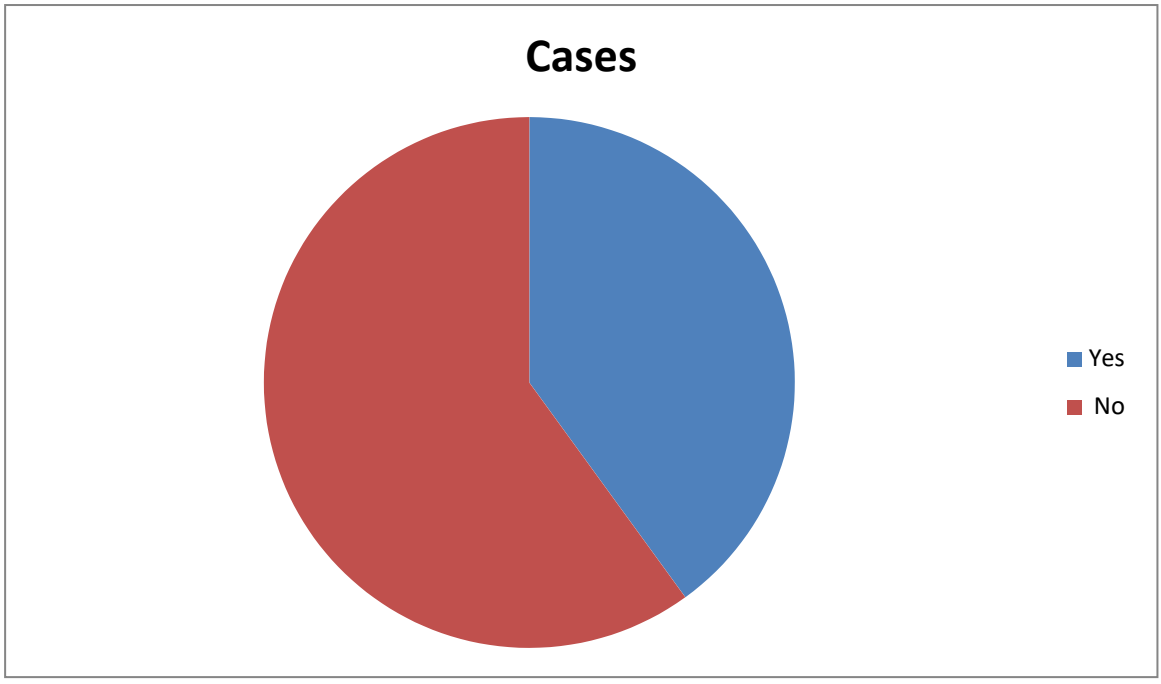

Table 9: Distribution of Outcome

	Cases	
	No	**%**
Recovered	86	
Death	14	
Total	100	100.0

Fig 9: Pie Diagram showing Distribution of Outcome

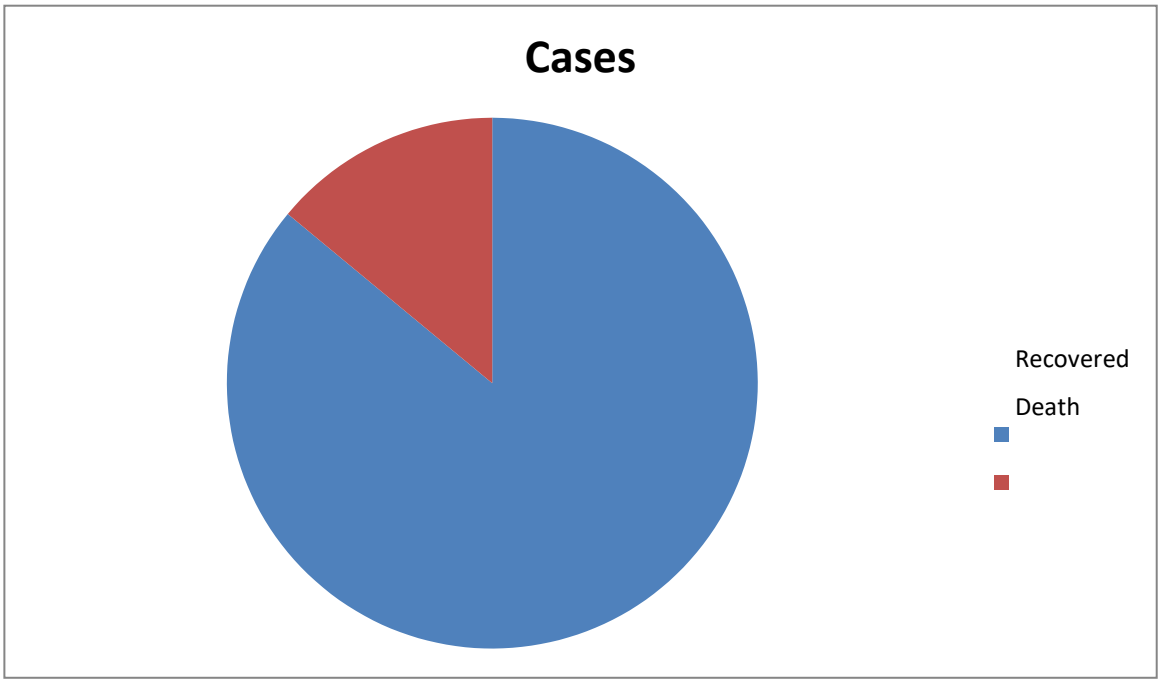

Table 10: Distribution of day of Death

	Cases	
	No	**%**
1	2	2
