B.L.D.E (DEEMED TO BE UNIVERSITY) VIJAYAPURA, KARNATAKA

Dissertation submitted to BLDE (Deemed to be University)

Vijayapura Karnataka

In partial fulfilment of the requirements for the award of the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

"CORRELATION OF SERUM POTASSIUM AND PLASMA CHOLINESTERASE IN ASSESSING THE SEVERITY OF ACUTE ORGANOPHOSPHATE POISONING"

BY

Dr. ESWARA SAI SONTINENI

Under the guidance of

Dr. PRAKASH .G. MANTUR

M.D (MEDICINE)

Professor,

Department of General Medicine

BLDE (DEEMED TO BE UNIVERSITY)

SHRI B .M PATIL MEDICAL COLLEGE HOSPITAL

& RESEARCH CENTRE VIJAYAPURA, KARNATAKA

2025

B.L.D.E DEEMED TO BE UNIVERSITY

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL

& RESEARCH CENTRE, VIJAYAPURA

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation/thesis entitled **"CORRELATION OF SERUM POTASSIUM AND PLASMA CHOLINESTERASE IN ASSESSING THE SEVERITY OF ACUTE ORGANOPHOSPHATE POISONING"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. PRAKASH.G. MANTUR, MD (**GENERAL MEDICINE) Professor, Department of Medicine, Shri B.M. Patil Medical College, Vijayapura, Karnataka.

Date: **Dr. ESWARA SAI SONTINENI**

Place: Vijayapura

B.L.D.E DEEMED TO BE UNIVERSITY

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL

& RESEARCH CENTRE, VIJAYAPURA

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"CORRELATION OF SERUM POTASSIUM AND PLASMA CHOLINESTERASE IN ASSESSING THE SEVERITY OF ACUTE ORGANOPHOSPHATE POISONING"** is a bonafide and genuine research work carried out by **Dr. ESWARA SAI SONTINENI** in partial fulfillment of the requirement for the degree of MD in General medicine.

Date:

Place: Vijayapura

Dr. PRAKASH.G. MANTUR, M.D
Professor,
Department of General Medicine
BLDE (Deemed) to be University,
Shri B.M.Patil Medical college, Hospital and
Research Center, Vijayapura, Karnataka

B.L.D.E DEEMED TO BE UNIVERSITY

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL

& RESEARCH CENTRE, VIJAYAPURA

ENDORSEMENT BY THE HOD, PRINCIPAL/HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"CORRELATION OF SERUM POTASSIUM AND PLASMA CHOLINESTERASE IN ASSESSING THE SEVERITY OF ACUTE ORGANOPHOSPHATE POISONING"** is a bonafide research work carried out by **Dr. ESWARA SAI SONTINENI** under the guidance of **Dr. PRAKASH.G. MANTUR** MD Professor, Department of Medicine, Shri B.M. Patil Medical College and Research Centre, Vijayapura.

Seal & Signature of HOD of Medicine

Dr. SANJEEVKUMAR N. BENTOOR
M. D. (Medicine)
BLDEDU's Shri B.M. Patil
Medical College, Hospital
& Research Centre, Vijayapura

Date:

Place: Vijayapura

Seal and signature of The Principal

Dr. ARAVIND V PATIL
M.S. (General Surgery)
BLDEDU's Shri B.M.Patil Medical
College, Hospital
& Research Centre, Vijayapura

Date:

Place: Vijayapura

B.L.D.E DEEMED TO BE UNIVERSITY

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL

& RESEARCH CENTRE, VIJAYAPURA

COPYRIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the BLDE Deemed to be University, Vijayapura, Karnataka shallhave the rights to preserve, use and disseminate this dissertation /thesis in print or electronic format for academic / research purpose.

Date: **Dr. ESWARA SAI SONTINENI**

Place: Vijayapura

ACKNOWLEDGEMENT

I have got no words to express my deep sense of gratitude and regards to my guide **Dr. PRAKASH. G. MANTUR,** M.D, Professor of Medicine, under whose inspiring guidance & supervision, I am studying and continuing to learn the art of medicine. His deep knowledge, devotion to work and zeal of scientific research makes him a source of inspiration not only for me but for others too. It is because of his generous help, expert and vigilant supervision, that has guided & helped me to bring out this work in the present form.

