"STUDY OF EFFECT OF SERUM POTASSIUM LEVELS ON PATIENTS WITH ORGANOPHOSPHATE POISONING PRESENTING TO KLES DR. PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI-A 1 YEAR OBSERVATIONAL STUDY"

BY

REGISTRATION NUMBER: BG0121001

DISSERTATION

Submitted to

KLE Academy of Higher Education and Research

Belagavi, Karnataka

In partial fulfilment of the requirements for the degree of

M. D. (Doctor of Medicine)

In

General Medicine

Department of General Medicine

Jawaharlal Nehru Medical College, KAHER

Belagavi-590010, Karnataka

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

ENDORSEMENT

This is to certify that the dissertation entitled **"STUDY OF EFFECT OF SERUM POTASSIUM LEVELS ON PATIENTS WITH ORGANOPHOSPHATE POISONING PRESENTING TO KLES DR. PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI- A 1 YEAR OBSERVATIONAL STUDY"** is a bonafide research done by **REGD NO. BG0121001**

DR. REKHA S. PATIL MD

Professor and Head

Department of General Medicine

J. N. Medical College

Nehru Nagar, Belagavi-10

Date : 28/06/2024

Place : Belagavi

DR.(MRS.) N.S. MAHANTSHETTI MD

Principal

J. N. Medical College

Nehru Nagar, Belagavi-10

PRINCIPAL
JAWAHARLAL NEHRU MEDICAL COLLEGE
BELAGAVI

Date : 28/06/2024

Place : Belagavi

UNDERTAKING

I, Regd No. BG0121001, hereby declare that the information and the data mentioned in my dissertation entitled **"STUDY OF EFFECT OF SERUM POTASSIUM LEVELS ON PATIENTS WITH ORGANOPHOSPHATE POISONING PRESENTING TO KLES DR. PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI- A 1 YEAR OBSERVATIONAL STUDY",** belongs to me and is original. I am aware of the definition of Plagiarism as detailed below:

- An act or instance of using are closely imitating the languages and thoughts of another author without authorization and the representation of the authors work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another's words, thoughts or ideas as one's own without any attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is an original one, and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then, I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the university.

Date: 28/06/2024

Place: BELAGAVI

REGD NO.: BG0121001

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH

JAWAHARLAL NEHRU MEDICAL COLLEGE

(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be-University)

(Recognized by National Medical Commission, New Delhi)

Accredited 'A+' Grade by NAAC (3rd Cycle) **Placed in Category 'A' by MoE (GoI)**

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 – 2471350 0831 – 2470759 www.jnmc.edu rincipal@jnmc.edu

Ref No: MDC/PG/ Date: 25-06-2024

"ACCEPTANCE LETTER"

The softcopy of thesis entitled: **"STUDY OF EFFECT OF SERUM POTASSIUM LEVELS ON PATIENTS WITH ORGANOPHOSPHATE POISONING PRESENTING TO KLES DR. PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI"** has been submitted for anti-plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **07%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER BELAGAVI J. N. MEDICAL COLLEGE

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BG0121001
Postgraduate Student,
2021-22 Batch,
Department of General Medicine
J. N. Medical College, Belagavi.

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 113 **Date: 27/09/2022**

To,

Regd. No. BG0121001
PG Student in General Medicine,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"STUDY OF EFFECT OF SERUM POTASSIUM LEVELS ONPATIENTS WITH ORGANOPHOSPHATE POISONING PRESENTING TO 'KLES DR. PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI- A 1 YEAR OBSERVATIONAL STUDY.",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

LIST OF ABBREVIATIONS

OP	Organophosphorus
OPC	Organophosphorus Compound
AChE	Acetylcholinesterase
WHO	World Health Organisation
LD50	Lethal Dose in 50% subjects
TESS	Toxic Exposure Surveillance System
BuChE	Butyrylcholinesterase
CNS	Central Nervous System
ARDS	Acute Respiratory Distress Syndrome
BMI	Body Mass Index
ICU	Intensive Care Unit
CYP450	Cytochrome P450
CBC	Complete Blood Cell Count
ECG	Electrocardiogram
POP	Peradeniya Organophosphorus Score
K+	Potassium level
PAM	Pralidoxime
PChE	Plasma cholinesterase
SChE	Serum cholinesterase
WBC	White Blood Cells
EchE	Erythrocyte cholinesterase
RR	Respiratory rate

ABSTRACT

Background: Organophosphate (OP) poisoning is a critical issue, especially in regions with extensive agricultural activities. These toxic compounds, widely used as pesticides, lead to severe neurotoxicity and substantial health risks. Understanding the biochemical profile (specially serum potassium levels) is essential for better management.

Objective: This study aims to evaluate the effect of serum potassium levels on mortality in organophosphate poisoning cases, providing comprehensive insights on better management.

Methodology: An observational study was conducted involving 60 patients admitted with organophosphate poisoning at KLEs Dr Prabhakar Kore Hospital, Belgaum over a course of 1 year (1stJanuary to 31stDecember, 2023). Data was collected and statistical analyses were performed to identify significant patterns and correlations.

Results:

- **Demographics:** Primarily affecting young adults aged 25 to 30 (46%), with a male predominance (73.3%). Phorate was the most common agent (70%). The majority of the cases were due to intentional self-poisoning.
- **Laboratory Findings:**Hypokalemia is present in 63.3% of organophosphate poisoning cases. Hypokalemia is most commonly observed in severe cases of organophosphate poisoning (42.2%).
- **Outcomes:**Death occurs in 15.79% of hypokalemic organophosphate poisoning cases compared to 0% of normokalemic cases.

Conclusion: Hypokalemia emerges as a significant complication, especially in severe cases, correlating with heightened mortality rates and increased ventilator requirements. Clinical features such as convulsions, fasciculations, and respiratory distress often accompany hypokalemia. In cases of organophosphorus poisoning, hypokalemia can be utilised as an efficient and dependable indicator of death and morbidity rates.These findings emphasize the critical importance of prompt recognition and management of hypokalemia in OPC poisoning cases to mitigate adverse outcomes and potentially reduce mortality rates.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

Introduction

Organophosphorus (OP) pesticide poisoning is a major concern across the world, especially in countries that are mainly dependent on agriculture. It is responsible for over 80% of the hospitalizations caused by pesticides.1 The primary tool for protecting crops and controlling pests in India is OP pesticide, due to the country's heavy reliance on agriculture. As a result, farmers who are unintentionally exposed to too much of these pesticides may experience negative health impacts. Nevertheless, it has also become a popular agent for self-inflicted poisoning due to its inexpensiveprice and simpleaccessibility.2

Nearly 300,000 individuals each year, primarily from rural areas, die as a result of pesticide self-poisoning. As much as 70% of cases occur in underdeveloped nations.3,4 A lack of nearby hospitals, an insufficient transportation system, a high patient-to-caregiver ratio, and, ultimately, an ineffective antidote might all contribute to the high fatality rate.The irreversible cholinesterase inhibitory effects of OP insecticides extend to their inhibition of pseudocholinesterase as well as cholinesterase.3,5 When cholinesterase is inhibited, acetylcholine builds up at synapses, which in turn causes overstimulation and interference with neurotransmission in the central nervous system and the periphery.6

One traditional route of absorption in cases of suicidal ideation is through the stomach mucosa, which is permeable to organophosphates. Organophosphate compounds are activated and detoxified in the liver, although they are mostly excreted by the kidneys.7 Muscarinic and nicotinic clinical symptoms include bradycardia, hypotension, increased salivation/lacrimation, excessive sweating, nausea, vomiting, diarrhoea, abdominal discomfort, faecal and urine incontinence, and hypotension. Anxiety, agitation, convulsions, miosis,

sleeplessness, coma, cheyne-stokes breathing, heart failure, and respiratory failure are all signs that originate in the central nervous system.

Within one to three days following an acute cholinergic crisis, patients often have intermediate syndrome, also known as type II paralysis. Intermediate syndrome rates range from 8 to 50 percent.8 Agricultural workers are at increased risk for organophosphate-induced delayed neuropathy, a condition that can be caused by chronic OP exposure.9

Acute OP poisoning treatment begins with stabilizing the patient's heart and lungs, then moves on to decontamination (i.e., removing any clothing that could be a source of ongoing exposure in occupational intoxication), irrigation of the skin and eyes, and finally, gastric lavage and activated charcoal to reduce the compound's absorption. Pralidoxime chloride, which restores activity to acetyl cholinesterase that has been suppressed, and atropine, an antagonist of peripheral and central muscarinic receptors, form the backbone of therapy. Antioxidants, magnesium sulphate, sodium bicarbonate, and other novel adjunct therapies have been studied for the treatment of OP poisoning in recent years.10 Paralysis of the respiratory muscles, bronchospasm, and bronchial secretions are common causes of death, along with cardiovascular and respiratory failure.

Problems in diagnosis or treatment that go unchecked might lead to deadly outcomes. Intensive care, atropine, and oxime cholinesterase reactivators are necessary for the challenging management of acute poisoning. The field of emergency medicine is unique in its ability to detect critical illnesses quickly, decontaminate patients, and provide definite treatment, all of which are essential to life.

Aim and Objectives

- To evaluate the serum potassium levels in organophosphate poisoning in patients presenting to KLES Dr. Prabhakar Kore Hospital & MRC, Belagavi.
- To evaluate the acid base interpretation in patients with acute organophosphate poisoning presenting to KLES Dr. Prabhakar Kore Hospital & MRC, Belagavi.

Review of Literature

The esterification reaction between phosphoric acid and alcohol produces organophosphates, a class of chemical compounds that includes many different types. Pesticides, herbicides, and nerve agents are some of the current uses for organophosphates in chemical warfare. Insecticides and herbicides are the most common routes of exposure for patients to organophosphates. Organophosphates cause an excess of the neurotransmitter acetylcholine by blocking the enzyme acetylcholinesterase (AChE). Cholinergic toxidrome is a symptom of an excess of acetylcholine in the body; it affects the central nervous system and nicotinic and muscarinic receptors. Symptoms might start appearing minutes after exposure and take up to a week to go away, depending on the chemical in question.11-15

Clinical issues associated to organophosphate poisoning have persisted in underdeveloped nations despite a decrease in instances in affluent nations owing to stronger limitations on the use of these chemicals.Pesticides are frequently used as a means of self-harm due to their lethality and widespread availability in the developing world. Therefore, developing nations that heavily depend on agriculture and often have less stringent pesticide regulations result in the majority of cases of organophosphate toxicity. Research indicates that deliberate poisoning leads to a higher mortality rate than accidental exposure to these compounds.16,17When it comes to organophosphate poisoning, the most common cause of mortality is respiratory failure due to bronchorrhea and bronchospasm. It is also well-documented that there is neurological consequences and chronic toxicity, including the intermediate syndrome. Healthcare providers in industrialized countries need to be able to identify this poison since it might be used as a weapon in wars and terrorist attacks.

History of Organophosphates Use

Although the first organophosphate pesticide was created in the mid-1800s, it wasn't until after WWII that it became widely used. Initially, in the 1930s, these compounds were used as insecticides before finding application as neurotoxins by the German military. The organophosphate chemicals sarin and VX were utilized by the Japanese cult Aum Shinrikyo in 1994 and 1995, marking the initial reported instances of VX's use as a terrorist agent. In February 1997, the first reported murder involving VX occurred with the assassination of Kim Jong-Nam at a Malaysian airport. In March 2018, the poisoning of Sergei and Yulia Skripal took place in England, leading to the hospitalization of a police officer who was also poisoned during this assassination attempt. The compound used in this event was the organophosphorus agent known as Novichok.18,19

Etiology

Organophosphate toxicity can occur either due to occupational or accidental exposure to pesticides, intentional self-harm exposure, or chemical warfare and terrorist attacks. Over 50,000 compounds have been developed and evaluated for pesticidal activity. In the United States, 37 organophosphate pesticides have been registered, all of which have the potential to cause toxicity. In the developing world, this number is higher due to less stringent regulation of these compounds. Organophosphate exposure can occur through inhalation, ingestion, or contact with skin. Although these compounds are readily absorbed in the body after inhalation and ingestion, systemic absorption following dermal exposure shows more significant variability.

The onset, severity, and duration of toxicity depend on the ingested amount, absorption route, and the specific pesticide's toxicokinetics.20 The compounds are categorized into five classes by

the World Health Organization (WHO), starting with "Extremely hazardous" and ending with "Active ingredients unlikely to present acute hazard in normal use." The data used to classify these substances is taken from the median lethal dosage (LD50), which is the oral fatal dose for 50% of the subjects exposed to the active component in rats. But there are certain substances in the same class that are more hazardous than others, and the LD50 categorization can't tell them apart.

Epidemiology

Organophosphate exposure incidents peaked in 1997 with 20,135 cases and subsequently declined, according to a study that examined the annual reports of the Toxic Exposure Surveillance System (TESS) maintained by the American Association of Poison Control Centers. The study ran from 1995 to 2004.21 No fatalities were recorded in the 2079 cases of organophosphate exposure documented in the 2020 annual report of the National Poison Data System.22 The United States Environmental Protection Agency's decision to phase out the use of organophosphate pesticides in residential settings is largely responsible for this significant decrease in organophosphate exposure. The project started in 2000 and ran till 2005.21 The data collected from poison control centers' monitoring programs might not be representative of the overall exposure rates in the US, though. Reason being, these numbers could be under- or overstated depending on the exact substance in question, as they are based on self-reports or reports from medical providers.

The overall exposure rate of organophosphate and its accompanying toxicity can be difficult to determine with any degree of accuracy. Approximately one-third of all suicides in 2007 were caused by pesticide poisoning, which impacted an estimated 371,594 people worldwide.17

In 1990, WHO estimated that there were 1 million unintentional pesticide poisonings, resulting in approximately 20,000 fatalities. A 2020 study estimated 740,000 unintentional pesticide poisonings, resulting in 7446 deaths across 141 countries.23 The actual extent of exposure and toxicity is likely higher due to inadequate reporting and limited statistical data.

Pathophysiology

Acetylcholine is a neurotransmitter with widespread functionality in the nervous system. Acetylcholine is present in parasympathetic and sympathetic ganglia, all postganglionic parasympathetic nerves, the postganglionic sympathetic nerve innervating sweat glands, and skeletal neuromuscular junctions. Upon depolarization of an axon, acetylcholine is released into the synaptic cleft, which activates the postsynaptic receptors, resulting in the propagation of an action potential. Carboxylic ester hydrolases metabolize acetylcholine into acetic acid and choline through hydrolysis. This process occurs rapidly, with choline being reabsorbed into the presynaptic nerve to be used for the synthesis of additional acetylcholine. The main enzymes responsible for this metabolism are AChE and butyrylcholinesterase (BuChE). AChE is located in nervous and skeletal tissues, as well as on erythrocyte membranes. BuChE is present in plasma and various organs such as the liver, heart, pancreas, and brain. However, the function of BuChE remains partially understood.

Organophosphate pesticides are characterized by their ability to inhibit carboxyl ester hydrolases, more specifically AChE inhibition. By phosphorylating the serine hydroxyl group on AChE, these pesticides render the enzyme inactive. The buildup of acetylcholine within the synapse causes an overstimulation of both nicotinic and muscarinic receptors, as AChE is crucial for acetylcholine breakdown.

When the neuromuscular junction's nicotinic receptors are overstimulated, it can cause fasciculations, myoclonic jerks, and, in the end, flaccid paralysis from depolarizing blocks. Hypertension, perspiration, rapid heart rate, and left shift leukocytosis are some of the symptoms that may be caused by the presence of nicotine receptors in the adrenal glands. 24 - 27 The symptoms caused by organophosphate poisoning are caused by the fact that it acts on muscarinic receptors. These effects, which are mediated by a G-protein-coupled receptor pathway, usually take longer to manifest than nicotinic receptor actions. Both the parasympathetic and sympathetic nervous systems can be found to have muscarinic receptors. When the sympathetic nervous system is activated, the sweat glands are overstimulated, leading to profuse perspiration. Heart, exocrine glands, and smooth muscle systems are among those that can be affected by the parasympathetic effects of organophosphate poisoning. Bradycardia, bronchorrhea, and bronchospasm are serious, sometimes fatal complications of muscarinic overstimulation that can make breathing difficult. 28

Excessive acetylcholine in the CNS can cause CNS depression, leading to coma and seizures. In cases where patients ingest agricultural pesticides, the presence of co-formulants and alcohol also poses a concern. Pesticides are frequently combined with solvents and surfactants to form an emulsifiable concentrate rather than being in a pure organophosphate form. The extent of the toxicity associated with co-formulants remains uncertain. Considering that organophosphate toxicity can induce CNS depression and coma, the risk of aspirating these solvents is a considerable concern. Reports of aspiration pneumonitis and adult respiratory distress syndrome (ARDS) have emerged in cases of organophosphate toxicity. However, it remains uncertain whether these conditions are caused by the compound itself or its aspiration.29

Toxicokinetics

Organophosphate pesticides can be absorbed through multiple routes, including inhalation, ingestion, ocular contact, and dermal exposure, and inhalation results in the quickest absorption.30 Although systemic absorption varies after dermal exposure, it can be heightened by various factors such as broken skin, dermatitis, and elevated environmental temperatures. Oral ingestion is often associated with intentional self-harm attempts in adults and can also be observed with accidental exposures in children.

The exact time for peak plasma concentration after organophosphate exposure is unknown. However, a study involving human volunteers revealed that the time to peak plasma concentrations occurred approximately 6 hours after orally ingesting very low doses of chlorpyrifos.31 Notably, these findings may not apply to all organophosphate compounds, especially in cases of high-volume ingestion, as seen in intentional self-harm attempts. Furthermore, the study involved pure chlorpyrifos, which differs from agricultural pesticides and may include additives impacting the absorption and distribution of the organophosphate. This study also utilized pure chlorpyrifos, in contrast to agricultural pesticides that may incorporate additives capable of influencing the absorption and distribution of the organophosphate.

Most organophosphates exhibit lipophilic properties and possess a high volume of distribution. They distribute rapidly into adipose tissue, the kidneys, and the liver. Their extensive distribution provides protection from metabolism. The level of lipophilicity and the patient's adipose tissue can affect the outcome after poisoning. A 2014 Korean study examined the outcomes of 112 acutely poisoned patients, of whom 40 individuals were dealing with obesity. Individuals with a body mass index (BMI) of more than 25 experienced prolonged mechanical ventilation, extended stays in the intensive care unit (ICU), and an increased total length of hospital admission.32

The mobilization of unmetabolized organophosphates from fat stores can trigger a cholinergic crisis. This phenomenon is associated with highly lipophilic compounds and typically does not manifest in individuals with low lipophilicity and smaller volumes of distribution. Organophosphates can have a direct inhibitory effect on the AChE enzyme without necessitating initial metabolism after absorption. These direct-acting compounds are called oxons and differ from other compounds known as thions, which require metabolic activation within the body to become active. Thion organophosphate compounds are activated by cytochrome P450 (CYP450) enzymes, primarily located in the liver and intestine. The specific CYP450 enzymes involved may vary depending on the concentration and type of the organophosphate.33

When an organophosphate binds to the enzyme AChE, it undergoes cleavage, forming a stable yet reversible bond and rendering the AChE inactive. Although a regeneration process may occur, it proceeds more slowly than the inhibition and may take hours to days to restore AChE function completely. During its inactive state, the enzyme can potentially undergo the aging process, in which the initial reversible bond becomes irreversible, and enzyme regeneration can no longer occur. The time frame of aging varies among different organophosphate compounds. The antidote pralidoxime accelerates acetylcholine regeneration and reduces the number of inactive enzymes available for aging. Pralidoxime is effective only before the aging process, which is time-sensitive and dependent on the specific organophosphate compound involved.34 Once aging takes place, AChE can no longer be regenerated, thereby necessitating de novo synthesis for enzyme replenishment.