2	4	4
3	4	4
4	3	3
5	1	1
Total	14	100.0

Fig 10: Graph showing day of death

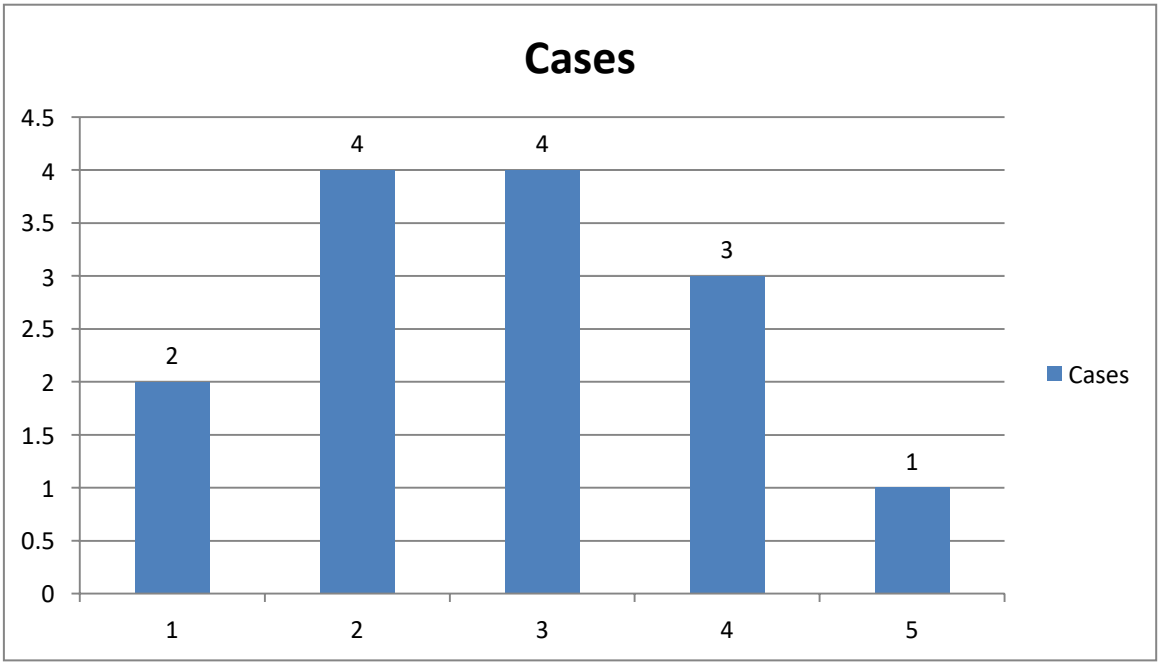

Table 11: Comparison of Age and Outcome

Age(years)	Outcome		Total	P value
	Alive	Dead		
	N (%)	N (%)		
<30	31 (36)	0	31	< 0.05*
>30	55 (64)	14 (100)	69	
Total	11	89	100	