My sincere thanks are due to **Dr. ARVIND PATIL** MS Principal, & **Dr. SANJEEVKUMAR N. BENTOOR M.D**, HOD, Department of General Medicine, Shri B. M. Patil Medical College, Vijayapura, for permitting me to conduct this study. I wish to acknowledge my professors and take this opportunity to express my deep sense of gratitude and sincere thanks to **Dr. R.C. BIDRI, Dr. M.S. MULIMANI, Dr. S. S. DEVARMANI, Dr. BADIGER SHARANABASAWAPPA, Dr. VIJAY KUMAR WARAD, Dr. R.M. HONNUTAGI**, **Dr. S.M. BIRADAR** for their supervision and timely advice.

I would be failing in my duty if I would not acknowledge my thanks to all the patients who were kind enough to help with this study. I would like to thank my parents **SONTINENI SRINIVASA RAO**, **SONTINENI LAKSHMI** and brother **Dr. HEMANTH VENKATA SAI** for their constant encouragement and moral support.

Dr. ESWARA SAI SONTINENI

ABSTRACT

Introduction:

Organophosphate (OP) compounds are widely used as pesticides, particularly in agricultural regions, and constitute a significant cause of poisoning worldwide. The toxicity primarily results from inhibition of acetylcholinesterase enzyme, leading to accumulation of acetylcholine at synapses and manifesting as a characteristic cholinergic toxidrome. While plasma cholinesterase levels have been traditionally used to confirm exposure and assess severity, their correlation with clinical outcomes is not always consistent. Recent studies have suggested alterations in serum electrolytes, particularly potassium, as potential markers of poisoning severity. This study aimed to correlate serum potassium and plasma cholinesterase levels with the clinical severity of acute organophosphate poisoning.

Methods:

This prospective study was conducted at Shri B.M. Patil Medical College, Hospital and Research Centre, Vijayapura, from May 2023 to December 2024. A total of 83 patients with acute organophosphate poisoning were included. Detailed clinical assessment, including Glasgow Coma Scale (GCS) scoring and pupillary examination, was performed at admission. Serum potassium and plasma cholinesterase levels were measured at admission and after 24 hours. Patients were classified as having severe or non-severe poisoning based on clinical parameters and requirement for mechanical ventilation. Statistical analysis was performed to assess the correlation between biochemical markers and poisoning severity.

Results:

The majority of patients (67.5%) were between 20-40 years of age, with a slight male predominance (51.8%). Common clinical features included vomiting (74.7%), muscle weakness (62.6%), and bronchospasm (30.1%). Severe poisoning was observed in 37.3% of patients, with 26.5% requiring mechanical ventilation. Patients with severe poisoning had significantly lower serum potassium levels both initially (3.32±0.59 vs 4.08±0.47 mEq/L, $p<0.001$) and at 24 hours (3.6±0.55 vs 4±0.34 mEq/L, $p=0.001$) compared to non-severe cases. Similarly, plasma cholinesterase levels were significantly lower in severe cases, both initially (1465.4±2336.2 vs 3455.4±2785.4, $p=0.001$) and at 24 hours (1633.5±2620.9 vs 3550.9±2794.6, $p=0.01$). A significant positive correlation was observed between serum potassium and acetylcholinesterase levels in both severe ($r=0.675$, $p=0.001$) and non-severe ($r=0.582$, $p=0.003$) poisoning cases. Patients requiring mechanical ventilation had markedly lower initial potassium levels (3.12±0.48 mEq/L vs. 3.97±0.54 mEq/L, $p<0.001$). There was a strong association between mortality and low serum potassium, with non-survivors having dramatically lower potassium levels (2.86±0.44 mEq/L) compared to survivors (3.94±0.51 mEq/L, $p<0.001$). Similarly, patients who died had profoundly reduced initial acetylcholinesterase levels (452.8±198.6) compared to those who were discharged (3212.9±2824.8, $p<0.001$). Severe poisoning was also associated with lower GCS scores ($p<0.001$) and smaller pupil sizes (1.48±0.67 vs 2.37±0.74 mm, $p<0.001$).

Conclusion:

Serum potassium levels show a significant correlation with the severity of acute organophosphate poisoning, comparable to the established marker plasma cholinesterase. The combination of these biochemical parameters with clinical indicators like GCS scores and pupillary changes provides a comprehensive approach to severity assessment. Serum potassium measurement can serve as a simple, cost-effective, and readily available tool for early risk stratification, particularly in resource-limited settings.