History and Physical Examination

When dealing with potential toxicity cases, it is important to consider the specific compound involved and the timing of exposure, especially in cases of intentional ingestion, as significant elements of the patient's medical history. Because various chemicals might have widely varying degrees of toxicity, it is important to try to keep the pesticide container intact so that a medical toxicologist or Poison Control Centre can collect this information if possible. Organophosphate compounds vary in their exposure routes, degrees or dosages, and the severity of toxicity, which in turn affects when symptoms begin and how long they last. The lipophilicity and other toxicokinetic properties of the chemical affect how long the toxicity lasts. The compound's release from fat storage might lead to repeated cholinergic effects in certain situations.35

In severe organophosphate toxicity, the prototypical patient may exhibit unresponsiveness, pinpoint pupils, muscle fasciculations, and diaphoresis. Additional symptoms can include emesis, diarrhea, excessive salivation, lacrimation, and urinary incontinence. In cases of intentional self-poisoning of organophosphates, the presence of a garlic or solvent odor may persist.

Evaluation

It is crucial to start therapy before laboratory confirmation of organophosphate poisoning since the diagnosis is based on clinical examination. When no prior exposure or consumption of organophosphates is apparent, a strong clinical suspicion for poisoning becomes even more important. Toxic effects are most often shown in patients with miotic pupils, sweating excessively, and difficulty breathing. An identifiable garlic or petroleum aroma is given off by certain organophosphates,which might be helpful in making a diagnosis.

It is possible to provide atropine as a trial if organophosphate poisoning is suspected but not proven. Suspicion of AChE inhibitor poisoning should be raised if symptoms improve after 0.6 to 1 mg of atropine. The sensitivity and specificity of this experiment might be difficult to evaluate, especially in situations of severe poisoning, due to the paucity of data. Hence, to solve this problem, further research is required. Patients with severe poisoning might not react to even a little dosage of atropine, leading to an inaccurate negative result.

While it's true that certain labs can detect cholinesterase activity directly, the findings may not be available quickly enough to inform treatment decisions when these tests are outsourced. Two cholinesterase enzymes that are frequently tested are BuChE and RBC AChE. Compared to RBC AChE activity, BuChE activity lacks specificity. Malnutrition, iron deficiency anaemia, liver disease, chronic sickness, and inherited enzyme failure can all lead to low BuChE activity. This test is already difficult to interpret since the amount of enzyme inhibition differs for each individual organophosphate poisoned and because there is a lack of data for many chemicals.

RBC AChE activity is believed to have a stronger correlation with the clinical features of organophosphate toxicity. In clinical settings, symptoms typically develop when more than 50% of this enzyme is inhibited, although this threshold can vary with specific compounds. Notably, it is essential to collect blood samples in appropriate tubes, as fluoride can deactivate the enzymes, potentially yielding falsely low activity levels.

Healthcare providers may order a range of essential laboratory tests, including specific diagnostic tests for organophosphate poisoning, as well as other tests to assess the patient's overall health. Some examples of these tests are a pregnancy test, a basic metabolic panel, tests for glucose levels, liver and kidney function, arterial blood gas analysis, and a complete blood

cell count (CBC). Sinus bradycardia, caused by parasympathetic activity, is usually shown on the electrocardiogram (ECG).

Treatment / Management

Oxygen administration, muscarinic antagonists (often atropine), fluids, and an oxime that removes the phosphate group from acetylcholinesterase are all part of the treatment plan.36 As needed, respiratory assistance is administered. After the patient has been completely stabilized and resuscitated, only then may gastric decontamination be addressed. Changes in atropine requirements, decreasing respiratory function due to intermediate syndrome, and recurring cholinergic characteristics occurring with fat-soluble organophosphorus should be closely monitored in patients following stabilization.37

Summary of treatment

Make sure the patient is breathing normally and that their blood is flowing properly. To lessen the likelihood of aspiration of gastric contents, place the patient in a left lateral posture, ideally with their head lower than their feet. If it is accessible, provide high flow oxygen. If the patient's airway or breathing is impaired, intubate them. Then, depending on the severity, have an intravenous line set up and bolus 1–3 milligrams of atropine. In order to maintain a systolic blood pressure more than 80 mm Hg and a urine output greater than

0.5 mL/kg/h, an infusion of 0.9% normal saline should be established.At the time of the first atropine dose, make sure to record the following vital signs: pulse rate, blood pressure, pupil size, sweating, and auscultatory findings. Then, inject 2 grams (or 250

mg) of pralidoxime chloride into a second cannula intravenously over 20 to 30 minutes. After that, administer 0.5 to 1 gramme (or 30 milligrams per hour) of pralidoxime (or 250 mg per hour of obidoxime) into a 0.9% normal saline solution.

Perform vital signs such as a pulse, blood pressure, perspiration, and chest sounds 5 minutes after administering atropine. Give double the first dosage of atropine if the patient does not show improvement after 5 minutes; keep monitoring their vitals and administer further doses of atropine as needed. Quit doubling the dosage once you see an improvement in the parameters. Although atropine will not remove specific sites of aspiration, it is important to provide boluses of the drug until the patient's heart rate reaches 80 beats per minute, systolic blood pressure reaches 80 mm Hg, and the chest is clear. Sweating often ends after a while. Since there are several potential causes of tachycardia, it is not a contraindication to atropine. Although dilated pupils are a typical side effect of atropine, this is not a good endpoint to utilize for starting therapy since there is a wait before the drug has its full impact. On the other hand, atropine poisoning is indicated by very dilated pupils.

Additional doses of atropine should be administered based on clinical judgement in cases when the patient's heart rate and blood pressure are slightly below their objectives, but the chest is clear. Additional atropine may not be necessary at this stage. The use of vasopressors may alleviate severe hypotension. At this time, it is unclear if vasopressors or greater dosages of atropine are more beneficial.

Initiate an atropine infusion and provide 10–20% of the whole dosage per hour until the patient is stable. Make sure to monitor the patient often to determine the appropriate

dosage of atropine. Cholinergic characteristics will return after a while if insufficient dosage is administered. Patients may experience agitation, pyrexia, and the development of absent bowel sounds and urine retention if administered in excess. Wait 30–60 minutes for these side effects to go away before restarting the infusion at a reduced pace if this occurs.

The patient should be extubated and the oxime infusion should be maintained until atropine is no longer needed, which should take 12 to 24 hours. Respiratory function should be monitored continuously. If a patient's vital capacity is less than 15 mL/kg or if their tidal volume is less than 5 mL/kg, or if they experience apneic episodes, or if their PaO2 is less than 8 kPa (60 mm Hg) on a FIO2 level more than 60%, intubation and ventilation should be performed.

Regularly ask aware patients to raise their head off the bed and hold it that way while pressing on their forehead to evaluate flexor neck strength. The patient is at danger for developing peripheral respiratory failure (intermediate syndrome) if there is any indication of weakness. In these cases, monitoring tidal volume should be done every four hours. A requirement for intubation and ventilation is indicated by values below 5 mL/kg.

Give benzodiazepines as prescribed and adjust the dosage of atropine as needed to treat agitation. Atropine, which blocks natural thermoregulatory responses like sweating, makes physical restraint of agitated patients in hot environments much riskier of severe hyperthermia. Therefore, it is crucial to have sufficient sedation. Recurring cholinergic crises caused by the release of fat-soluble organophosphorus from fat storage should be closely monitored. Ingesting certain organophosphorus can cause

such crises to last for days or even weeks. Patients who have cholinergic side effects again will require atropine and oxime retreatment.

There is a lack of data due to the small number of randomized studies conducted on this type of poisoning.36 There were no clinical trials conducted before the fast introduction of atropine and oximes to clinical practice in the 1950s. Consequently, the best treatment plans for each medication are unknown to us. The optimal method of administering the main therapies is still up for debate and varies greatly in practice, which makes it difficult to conduct trials of alternative interventions. This variation reduces the external validity of the study and makes it harder to create a generally approved study methodology.

Results and treatment efficacy

The reported case fatality rates by hospitals differ significantly, with one German critical care unit reporting 40% and the Poison Control Centre at Mach Mai hospital in Hanoi, Vietnam, reporting 1·85% (Pham Due, Personal Communication).38,39

It is tempting to compare the efficacy of treatments provided at various institutions due to the paucity of randomized studies. Many factors, unfortunately, complicate such comparisons.

Factors affecting outcome in organophosphorus pesticide self-poisoning

- The oral LD50 in rats is a common metric for toxicity ratings. This scale can generally distinguish between pesticides that are quite safe and those that are very toxic; for

instance, temephos (LD50 8600 mg/kg,40 WHO: unlikely to produce acute danger) has not been linked to fatalities, whereas parathion (LD50 13 mg/kg,40 WHO: Class IA) is extremely toxic.

- Impurities: pesticides produced recently are evaluated for their toxicity by the World Health Organisation (WHO). Chemical reactions involving pesticides stored in heated environments can produce harmful byproducts. The late 1970s saw the deaths of pesticide sprayers in Pakistan who were using malathion; similar incidents with diazinon and dimethoate have also been reported.41–44
- Toxic effects of pesticides are conditional on their formulation, which in turn are dependent on the organophosphorus and the country of production. For instance, in Sri Lanka, malathion is sold as a 3% powder, whereas in Burma, it is sold as an 80% solution.
- Subgroups of alkyl: The majority of pesticides are dimethyl organophosphorus, which has two methyl groups connected to the phosphate by oxygen atoms, or diethyl organophosphates, which have two ethyl groups. For oximes to be successful in treating dimethyl poisoning, patients need to be administered promptly since acetylcholinesterase ageing occurs significantly faster for dimethyl poisoning compared to diethyl poisoning. An unusual structural feature of some pesticides is the presence of an additional alkyl group (such as propyl in profenofos) linked to the phosphate group by means of a sulphur atom. It is quite unlikely that oximes will be effective against these organophosphorus insecticides, and acetylcholinesterase ages much more rapidly.
- Activation is required. Cytochrome P450 enzymes in the liver and intestines desulphurize many substances that are inactive thioates (with a phosphorus atom connected to it via a

double bond). When organophosphorus concentrations are high, as they are during self-poisoning, the P450 3A4 enzyme appears to be the most active.45

- The rate of activation and inhibition of AChE. Different pesticides have different activation rates for thioate organophosphates.45,46 The oxons of organophosphorus pesticides also vary greatly in the rate at which they inhibit acetylcholinesterase.
- How long an impact lasts—the solubility in fat and half-life. After absorption, a significant portion of some organophosphorus pesticides, such as thioate insecticides like fenthion, are transported to fat reserves. The early cholinergic symptoms are often moderate, and it appears to lower the peak blood organophosphorus concentration. Recurrent cholinergic symptoms, which can persist for many days or weeks, are caused by the subsequent sluggish redistribution and activation. These organophosphorus often cause peripheral respiratory failure, which is likely caused by the ongoing suppression of acetylcholinesterase. Oximes may potentially be useful for several days in these people as ageing does not begin until acetylcholinesterase inhibition is obstructive. Other organophosphorus compounds, such as dichlorvos, do not require activation, are not fat soluble, and may have a substantially shorter half-life and far quicker start of action. By utilising the Kow (logarithm octanol/water coefficient) as a grading system, fat solubility may be classified as either not fat soluble (less than 1·0) or extremely fat soluble (greater than 4·0). 47

Organophosphorus poisoning might start acting up quickly after intake, depending on the aforementioned circumstances. There is a higher risk of hypoxic brain injury and aspiration if symptoms and respiratory arrest begin early after ingesting an oxon organophosphorus, which quickly inhibits acetylcholinesterase. In as little as twenty minutes, the thioate organophosphorus

parathion can be converted to paraoxon, rendering the patient comatose. Dimethoate and fenthion are additional thioate organophosphorus poisons whose symptoms manifest later, providing patients more time to reach the hospital.

For example, despite the fact that many poisoning textbooks describe organophosphorus pesticides as though they were chemically equivalent, there are important chemistry-based variations that affect how well treatments work.48 Ingested pesticides determine factors such as the number of patients who make it to the hospital, their severity upon admission, the success of oxime therapy, the probability of future cholinergic crises, and the necessity of respiratory assistance. The significance of randomized trials in determining the efficacy of treatments for particular pesticides is reinforced by this variance.

Initial stabilization

An immediate medical crisis exists in cases of severe acute organophosphorus pesticide exposure. Proper breathing, circulation, and maintenance of a patent airway are essential components of effective treatment. It is ideal to administer oxygen as soon as possible. Contrary to popular belief, there is insufficient evidence to back the idea that atropine should not be administered prior to the availability of oxygen.49,50Early use of atropine to patients with pesticide poisoning can decrease secretions and improve respiratory function, which is especially important in hospitals without access to oxygen. The patient has to be positioned with their neck extended in the left lateral position. This posture lessens the likelihood of aspiration, aids in maintaining an open airway, and may lessen the rate of pyloric emptying and poison

absorption.51,52 As part of supportive care, it is important to provide fluids and monitor blood glucose levels.

When stabilizing patients who have been poisoned with organophosphorus, healthcare professionals are believed to be at danger of poisoning.53,54 There have been reports of this kind of poisoning in a small number of Western hospitals, but no studies have demonstrated that healthcare workers' acetylcholinesterase or butyrylcholinesterase inhibitory effects are due to high levels of organophosphorus exposure.55 Some side effects, such nausea and headaches, might be anxiety or exposure to the pesticide's organic solvent (like xylene).55,56

Annually, basic hospitals across Asia treat hundreds of thousands of patients with severe organophosphorus poisoning; neither patients nor healthcare providers are protected from subsequent poisoning since no additional measures are taken. Hospital staff's reluctance to treat pesticide poisoning patients endangers such patients. The effects of solvents and pesticides should be minimized by following guidelines that call for maximum ventilation, regular crew rotation, and universal precautions.55

Muscarinic antagonist drugs

Other muscarinic antagonists have been investigated in animal studies, while atropine is still the cornerstone of treatment globally.49 Their ability to enter the central nervous system is a key distinction among these medications.57 While neither glycopyrronium bromide nor hyoscine methobromide penetrate the central nervous system, hyoscine does so very well; atropine does so, but not to the same extent as hyoscine.

When patients are given an excessive amount of atropine, the most serious side effect they may experience is anticholinergic delirium.49 Consequently, glycopyrronium is preferred by certain doctors as a means of treating the peripheral effects of organophosphorus that does not lead to disorientation. Patients suffering from the cholinergic syndrome often have decreased respiration and unconsciousness, and this medication may not be able to help because of its weak central nervous system penetration. Although it lacked the ability to discern subtle variations between treatments, a small randomized controlled study comparing glycopyrronium and atropine found no statistically significant difference in mortality or ventilation rates.58 Despite little peripheral indications, a patient with significant extrapyramidal characteristics was effectively treated with hyoscine.59 It seems to be more effective than atropine in controlling seizures caused by inhaled organophosphorus nerve agents, according to animal studies.60 Organophosphorus poisoning does not typically cause seizures or extrapyramidal symptoms. Because of its accessibility, low cost, and modest CNS penetration, atropine will likely continue to be the antimuscarinic drug of choice until alternative muscarinic antagonists prove to have a higher benefit-to-harm ratio in high-quality randomized studies. To the best of our knowledge, no randomized controlled studies have evaluated loading or continuation atropine regimens against one another.

Improving cardiac and respiratory function and reversing cholinergic characteristics are the primary goals of early treatment. To quickly reverse bronchospasm and bronchorrhoea, they employ a regimen of doubling dosages in an effort to raise the pulse rate and systolic blood pressure over 80 mm Hg. With this protocol, a patient can get as much as 70 mg of atropine in stages in under 30 minutes, leading to fast stabilization with little atropine toxicity risk. While one research out of southern India found that atropine infusions were more effective than

multiple bolus dosages, the study's reliance on historical controls casts doubt on the reliability of its results. A key advantage for hospitals with limited personnel is that infusions might decrease changes in blood atropine levels, which would mean less need for regular patient observation. 61

Oximes

When organophosphorus inhibits acetylcholinesterase, oximes can restore its activity. Midway through the 1950s, Wilson and colleagues found paratoxime, and it was quickly and effectively used in clinical practice to treat parathion toxicity. Despite the development of other oximes like obidoxime and trimedoxime, pralidoxime is still the most commonly used one. Iodide, chloride, methylsulfate, and mesylate are its four salts.62 Methylsulfate and mesylate are mostly utilised in France, Belgium, and the UK, while the chloride and iodide compounds find widespread application. In comparison to iodide, the chloride salt offers several benefits, the most notable of which is its lower molecular weight (173 vs. 264), which results in 1.5 times the amount of active chemical per gramme of salt. Patients can potentially have thyroid toxicity when using pralidoxime iodide at high dosages over an extended length of time.63

Although pralidoxime was first shown to help with parathion overdose, many Asian practitioners are still not convinced of its efficacy.64–66 Specifically, two randomized controlled studies conducted in the early 1990s in Vellore, India, found that pralidoxime infusions at low doses might be harmful.67,68 A problem with the trial's design (such as an inefficient dosage or an unfair allocation) might explain why there was no therapeutic benefit. It is also possible that the patients visited at this hospital are not responding to pralidoxime, either because of the specific pesticide they swallowed, the amount they consumed, or the length of time it takes until they are given pralidoxime.69,70

Three meta-analyses including pralidoxime have been published: one from the Cochrane Collaboration and two others.71–73Despite including two randomized controlled trials, the Cochrane review failed to find any conclusive evidence of benefit or harm.68,69 We can't be too sure that oximes are dangerous because past meta-analyses mixed randomized controlled trials with observational studies that weren't randomized or weren't controlled historically. 64,67,68,74–81

After these meta-analyses were finished, researchers in Baramati, India, randomized 200 patients with moderate organophosphorus poisoning (not including very sick patients) to see how 200 mg of very-high-dose pralidoxime iodide (2 g loading dose, then 1 g hourly or every 4 hours for 48 hours, then 1 g every 4 hours until recovery) affected them.82 Case fatality was 1% lower in the high-dose regimen group compared to 8% in the low-dose group, pneumonia was 8% lower in the high-dose group compared to 35% lower in the low-dose group, and the median number of days spent on mechanical ventilation was 5 days instead of 10 days. There were no controlled laboratory investigations to determine the specific pesticide used, the extent of baseline acetylcholinesterase inhibition, or the rate of reversal.63 On the other hand, if patients are treated early and get adequate supportive care, this study implies that big doses of pralidoxime might be beneficial.

It appears that the capacity to restore acetylcholinesterase inhibition with oximes differs depending on the pesticide consumed, according to observational studies of pralidoxime and obidoxime. 69,83 When it comes to acetylcholinesterase inhibitors, oximes tend to work well with diethyl pesticides like quinalphos and parathion, but they don't seem to have much of an impact with dimethyl organophosphoruses like monocrotophos or oxydemeton-methyl. Oximes have no effect on acetylcholinesterase inhibition by S-alkyl-linked organophosphorus, as is

shown with profenofos. The fact that various herbicides promote acetylcholinesterase ageing at different rates likely contributes to the observed discrepancy. Surprisingly, high-dose pralidoxime had no discernible effect in the Baramati investigation on mild cases of dimethyl or diethyl organophosphorus poisoning. To determine if this advantage is still there for severe poisoning, moreinvestigations are required.82

Following poisoning, oximes completely reactivated acetylcholinesterase in A quinalphos, a diethyl pesticide; partially in B oxydemeton-methyl, a dimethyl pesticide; and completely in C profenofos, an S-alkyl pesticide. An arrow pointing to the timing of the first pralidoxime dosage is displayed. Around 600 mU/μmol Hb is the normal range for acetylcholinesterase activity. In all three instances, in-vitro acetylcholinesterase activity was measured upon admission, when the initial dosage of pralidoxime was administered, to determine the amount of enzyme that could be reactivated, or how much had not yet aged. Approximately 85% (A), 50% (B), and 5% (C) of the enzyme was not aged. Within four hours, every patient had made it to the hospital.

Reactions of acetylcholinesterase after inhibition with organophosphorus

There is a gradual yet spontaneous reactivation of inhibited acetylcholinesterase. Depending on the organophosphorus, the reactivation half-life can be anywhere from 1 hour for dimethyl to 30 hours for diethyl. Oximes expedite this reactivation process. Regrettably, freshly reactivated acetylcholinesterase will be quickly reinhibited if there is a large quantity of organophosphorus. The relative amounts and affinities of the organophosphorus and oxime, as well as the kind of organophosphorus, determine whether inhibition or reactivation is more prevalent. Loss of one of the two alkyl groups connected to the bound phosphate might cause inhibited acetylcholinesterase to age as well. It is not possible to use oximes to reawaken aged

acetylcholinesterase. Depending on the inhibiting pesticide, the half-life of ageing can be anywhere from 3 hours for dimethyl to 33 hours for diethyl. Accordingly, there are significant clinical implications of ageing.