*P value <0.05 Significant using Chi Square Test/ Fischers Exact Test

Table 12: Comparison of Sex and Outcome

*

SEX	Outcome		Total	P value
	Alive	Dead		
	N (%)	N (%)		
Male	29 (33.7)	5 (35.7)	34	0.884 >0.05
Female	57 (66. 3)	9 (64.3)	66	
Total	86	14	100	

P value >0.05 Not significant (NS) using Chi Square Test

Table 13: Comparison of Mode of Ingestion and Outcome

Mode	Outcome		Total	P value
	Alive	Dead		
	N (%)	N (%)		
Oral	84 (97.7)	14 (100)	98	>0.05
Aural	2 (2.3)	0	2	
Total	86	14	100	

P value >0.05 Not significant (NS) using Chi Square Test

Table 14: Comparison of amount of intake and Outcome

Quantity	Outcome		Total	P value
	Alive	Dead		
	N (%)	N (%)		
<25	40 (46.5)	0	40	< 0.05*
25-50	23 (26.7)	4 (28.6)	27	
50-75	7 (8.1)	0	7	
75-100	16 (18.6)	10 (71.4)	26	
Total	86	14	100	

*P value <0.05 significant (S) using Chi Square Test

Table 15: Comparison of Day of Presentation and Outcome

Age (yrs)	Outcome		Total	P value
	Alive	Dead		
	N (%)	N (%)		
<1hr	12 (14)	0	12	<0.05*
1-2hr	25(29.1)	0	25	
2-3hr	32 (37.2)	0	32	
3-4hr	14 (16.3)	2 (14.3)	16	
4-5hr	1 (1.2)	2 (14.3)	3	
>5hr	2 (2.4)	10 (71.4)	12	
Total	86	14	100	

*P value <0.05 significant (S) using Chi Square Test

Table 16: Comparison of Composition and Outcome

Age (yrs)	Outcome		Total	P value
	Alive	Dead		
	N (%)	N (%)		
Chlorpyrifos	36 (41.9)	6 (42.9)	42	>0.05
Dichlorvas	2 (2.3)	0	2	
Dimethoate	16 (18.6)	0	16	
Monochrotophos	17 (19.8)	8 (57.1)	25	
Parathion	11 (12.8)	0	11	
Phorate	1 (1.2)	0	1	
Profenofos	3 (3.5)	0	3	
Total	86	14	100	

P value >0.05 Not significant (NS) using Chi Square Test

Table 17: Comparison of pop score and Outcome

POP	Outcome		Total	P value
	Alive	Dead		
	N (%)	N (%)		
Mild	49 (57)	0	49	<0.05*
Moderate	24 (27.9)	0	24	
Severe	13 (15.1)	14 (100)	27	
Total	86	14	100	

*P value <0.05 significant (S) using Chi Square Test

Table 18: Comparison of Respiratory failure and Outcome

	Outcome		Total	P value
	Alive	Dead		
	N (%)	N (%)		
Yes	16 (18.6)	14 (100)	30	<0.05*
No	70 (81.4)	0	70	
Total	11	89	100	

*P value <0.05 significant (S) using Chi Square Test

Table 19: Comparison of deranged LFT and Outcome

Age (yrs)	Outcome		Total	P value
	Alive	Dead		
	N (%)	N (%)		
Yes	26 (30.2)	14 (100)	40	< 0.05*
No	60 (69.8)	0	60	
Total	86	14	100	

*P value <0.05 significant (S) using Chi Square Test

Table 20: Distribution of Mean CPK among Study Subjects

Parameter	CPK		T score	P value
	DAY 1	DAY 3		
	Mean ± SD	Mean ± SD		
CPK	**154.09 ± 93**	**119.33 ± 109**	5.67	**< 0.05***