Keywords:

Organophosphate poisoning, Serum potassium, Plasma cholinesterase, Glasgow Coma Scale, Hypokalemia, Poisoning severity, Acetylcholinesterase, Miosis, Mechanical ventilation, Prognostic markers.

ABBREVIATIONS

Ach	:	Acetylcholine
AChE	:	Acetylcholinesterase
ANOVA	:	Analysis of Variance
APACHE	:	Acute Physiology and Chronic Health Evaluation
BChE	:	Butyrylcholinesterase (Plasma Cholinesterase)
ChE	:	Cholinesterase
CNS	:	Central Nervous System
COPD	:	Chronic Obstructive Pulmonary Disease
ECG	:	Electrocardiogram
GCS	:	Glasgow Coma Scale
HR	:	Heart Rate
ICU	:	Intensive Care Unit
IM	:	Intramuscular
IV	:	Intravenous
K+	:	Potassium
mEq/L	:	Milliequivalents per Liter
Na+	:	Sodium
Na+-K+ ATPase	:	Sodium-Potassium Adenosine Triphosphatase
OP	:	Organophosphate
PAM	:	Pralidoxime (Pyridine-2-aldoxime methochloride)
PChE	:	Plasma Cholinesterase
PNS	:	Peripheral Nervous System
POP Scale	:	Peradeniya Organophosphorus Poisoning Scale
PSS	:	Poisoning Severity Score

RBC	:	Red Blood Cell
RR	:	Respiratory Rate
SD	:	Standard Deviation
SPSS	:	Statistical Package for the Social Sciences
WHO	:	World Health Organization

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Organophosphate (OP) poisoning represents one of the most significant causes of pesticide-related morbidity and mortality worldwide, particularly in developing countries where agricultural practices are prevalent and regulations may be less stringent.[1] The World Health Organization estimates that approximately three million cases of pesticide poisoning occur annually, with organophosphates accounting for a substantial proportion of these incidents, resulting in an estimated 300,000 deaths.[2]

The toxicological mechanism of organophosphate compounds primarily involves the inhibition of acetylcholinesterase (AChE), leading to the accumulation of acetylcholine at synaptic junctions and neuromuscular junctions. This accumulation results in continuous stimulation of cholinergic receptors, manifesting as a characteristic cholinergic crisis.[3] The clinical presentation encompasses a wide spectrum of symptoms affecting multiple organ systems, including the central nervous system, autonomic nervous system, and neuromuscular junctions, making early recognition and accurate assessment of severity crucial for appropriate management.

Plasma cholinesterase levels have traditionally served as a biochemical marker for diagnosing and monitoring OP poisoning. This enzyme, also known as butyrylcholinesterase or pseudocholinesterase, shows a rapid decline in activity following OP exposure, making it a valuable diagnostic tool.[4] However, the relationship between plasma cholinesterase levels and clinical severity has shown varying degrees of correlation in different studies, suggesting the need for additional parameters to enhance prognostic accuracy.

The role of electrolyte disturbances, particularly serum potassium alterations, has gained increasing attention in recent years. Potassium homeostasis can be significantly disrupted in OP poisoning through multiple mechanisms, including altered

cellular membrane permeability, autonomic dysfunction, and respiratory compromise.[5] Early studies have suggested that hyper- or hypokalemia may correlate with the severity of poisoning and could potentially serve as a prognostic indicator when evaluated alongside other clinical and biochemical parameters.

The complex pathophysiology of OP poisoning involves multiple mechanisms that can affect both cholinesterase activity and potassium homeostasis. The inhibition of acetylcholinesterase leads to excessive cholinergic stimulation, which can cause increased secretions, bronchospasm, bradycardia, and muscle fasciculations. These manifestations can indirectly influence potassium levels through various mechanisms, including respiratory acidosis, tissue hypoxia, and altered cellular transport.[6]

Furthermore, the autonomic effects of OP poisoning can lead to significant cardiovascular complications, which may be exacerbated by electrolyte disturbances. Studies have shown that patients with severe OP poisoning often exhibit ECG changes, some of which may be attributed to potassium abnormalities.[7] Understanding the relationship between serum potassium levels and poisoning severity could therefore have important implications for cardiac monitoring and management strategies.