About half of the acetylcholinesterase will have aged and become insensitive to oximes by the time a patient who has consumed a dimethyl insecticide arrives at the hospital three hours after ingestion. Oximes will not work on a patient who arrives after 12 hours since their acetylcholinesterase is around 94% old. Transferring patients to a secondary hospital for oxime administration is a typical occurrence. Because oximes can be effective for up to five days after consumption and diethyl insecticides require 33 hours to achieve 50% inhibition, the situation is improved.

Atypical organophosphorus poisoning, especially with compounds like profenofos that lack both methyl and ethyl groups, appears to accelerate the ageing process. Since the half-life of oximes appears to be significantly less than 1 hour, they will not work at all if the patient arrives more than an hour or two after taking them.

It is important to consider this variation in pesticide responses when interpreting clinical information pertaining to oximes.69 The pesticide re-inhibits any acetylcholinesterase that the oximes reactivate, which limits the clinical consequences. High blood concentrations of organophosphorus after a substantial dosage can further mitigate these effects. If a patient has a serious consequence, like aspiration pneumonia or hypoxic brain damage, before therapy, oximes will not improve outcomes. Parathion and dichlorvos are two examples of fast-acting pesticides that are known to cause these kinds of side effects.

Whenever a patient is experiencing symptoms and requires atropine, the World Health Organization suggests administering an oxime.84 The administration of a loading dose of pralidoxime chloride or obidoxime, followed by a continuous infusion, is necessary to guarantee a therapeutic concentration. Quickly administering the oxime loading dosage as a bolus raises the risk of vomiting (and aspiration), tachycardia, and diastolic hypertension; hence, this approach should not be used.

Benzodiazepines

It is common for patients who have been poisoned with organophosphorus to experience agitated delirium. The pesticide, atropine toxicity, hypoxia, alcohol consumption, poisoning, and medical issues all play a role in the complicated aetiology. Although treating or preventing underlying causes is the cornerstone of care, medication is necessary for certain individuals. Diazepam is useful for treating people who are extremely disturbed.

While diazepam is the drug of choice for treating seizures, it is unusual for individuals with pesticide exposure who are adequately oxygenated to experience seizures.48 It appears that organophosphorus nerve agents, such soman and tabun, are associated with an increased risk of seizures.85 Although there is a dearth of human trials on diazepam, animal research suggests that it mitigates brain damage86 and protects against respiratory failure and mortality 87.

Gastrointestinal decontamination

Unfortunately, resuscitation and antidote administration are frequently postponed in favor of gastric lavage when poisoned patients arrive at the hospital.88 Gastric cleansing has not been shown to help organophosphorus poisoning patients in any way.36 Patients should not undergo

gastric decontamination until they have been stabilized and treated with oxygen, atropine, and an oxime.88

The lack of randomized controlled trials to establish the effectiveness of gastric lavage as a cleaning method for organophosphorus poisoning has not stopped its widespread use.36 Organophosphorus absorption rate from the human intestines is unknown; however, the quick start of poisoning in animals89 and humans39 indicates that absorption is quick, happening within minutes of swallowing, for some pesticides. Thus, the window of opportunity for efficient lavage is likely to be brief. Lavage should only be considered in cases when the patient comes within one hour of swallowing the poison, according to guidelines for the treatment of medication self-poisoning.90 Lavage should likely only be done for intubated or cooperative patients who appear shortly after ingesting a large amount of hazardous pesticide; however, the applicability of these recommendations to organophosphorus poisoning is debatable. 91 It is quite improbable that large quantities of organophosphorus would remain in the stomach following a single lavage, however repeated gastric lavages are advised in China to eliminate any leftover pesticides.

Organophosphorus pesticide poisoning is not an appropriate indication for ipecacuanha-induced emesis.36,93If ipecacuanha is administered to a patient who has been poisoned with organophosphorus, they run the danger of becoming unconscious quickly and perhaps aspirating. By forcing fluid down the pylorus and into the small bowel, mechanically inducing emesis with large amounts of water likely increases the rate of absorption.93

More than a thousand pesticide poisoned individuals were included in a randomized controlled study in Sri Lanka that compared the efficacy of single and repeated doses of super- activated charcoal to a placebo. Neither regimen was shown to provide a meaningful advantage.94 The

lack of impact in patients might be attributed to the pesticide's quick absorption into the blood, as activated charcoal binds organophosphorus in vitro. 95Another possible cause of fatal occurrences is when the dosage is excessively high in relation to the amount of charcoal provided, when the charcoal is administered at the wrong time, or when the solvent prevents proper binding. There is no proof that activated charcoal helps those who have poisoning from pesticides.

Other therapies

The therapeutic methods that are now in use are somewhat limited.96 Research on a number of novel treatments has shown conflicting findings. But maybe in the future we'll find a number of inexpensive medicines that operate at different locations that could supplement the ones we have currently.

In order to improve function at neuromuscular junctions and prevent central nervous system overstimulation caused by NMDA receptor activation, magnesium sulphate inhibits ligand-gated calcium channels, which in turn reduces acetylcholine release from pre-synaptic terminals.97 Magnesium sulphate was shown to lower death in a trial involving individuals poisoned with organophosphorus pesticides (0/11 0% versus 5/34 14.7 percent; $p<0.01$).98 Be cautious when interpreting these results, though, because the trial was small, the allocation was not randomized (every fourth patient got the intervention), and the journal didn't fully detail the dosage of magnesium sulphate used and other parts of the methodology.

Another drug that decreases acetylcholine production and release from presynaptic terminals is clonidine, an alpha2-adrenergic receptor agonist. Treatment with clonidine, particularly when

combined with atropine, has been beneficial in animal tests, although its effects on humans remain unclear.99

In cases of organophosphorus poisoning, sodium bicarbonate is occasionally administered to patients in Brazil and Iran instead of oximes.According to some reports, dogs can benefit from high blood pH levels (up to 7.45–7.55) for reasons that are still unclear. On the other hand, a Cochrane review found that there is currently not enough evidence to determine if sodium bicarbonate should be used to treat organophosphorus poisoning in humans. Other treatments may work more effectively if organophosphorus is removed from the blood. Haemofiltration may be helpful following dichlorvos poisoning; the drug has low fat solubility and should thus have a small volume of distribution; however, the exact roles of haemodialysis and haemofiltration are still unclear. The study was conducted in China and was not randomized. To develop solid evidence-based therapy recommendations, randomized controlled trials are required, although a comprehensive evaluation of various medicines for organophosphorus poisoning is now under progress.

Butyrylcholinesterase reduces the quantity of organophosphorus in plasma that can inhibit acetylcholinesterase in synapses by scavenging it.105 The cloned enzyme is now being studied for potential use in military applications, namely in the injection of soldiers before to their exposure to organophosphorus nerve gases.106 We can't tell when someone is going to swallow the pesticide, thus this preventative measure isn't workable for organophosphorus poisoning. Turkish medical professionals have treated poisoned patients with butyrylcholinesterase in fresh frozen plasma, according to 107 reports. Although there was a benefit shown in a small controlled trial (12 patients given fresh frozen plasma with 21 control patients), the experiment was not randomized, and the decisions regarding allocation were not made apparent.

Measurement System for Peradeniya Organophosphorus Poisoning (POP) A scoring system called the Peradeniya Organophosphorus Poisoning (POP) Scale was established in 1993 by N Senanayake, H J de Silva, and L Karalliedde. As parameters, we take OP poisoning symptoms that are commonly seen in patients and rate them on a scale from 0 to 2.

Peradeniya Organophosphorus Poisoning Score:

	Clinical criteria	Score
Pupil size	>2 mm	0
	<2 mm	1
	Pin-point	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 or continuous	1
	Both, generalized and continuous	2
Level of consciousness	Conscious and rationale	0
	Impaired response to verbal commands	1
	No response to verbal commands	2
Seizures	Absent	0
	Present	1

According to research by Syed M. Ahmed et al., 79.86% of the instances were considered suicidal, while the other cases were classified as accidental. The time that patients were on mechanical ventilation ranged from just 48 hours to over 7 days. For patients who needed mechanical breathing for longer than 7 days, for 5 to 7 days, and for 2 to 4 days, the corresponding mortality rates were 33.3%, 7.2%, and 100%. None of the thirteen patients who experienced a lag period of more than six hours did not make it. When PAM was initiated 6 to 12 hours after poisoning, 17.1% of patients died; when it was initiated 13 to 24 hours after poisoning, 28.1% of patients died. The duration of mechanical breathing and total dosage of PAM were positively correlated with the lag time of commencing PAM, with a statistically

significant connection ($P < 0.0001$). Independent predictors of mortality did not include age, lag time, poisoning severity, or length of breathing. The total number of fatalities was 18.6%.108

A total of 45 out of 46 cases (97.83%) were determined to be suicidal, according to research by Gurulingappa Patil et al. Nine patients out of forty-six required intubation and mechanical ventilation. The time that patients were on mechanical ventilation ranged from just 48 hours to over 7 days. In the groups that needed mechanical ventilation for more than 7 days, 2 to 7 days, and less than 2 days, the mortality rate was 50%, 0%, and 100%, respectively. Age, poisoning severity, cholinesterase levels, and length of breathing were all factors in mortality, but none of them were independent predictors. The overall death rate for those who needed mechanical breathing assistance was 22.22 percent.109

In their investigation, Mohit Desai et al. found that 24 individuals suffered from hypokalemia. Twenty individuals (51.3%) out of 39 male patients had hypokalemia. Hypokalemia was seen in 4 individuals (36.4%) out of 11 female patients. Fourteen out of fifty patients, or 28%, developed hyponatremia, defined as a sodium level below 135 meq/dl. Hyponatremia was associated with hypokalemia in 6 individuals (42.9 percent). Six instances of hyponatremia constitute 25% of the twenty-four individuals diagnosed with hypokalemia. In cases of organophosphorus chemical poisoning, hypokalemia considerably raises the risk of both illness and death. In cases of organophosphorus chemical poisoning, hypokalemia can be utilised as an economical and dependable indicator of death and morbidity.110

Thirty patients—22 males and 8 females—involved in the study were participants in the investigation by Vandana Dandekar et al. Thirty patients were categorized using the Proud foot method for OPC poisoning based on their clinical presentation. Hypokalemia occurred in 13.4%

of the patients (68.4% of the total). A mean blood K+ level of 2.90 ± 0.11 was associated with the development of fasciculations and muscle weakness. When the average serum K+ levels were around 0.10 mmol/L, ventilator support became necessary. When the average serum K+ level dropped to 2.7 ± 0.06 mmol/L, death was recorded. There was a substantial ($P < 0.001$) correlation between the level of serum K+ and the ventilator support and death. Patients with hypokalemia and severely decreased PChE who also had respiratory distress were more likely to die. A dramatic drop in serum K+ and PChE levels was associated with severe OP poisoning symptoms. Therefore, these biochemical results should be supported as indicators of the seriousness of op poisoning. If a patient has hypokalemia after being poisoned with OPC, medical toxicologists and clinicians should be concerned since it indicates a dismal prognosis.111

Researchers Bijush Difoesa et al. found that out of 108 patients admitted with organophosphorus poisoning, 72% were discharged with normal serum potassium levels upon admission, while 22% ($n = 22$) of the patients who passed away had hypokalemia upon admission. A chi-square value of 0.001 ($P < 0.05$) indicates that there is a statistically significant connection between serum potassium and outcome. According to the Peradeniya OP poisoning scale, there was a strong correlation between the severity of acute organophosphate poisoning and the serum potassium level on the day of admission. A worse prognosis was observed in patients whose serum potassium levels were lower at admission. There was a strong correlation between low serum potassium levels and the requirement for a ventilator. Organophosphorus toxicity can thus be predicted by serum potassium levels. There will be a decrease in mortality rates and better patient triage as a result of this. 112

Materials and Methods

Source of data: Patients above the age of 18 years admitted to KLEs Dr.Prabhakar Kore Hospital and MRC, with diagnosis of organophosphate poisoning during the 1-year period from January 2023 to December 2023.

Study design: Hospital based study

Study period: January 2023 to December 2023

Sample size: The minimum sample size formula based on prevalence rate is

$$n = \frac{z_\alpha{}^2 P(1-P)}{d^2}$$

where P is the prevalence rate and d is the percentage likely difference in the prevalence.

z_α is linked with the level of significance. For 5% level of the significance $z_\alpha = 1.96$.

The parameter considered in the calculation is the prevalence rate of most common type of OP consumed which is Monocrotophose which is at 62%.111

With P = 62% and d = 20% of P = 12.4%, the sample size is 59.

To round off sample size was increased to 60.

Sampling technique: Purposive sampling. Patients of age 18 and older with diagnosis of organophosphate poisoning was included in the study.

Inclusion criteria:

- Adult patients (aged 18 and older) with a confirmed diagnosis of organophosphate poisoning. The diagnosis of acute organophosphate poisoning was established using the following criteria:
    - A documented history of exposure to organophosphates
    - Clinical presentation with muscarinic or nicotinic symptoms indicative of organophosphate poisoning, such as pinpoint pupils (miosis), excessive salivation, or muscle twitching (fasciculations)
    - A decreased level of plasma cholinesterase
- To be eligible for inclusion in this study, patients must meet all three criteria and must have been admitted to the hospital within 24 hours of ingesting the organophosphate poison.

Exclusion criteria:

- Patients who have already received PAM.
- Patients with double insecticide or multiple poisoning with other drugs.
- Patients having previous illnesses which can lead to hypokalemia (eg. diuretic use, insulin overdose, laxative abuse, acidosis, corticosteroids)

Data collection procedure:

Blood investigations of patients with organophosphate poisoning was sent on the day of admission, serially on subsequent days and on the day of discharge. Blood investigations required are serial serum potassium levels and serum acetylcholinesterase levels at the time of admission and discharge.

Investigations required in patients:

- Plasma cholinesterase concentrations
- Biochemistry including:
    - Serial serum potassium levels
    - White blood cell (WBC) counts
    - Arterial blood gas analysis

Budget analysis:

- Blood investigations – 50,000
- Printing and copying supplies – 12,000
- Miscellaneous – 10,000
- Total – 72,000

Statistical Analysis

All the collected data was entered in Microsoft Excel sheet and then transferred to SPSS software ver. 22 for analysis. Qualitative data was presented as frequency and percentages and analyzed using chi-square test. Quantitative data was presented as mean and SD and compared by t-test. P-value < 0.05 was taken as level of significance.

Results

Table no 1: Age distribution amongst study population

Age group	No of Patients (n)	Percent (%)
Less than 25 years	11	19
25 to 30 years	28	46
31 to 40 years	11	17
41 to 50 years	5	9
more than 50 years	5	9
Total	60	100

As seen in the above table, most of the study population belongs to the age group of 25 to 30 years (46%) followed by Less than 25 years (19%) and 30 to 40 years (17%).

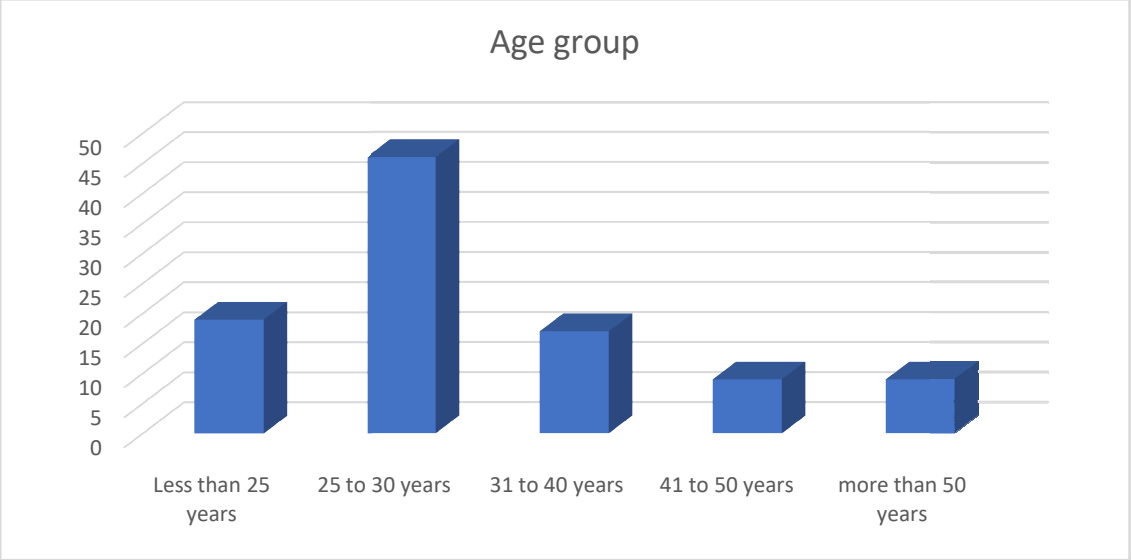

Table no 2: Gender distribution amongst study population

Gender	Frequency (n)	Percent (%)
Female	16	26.7
Male	44	73.3
Total	60	100

As seen in the above table, there was male predominance (73.3%) as compared to female (26.7%).

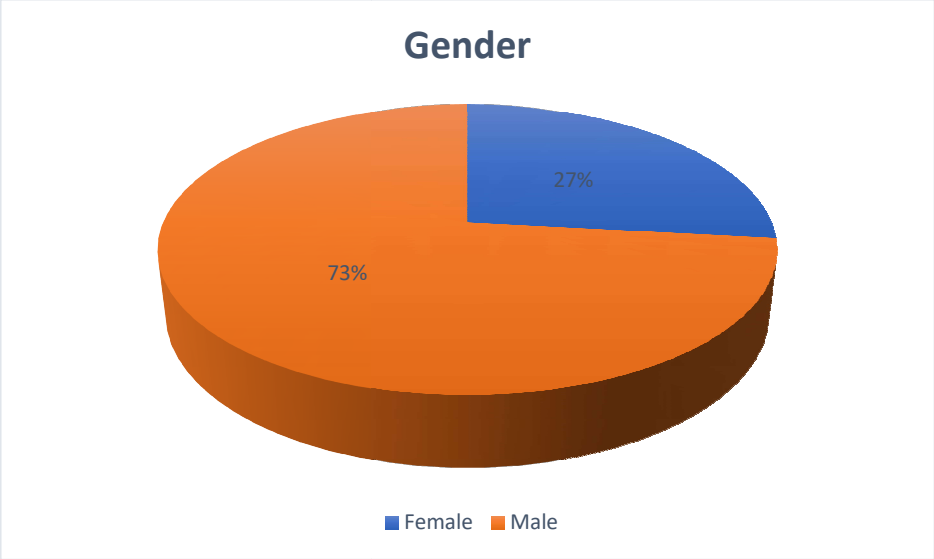

Table no 3: Type of OPC compound

Type of OPC compound	Frequency (n)	Percent (%)
Dichlorphos	2	3.3
Dimethoate	4	6.7
Parathion	2	3.3
Phorate	42	70
Unknown	10	16.7
Total	60	100

As seen in the above table, phorate (70%) was the most common type of OPC poisoning followed by dimethoate (6.7%), dichlorphos (3.3%) and parathion (3.3%)

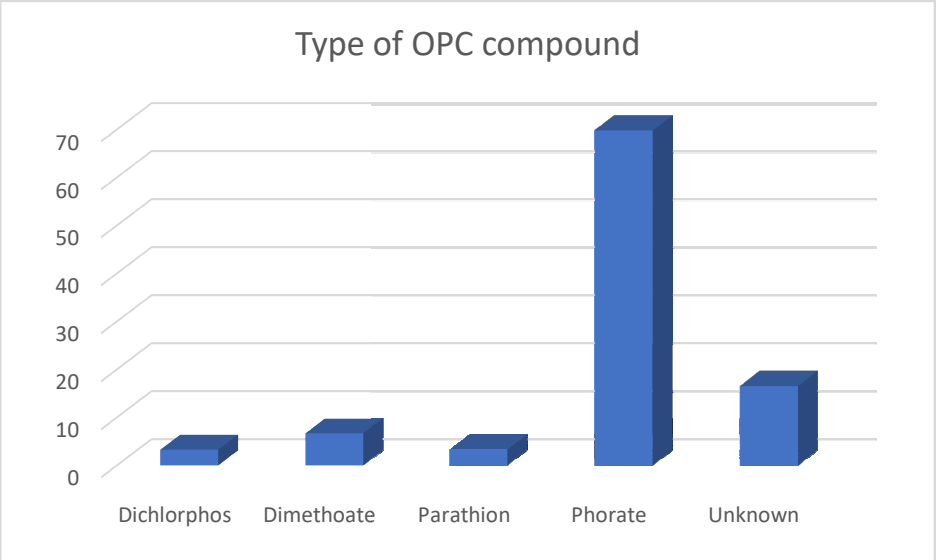

Table no 4: Incidence of Hypokalemia

Hypokalemia	Frequency (n)	Percent (%)
Absent	22	36.7
Present	38	63.3
Total	60	100

As seen in the above table, Hypokalemia was observed in 63.3% of OPC poisoning cases.