*p <0.05 –Significant by Applying Students T –Test

Clinical Severity and Mean creatine Kinase

Parameter	CPK		P value
	DAY 1	DAY 3	
	Mean ± SD	Mean ± SD	
Mild	**74 ± 18**	**50.14 ± 12**	**< 0.05***
Moderate	**192.9 ± 22**	**113.5 ± 26**	
Severe	**304.9 ± 53**	**279.6 ± 121.5**	

*p <0.05 –Significant by Applying ANOVA

DISCUSSION

❖ DISCUSSION

- An observational study conducted in Department of general medicine, Government Thiruvarur Medical college. The study enrolled 100 patients admitted with OPC poisoning after applying inclusion and exclusion criteria. The study period was 1 year from march 2021 to march 2022.

- Out of 100 patients, the study population comprised of 34% females and 66% males. This finding is consistent with study conducted by S.Shivakumar and K.Raghavan et al of Tamilnadu who reported 165 cases of OPC poisoning with male predominance in sex distribution. Kuntal Battacharya et al from Kolkata showed male predominance. A similar pattern of male dominance observed in Mangalore and Srilanka case series. In South India, as males are more involved in spraying pesticides.

- The mean age of study population was37.7 years and Standard deviation of 13.

- The predominant age group of the study population were in the age group of 31-40 years . 91% of cases with age <30 years observed by Karalliede L., Senanayake N. et al of Srilanka. A study in Mangalore showed more in cases in 20-30 years (36.6%) This study shows that the target age group in younger age and the need for improving the management protocol and decreasing the mortality.

- The most common OP compound ingested was chlorpyriphos. Study by bhattacharya etal described the most frequent compound as Chlorpyrifos. The mortality rate in our study paradeniy14 %. Sundaram et al study showed mortality rate of 22.5%. In our study, POP scoring was much reliable and was correlated with duration of presentation,outcome, levels of mean Creatine Kinase. The initial serum CK with Clinical severity by POP scoring and outcome study was correlated by Kunal Bhattacharya et al of Kolkata. Mild POP score seen in 49%, Moderate POP score is 24%

- The initial rise in Serum CK in severe acute OPC poisoning is probably due to the presence of muscle fiber necrosis. This occurred even before the development of Intermediate syndrome in which CK level is expected to rise. Hence it can be used as predictor for IMS. With good management, CK levels may be reduced to normal within 5 days.

- In our study, raised serum CK levels significantly correlated with initial clinical severity by POP scoring, increasing atropine requirement, hospital stay duration, IMS, complications like arrhythmias, renal failure, pancreatitis, coma and outcome and it more significantly correlated with the initial serum levels at admission. Hence, we have concluded that levels of Serum CK can be used as parameters for assessing the severity and Outcome of Acute OPC poisoning replacing Serum AchE levels

CONCLUSION

➢ CONCLUSION:

OPC poisoning is one of the leading causes of death in India. In our study,

- There is a male preponderance
- Age group between 31 to 40 years are more commonly encountered in poisoning
- Most common compound was chlorpyriphos
- Quantity of the poison consumed corelates with the severity of outcome
- The mortality rate is 14%
- POP Scoring was found to be a reliable scale in assessing severity
- The serum levels of CK significantly corelated with the severity and outcome
- Hence, we conclude that serial CK monitoring can be used as a parameter for assessing the severity and outcome of acute OPC poisoning.

LIMITATIONS OF THE STUDY

➢ LIMITATIONS OF THE STUDY

- Due to financial constraints the measurements of CK may not be feasible in all the health care set up.

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BIBLIOGRAPHY

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ANNEXURES

❖ PROFORMA

- NAME:
- AGE:
- SEX:
- IP NO:
- ADDRESS:
- CONTACT NO:
- SOCIAL CLASS:

- PRESENTING ILLNESS:

- GENERAL EXAMINATION:
- VITALS:

  PR:
  BP:
  RR:
  TEMP:

- CLINICAL EXAMINATION:

  Consciousness
  Pulse rate
  Respiratory rate
  Miosis
  Fasciculation
  Any other clinical feature if present

- SYSTEMIC EXAMINATION:

  CVS
  RS
  P/A
  CNS

- INVESTIGATIONS:

  COMPLETE BLOOD COUNT
  LIVER FUNCTION TEST
  RENAL FUNCTION TEST
  SERUM ELECTROLYTES

BLOOD GLUCOSE LEVELS
URINE ROUTINE
SERUM CREATININE PHOSPHOKINASE VALUES

POP SCORE

SERIAL ESTIMATION OF SERUM CREATINE PHOSPHOKINASE

	DAY 1 U/L	**DAY 2 U/L**
SERUM CREATINE KINASE		
CLINICAL SCORING		
ATROPINE REQUIREMENT		
COMPLICATIONS IF ANY		
DURATION OF THE HOSPITAL		

INFORMATION SHEET

Place of study:
Government Thiruvarur medical college and hospital, Thiruvarur-610004

Name of the investigator:
Dr. R. Divya

Name of the participant:

Age:

Sex:

IP Number:

Study title:

CREATININE PHOSPHOKINASE AS AN PROGNOSTIC INDICATOR IN ACUTE ORGANOPHOSPHOROUS COMPOUND POISONING.

- AIM:

- ✓ To estimate the creatinine phosphokinase levels in acute organophosphorous poisoning cases at the time of admission and serially on day 3

- ✓ To study correlation of creatinine phosphokinase level in acute organophosphorous poisoning based on paradeniya opc poisoning scoring scale for prediction of prognosis.

- ✓ Prestructured proforma will be used to record the relevant information.

- ✓ CAN I REFUSE TO PARTICIPATE IN THE STUDY?

  Participation in the study is purely voluntary. You may refuse to participate or withdraw from the study any time. In both the cases the treatment and care for the receive from this hospital will not be affected in any manner.

- ✓ BENEFITS AND HARMS OF PARTICIPATING IN THE STUDY:

  The participants will be benefited by knowing the status etiology can be identified and it can be corrected. By the Way of participating in the study, patient is contributing to updation of science which may benefit him/her and all other patients suffering due to this illness in future.

- ✓ CONFIDENTIALITY:

  The data collected from the patient will be used for study purpose only. The results of the study will be published. Personal information of the participant will be kept confidential. There will not be any disclosure about patient information without permission.

- ✓ SUBJECT RIGHTS:

  If you wish further information regarding your rights as a research participant, you may contact the principal investigator in the mobile number

❖ PATIENT CONSENT FORM

Participants name:

Address:

Title of the study:

Serum creatinine phosphokinase as an prognostic indicator in acute organophosphorus compound poisoning.

- ✓ The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purposes.i have been given an information sheet giving details of the study. Ifully consent to participate in the above study

Signature of the participant: Date:

Signature of the witness: Date:

Signature of the investigator: Date:

PLAGIARISM CERTIFICATE

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DISSERTATION
on
SERUM CREATINE PHOSPHOKINASE AS AN PROGNOSTIC INDICATOR IN
ACUTE
ORGANOPHOSPHORUS POISONING
Submitted
in partial fulfilment of requirements
for

PLAGIARISM CERTIFICATE

This is to certify that the dissertation work titled **"SERUM CREATINE PHOSPHOKINASE AS AN PROGNOSTIC INDICATOR IN ACUTE ORGANOPHOSPHORUS POISOING "** of the candidate **Dr. R.DIVYA** with registration number **200120109002** for the award of **M.D. GENERAL MEDICINE** in the branch of **I**. 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 results show **9 percentage** of plagiarism in the dissertation.