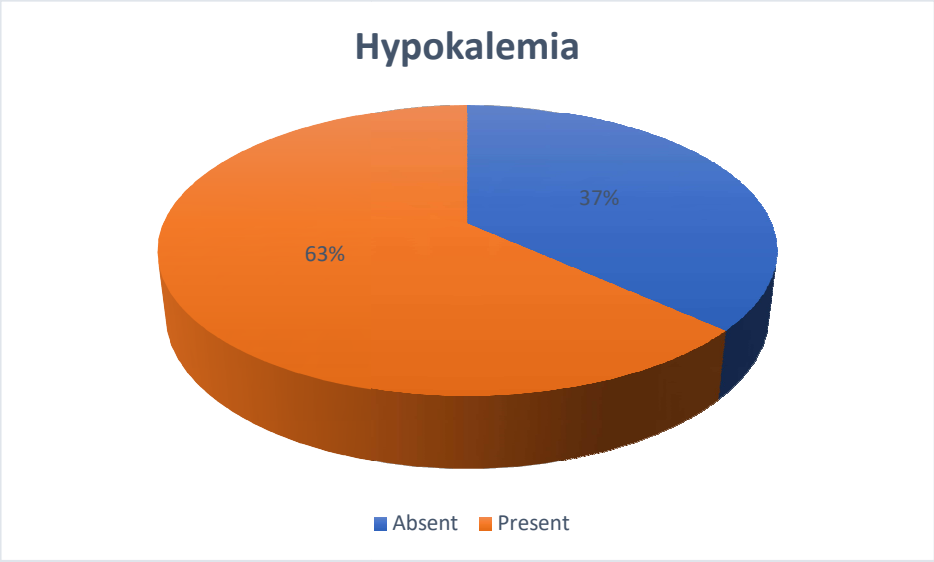

Table no 5: Type of OPC compound vs Hypokalemia

			Hypokalemia		Total
			No	Yes	
Type of OPC compound	Dichlorphos	Count	2 (9.10%)	0 (0.00%)	2 (3.30%)
	Dimethoate	Count	2 (9.10%)	2 (5.30%)	4 (6.70%)
	Parathion	Count	0 (0.00%)	2 (5.30%)	2 (3.30%)
	Phorate	Count	16 (72.70%)	26 (68.40%)	42 (70.00%)
	Unknown	Count	2 (9.10%)	8 (21.10%)	10 (16.70%)
Total		Count	22 (100.00%)	38(100.00%)	60(100.00%)

P value -0.545

As seen in the above table, hypokalemia was observed most commonly in phorate OPC poisoning cases (68.4%) followed by parathion (5.3%) and dimethoate (5.3%) though the difference was statistically insignificant.

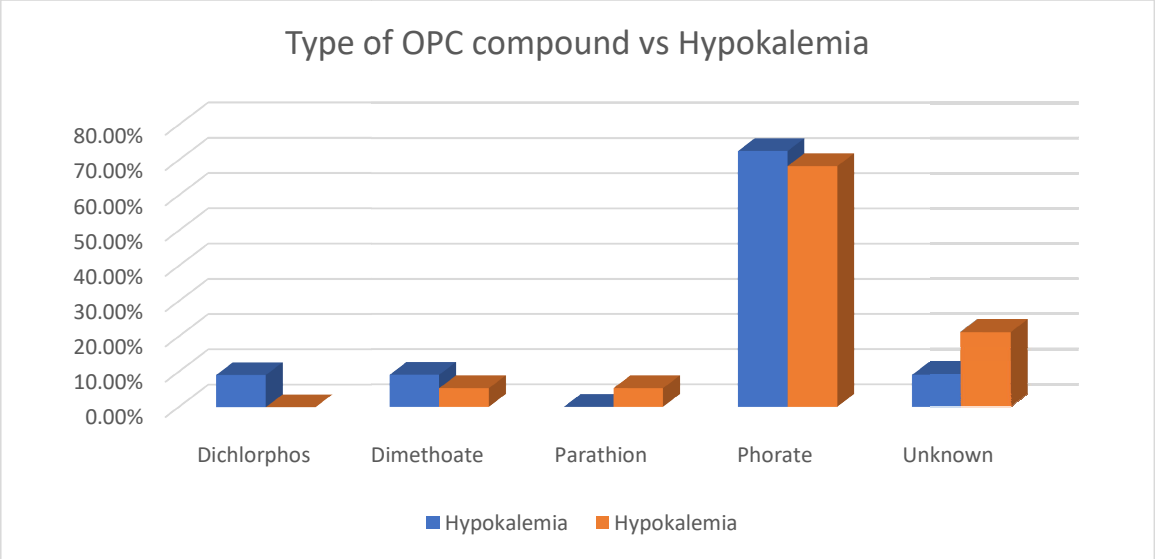

Table no 6: Grade (POP Score) of OPC poisoning vs Hypokalemia

			Hypokalemia		Total
			No	Yes	
Grade of OPC poisoning	Mild	Count	8 (36.40%)	8 (21.10%)	16 (26.70%)
	Moderate	Count	10 (45.50%)	14 (36.80%)	24 (40.00%)
	Severe	Count	4 (18.20%)	16 (42.10%)	20 (33.30%)
Total		Count	22 (100.00%)	38 (100.00%)	60 (100.00%)

P value -0.01

As seen in the above table, hypokalemia was observed most commonly in severe grade of OPC poisoning cases (42.2%) followed by moderate grade (36.8%) and mild (21.1%) though the difference was statistically significant.

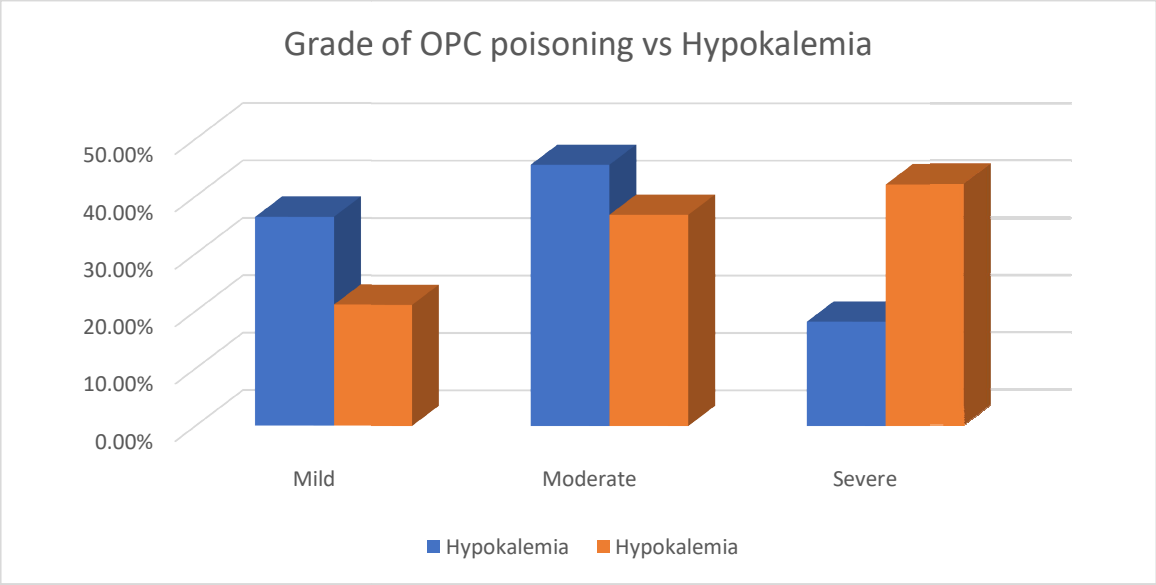

Table no 7: Requirement of ventilator vs Hypokalemia

			Hypokalemia		Total
			No	Yes	
Requirement of ventilator	No	Count	21 (90.90%)	20 (52.60%)	41 (66.70%)
	Yes	Count	1 (9.10%)	18 (47.40%)	19 (33.30%)
Total		Count	22 (100.00%)	38 (100.00%)	60 (100.00%)

P value -0.03

As seen in the above table, ventilatorrequirement was observed in 47.4% ofHypokalemic OPC poisoning cases as compared to 9.1% of normokalemic OPC poisoning cases and the difference was statistically significant.

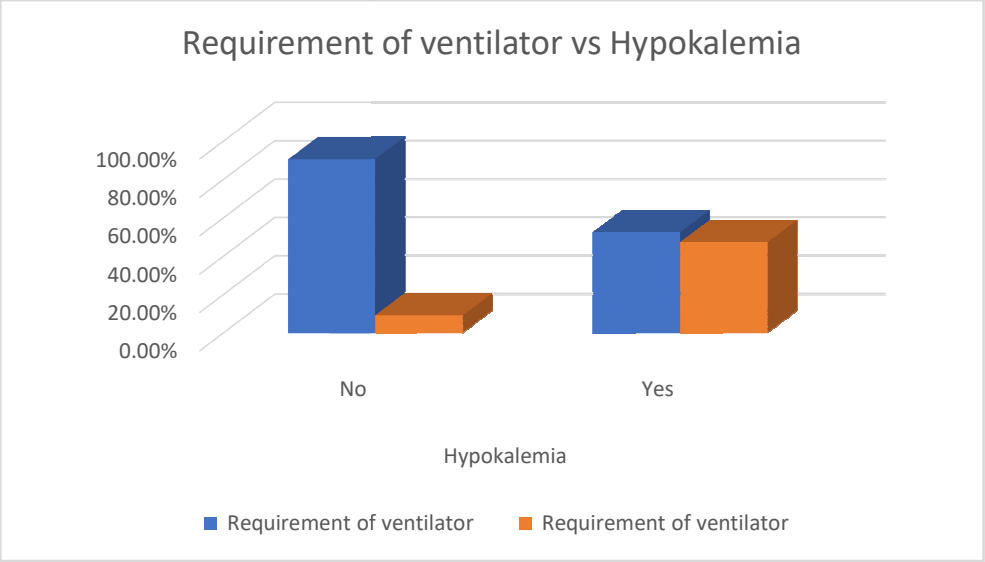

Table no 8: Convulsion vs Hypokalemia

			Hypokalemia		Total
			No	Yes	
Convulsion	No	Count	20 (90.90%)	29 (76.32%)	49 (81.67%)
	Yes	Count	2 (9.10%)	9 (23.68%)	11 (18.33%)
Total		Count	22 (100.00%)	38 (100.00%)	60 (100.00%)

P value -0.256

As seen in the above table, convulsion was observed in 23.68% of Hypokalemic OPC poisoning cases as compared to 9.1% of normokalemic OPC poisoning cases and the difference was statistically insignificant.

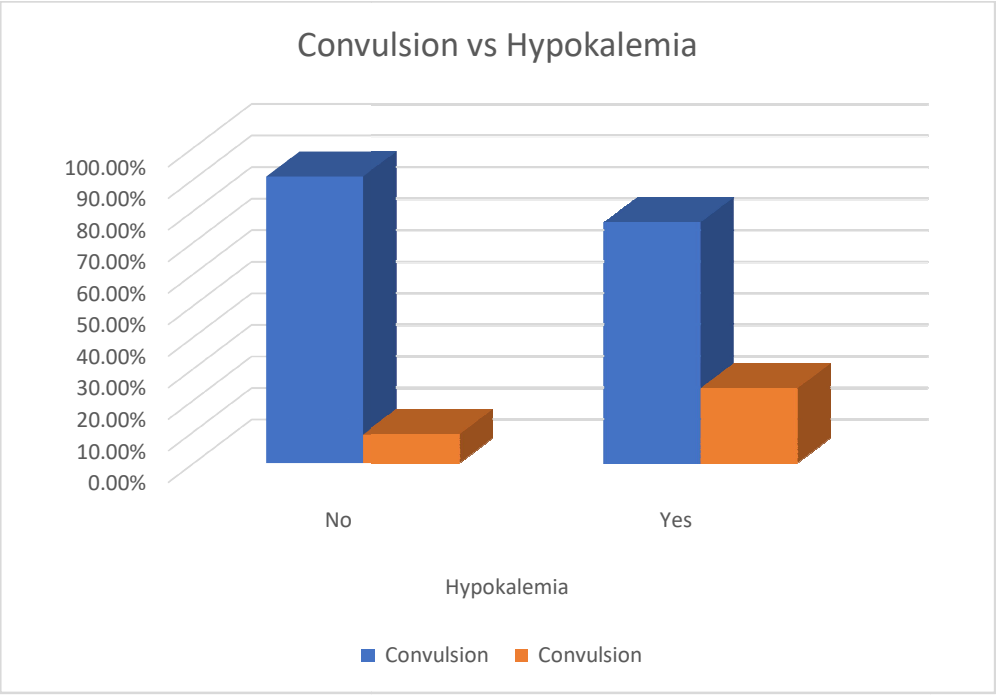

Table no 9: Fasciculation vs Hypokalemia

			Hypokalemia		Total
			No	Yes	
Fasciculation	No	Count	9 (40.91%)	7 (18.42%)	16 (26.67%)
	Yes	Count	13 (59.09%)	31 (81.58%)	44 (73.33%)
Total		Count	22 (100.00%)	38 (100.00%)	60 (100.00%)

P value -0.160

As seen in the above table, fasciculationswere observed in 81.58% of Hypokalemic OPC poisoning cases as compared to 59.09% of normokalemic OPC poisoning cases and the difference was statistically insignificant.

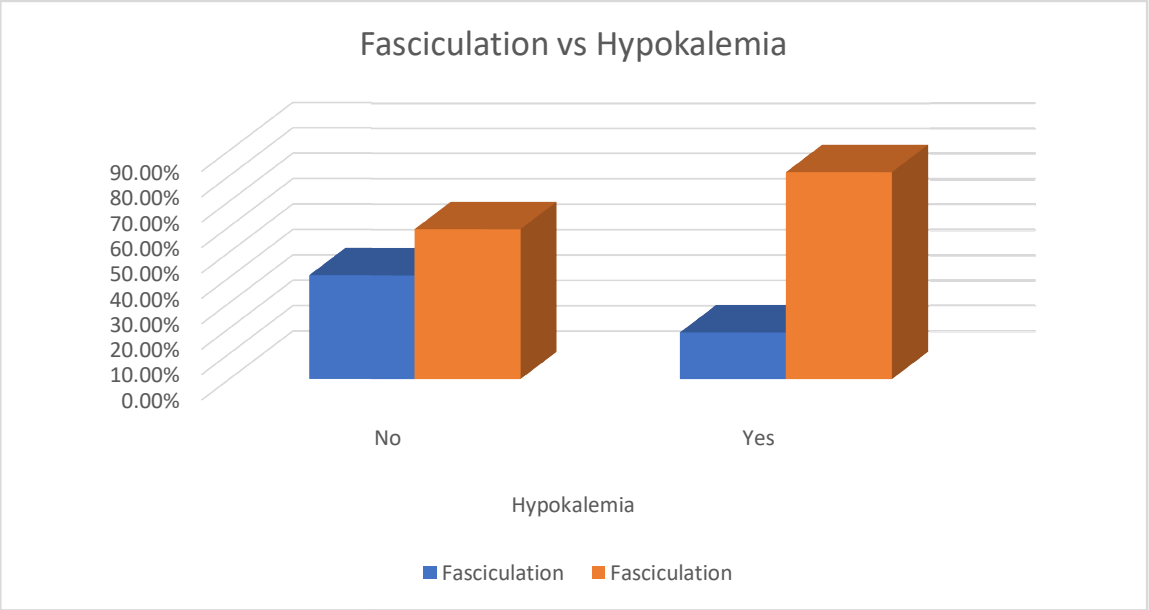

Table no 10: Respiratory Distress RR> 24 vs Hypokalemia

			Hypokalemia		Total
			No	Yes	
Respiratory Distress RR> 24	No	Count	17 (77.27%)	16 (42.11%)	33 (55.00%)
	Yes	Count	5 (22.73%)	22 (57.89%)	27 (45.00%)
Total		Count	22 (100.00%)	38 (100.00%)	60 (100.00%)

P value -0.177

As seen in the above table, Respiratory distress (RR> 24) was observed in 57.89% of Hypokalemic OPC poisoning cases as compared to 22.73% of normokalemic OPC poisoning cases and the difference was statistically insignificant.

.

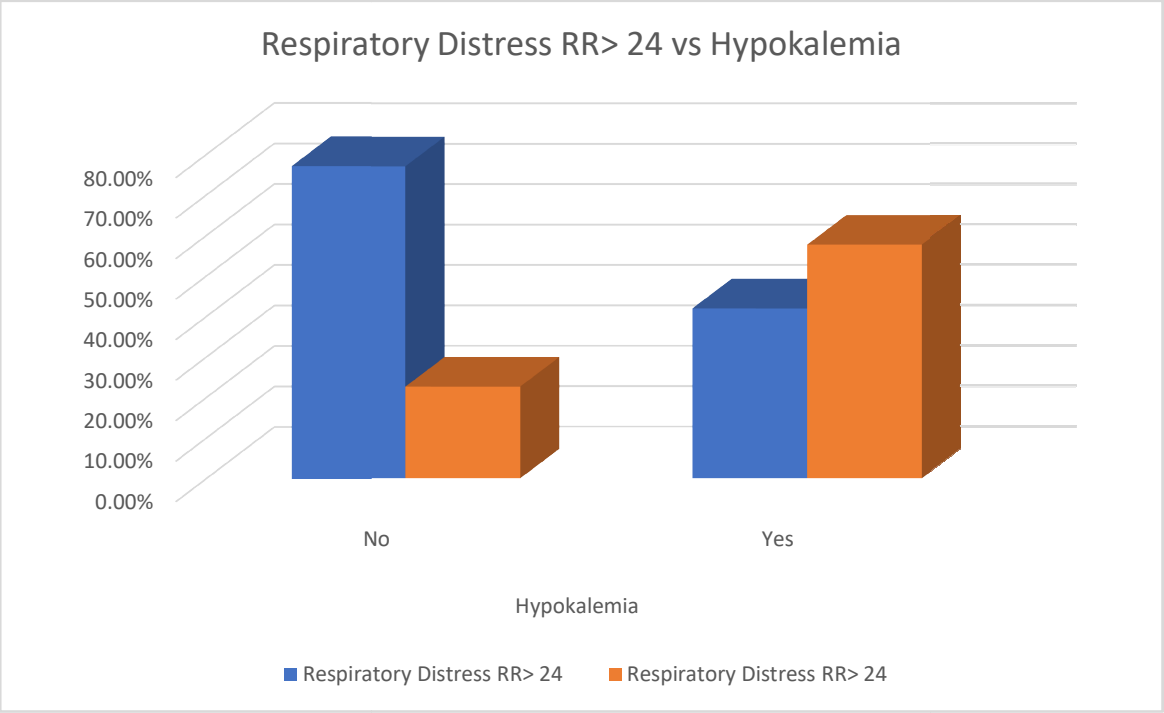

Table no 11: Mortality vs Hypokalemia

			Hypokalemia		Total
			No	Yes	
Death	No	Count	22 (100.00%)	32 (84.21%)	54 (90.00%)
	Yes	Count	0 (0.00%)	6 (15.79%)	6 (10.00%)
Total		Count	22 (100.00%)	38 (100.00%)	60 (100.00%)

P value -0.03

As seen in the above table, death was observed in 15.79% ofHypokalemic OPC poisoning cases as compared to 0 % of normokalemic OPC poisoning cases and the difference was statistically significant.

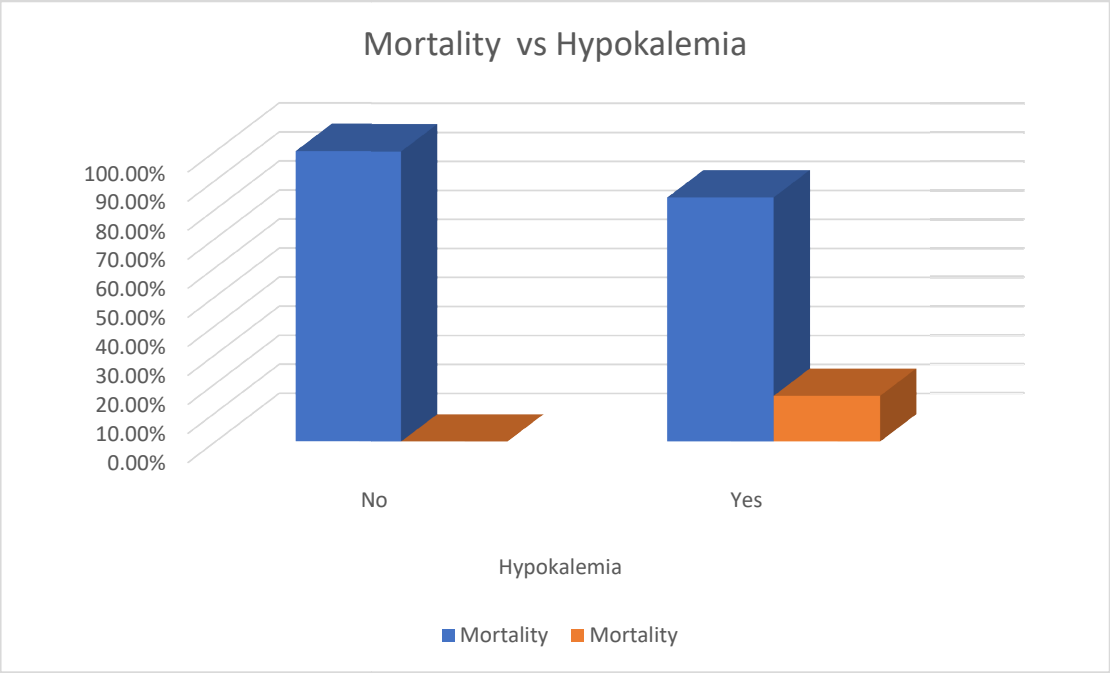

Table no 12: Duration of hospital stay vs Hypokalemia

			Hypokalemia		Total
			No	Yes	
Duration of hospital stay	less than 10 days	Count	17 (77.27%)	28 (73.68%)	45 (75.00%)
	more than 10 days	Count	5 (22.73%)	10 (26.32%)	15 (25.00%)
Total		Count	22 (100.00%)	38 (100.00%)	60 (100.00%)

P value -0.624

As seen in the above table, more than 10 days was observed in 26.32% of Hypokalemic OPC poisoning cases as compared to 22.73% of normokalemic OPC poisoning cases and the difference was statistically insignificant.

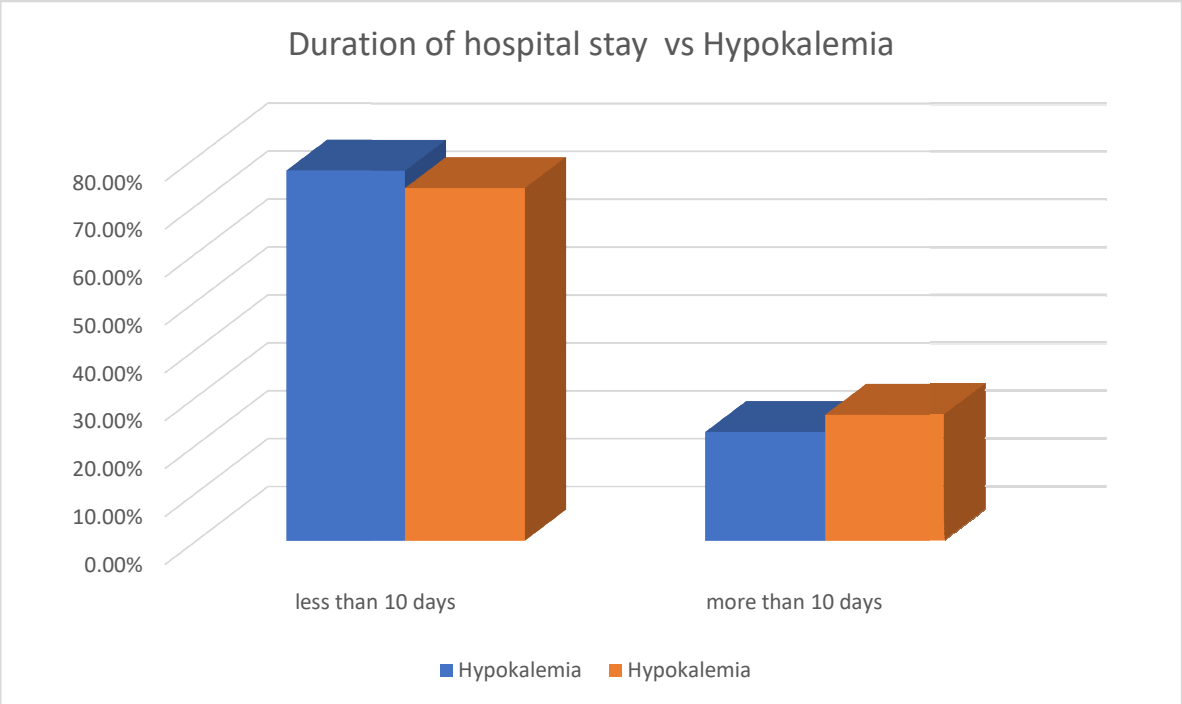

Table no 13: SerumCholinesterase levels vs Hypokalemia

S.Che LEVELS	Hypokalemia		Total
	No	Yes	
<100 (SEVERE)	1(5%)	20 (52%)	21 (35%)
100-200 (MODERATE)	1(5%)	12(32%)	13 (21.7%)
200-400 (MILD)	8(35%)	4(11%)	12 (20%)
>400 (NORMAL)	12(57%)	2(5%)	14 (23.3%)
TOTAL	22(100%)	38(100%)	60(100%)

Chi square test, P value-0.001

The data reveals a significant association between Serum cholinesterase (S.Che) levels and hypokalemia, with a statistically significant P value of 0.001. Among patients with severe hypokalemia (S.cholinesterase<100 mg/dL), 52% had hypokalemia compared to only 5% without it, while in the normal S. cholinesterase group (>400 mg/dL), only 5% had hypokalemia and 57% did not. This trend shows that lower S. cholinesterase levels are associated with a higher prevalence of hypokalemia, highlighting a statistically significant relationship between decreased Serum Cholesterol levels and the occurrence of hypokalemia.

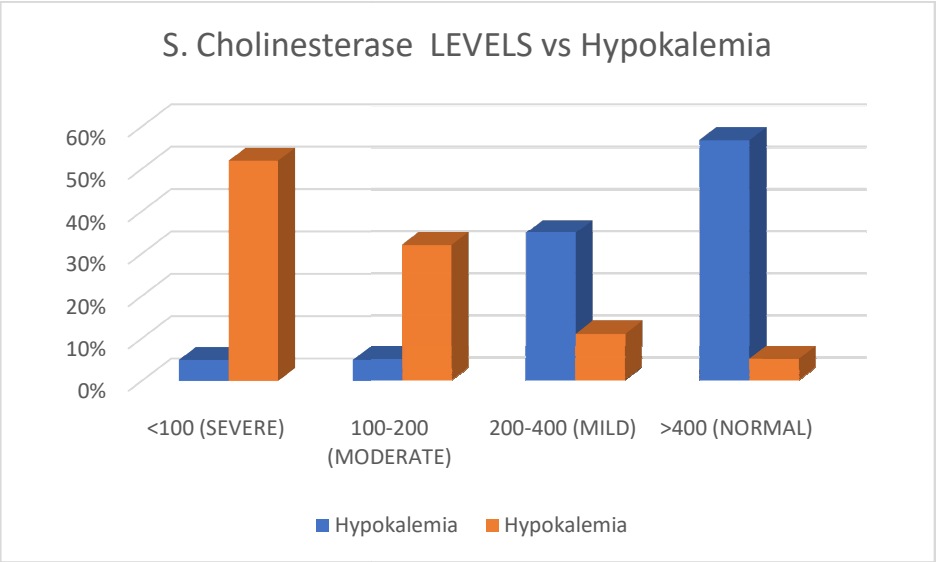

Table no 14: Serumcholinesterase levels

Cholinesterase level	Mean ±SD
Cholinesterase on admission	1978.82±1878.22
Cholinesterase at discharge	473±129

The data on cholinesterase levels indicates a notable change between admission and discharge. On admission, the mean cholinesterase level was 1978.82 ± 1878.22 units, whereas at discharge, the mean level significantly decreased to 473 ± 129 units. This substantial reduction in cholinesterase levels from admission to discharge suggests a significant physiological or therapeutic impact during the hospital stay.

Table no 15: Various Parameters

Parameters	**Mean±SD**
Age (years)	35.05±15.17
Pulse	86.18±16.30
Systolic BP	119.51±14.06
Diastolic BP	75.57±9.57
RR	22.03±3.31
Temperature	99.49±1.61

The table presents the mean and standard deviation for various patient parameters: the mean age is 32.05 ± 15.17 years, the mean pulse rate is 89.18 ± 16.30 beats per minute, the mean systolic blood pressure (BP) is 118.51 ± 14.06 mmHg, the mean diastolic BP is 74.57 ± 9.57 mmHg, the mean respiratory rate (RR) is 21.03 ± 3.31 breaths per minute, and the mean body temperature is 98.49 ± 1.61°F.

Table no 16: WBC count vs Hypokalemia

WBC level	Hypokalemia		Total
	No	Yes	
<10k	21(95%)	10 (32%)	31 (52%)
10k-15k	1(5%)	21 (52%)	21 (35%)
more than 15k	0(0%)	8(16%)	8 (13%)
Total	22(100%)	38(100%)	60(100%)

The table shows the distribution of white blood cell (WBC) levels in patients and their association with hypokalemia. Among patients with WBC levels less than 10,000/µL, 95% did not have hypokalemia, while 32% did, constituting 52% of the total patients. In the 10,000-15,000/µL WBC range, 5% did not have hypokalemia, whereas 52% did, representing 35% of the total. For WBC levels greater than 15,000/µL, none were without hypokalemia, while 16% had hypokalemia, making up 13% of the total patients. Overall, 37% of the patients did not have hypokalemia, while 63% did, indicating a significant association between higher WBC levels and the presence of hypokalemia.

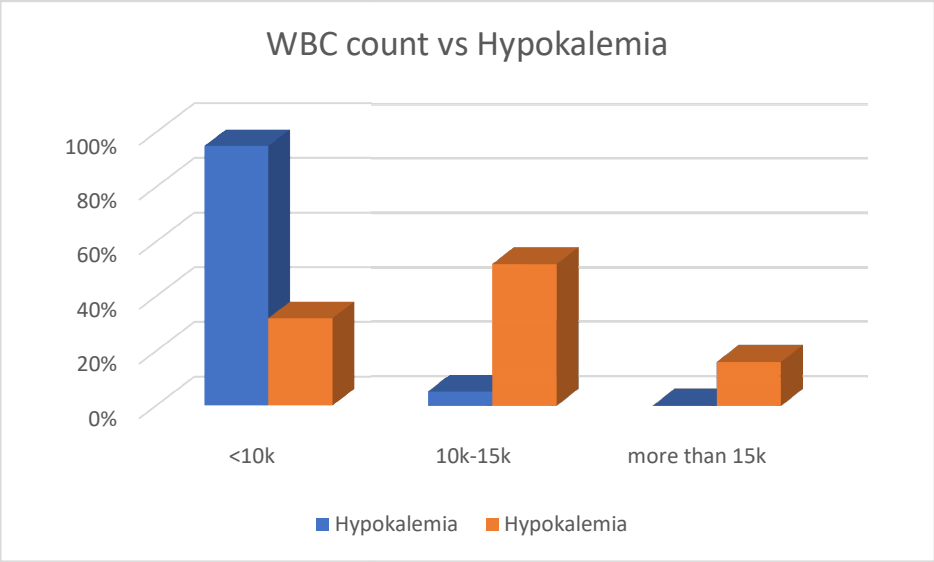

Table no 17: Arterial blood gas at various interval

Arterial blood gas	At admission	At discharge	P value
pH value	7.10 + 0.17	7.36 + 0.14	0.01
Pco2, mm Hg	40.9 + 13.0	32.7 + 8.4	0.04
HCO3 (mmol/L)	16.8 + 7.5	20.4 + 4.6	0.03

The table shows significant improvements in arterial blood gas (ABG) values from admission to discharge, with the pH value increasing from 7.18 ± 0.17 to 7.36 ± 0.14 (P = 0.01), indicating a normalization of acid-base balance. Pco2 levels decreased from 40.9 ± 13.0 mm Hg to 32.7 ± 8.4 mm Hg (P = 0.04), suggesting improved respiratory function or a reduction in respiratory acidosis. Additionally, HCO3 levels rose from 16.8 ± 7.5 mmol/L to 20.4 ± 4.6 mmol/L (P = 0.03), indicating a correction of metabolic acidosis. All changes are statistically significant, with P values less than 0.05.

Discussion

In India and other developing nations where agriculture is the main industry, organophosphorus insecticides are among the leading causes of poisoning-related illness and death.113,114

OP compounds represent a growing threat to human health as their usage grows in both agriculture and industry, thanks to their widespread availability and inexpensive cost. Organophosphorus poisoning is becoming an increasingly serious problem in developing nations' healthcare systems in the modern era.115,116 There are a number of carboxylic ester hydrolases that organophosphorus pesticides can permanently block. These include acetylcholinesterase (AChE), erythrocyte cholinesterase (EChE), plasma butyl cholinesterase (BuChE), and other non-specific proteases. In the central and autonomic nervous systems, as well as in the skeletal neuromuscular junction, these chemicals primarily cause toxicity by overstimulation of muscarinic and nicotinic cholinergic receptors with additional acetylcholine.117 Potassium is an essential biological component. Primarily, it facilitates glomerulo-tubular renal function, conduction of nerve impulses, and contraction of skeletal muscles and the heart.118

As previously shown by Tripathy et al., hypokalemia is among the most prevalent electrolyte abnormalities linked to organophosphorus poisoning.119,120 Organophosphorus poisoning is characterized by hypokalemia, which he also showed signs of having. 119 As a result of its negative impacts on heart rhythm, blood pressure, and cardiovascular mortality, hypokalemia is linked to a tenfold increase in the risk of mortality in hospitalized patients. 120 Because serum potassium levels are typically out of whack, it is cost-effective to estimate these values. An inexpensive and readily available prognostic diagnostic for acute organophosphorus exposure is serum potassium. 119

Ages 25–30 make up 46% of the sample, with those under 25 making up 19% and those between 30 and 40 making up 17%. The majority of the patients in the research were in the 20- to 40-year-old age bracket, and the average age of the participants was 29.06 ± 11.9 years (ranging from 16 to 60 years), which aligns with the results found by Bijush Difoesa et al.112 There were more men than women in this research (73.3% vs. 26.7%). The gender ratio is 1:3.2 in the study of Tanveer Hassan Banday et al. as well. 121 Males were more likely to experience poisoning than females (76.6% vs. 23.3%).121 Another group that noticed a similar pattern was Aziza et al. and Safdar et al.122,123 Nonetheless, the current study's female-to-male ratio differs significantly from that of Ather et al., which is 1:1, and Tall et al., which is 1:1.8.124,125 Hypokalemia was noted in 63.3% of the patients with OPC poisoning in this investigation. Similarly, 15.03% of patients were found to have hypokalemia in the research by Tanveer Hassan Banday et al.121

Phorate accounted for 70% of OPC poisoning cases in this investigation, with dimethoate at 6.7%, dichlorphos at 3.3%, and parathion at 3.3%.The current investigation found that hypokalemia was most usually reported in instances of phorate OPC poisoning (68.4%), followed by parathion (5.3%), and dimethoate (5.3%), but there was no statistically significant difference between the three.

Although there was a statistically significant difference, hypokalemia was seen most frequently in instances of severe OPC poisoning (42.2%), intermediate grade (36.8%), and mild (21.1%). Syed M. Ahmed et al. found similar results: 42 patients (48.8% of the total) experienced severe poisoning, 30 (34.9%) moderate poisoning, and 14 (16.3%) light poisoning out of 86 patients. The current investigation found that although there was no statistically significant difference between hypokalemic and normokalemic OPC poisoning patients, 52.6% of hypokalemic cases

and 27.3% of normokalemic individuals had respiratory distress (RR> 24). 108 total A much higher percentage of patients requiring a ventilator were found to have hypokalemic OPC poisoning (47.4% vs. 9.1% in normokalemic OPC poisoning). Banday et al. found that 39.8% of OPC patients required a ventilator, which is consistent with our results.121 Mahadeshwara Prasad et al. found a similar result when they combined respiratory distress with mechanical ventilation, which was statistically significant (p value < 0.001).126

There was no statistically significant difference between the 23.68% and 81.58% of hypokalemic OPC poisoning patients that exhibited fasciculation and convulsions, respectively, in this investigation. There was no statistically significant difference in the proportion of hypokalemic OPC poisoning cases (50.89% vs. 22.73% of normokalemic OPC poisoning cases) that reported respiratory distress (RR> 24). In 10% of cases with OPC poisoning, fasciculation was seen, which is in line with the results of the research by Indranil Banerjee et al.127 As compared to normokalemic OPC poisoning cases, 15.79% of hypokalemic OPC poisoning patients had a fatality rate, which was statistically significant. Patients having an average potassium level of 2.90 +/- 0.057 meq/dl (p value < 0.001) died in the study conducted by D.R. Mahadeshwara Prasad et al. According to the Proudfoot classification, 126 severe patients (61.9%) in the study by D. R. Mahadeshwara Prasad et al. were found to have hypokalemia. Hypokalemia was associated with a greater fatality rate in our research compared to OP poisoning subjects with normal potassium levels. A greater frequency of heart disorders linked to low potassium levels may explain the increased mortality. It was discovered that gastric lavage, an early method of decontaminating the stomach, can help reduce the degree of poisoning in patients who arrive to the emergency room soon after consuming OP pesticides. Having said that, it is not entirely apparent how useful stomach lavage is for late presentation following

poison administration or for repeated lavages.128 An uncommon but significant consequence of organophosphorus exposure is renal damage. Treatment entirely resolves hypokalemia-associated ventricular arrhythmia, a renal complication that can develop from renal tubular acidosis or another cause.129 Potential side effects of organophosphorus poisoning or long-term exposure to these pesticides include inflammation of the liver and pancreas, as well as problems with the central and peripheral neurological systems. When someone is poisoned with organophosphorus, their nervous system often malfunctions due to an excess of acetylcholine at the neuromuscular junction. However, weakness in the muscles of the head and neck can also be brought on by neuromuscular involvement caused by an excess of acetylcholine.130 Intermediate syndrome is the weakening of the muscles that often manifests between twenty-four and ninety- six hours after the acute cholinergic syndrome has resolved as a result of taking atropine. Muscles innervated by the central nervous system are the most usually affected, followed by the muscles of the proximal limbs and neck.

The current study found that 26.32% of hypokalemic OPC poisoning cases and 22.73% of normokalemic OPC poisoning cases had symptoms for more than 10 days, while there was no statistically significant difference between the two groups. According to a research by Indukuri Devi Amrutha et al., 31.6% of instances of OPC poisoning required a hospital stay of more than 10 days, while 18.2% of cases of normokalemic OPC poisoning required the same amount of time, but the difference was not statistically significant. 131

In the present study, the data reveals a significant association between Serum Cholinesterase (AChE) levels and hypokalemia, with a statistically significant P value of 0.001. Among patients with severe hypokalemia (S. Cholinesterase <100 mg/dL), 52% had hypokalemia compared to only 5% without it, while in the normal S. Cholinesterase group (>400

mg/dL), only 5% had hypokalemia and 57% did not. This trend shows that lower S. Cholinesterase levels are associated with a higher prevalence of hypokalemia, highlighting a statistically significant relationship between decreased Serum Cholesterol levels and the occurrence of hypokalemia.