Guide & Supervisor sign with Seal

ETHICAL COMMITEE CERTIFICATE

Govt. Thiruvarur Medical College & Hospital, Thiruvarur,Tamil Nadu, India- 610004

CERTIFICATE OF ETHICS APPROVAL

Ref .No: 2858 /M.E/2021

This is to certify that, the verified project no: 004/IEC/GTMC/2022 entitled "**SERUM CREATININE PHOSPHOKINASE AS AN PROGNOSTIC INDICATOR IN ACUTE ORGANOPHOSPHORUS COMPOUND POISONING.**" submitted by **Dr.R.DIVYA.,** is **APPROVED** by Institution ethics committee, at its meeting held on 26/2/2022 under the following terms and condition.

a)If any modification of Post Ethical approved research protocol done by the principal investigator, it will be mandatory to submit the modified details within seven days to the IEC

b) Any serious adverse event occurring during the course of the study should be reported to the IEC within a period of seven days

c) A yearly progress report of the project has to be submitted to the IEC for review.

d) This approval is valid for three years or the duration of the project whichever is less

The IEC has right to give a letter to stop/ terminate this study at any time.

Dr.S.Rajendran., MS
Member secretary
Institution ethics committee
Govt.Thiruvarur Medical college
Thiruvarur

Copy to:

1. Principal investigator
2. Office copy of IEC

MASTERCHART

S.NO	AGE	SEX	MODE	QUANTITY	COMPOSITIO	TIME OF PRE	POP SCORE	CPK DAY 1	CPK DAY 3	RESPIRATOR	deranged LF	OUTCOME	DAY OF DEATH
1		32M	oral	100ml	Profenofos	4 hrs	severe	306	176	yes	yes	RECOVERED	
2		24F	oral	25 ml	chlorpyrifos	3 hrs	mild	56	42			RECOVERED	
3		44M	oral	75ml	monochroto	2 hrs	moderate	196	102			RECOVERED	
4		28M	oral	25ml	profenofos	3 hrs	mild	75	56			RECOVERED	
5		33M	oral	100ml	chlorpyrifos	2 hrs	severe	322	211	yes	yes	RECOVERED	
6		18F	oral	25ml	chlorpyrifos	3 hrs	mild	63	36			RECOVERED	
7		43M	oral	100ml	monochroto	8 hours	severe	275	398	YES	YES	DEATH	day 4
8		34M	oral	100 ml	dimethoate	2 hours	moderate	183	65		yes	RECOVERED	
9		29M	oral	25ml	chlorpyrifos	3 hrs	mild	54	37			RECOVERED	
10		32F	oral	100ml	chlorpyrifos	6 hrs	severe	296	428	yes	yes	DEATH	day 3
11		35M	oral	25ml	profenofos	3 hrs	mild	66	65			RECOVERED	
12		21M	oral	25ml	chlorpyrifos	4 hrs	mild	77	44			RECOVERED	
13		65M	oral	50ml	chlorpyrifos	3 hrs	mild	52	39			RECOVERED	
14		33F	oral	100ml	chlorpyrifos	4 hrs	severe	286	176	yes	yes	RECOVERED	
15		47F	oral	25ml	dimethoate	2 hrs	mild	52	37			RECOVERED	
16		28M	oral	50ml	monochroto	2 hrs	moder	211	120			RECOVERED	
17		33F	oral	25ml	chlorpyrifos	3 hrs	mild	67	60			RECOVERED	
18		36M	oral	100ml	monochroto	4 hrs	severe	305	369	yes	yes	DEATH	day 5
19		26F	oral	25ml	monochroto	1 hr	mild	52	51			RECOVERED	
20		52F	Oral	50ml	chlorpyrifos	3 hrs	mild	47	67			RECOVERED	
21		22M	oral	25ml	chlorpyrifos	4 hrs	mild	68	48			RECOVERED	
22		34F	oral	75ml	dimethoate	2 hrs	moderate	279	142	yes	yes	RECOVERED	
23		56F	oral	25 ml	parathion	2 hrs	mild	78	49			RECOVERED	
24		23M	oral	25ml	monochroto	3 hrs	mild	61	42			RECOVERED	
25		37M	oral	100ml	chlorpyrifos	4 hrs	severe	218	114	yes	yes	RECOVERED	
26		23F	oral	25ml	phorate	3 hrs	mild	85	75			RECOVERED	
27		34F	oral	25ml	monochroto	1 hr	mild	75	68			RECOVERED	
28		42M	oral	100ml	dimethoate	4 hrs	severe	298	142	yes	yes	RECOVERED	
29		38M	oral	75ml	dimethoate	5 hrs	moderate	195	118			RECOVERED	
30		26M	oral	25ml	dichlorvas	2 hrs	mild	100	50			RECOVERED	
31		22F	oral	75ml	chlorpyrifos	2 hrs	moderate	182	127		yes	RECOVERED	
32		35F	oral	25ml	parathion	1 hr	mild	95	63			RECOVERED	
33		73M	oral	100ml	monochroto	6 hrs	severe	359		yes	yes	DEATH	day 1
34		19M	oral	25ml	monochroto	2 hrs	mild	58	45			RECOVERED	
35		39M	oral	25ml	chlorpyrifos	2 hrs	mild	61	37			RECOVERED	
36		22M	oral	25ml	dimethoate	1 hr	mild	72	54			RECOVERED	
37		28F	oral	50ml	chlorpyrifos	4 hrs	moderate	158	135		yes	RECOVERED	
38		35F	oral	25ml	dimethoate	10 hrs	mild	68	57			RECOVERED	
39		58F	oral	50ml	monochroto	6 hrs	severe	399		yes	yes	DEATH	day 2
40		25M	oral	100ml	chlorpyrifos	3 hrs	severe	217	185	yes	yes	RECOVERED	