In the present study, the data on cholinesterase levels indicates a notable change between admission and discharge. On admission, the mean cholinesterase level was 1978.82 ± 1878.22 units, whereas at discharge, the mean level significantly decreased to 473 ± 129 units. This substantial reduction in cholinesterase levels from admission to discharge suggests a significant physiological or therapeutic impact during the hospital stay.

In the present study, deviation for various patient parameters: the mean age is 32.05 ± 15.17 years, the mean pulse rate is 89.18 ± 16.30 beats per minute, the mean systolic blood pressure (BP) is 118.51 ± 14.06 mmHg, the mean diastolic BP is 74.57 ± 9.57 mmHg, the mean respiratory rate (RR) is 21.03 ± 3.31 breaths per minute, and the mean body temperature is 98.49 ± 1.61°F.

In the present study, among patients with WBC levels less than 10,000/µL, 90% did not have hypokalemia, while 32% did, constituting 52% of the total patients. In the 10,000-15,000/µL WBC range, 5% did not have hypokalemia, whereas 52% did, representing 35% of the total. For WBC levels greater than 15,000/µL, none were without hypokalemia, while 16% had hypokalemia, making up 13% of the total patients. Overall, 37% of the patients did not have hypokalemia, while 63% did, indicating a significant association between higher WBC levels and the presence of hypokalemia.

There were significant improvements in arterial blood gas (ABG) values from admission to discharge, with the pH value increasing from 7.18 ± 0.17 to 7.36 ± 0.14 (P = 0.01), indicating a normalization of acid-base balance. Pco2 levels decreased from 40.9 ± 13.0 mm Hg to 32.7 ± 8.4

mm Hg (P = 0.04), suggesting improved respiratory function or a reduction in respiratory acidosis. Additionally, HCO3 levels rose from 16.8 ± 7.5 mmol/L to 20.4 ± 4.6 mmol/L (P = 0.03), indicating a correction of metabolic acidosis. All changes are statistically significant, with P values less than 0.05.

Summary

- The majority of the study population falls within the age group of 25 to 30 years (46%), followed by those under 25 years (19%) and 30 to 40 years (17%).
- There is a higher prevalence of males (73.3%) compared to females (26.7%).
- Phorate is the most common type of organophosphate poisoning (70%), followed by dimethoate (6.7%), dichlorvos (3.3%), and parathion (3.3%).
- Hypokalemia is present in 63.3% of organophosphate poisoning cases, with the highest incidence in phorate poisoning cases (68.4%), followed by parathion (5.3%) and dimethoate (5.3%), though the differences are not statistically significant.
- Hypokalemia is most commonly observed in severe cases of organophosphate poisoning (42.2%), followed by moderate (36.8%) and mild cases (21.1%), but these differences are not statistically significant.
- Ventilator requirement is observed in 47.4% of hypokalemic organophosphate poisoning cases compared to 9.1% of normokalemic cases, a difference that is statistically significant.
- Convulsions occur in 23.68% of hypokalemic organophosphate poisoning cases compared to 9.1% of normokalemic cases, although this difference is not statistically significant.
- Fasciculations are present in 81.58% of hypokalemic organophosphate poisoning cases compared to 59.09% of normokalemic cases, with no statistically significant difference.

- Respiratory distress (RR > 24) is observed in 57.89% of hypokalemic organophosphate poisoning cases compared to 22.73% of normokalemic cases, but the difference is not statistically significant.
- Death occurs in 15.79% of hypokalemic organophosphate poisoning cases compared to 0% of normokalemic cases, and this difference is statistically significant.
- Hospital stays exceeding 10 days are observed in 26.32% of hypokalemic organophosphate poisoning cases compared to 22.73% of normokalemic cases, with no statistically significant difference.
- Significant improvements in arterial blood gas (ABG) values from admission to discharge were observed, with pH values increasing from 7.18 ± 0.17 to 7.36 ± 0.14 (P = 0.01), indicating a normalization of acid-base balance. Pco2 levels decreased from 40.9 ± 13.0 mm Hg to 32.7 ± 8.4 mm Hg (P = 0.04), suggesting improved respiratory function or a reduction in respiratory acidosis. Additionally, HCO3 levels rose from 16.8 ± 7.5 mmol/L to 20.4 ± 4.6 mmol/L (P = 0.03), indicating a correction of metabolic acidosis. All changes are statistically significant, with P values less than 0.05.

Conclusion

The study underscores the prevalence of OPC poisoning, with phorate being the most common agent, primarily affecting young adults aged 25 to 30, with a male predominance. Hypokalemia emerges as a significant complication, especially in severe cases and those poisoned by phorate, correlating with heightened mortality rates and increased ventilator requirements. Clinical features such as convulsions, fasciculations, and respiratory distress often accompany hypokalemia. In cases of organophosphorus chemical poisoning, hypokalemia considerably raises the risk of both illness and death. In cases of organophosphorus chemical poisoning, hypokalemia can be utilised as an efficient and dependable indicator of death and morbidity rates.These findings emphasize the critical importance of prompt recognition and management of hypokalemia in OPC poisoning cases to mitigate adverse outcomes and potentially reduce mortality rates. Further investigation into strategies for early detection and intervention targeting hypokalemia could substantially enhance patient outcomes in this population.

STRENGTHS AND LIMITATIONS

STRENGTHS OF THE STUDY:

- In clinical settings, we tend to be inclined towards the neurological manifestations of organophosphate poisoning, which predicts morbidity rather than the mortality. This study takes into account the biochemical parameters (specially serum potassium levels) which get deranged in OP poisoning,which is an important and direct predictor of mortality in such patients.
- Serum potassium levels have been found to serve as a surrogate marker in predicting mortality.

LIMITATIONS OF THE STUDY:

- This study has taken into account the serum AChE levels instead of the true RBC cholinesterase levels due to lack of available test kits throughout the country; which might have impaired the overall results
- Duration of hospital stay was less than ten days for a significant number of people due to patients being shifted elsewhere (to other hospitals) for further care and management, giving us a wrong interpretation of the same.

References

1. Proudfoot AT. Salicylates and salicylamides. In: Haddad LM, Winchester JF, editors. *Clinical Management of Poisoning and Drug Overdose.* 3rd ed. Philedelphia: WB Saunders; 1983. pp. 575–86.
2. South-East Asia: World Health Organisation; 2009. WHO. Health implications from monocrotophos use: A review of the evidence in India; pp. 1–60.
3. Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. *Q J Med.* 2000;93:715–31.
4. Eddleston M, Phillips MR. Self poisoning with pesticides. *BMJ.* 2004;328:42–4.
5. Buckley NA, Karalliedde L, Dawson A, Senanayake N, Eddleston M. Where is the evidence for the management of pesticide poisoning – is clinical toxicology fiddling while the developing world burns? *J Toxicol Clin Toxicol.* 2004;42:113–6.
6. Haddad LM. A general approach to the emergency management of poisonings. In: Haddad LM, Winchester JF, editors. *Clinical Management of Poisonings and Drug Overdose.* 3rd ed. Philadelphia, PA: WB Saunders; 1983. pp. 4–18.
7. Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. *QJM.* 2000;93:715–31.
8. Yang CC, Deng JF. Intermediate syndrome following organophosphate insecticide poisoning. *J Chin Med Assoc.* 2007;70:467–72.
9. Emerick GL, Peccinini RG, de Oliveira GH. Organphosphorus-induced delayed neuropathy: A simple and efficient therapeutic strategy. *Toxicol Lett.* 2010;192:238–44.

10. Pajoumand A, Shadnia S, Rezaie A, Abdi M, Abdollahi M. Benefits of magnesium sulfate in the management of acute human poisoning by organophosphorus insecticides. *Hum Exp Toxicol.* 2004;23:565–9.
11. Verheyen J, Stoks R. Current and future daily temperature fluctuations make a pesticide more toxic: Contrasting effects on life history and physiology. Environ Pollut. 2019 May;248:209-218.
12. Aroniadou-Anderjaska V, Figueiredo TH, Apland JP, Braga MF. Targeting the glutamatergic system to counteract organophosphate poisoning: A novel therapeutic strategy. Neurobiol Dis. 2020 Jan;133:104406.
13. Dagg K, Irish S, Wiegand RE, Shililu J, Yewhalaw D, Messenger LA. Evaluation of toxicity of clothianidin (neonicotinoid) and chlorfenapyr (pyrrole) insecticides and cross-resistance to other public health insecticides in Anopheles arabiensis from Ethiopia. Malar J. 2019 Feb 22;18(1):49.
14. Mendes PA, Pereira TC, Pina R, Santos R. Chlorpyrifos-Induced Delayed Neurotoxicity with A Rare Presentation of Flaccid Quadriplegia: A Diagnostic Challenge. Eur J Case Rep Intern Med. 2018;5(1):000751.
15. Chen KX, Zhou XH, Sun CA, Yan PX. Manifestations of and risk factors for acute myocardial injury after acute organophosphorus pesticide poisoning. Medicine (Baltimore). 2019 Feb;98(6):e14371.
16. Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. QJM. 2000 Nov;93(11):715-31.
17. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: systematic review. BMC Public Health. 2007 Dec 21;7:357.

18. Kloske M, Witkiewicz Z. Novichoks - The A group of organophosphorus chemical warfare agents. Chemosphere. 2019 Apr;221:672-682.
19. Hulse EJ, Haslam JD, Emmett SR, Woolley T. Organophosphorus nerve agent poisoning: managing the poisoned patient. Br J Anaesth. 2019 Oct;123(4):457-463.
20. Kamanyire R, Karalliedde L. Organophosphate toxicity and occupational exposure. Occup Med (Lond). 2004 Mar;54(2):69-75.
21. Sudakin DL, Power LE. Organophosphate exposures in the United States: a longitudinal analysis of incidents reported to poison centers. J Toxicol Environ Health A. 2007 Jan 15;70(2):141-7.
22. Gummin DD, Mowry JB, Beuhler MC, Spyker DA, Bronstein AC, Rivers LJ, Pham NPT, Weber J. 2020 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 38th Annual Report. Clin Toxicol (Phila). 2021 Dec;59(12):1282-1501.
23. Boedeker W, Watts M, Clausing P, Marquez E. The global distribution of acute unintentional pesticide poisoning: estimations based on a systematic review. BMC Public Health. 2020 Dec 07;20(1):1875.
24. Sikary AK. Homicidal poisoning in India: A short review. J Forensic Leg Med. 2019 Feb;61:13-16.
25. Dardiotis E, Aloizou AM, Siokas V, Tsouris Z, Rikos D, Marogianni C, Aschner M, Kovatsi L, Bogdanos DP, Tsatsakis A. Paraoxonase-1 genetic polymorphisms in organophosphate metabolism. Toxicology. 2019 Jan 01;411:24-31.

26. Jokanović M. Neurotoxic effects of organophosphorus pesticides and possible association with neurodegenerative diseases in man: A review. Toxicology. 2018 Dec 01;410:125-131.
27. aughton SX, Terry AV. Neurotoxicity in acute and repeated organophosphate exposure. Toxicology. 2018 Sep 01;408:101-112.
28. Rusyniak DE, Nañagas KA. Organophosphate poisoning. Semin Neurol. 2004 Jun;24(2):197-204.
29. Eddleston M. The pathophysiology of organophosphorus pesticide self-poisoning is not so simple. Neth J Med. 2008 Apr;66(4):146-8.
30. Kwong TC. Organophosphate pesticides: biochemistry and clinical toxicology. Ther Drug Monit. 2002 Feb;24(1):144-9.
31. Nolan RJ, Rick DL, Freshour NL, Saunders JH. Chlorpyrifos: pharmacokinetics in human volunteers. Toxicol Appl Pharmacol. 1984 Mar 30;73(1):8-15.
32. Lee DH, Jung KY, Choi YH, Cheon YJ. Body mass index as a prognostic factor in organophosphate-poisoned patients. Am J Emerg Med. 2014 Jul;32(7):693-6.
33. .Buratti FM, Volpe MT, Meneguz A, Vittozzi L, Testai E. CYP-specific bioactivation of four organophosphorothioate pesticides by human liver microsomes. Toxicol Appl Pharmacol. 2003 Feb 01;186(3):143-54.
34. Eyer P. The role of oximes in the management of organophosphorus pesticide poisoning. Toxicol Rev. 2003;22(3):165-90.
35. Merrill DG, Mihm FG. Prolonged toxicity of organophosphate poisoning. Crit Care Med. 1982 Aug;10(8):550-1.

36. Eddleston M, Singh S, Buckley N. Organophosphorus poisoning (acute) *Clin Evid.* 2005;**13**:1744–1755.

37. Eddleston M, Dawson A, Karalliedde L. Early management after self-poisoning with an organophosphorus or carbamate pesticide—a treatment protocol for junior doctors. *Crit Care.* 2004;**8**:R391–R397.

38. Zilker T, Hibler A. Treatment of severe parathion poisoning. In: Szinicz L, Eyer P, Klimmek R, editors. *Role of oximes in the treatment of anticholinesterase agent poisoning.* Spektrum, Akademischer Verlag; Heidelberg: 1996. pp. 9–17.

39. Eyer F, Meischner V, Kiderlen D. Human parathion poisoning. A toxicokinetic analysis. *Toxicol Rev.* 2003;**22**:143–163.

40. WHO . *WHO recommended classification of pesticides by hazard and guidelines to classification 2000–2001. WHO/PCS/01.4.* World Health Organization; Geneva: 2001.

41. Baker EL, Warren M, Zack M. Epidemic malathion poisoning in Pakistan malaria workers. *Lancet.* 1978;**1**:31–34.

42. Aldridge WN, Miles JW, Mount DL, Verschoyle RD. The toxicological properties of impurities in malathion. *Arch Toxicol.* 1979;**42**:95–106.

43. Soliman SA, Sovocool GW, Curley A, Ahmed NS, El-Fiki S, El-Sebae SK. Two acute human poisoning cases resulting from exposure to diazinon transformation products in Egypt. *Arch Environ Health.* 1982;**37**:207–212.

44. Meleney WP, Peterson HO. The relationship of shelf age to toxicity of dimethoate to sheep. *J Am Vet Med Assoc.* 1964;**144**:756–758.

45. Buratti FM, Volpe MT, Fabrizi L, Meneguz A, Vittozzi L, Testai E. Kinetic parameters of OPT pesticide desulfuration by c-DNA expressed human CYPs. *Environ ToxicolPharmacol.* 2002;**11**:181–190.

46. Buratti FM, Volpe MT, Meneguz A, Vittozzi L, Testai E. CYP-specific bioactivation of four organophosphorothioate pesticides by human liver microsomes. *Toxicol Appl Pharmacol.* 2003;**186**:143–154.

47. Benfenati E, Gini G, Piclin N, Roncaglioni A, Vari MR. Predicting log P of pesticides using different software. *Chemosphere.* 2003;**53**:1155–1164.

48. Wadia RS, Bhirud RH, Gulavani AV, Amin RB. Neurological manifestations of three organophosphate poisons. *Indian J Med Res.* 1977;**66**:460–468.

49. Heath AJW, Meredith T. Atropine in the management of anticholinesterase poisoning. In: Ballantyne B, Marrs T, editors. *Clinical and experimental toxicology of organophosphates and carbamates.* Butterworth Heinemann; Oxford: 1992. pp. 543–554.

50. Reigart JR, Roberts JR. *Recognition and management of pesticide poisonings.* 5th edn. Office of Pesticide Programs, US Environmental Protection Agency; Washington DC: 1999. Organophosphate insecticides; pp. 34–47.

51. Vance MV, Selden BS, Clark RF. Optimal patient position for transport and initial management of toxic ingestions. *Ann Emerg Med.* 1992;**21**:243–246.

52. Anvari M, Horowitz M, Fraser R. Effects of posture on gastric emptying of nonnutrient liquids and antropyloroduodenal motility. *Am J Physiol.* 1995;**268**(5 Pt 1):G868–G871.

53. Geller RJ, Singleton KL, Tarantino ML. Nosocomial poisoning associated with emergency department treatment of organophosphate toxicity—Georgia, 2000. *MMWR Morb Mortal Wkly Rep.* 2001;**49**:1156–1158.

54. Stacey R, Morfey D, Payne S. Secondary contamination in organophosphate poisoning: analysis of an incident. *Q J Med.* 2004;**97**:75–80.

55. Little M, Murray L. Consensus statement: Risk of nosocomial organophosphate poisoning in emergency departments. *Emerg Med Australasia.* 2004;**16**:456–458.

56. Roberts D, Senarathna L. Secondary contamination in organophosphate poisoning. *Q J Med.* 2004;**97**:697–698.

57. Brown JH, Taylor P. Muscarinic receptor agonists and antagonists. In: Hardman JG, Limbird LE, editors. *Goodman and Gilman's The pharmacological basis of therapeutics.* 10th edn. McGraw-Hill; New York: 2001. pp. 155–173.

58. Bardin PG, van Eeden SF. Organophosphate poisoning: grading the severity and comparing treatment between atropine and glycopyrrolate. *Crit Care Med.* 1990;**18**:956–960.

59. Kventsel I, Berkovitch M, Reiss A, Bulkowstein M, Kozer E. Scopolamine treatment for severe extra-pyramidal signs following organophosphate (chlorpyrifos) ingestion. *Clin Toxicol.* 2005;**43**:877–879.

60. McDonough JH, Zoeffel LD, McMonagle J, Copeland TL, Smith CD, Shih TM. Anticonvulsant treatment of nerve agent seizures: anticholinergics versus diazepam in soman-intoxicated guinea pigs. *Epilepsy Res.* 2000;**38**:1–14.

61. Sunder Ram J, Kumar SS, Jayarajan A, Kuppuswamy G. Continuous infusion of high doses of atropine in the management of organophosphorus compound poisoning. *J Assoc Physicians India.* 1991;**39**:190–193.
62. Bismuth C, Inns RH, Marrs TC. Efficacy, toxicity and clinical uses of oximes in anticholinesterase poisoning. In: Ballantyne B, Marrs T, editors. *Clinical and experimental toxicology of organophosphates and carbamates.* Butterworth Heinemann; Oxford: 1992. pp. 555–577.
63. Eyer P, Buckley NA. Pralidoxime for organophosphate poisoning. *Lancet.* 2006;**368**:2110–2111.
64. de Silva HJ, Wijewickrema R, Senanayake N. Does pralidoxime affect outcome of management in acute organophosphate poisoning? *Lancet.* 1992;**339**:1136–1138.
65. Singh S, Batra YK, Singh SM, Wig N, Sharma BK. Is atropine alone sufficient in acute severe organophosphate poisoning? Experience of a North West Indian hospital. *Int J Clin Pharmacol Ther.* 1995;**33**:628–630.
66. Peter JV, Cherian AM. Organic insecticides. *AnaesthIntens Care.* 2000;**28**:11–21.
67. Johnson S, Peter JV, Thomas K, Jeyaseelan L, Cherian AM. Evaluation of two treatment regimens of pralidoxime (1 gm single bolus dose *vs* 12 gm infusion) in the management of organophosphorus poisoning. *J Assoc Physicians India.* 1996;**44**:529–531.
68. Cherian AM, Peter JV, Samuel J. Effectiveness of P2AM (PAM -pralidoxime) in the treatment of organophosphorus poisoning. A randomised, double blind placebo controlled trial. *J Assoc Physicians India.* 1997;**45**:22–24.
69. Eddleston M, Szinicz L, Eyer P, Buckley N. Oximes in acute organophosphorus pesticide poisoning: a systematic review of clinical trials. *Q J Med.* 2002;**95**:275–283.

70. Peter JV, Moran JL. Role of oximes in human organophosphate poisoning—a critical look at the evidence. In: Nayyar V, editor. *Critical Care Update 2004*. Jaypee; New Delhi: 2004. pp. 153–163.
71. Buckley NA, Eddleston M, Szinicz L. Oximes for acute organophosphate pesticide poisoning. *Cochrane Database Syst Rev.* 2005;**1** CD005085.
72. Peter JV, Moran JL, Graham P. Oxime therapy and outcomes in human organophosphate poisoning: an evaluation using meta-analytic techniques. *Crit Care Med.* 2006;**34**:502–510.
73. Rahimi R, Nikfar S, Abdollahi M. Increased morbidity and mortality in acute human organophosphate-poisoned patients treated by oximes: a meta-analysis of clinical trials. *Hum Exp Toxicol.* 2006;**25**:157–162.
74. Duval G, Rakouer JM, Tillant D, Auffray JC, Nigond J, Deluvallee G. Intoxications aiguës par insecticides à action anticholinestérasique. Evaluation de l'efficacité d'un réactivateur des cholinestérases, le pralidoxime. *J Toxicol Clin Exp.* 1991;**11**:51–58.
75. Abdollahi M, Jafari A, Jalali N, Balali-Mood M, Kebriaeezadeh A, Nikfar S. A new approach to the efficacy of oximes in the management of acute organophosphate poisoning. *Iranian J Med Sci.* 1995;**20**:105–109.
76. Balali-Mood M, Shariat M. Treatment of organophosphate poisoning. Experience of nerve agents and acute pesticide poisoning on the effects of oximes. *J Physiol (Paris)* 1998;**92**:375–378.
77. Sungur M, Guven M. Intensive care management of organophosphate insecticide poisoning. *Crit Care.* 2001;**5**:211–215.

78. Chugh SN, Aggarwal N, Dabla S, Chhabra B. Comparative evaluation of atropine alone and atropine with pralidoxime (PAM) in the management of organophosphorus poisoning. *J Indian Acad Clin Med.* 2005;**6**:33–37.
79. Cherian MA, Roshini C, Visalakshi J, Jeyaseelan L, Cherian AM. Biochemical and clinical profile after organophosphorus poisoning—a placebo-controlled trial using pralidoxime. *J Assoc Physicians India.* 2005;**53**:427–431.
80. Grimes DA, Schulz KF. An overview of clinical research: the lay of the land. *Lancet.* 2002;**359**:57–61.
81. Grimes DA, Schulz KF. Bias and causal associations in observational research. *Lancet.* 2002;**359**:248–252.
82. Pawar KS, Bhoite RR, Pillay CP, Chavan SC, Malshikare DS, Garad SG. Continuous pralidoxime infusion versus repeated bolus injection to treat organophosphorus pesticide poisoning: a randomised controlled trial. *Lancet.* 2006;**368**:2136–2141.
83. Thiermann H, Szinicz L, Eyer F. Modern strategies in therapy of organophosphate poisoning. *Toxicol Lett.* 1999;**107**:233–239.
84. WHO International Programme on Chemical Safety . *Poisons information monograph G001. Organophosphorus pesticides.* World Health Organization; Geneva: 1999.
85. Sidell FR. Nerve agents. In: Sidell FR, Takafuji ET, Franz DR, editors. *Medical aspects of chemical and biological warfare.* Borden Institute, Walter Reed Army Medical Center; Washington, DC: 2006. pp. 129–179.
86. Murphy MR, Blick DW, Dunn MA. Diazepam as a treatment for nerve agent poisoning in primates. *Aviat Space Environ Med.* 1993;**64**:110–115.

87. Dickson EW, Bird SB, Gaspari RJ, Boyer EW, Ferris CF. Diazepam inhibits organophosphate-induced central respiratory depression. *Acad Emerg Med.* 2003;**10**:1303–1306.

88. Eddleston M, Haggalla S, Reginald K. The hazards of gastric lavage for intentional self-poisoning in a resource poor location. *ClinToxicol.* 2007;**45**:136–143.

89. Kramer RE, Ho IK. Pharmacokinetics and pharmacodynamics of methyl parathion. *Chinese Med J (Taipei)* 2002;**65**:187–199.

90. American Academy of Clinical Toxicology and European Association of Poison Centres and Clinical Toxicologists Position paper: gastric lavage. *J Toxicol Clin Toxicol.* 2004;**42**:933–943.

91. Bhattarai MD. Gastric lavage is perhaps more important in developing countries. *BMJ.* 2000;**320**:711.

92. Gu YL, Wan WG, Xu ML, Zou HJ. Gastric lavage for organophosphate pesticide poisoned patients in Chinese *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi.* 2004;**22**:388–390.

93. American Academy of Clinical Toxicology and European Association of Poisons Centres and Clinical Toxicologists Position paper: ipecac syrup. *J Toxicol Clin Toxicol.* 2004;**42**:13343.

94. Eddleston M, Juszczak E, Buckley NA. Randomised controlled trial of routine single or multiple dose superactivated charcoal for self-poisoning in a region with high mortality. *Clin Toxicol.* 2005;**43**:442–443.

95. Guven H, Tuncok Y, Gidener S. In vitro adsorption of dichlorvos and parathion by activated charcoal. *J Toxicol Clin Toxicol.* 1994;**32**:157–163.

96. Buckley NA, Roberts DM, Eddleston M. Overcoming apathy in research on organophosphate poisoning. *BMJ.* 2004;**329**:1231–1233.

97. Singh G, Avasthi G, Khurana D, Whig J, Mahajan R. Neurophysiological monitoring of pharmacological manipulation in acute organophosphate poisoning. The effects of pralidoxime, magnesium sulphate and pancuronium. *Electroencephalogr Clin Neurophysiol.* 1998;**107**:140–148.

98. Pajoumand A, Shadnia A, Rezaie A, Abdi M, Abdollahi M. Benefits of magnesium sulfate in the management of acute human poisoning by organophosphorus insecticides. *Hum Exp Toxicol.* 2004;**23**:565–569.

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

100. Wong A, Sandron CA, Magalhaes AS, Rocha LCS. Comparative efficacy of pralidoxime vs sodium bicarbonate in rats and humans severely poisoned with O-P pesticide. *J Toxicol Clin Toxicol.* 2000;**38**:554–555.

101. Balali-Mood M, Ayati MH, Ali-Akbarian H. Effect of high doses of sodium bicarbonate in acute organophosphorous pesticide poisoning. *Clin Toxicol.* 2005;**43**:571–574.

102. Cordoba D, Cadavid S, Angulo D, Ramos I. Organophosphate poisoning—modication of acid-base equilibrium and use of sodium bicarbonate as an aid in the treatment of toxicity in dogs. *Vet Hum Toxicol.* 1983;**25**:1–3.

103. Roberts D, Buckley NA. Alkalinisation for organophosphorus pesticide poisoning. *Cochrane Database Syst Rev.* 2005;**1** CD004897.

104. Peng A, Meng FQ, Sun LF, Ji Z-S, Li YH. Therapeutic efficacy of charcoal hemoperfusion in patients with acute severe dichlorvos poisoning. *Acta Pharmacol Sin*. 2004;**25**:15–21.

105. Chambers JE, Oppenheimer SF. Organophosphates, serine esterase inhibition, and modeling of organophosphate toxicity. *Toxicol Sci.* 2004;**77**:185–187.

106. Rosenberg Y, Luo C, Ashani Y. Pharmacokinetics and immunologic consequences of exposing macaques to purified homologous butyrylcholinesterase. *Life Sci.* 2002;**72**:125–134.

107. Guven M, Sungur M, Eser B, Sary I, Altuntas F. The effects of fresh frozen plasma on cholinesterase levels and outcomes in patients with organophosphate poisoning. *J Toxicol Clin Toxicol.* 2004;**42**:617–623.

108. Ahmed SM, Das B, Nadeem A, Samal RK. Survival pattern in patients with acute organophosphate poisoning on mechanical ventilation: A retrospective intensive care unit-based study in a tertiary care teaching hospital. Indian J Anaesth. 2014 Jan;58(1):11-7.

109. Patil G, Murthy N, Nikhil M. Contributing Factors for Morbidity and Mortality in Patients with Organophosphate Poisoning on Mechanical Ventilation: A Retrospective Study in a Teaching Hospital. J Clin Diagn Res. 2016 Dec;10(12):UC18-UC20.

110. Mohit Desai et al., A Study Of Hypokalemia In Organophosphorus Poisoning In Dahod Gujarat, Paripex - Indian Journal Of Research,2021 ; Volume - 10 : Issue - 09

111. Vandana Dandekar et al, Evaluation of Serum Potassium Levels as Prognostic Marker in Acute Organophosphorus Poisoning in a Tertiary Care Centre, JMSCR, November 2019 Volume 07 Issue11: Page 347-354|

112. Difoesa B, Sharma DJ, Hari S, et al. Significance of serum potassium level at admission in organophosphorus poisoning and impact on outcome - a hospital-based study from north east India. J Evid Based Med Healthc 2021;8(36):3282-3287.

113. Sinha US, Kapoor AK, Agnihotri AK, et al. A profile of the poisoning cases which were admitted to the SRN Hospital, Allahabad, with a special reference to aluminium phosphide poisoning. J Forensic Med Toxicol 1999;6(1):40-43.

114. Dash SK, Mohanty MK, Patnaik KK, et al. The socio demographic profile of the poisoning cases. J Indian Acad Forensic Med 2005;3:133-138.

115. Hayes MM, Vander Westhuizen NG, Gelfanol M. Organophosphate poisoning in Rhodesia. S Afr Med J 1978;54;230-234.

116. Jeyaratnam J. Acute pesticide poisoning: a major global health problem. World Health Stat Q 1990;43(3):139- 144.

117. Clark RF. Insecticides: organic phosphorus compounds and carbamates. Chap - 109. In: Flomenbaum NE, Goldfrank LR, Hoffman RS, et al, eds. Goldfrank's toxicologic emergencies. 8 thedn. New York: McGraw Hill 2006:1497-1513.

118. Tetzlaff JE, O'Hara JF, Walsh MT. Potassium and anaesthesia. Can J Anaesth 1993;40(3):227–246.

119. Mount DB. Fluid and electrolyte disturbances. In: Kasper D, Fauci A, Hauser S, et al, eds. Harrison's principles of internal medicine. Vol. 1. New York: McGraw-Hill Education 2018;304-306.

120. Tripathy SK, Rout PK, Debta N, et al. Study of clinical profile of organophosphorus poisoning with special reference to electrocardiographic changes and electrolyte derangement. Int J Adv Med 2018;5(1):50- 56.

121. Banday TH, Tathineni B, Desai MS, Naik V. Predictors of Morbidity and Mortality in Organophosphorus Poisoning: A Case Study in Rural Hospital in Karnataka, India. N Am J Med Sci. 2015 Jun;7(6):259-65.

122. Safdar A, Saeed A, Muhammad NR. Organophosphorus poisoning: Emergency management in intensive care unit. *Prof.* 2003;10:308–14.

123. Hussain AM, Sultan ST. Organophosphorus insecticide poisoning: Management in surgical intensive care unit. *J Coll Physicians Surg Pak.* 2005;15:100–2.

124. Ather NA, Ara J, Khan EA, Sattar RA, Durrani R. Acute organophosphate insecticide poisoning. *J Surg Pak.* 2008;13:71–4.

125. Tall A, Ly AB, Nabeth P, Sall AA, Sarr FD, Faye J, et al. Epidemiological investigation of death cases by pesticide poisoning. *J Agric Biotechnol Sustainable Develop.* 2010;2:51–5.

126. Mahadeshwara P, Gouda HS, Hallikeri VR. Plasma cholinesterase: double-edged parameter in the diagnosis of acute organophosphorus poisoning. Med Sci Law. 2010 Jul;50(3):159-60.

127. Banerjee I, Tripathi S, Roy AS. Clinico-epidemiological characteristics of patients presenting with organophosphorus poisoning. N Am J Med Sci. 2012 Mar;4(3):147-50.

128. Eddleston M, Buckley NA, Eyer P, et al. Management of acute organophosphorus pesticide poisoning. Lancet 2008;371(9612):597-607.

129. Chowdhury FR, Bari MS, Alam MMJ, et al. Organophosphate poisoning presenting with muscular weakness and abdominal pain--a case report. BMC Res Notes 2014;7:140.

130. Jayawardane P, Dawson AH, Weerasinghe V, et al. The spectrum of intermediate syndrome following acute organophosphate poisoning: a prospective cohort study from Sri Lanka. PLoS Med 2008;5(7):e147.

131. Indukuri Devi Amrutha, Maricherla Goutham Kumar Gandhi, Dr Prahallad Chandra Mishra, Evaluation Of Serum Potassium Levels As A Prognostic Marker In Acute Organophosphorus Poisoning In Tertiary Care Centre, IOSR Journal of Dental and Medical Sciences,2023; Volume 22, Issue 12 Ser.6 : PP 40-42

ANNEXURE I

INFORMED CONSENT FORM

Dear Mr. /Mrs. /Dr. _____, you are kindly requested to enroll yourself in a research study titled, "Study of effect of serum potassium levels on patients with organophosphate poisoning". You have been requested to participate in this as you fit into the laid out criteria for a study 'subject'/ participant. Your participation in study is voluntary. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY: "Study of effect of serum potassium levels on patients with organophosphate poisoning"

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR:

NAME OF THE GUIDE/C0-INVESTIGATOR:

PURPOSE OF THE STUDY:

- To study the effect of serum potassium levels on organophosphate poisoning in patients presenting to KLES Dr. Prabhakar Kore Hospital & MRC, Belagavi.

PROCEDURES INVOLVED: If you agree to enroll yourself in my study, you will be interviewed regarding your present, past and family history then you will be clinically examined in detail and investigated accordingly. Then you will be subjected to a few blood investigations, namely :

- Plasma cholinesterase concentrations
- Biochemistry including :

❖ Serial serum potassium levels

❖ White blood cell (WBC) counts

❖ Arterial blood gas analysis

RISKS AND BENEFITS: There are no potential risks involved in this study. Benefits of taking part in this research: By taking part in this study, prognosis and risk of outcome can be detected with the help of the investigations.

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY: Taking part in the study is voluntary. You may choose not to enroll yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES: Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY: All data collected or disclosed by you during the course of participation of study, will be kept fully confidential. If, however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your informed & written consent. The only people to know that you are a research subject are

members of the research team. No information about you will be disclosed to other without your written permission except: In emergency to protect your rights AND welfare. If required by law.

AUTHORIZATION TO PUBLISH RESULT: The results of the study may be used to publish an article. When the results of research published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION: No additional costs shall be incurred upon you for the purpose of this study. It is purely being done with the idea of research and all the cost of study will be borne by the investigator.

COMPENSATION: In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, or you will be given information about where to receive medical care. However, no reimbursement, compensation or free medical care will be given.

सूचितसहमति

प्रियश्री / श्रीमती /

डॉ।_____,आपसेविनम्रअनुरोधहैकिआपस्वयंकोएकशोधअध्ययनमेंनामांकित करें,"ऑर्गेनोफॉस्फेटविषाक्ततावालेरोगियोंपरसीरमपोटेशियमकेस्तरकेप्रभावकाअध्ययन"I

आपसेयहअनुरोधकियागयाहैकिआपइसमेंएकअध्ययन'विषय' /प्रतिभागीकेनिर्धारितमानदंडोंमेंफिटहों।

अध्ययनमेंआपकीभागीदारीस्वैच्छिकहै।अध्ययनकेदौरानआपसेकुछप्रश्नपूछेजाएंगेऔरआपअपनेज्ञानकासबसेअच्छाजवाबदेनेवालेहैं।अध्ययनमेंभागलेनेयानलेनेकाआपकानिर्णयकिसीभीरूपमेंआपकेउपचारकोप्रभावितनहींकरेगा।यदिआपभागलेनेकानिर्णयलेतेहैंतोआपकिसी भीसमयवापसलेनेकेलिएस्वतंत्रहैं।

अध्ययनकाशीर्षक:

"ऑर्गेनोफॉस्फेटविषाक्ततावालेरोगियोंपरसीरमपोटेशियमकेस्तरकेप्रभावकाअध्ययन"

अध्ययनकाउद्देश्य:

- केएलईएसडॉ.

प्रभाकरकोरेअस्पतालऔरएमआरसी,बेलागवीमेंउपस्थितरोगियोंमेंऑर्गनोफॉस्फेटविषाक्तापरसीरमपोटेशियमकेस्तरकेप्रभावकाअध्ययनकरना।

शामिलप्रक्रियाएं:

यदिआपमेरेअध्ययनमेंअपनानामांकनकरनेकेलिएसहमतहैं,तोआपकेवर्तमान,अतीतऔरपारिवारिकइतिहासकेबारेमेंआपकासाक्षात्कारलियाजाएगा,फिरआपकीविस्तारसेचिकित्सकीयजांचकीजाएगीऔरतदनुसारजांचकीजाएगी।फिरआपकोकुछरक्तजांचोंकेअधीनकियाजाएगा,अर्थात्:

प्लाज्माचोलिनेस्टरेज़सांद्रता

जैवरसायनसहित:

- सीरियलसीरमपोटेशियमकास्तर
- श्वेतरक्तकोशिका (डब्ल्यूबीसी) मायनेरखतीहै
- धमनीरक्तगैसविश्लेषण

जोखिमऔरलाभ:

इसअध्ययनमेंकोईसंभावितजोखिमशामिलनहींहैं।इसशोधमेंभागलेनेकेलाभ:

इसअध्ययनमेंभागलेकरजांचकीसहायतासेपूर्वानुमानऔरपरिणामकेजोखिमकापतालगायाजासकताहै।

अध्ययनसेस्वैच्छिकभागीदारी / निकासी:

अध्ययनमेंभागलेनास्वैच्छिकहै।आपइसअध्ययनमेंखुदकोनामांकितनहींकरनेकाविकल्पचुनसकतेहैंऔरबीचमेंकभीभीअध्ययनछोड़नेकाविकल्पचुनसकतेहैं।

विकल्प:

अध्ययनमेंभागलेनेकेबारेमेंआपकानिर्णयकेएलईएसडॉ।प्रभाकरकोरेअस्पतालऔरचिकित्साअनुसंधानकेंद्र,बेलगाममेंआपकेलिएपेशकीगईवर्तमानयाभविष्यकीस्वास्थ्यदेखभालसेवाओंकोनहींबदलेगा।यदिआपचाहें,तोआपकोअध्ययनसेबाहररखाजाएगाऔरआपकेसभीविवरणोंकोगोपनीयरखाजाएगाऔरआपकोप्रबंधनकीनियमितलाइनमिलजाएगी।

गोपनीयताऔरगोपनीयता :

[illegible]
[illegible]
[illegible],
[illegible]
केवलयहजाननेकेलिएकिआपएकशोधविषयहैं,अनुसंधानटीमकेसदस्यहैं।आपकेलिखितअनुमतिकेबिनाआपकेबारेमेंकोईभीजानकारीकाखुलासानहींकियाजाएगा :

- [illegible]
- [illegible]

परिणामप्रकाशितकरनेकेलिएप्राधिकरण :

अध्ययनकेपरिणामोंकाउपयोगएकलेखप्रकाशितकरनेकेलिएकियाजासकताहै।जबएकसम्मेलनमेंप्रकाशितयाचर्चाकीगईशोधकेपरिणाम, कोईभीजानकारीप्रदर्शितनहींकीजाएगीजोआपकीपहचानकाखुलासाकरेगी।इसअध्ययनकेसंबंधमेंप्राप्तकीगईकोईभीजानकारीऔरजिसेआपकेसाथपहचानाजासकताहै,गोपनीयरहेगी।

भागीदारीकेलिएवित्तीयप्रोत्साहन :

इसअध्ययनकेउद्देश्यसेआपकेऊपरकोईअतिरिक्तलागतनहींलगेगी।
यहविशुद्धरूपसेअनुसंधानकेविचारकेसाथकियाजारहाहैऔरअध्ययनकासाराखर्चअन्वेषकद्वारावहनकियाजाएगा।

भरपाई :

इसअध्ययनमेंभागलेनेकेपरिणामस्वरूपआपघायलहोजातेहैं,तोकेएलईएसडॉ।प्रभाकरकोरेअस्पतालऔरचिकित्साअनुसंधानकेंद्र,बेलगाममेंउपचारकीपेशकशकीजाएगी,याआपकोचिकित्सादेखभालकहाँप्राप्तहोगी,इसकेबारेमेंजानकारीदीजाएगी।हालांकि,कोईप्रतिपूर्ति,मुआवजायामुफ्तचिकित्सादेखभालनहींदीजाएगी।

सहमतिपत्र

मैंस्वेच्छासेनीचेहस्ताक्षरकरकेइसअध्ययनमेंभागलेनेकेलिएसहमतहूं।मैंकिसीभीसमयवापसलेसकताहूं।मैंइसफॉर्मपरहस्ताक्षरकरकेअपने किसीभीकानूनीअधिकारकोनहींछोड़रहाहूं।नीचेदिएगएमेरेहस्ताक्षरसेसंकेतमिलताहैकिमैंनेइससहमतिफॉर्मकोपढ़ाहै,यायहमेरेलिएपढ़ाग याहै,यहसहमतिफॉर्मऔरउत्तरदिएगएप्रश्नोंकेउत्तरहैं

तिभागीयाकानूनीरूपसेअधिकृतप्रतिनिधिकाहस्ताक्षर / बायाँअंगूठाप्रिंट

प्रतिभागीकानाम: ....।

हस्ताक्षर / बाएंअंगूठेकानिशान: ....।

प्रतिभागीका

कानूनीरूपसेअधिकृतकानाम: ....।

प्रतिनिधि / अभिभावक

हस्ताक्षर / बाएंअंगूठेकानिशान: ....।

साक्षीकानाम: ....।

हस्ताक्षर / बाएंअंगूठेकानिशान: ....।

अन्वेषककानामऔरहस्ताक्षर: ....।

दिनांक:

जगह:

CONSENT FORM

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

……..…….. …..……

Name of the Participant Signature of the participant or Left-Hand Thumb impression

…… ……

Name of Investigator Signature of investigator or Left-Hand Thumb impression

……. ……..