41	47M	oral	25ml	chlorpyrifos	2 hrs	mild	62	39			RECOVERED		
42	32M	oral	50ml	monochroto	4 hrs	moderate	190	165			RECOVERED		
43	24M	oral	25ml	dimethoate	3 hrs	mild	42	37			RECOVERED		
44	48M	oral	100ml	monochroto	4 hrs	severe	289	199	yes	yes	RECOVERED		
45	33M	AURAL	50ml	chlorpyrifos	2 hrs	moderate	197	165		yes	RECOVERED		
46	42F	oral	50ml	chlorpyrifos	2 hrs	moderate	179	148		yes	RECOVERED		
47	37M	oral	25ml	chlorpyrifos	3 hrs	mild	47	34			RECOVERED		
48	55M	oral	100ml	monochroto	6 hrs	severe	314	448	yes	yes	DEATH	day 3	
49	25M	oral	75ml	chlorpyrifos	3 hrs	mild	57	55			RECOVERED		
50	48M	oral	100ml	monochroto	3 hrs	severe	316	216	yes	yes	RECOVERED		
51	32M	oral	25ml	dimethoate	1 hr	mild	56	45			RECOVERED		
52	28M	oral	100ml	chlorpyrifos	3 hrs	severe	211	176	yes	yes	RECOVERED		
53	49M	oral	25ml	parathion	1 hr	mild	75	47			RECOVERED		
54	37M	oral	50ml	parathion	2 hrs	mild	89	47			RECOVERED		
55	25F	oral	25m	chlorpyrifos	3 hrs	mild	95	45			RECOVERED		
56	19M	oral	50ml	monochroto	4 hrs	moderate	180	118			RECOVERED		
57	26F	oral	25ml	dichlorvas	3 hrs	mild	52	45			RECOVERED		
58	39M	oral	100ml	chlorpyrifos	4 hrs	severe	338	215	yes	yes	RECOVERED		
59	56F	oral	25ml	chlorpyrifos	1 hr	mild	45	35			RECOVERED		
60	38M	oral	50ml	monochroto	3 hr	moderate	192	132			RECOVERED		
61	22F	oral	50ml	monochroto	2 hr	moderate	200	130			RECOVERED		
62	46M	oral	75ml	chlorpyrifos	2 hr	moderate	192	100		yes	RECOVERED		
63	63F	oral	100ml	chlorpyrifos	6 hrs	severe	350	472	yes	yes	DEATH	day 4	
64	36F	oral	25ml	monochroto	1hr	mild	67	38			RECOVERED		
65	62M	oral	50ml	chlorpyrifos	5hrs	severe	372		yes	yes	DEATH	day 2	
66	33M	oral	50ml	monochroto	3 hrs	moderate	192	97		yes	RECOVERED		
67	26M	oral	100ml	chlorpyrifos	3 hrs	moderate	217	85		yes	RECOVERED		
68	35F	oral	50ml	parathion	1 hr	mild	79	46			RECOVERED		
69	58M	oral	50ml	monochroto	6 hrs	severe	376	435	yes	yes	DEATH		
70	45F	oral	25ml	dimethoate	1 hr	mild	59	43			RECOVERED		
71	56F	oral	75ml	chlorpyrifos	2 hr	moderate	175	90			RECOVERED		
72	26F	oral	25ml	chlorpyrifos	3 hr	mild	75	50			RECOVERED		
73	27M	AURAL	50ml	parathion	2 hrs	moderate	178	85	yes	yes	RECOVERED		
74	53M	oral	25ml	chlorpyrifos	2 hrs	mild	86	45			RECOVERED		
75	45M	oral	50ml	dimethoate	4 hrs	mild	95	45			RECOVERED		
76	36M	oral	100ml	monochroto	4 hrs	severe	330	476	yes	yes	DEATH	day 3	
77	32F	oral	25ml	chlorpyrifos	2 hrs	mild	90	85			RECOVERED		
78	68M	oral	50ml	parathion	3 hrs	moderate	180	115		yes	RECOVERED		
79	65F	oral	50ml	monochroto	10 hrs	severe	275	320	yes	yes	DEATH	day 4	
80	52M	oral	100ml	dimethoate	6 hrs	moderate	190	90			RECOVERED		

S.NO	AGE	SEX	MODE	QUANTITY	COMPOSITIO	TIME OF PRE	POP SCORE	CPK DAY 1	CPK DAY 3	RESPIRATO	deranged LF	OUTCOME	DAY OF DEATH
81	27	F	oral	50ml	chlorpyrifos	3 hrs	moderate	180	82		yes	RECOVERED	
82	57	M	oral	25ml	chlorpyrifos	3 hrs	mild	92	50			RECOVERED	
83	47	F	oral	100ml	chlorpyrifos	5 hrs	severe	390		yes	yes	DEATH	day 2
84	39	M	oral	50ml	parathion	3 hrs	mild	98	45			RECOVERED	
85	32	F	oral	25ml	parathion	2 hrs	mild	95	43			RECOVERED	
86	57	M	oral	100ml	dimethoate	3 hrs	severe	298	165	yes	yes	RECOVERED	
87	28	F	oral	25ml	monochroto	4 hrs	mild	112	65			RECOVERED	
88	67	M	oral	100ml	chlorpyrifos	3 hrs	severe	228	197	yes	yes	RECOVERED	
89	33	M	oral	25ml	parathion	1 hr	mild	89	45			RECOVERED	
90	36	M	oral	50ml	dimethoate	3 hrs	mild	98	37			RECOVERED	
91	42	M	oral	100ml	chlorpyrifos	6 hrs	severe	335		yes	yes	DEATH	day 1
92	19	M	oral	25ml	monochroto	3 hrs	mild	88	66			RECOVERED	
93	48	M	oral	25ml	monochroto	4 hrs	mild	118	72			RECOVERED	
94	36	M	oral	50ml	chlorpyrifos	3 hrs	moderate	190	125			RECOVERED	
95	32	M	oral	50ml	dimethoate	2 hrs	moderate	197	110			RECOVERED	
96	22	M	oral	50ml	parathion	2 hrs	mild	90	75			RECOVERED	
97	38	M	oral	100ml	dimethoate	2 hrs	moderate	198	80	yes	yes	RECOVERED	
98	32	M	oral	25ml	chlorpyrifos	1 hr	mild	97	56			RECOVERED	
99	26	M	oral	50ml	chlorpyrifos	3 hr	severe	210	260	yes	yes	RECOVERED	
100	47	M	oral	100ml	chlorpyrifos	6 hrs	severe	320	375	yes	yes	DEATH	day 3