Name of Witness Signature of Witness or Left-Hand Thumb impression

Date:

Place:

ANNEXURE II

PROFORMA

CASE NO:
NAME:
AGE/SEX:
IP NO.:
ADDRESS:
OCCUPATION:
COMPLAINTS AT PRESENTATION:
Past history:
Family history:
Personal history:
Treatment history:

PHYSICAL EXAMINATION:
GENERAL CONDITION:
- PALLOR- YES/NO
- ICTERUS-YES/NO
- LYMPHADENOPATHY-YES/NO
- CYANOSIS- YES/NO
- CLUBBING-YES/NO
- EDEMA-YES/NO

VITALS:
- TEMPERATURE:
- PULSE:
- RESPIRATORY RATE:
- BLOOD PRESSURE:

SYSTEMIC EXAMINATION:
R. S.:
C.V.S.:
C.N.S.:
P.A.:

INVESTIGATIONS:
WBC COUNTS
POTASSIUM LEVELS
ABG
ECG
PLASMA CHOLINESTERASE LEVEL

ANNEXURE III

MASTER CHART

MASTERCHART KEY

OPC: Organophosphorus Compound

BP: Blood pressure

RR: Respiratory Rate

AChE: Acetylcholinesterase

WBC: White blood count

HCO3-: Bicarbonate

Sr No	IP No	Patient's Name	Age	Age group	Sex	Address	Type of OPC	rade of OP poisonir	Pulse	Systolic pp	Diastolic pp	RR	ESPI DISTRESS RR>	Temperat ure	Hypokalemia	Potassium level	AChE level (admissic	AChE level (discharg	WBC leve	H (Admissio	co2 (Admission	CO3 (Admissio	H (Discharge	co2 (Discharge	CO3 (Discharge	equirement of ventilat	Convulsion	Fasciculation	Death	uration of hospital stay
1	1E+06	RURAJ BASAVARAJ RUKL	20	ess than 25 year	M	CHIKKODI	Unknown	mild	100	136	78	20	No	97.4	Yes	3	2283	611	<10k	7.12	33	17	7.29	25	21	no	N	Yes	no	less than 10 days
2	1E+06	HIDHAR SHIVAPPA HALAI	39	31 to 40 years	M	IIREBAGEWAI	Phorate	moderate	102	132	82	20	yes	98.6	Yes	2.7	1927	255	10k-15k	7.21	34	18	7.38	26	22	no	Y	Yes	no	less than 10 days
3	1E+06	HOK SHANKARAPPA BAN/	36	31 to 40 years	M	BELAGAVI	Dimethoate	moderate	98	132	86	26	yes	97	Yes	2.8	2773	272	10k-15k	7.1	44	14	7.27	36	18	yes	N	Yes	no	more than 10 days
4	1E+06	ARESHWAR APPASAB KH(	22	ess than 25 year	M	KHANAPUR	Dichlorphos	mild	102	134	82	20	yes	98.6	No	4	2485	813	<10k	7.25	35.9	14.7	7.42	27.9	18.7	no	N	Yes	no	less than 10 days
5	1E+06	HIMANA APPAJI NITTURK/	65	hore than 50 yea	M	GOKAK	Phorate	mild	102	128	84	24	No	101	Yes	3	2221	549	<10k	7.128	34.1	15	7.298	26.1	19	no	N	No	no	less than 10 days
6	1E+06	ANIL RAGHU CHANDGADI	40	31 to 40 years	M	BELAGAVI	Phorate	mild	106	134	80	20	yes	97	No	3.7	2280	608	<10k	7.1	44	20.3	7.27	36	24.3	no	N	No	no	more than 10 days
7	1E+06	SANJAY PUNDALIK JOSHI	50	41 to 50 years	M	BELAGAVI	Unknown	mild	106	130	78	24	No	98.6	Yes	3.3	2022	350	<10k	7.1	23	16	7.27	15	20	no	N	Yes	no	less than 10 days
8	1E+06	IRAJ BALAGOUDA DEMA	20	ess than 25 year	M	NIPPANI	Phorate	mild	102	132	80	26	No	97.4	No	4.3	1834	162	<10k	7.12	28	20	7.29	20	24	no	N	Yes	no	less than 10 days
9	1E+06	SHILPA PADEPPA PATTED	24	ess than 25 year	F	BELAGAVI	Phorate	severe	88	120	82	14	yes	98.4	Yes	2.5	2224	552	10k-15k	7.12	34	16	7.29	26	20	yes	N	Yes	no	less than 10 days
10	1E+06	RENUKA MARUTI SHIRAI	19	ess than 25 year	F	BELAGAVI	Phorate	moderate	102	134	82	18	yes	98.6	Yes	2.7	2742	271	10k-15k	7.1	34	18	7.27	26	22	yes	N	No	no	more than 10 days
11	1E+06	APPA CHANDRAPPA KUNI	62	hore than 50 yea	M	CHANDGADH	Unknown	mild	102	128	82	20	No	101.2	Yes	3.4	1737	65	<10k	7.1	36	18	7.27	28	22	no	N	No	no	less than 10 days
12	1E+06	YYA GURUPADAYYA HIRI	38	31 to 40 years	M	BAILHONGAL	Phorate	mild	102	128	82	22	yes	99.4	Yes	3	2420	748	<10k	7.1	36	20	7.27	28	24	no	N	No	no	less than 10 days
13	1E+06	EGANA OMKAR HONGEK/	35	31 to 40 years	F	HUKKERI	Unknown	mild	84	132	82	24	No	97.4	No	4.3	2352	680	<10k	7.11	33	18	7.28	25	22	no	N	Yes	no	less than 10 days
14	1E+06	SHANKAR SOMAPPA PUJEI	26	25 to 30 years	M	KHANAPUR	Unknown	severe	88	144	82	22	yes	98.6	Yes	1.8	2223	551	>15k	7.11	44	15.2	7.28	36	19.2	yes	Y	Yes	yes	less than 10 days
15	1E+06	MKAR LAXMAN HONGEK/	38	31 to 40 years	M	HUKKERI	Phorate	severe	102	144	84	20	yes	101	Yes	2.4	2673	453	10k-15k	7.1	33.4	18	7.27	25.4	22	yes	N	Yes	no	more than 10 days
16	1E+06	ANTOSH ISHAWAR JANTA	38	31 to 40 years	M	BAILHONGAL	Unknown	severe	102	144	82	24	yes	102.6	Yes	2.3	2823	374	>15k	7.11	33	18	7.28	25	22	yes	N	Yes	yes	less than 10 days
17	1E+06	NATH YALLAPPA SOMAN/	25	25 to 30 years	M	BELAGAVI	Phorate	severe	88	144	84	26	yes	99	Yes	2.5	1755	183	10k-15k	7.11	35	24	7.28	27	28	yes	N	Yes	no	less than 10 days
18	1E+06	SACHIN ANAND GODEKAI	27	25 to 30 years	M	SANKESHWAI	Phorate	severe	86	132	80	18	yes	100	Yes	1.5	2385	713	10k-15k	7.28	38	18	7.45	30	22	yes	Y	Yes	yes	less than 10 days
19	1E+06	SNEHA MOHAN KINIKAR	19	ess than 25 year	F	GOKAK	Phorate	mild	102	132	82	20	No	98	Yes	3.1	2733	726	<10k	7.2	37	20	7.37	29	24	no	N	Yes	no	less than 10 days
20	1E+06	ANAND SHANKAR KHOT	24	ess than 25 year	M	NIPPANI	Phorate	mild	102	132	90	18	No	98.6	No	4	2501	829	<10k	7.34	38	18	7.51	30	22	no	N	Yes	no	less than 10 days
21	1E+06	A MOHAMMAD RAFIQ PE	24	ess than 25 year	F	CHIKKODI	Dimethoate	mild	98	134	72	22	No	99	No	3.6	2732	726	<10k	7.3	44	15	7.47	36	19	no	N	Yes	no	more than 10 days
22	1E+06	LAXMAN BALLAPPA DOO(	32	31 to 40 years	M	HUKERI	Phorate	mild	100	140	80	22	No	98	No	3.6	1679	324	<10k	7.24	42	18	7.41	34	22	no	N	Yes	no	less than 10 days
23	1E+06	IRAPPA RAMAPPA BELAC	49	41 to 50 years	M	CHIKKODI	Unknown	severe	100	130	82	18	yes	98.6	Yes	2.4	2286	614	>15k	7.01	40	14	7.18	32	18	yes	Y	Yes	no	more than 10 days
24	1E+06	EENA MAHANTESH BISKC	35	31 to 40 years	F	BELAGAVI	Phorate	mild	92	134	80	24	No	99.4	Yes	3	2243	571	<10k	7.28	40	17	7.45	32	21	no	Y	Yes	no	less than 10 days
25	1E+06	AZIRUL ASLAM INAMDAI	40	31 to 40 years	F	GOKAK	Unknown	mild	106	140	86	26	No	98	Yes	3	2922	272	<10k	7.3	44	15	7.47	36	19	no	N	Yes	no	less than 10 days
26	1E+06	H SHANKARAPPA DHULAI	18	ess than 25 year	M	NIPPANI	Parathion	severe	102	130	86	18	yes	102	Yes	2.3	2221	549	10k-15k	7.32	45	15	7.49	37	19	yes	N	Yes	yes	more than 10 days
27	1E+06	AJ SHANKARAPPA DHULA	20	ess than 25 year	M	BELAGAVI	Phorate	mild	100	134	86	24	yes	99.4	No	3.6	2200	528	<10k	7.3	45	15	7.47	37	19	no	N	Yes	no	more than 10 days
28	1E+06	SHNAVI GANPATSING RAJ	19	ess than 25 year	F	HUKKERI	Phorate	moderate	102	144	80	22	No	101.6	Yes	3	2050	378	10k-15k	7.43	35	15	7.6	27	19	no	N	Yes	no	less than 10 days
29	1E+06	HAHAPURI GOPAL JINNAPI	55	hore than 50 yea	M	SANKESHWAI	Phorate	moderate	92	140	78	20	No	101.2	Yes	3.5	2922	372	10k-15k	7.2	37	15	7.37	29	19	no	N	Yes	no	less than 10 days
30	1E+06	D BHARAMAPPA BALEKU	21	ess than 25 year	M	KHANAPUR	Phorate	moderate	102	134	82	26	No	98	No	4.6	2541	869	<10k	7.34	38	18	7.51	30	22	no	Y	Yes	no	less than 10 days
31	1E+06	PUNDALIK GAWADU PATII	35	31 to 40 years	M	BELAGAVI	Unknown	moderate	100	136	78	18	No	98.6	Yes	3	2442	770	<10k	7.3	44	15	7.47	36	19	no	N	Yes	no	less than 10 days
32	1E+06	ESH HANAMANTAPPA SAL	36	31 to 40 years	M	BELAGAVI	Phorate	moderate	102	132	82	24	yes	97.6	Yes	2.7	1782	110	10k-15k	7.24	42	18	7.41	34	22	yes	Y	Yes	no	less than 10 days
33	1E+06	RIKANT SURENDRA SHET	40	31 to 40 years	M	IIREBAGEWAI	Dimethoate	moderate	98	132	86	19	yes	98.6	Yes	2.8	2292	620	10k-15k	7.28	38	24	7.45	30	28	no	N	Yes	no	less than 10 days
34	1E+06	HIDAPPA BASAPPA ANKAI	24	ess than 25 year	M	BELAGAVI	Dichlorphos	severe	102	134	92	22	yes	97.6	No	4	1183	535	<10k	7.26	44	22	7.43	36	26	yes	N	Yes	no	less than 10 days
35	1E+06	NIDA ISAK SAYYED	25	25 to 30 years	F	KHANAPUR	Phorate	moderate	102	128	82	27	No	98.6	No	3.6	1804	641	<10k	7.28	43	18	7.45	35	22	no	N	No	no	less than 10 days
36	1E+06	HANDRAKANT AJIT SOUD	20	ess than 25 year	M	GOKAK	Phorate	severe	106	144	80	22	yes	99.4	No	3.7	1103	425	<10k	7.3	44	15	7.47	36	19	no	N	No	no	more than 10 days
37	1E+06	ADITYA NARSING KURADI	19	ess than 25 year	M	BELAGAVI	Phorate	moderate	106	130	78	26	No	98	Yes	3.3	1404	242	10k-15k	7.26	40	19	7.43	32	23	no	N	No	no	less than 10 days
38	1E+06	SHIVANI RAMADAS PATIL	23	ess than 25 year	F	BELAGAVI	Phorate	moderate	102	136	80	26	No	98.6	No	4.3	1101	636	<10k	7.3	42	20	7.47	34	24	no	N	Yes	no	less than 10 days
39	1E+06	IESH SIDAPPA NEELANNA	40	31 to 40 years	M	CHIKKODI	Phorate	severe	88	120	94	26	yes	97.4	Yes	2.5	1671	289	>15k	7.1	40	21	7.27	32	25	yes	N	Yes	no	less than 10 days
40	1E+06	ASHRAM GUNDUJI SAKRO.	60	hore than 50 yea	M	BELAGAVI	Phorate	moderate	100	134	92	22	yes	103	Yes	2.7	2288	616	<10k	7.14	42	20	7.31	34	24	yes	N	No	no	less than 10 days
41	1E+06	KSHI BASAVARAJ DEVAR/	35	31 to 40 years	F	BELAGAVI	Phorate	moderate	100	140	92	13	No	98	No	3.6	1901	229	<10k	7.15	41	17	7.32	33	21	no	N	No	no	less than 10 days
42	1E+06	RAMAVVA ARJUN KATTI	32	31 to 40 years	M	CHANDGADH	Phorate	moderate	102	128	94	20	No	97.4	No	3.6	2271	599	<10k	7.19	44	15	7.36	36	19	no	N	No	no	more than 10 days
43	1E+06	VACHANDRA ALLAPPA DI	28	25 to 30 years	M	BAILHONGAL	Dimethoate	moderate	84	128	92	20	No	98.6	No	4.3	2222	550	<10k	7.18	41	15	7.35	33	19	no	N	No	no	less than 10 days
44	1E+06	MI BALLAPPA KALASANN/	56	hore than 50 yea	F	HUKKERI	Phorate	severe	88	128	94	26	yes	97	Yes	1.8	2802	155	>15k	7.14	42	15	7.31	34	19	yes	Y	Yes	yes	more than 10 days
45	1E+06	PPA PUNDALIK TADASAN	48	41 to 50 years	M	KHANAPUR	Phorate	severe	100	134	94	20	yes	98.6	Yes	2.4	2404	732	10k-15k	7.18	41	14	7.35	33	18	yes	N	Yes	no	more than 10 days
46	1E+06	A MOHAMMAD RAFIQ BAC	22	ess than 25 year	F	HUKKERI	Unknown	severe	100	134	82	24	yes	101	Yes	2.3	2243	571	>15k	7.19	45	12	7.36	37	16	yes	N	Yes	no	more than 10 days
47	1E+06	SADASHIV BABU KUMBAI	57	hore than 50 yea	M	BAILHONGAL	Phorate	severe	88	134	82	20	yes	97	Yes	2.5	2352	680	10k-15k	7.13	45	16	7.3	37	20	yes	N	Yes	no	less than 10 days
48	1E+06	KSHATA GOPAL KALLOLI	29	25 to 30 years	F	BELAGAVI	Phorate	severe	86	132	90	24	yes	98.6	Yes	1.5	2841	266	10k-15k	7.11		15	7.28	36	19	yes	Y	Yes	yes	less than 10 days
49	1E+06	MED SHAHID BASHIRAHMI	38	31 to 40 years	M	SANKESHWAI	Phorate	moderate	100	132	82	26	No	97.4	Yes	3.1	2201	529	10k-15k	7.17	45	15	7.34	37	19	no	N	Yes	no	less than 10 days
50	1E+06	APNA VIDYADHAR KABB	33	31 to 40 years	F	GOKAK	Phorate	severe	100	132	90	14	No	98.4	No	4	2718	452	>15k	7.31	45	14	7.48	37	18	no	N	No	no	less than 10 days
51	1E+06	YADHAR NEMINATH KAB	35	31 to 40 years	M	NIPPANI	Unknown	moderate	98	134	92	18	No	98.6	No	3.6	1684	372	<10k	7.19	41	13	7.36	33	17	no	N	No	no	less than 10 days
52	1E+06	AVEEN SHEKHAR RABAK/	44	41 to 50 years	M	CHIKKODI	Phorate	severe	100	140	92	20	No	101.2	No	3.6	1113	142	<10k	7.11	45	15	7.28	37	19	no	N	No	no	less than 10 days
53	1E+06	NTOSH SHETTEPPA MAGD	19	ess than 25 year	M	HUKKERI	Phorate	severe	100	140	92	22	No	99.4	Yes	2.4	1141	263	10k-15k	7.14	45	15	7.31	37	19	no	Y	Yes	no	more than 10 days
54	1E+06	ANIL VASANT BOKADE	28	25 to 30 years	M	CHIKKODI	Phorate	moderate	92	134	96	24	No	97.4	Yes	3	1481	263	10k-15k	7.11	41	18	7.28	33	22	no	N	No	no	less than 10 days
55	1E+07	VISHWAS SURESH MALI	26	25 to 30 years	M	BELAGAVI	Phorate	moderate	100	130	92	22	No	98.6	Yes	3	1744	748	10k-15k	7.15	45	17	7.32	37	21	no	N	Yes	no	less than 10 days
56	1E+06	HA LAGAMANNA GADDI	43	41 to 50 years	F	GOKAK	Parathion	severe	100	140	82	18	No	97.4	Yes	2.3	1080	192	>15k	7.16	44	16	7.33	36	20	no	N	Yes	no	more than 10 days
57	1E+07	PPA DEVAPPA MARYALLA	53	hore than 50 yea	M	BELAGAVI	Phorate	moderate	98	134	96	24	No	97.8	No	3.6	2260	588	<10k	7.11	41	15	7.28	33	19	no	N	Yes	no	less than 10 days
58	1E+07	RAJ ADIVEPPA KANCHAGA	32	31 to 40 years	M	BELAGAVI	Unknown	moderate	98	130	96	16	No	98.2	Yes	3	2642	970	10k-15k	7.1	44	17	7.27	36	21	no	N	Yes	no	less than 10 days
59	1E+07	UR MAHADEV MUNGARW	33	31 to 40 years	M	HUKKERI	Phorate	moderate	92	140	96	27	No	101	No	3.6	2342	670	<10k	7.11	45	17	7.28	37	21	no	N	Yes	no	less than 10 days
60	1E+07	UTTAPPA SATYAPPA GHAS	22	ess than 25 year	M	SANKESHWAI	Phorate	moderate	102	134	82	20	No	97.4	No	4.6	2202	530	<10k	7.16	45	13	7.33	37	17	no	Y	Yes	no	less than 10 days