The assessment of OP poisoning severity has traditionally relied on clinical scoring systems, such as the Peradeniya Organophosphorus Poisoning (POP) scale and the Modified Glasgow Coma Scale. While these scoring systems provide valuable clinical information, the integration of biochemical parameters could potentially enhance their predictive value.[8] The correlation between serum potassium and plasma cholinesterase levels, if established, could provide a more comprehensive approach to severity assessment.

Recent research has suggested that the combined evaluation of multiple parameters, including clinical features, cholinesterase levels, and electrolyte status, may

provide better prognostic information than any single parameter alone.[9] This multimodal approach to severity assessment could potentially improve risk stratification and guide more targeted therapeutic interventions. Understanding the relationship between these parameters could also help identify patients at higher risk of complications and poor outcomes.

The timing of biochemical measurements in relation to poisoning onset and treatment initiation also presents an important consideration. While plasma cholinesterase levels typically show early depression following OP exposure, the temporal pattern of potassium alterations and their relationship to clinical severity may vary. Additionally, therapeutic interventions, particularly atropine and oxime therapy, may influence both cholinesterase reactivation and electrolyte balance.[10]

This research aims to investigate the correlation between serum potassium levels and plasma cholinesterase activity in acute OP poisoning, with particular emphasis on their combined utility in assessing poisoning severity. By examining these relationships, we hope to contribute to the development of more comprehensive and accurate methods for evaluating OP poisoning severity, ultimately leading to improved patient outcomes through more targeted therapeutic approaches.

AIM & OBJECTIVES

Objectives:

1. To evaluate the levels of serum potassium and plasma cholinesterase in Acute Organophosphorous poisoning and
2. To correlate them in assessing the severity of acute organophosphorous poisoning

REVIEW OF LITERATURE

ORGANOPHOSPHOROUS (OP) POISONING

EPIDEMIOLOGY

Global Status: More than 80% of hospitalisations connected to pesticides are caused by the organophosphate chemicals, which are most frequently linked to serious human poisoning.[11"]Because of their unstable chemical structure, which causes rapid hydrolysis and little long-term accumulation in the environment, organophosphate insecticides have grown in popularity for both home and agricultural use, in contrast to the days when chlorinated hydrocarbon compounds like DDT were widely used.[12] Figure I displays the chemical structures of a few OP compounds. However, more human poisonings have occurred as a result of this broad use. According to estimates from the Environmental Protection Agency until the 1970s, pesticide poisoning in the US required 3,000 hospitalisations annually, with a 10% death rate for adults and 50% for children.[13] According to data from the American Association of Poison Control Centres, 33,000 of the 77,000 insecticide exposures nationwide in 1983 were organophosphates. [14] According to a 2020 study, there were 740,000 unintended pesticide poisonings in 141 nations, which led to 7446 fatalities. Because of poor reporting and a lack of statistical data, the true level of exposure and toxicity is probably higher."[15]

National status: Given that agriculture accounts for 22% of India's GDP and provides a living for about 70% of its workforce, the significance of pesticides in the country is clear. Almost 60% of the global agrochemical market is controlled by the top five multinational corporations as a result of industry consolidation. With over 400 formulators and 30 to 40 major producers, the Indian sector is highly fragmented. In 2006, insecticides accounted for 67% of all pesticide usage, indicating a lopsided use

pattern. Due to ease access to extremely dangerous goods and little risk knowledge, particularly among women and children, the potential negative effects of pesticide exposure on human health are probably greater in nations like India. “Because of easy access for kids and inadequate labelling, overexposure to pesticides can happen before spraying, during mixing, during spraying, and after spraying operations. Bystanders and spray operators may be impacted. Intentional pesticide poisonings are more common when highly toxic chemicals are readily available and inexpensive.”[16] Poisoning has grown in concern during the last ten years, both in India and internationally. 38 Poisoning is only a 1–2% cause of death in developed nations, but it is the fourth leading cause of death in developing nations like India, with rates ranging from 15–30%, particularly in rural areas. [17] According to WHO estimates, pesticides are currently the most popular way for people to commit suicide globally. “In 2016, the suicide death rate was 16.5 per 100,000, compared to the global average of 10.5 per 100,000. The elderly, those with special needs, and those aged 15 to 29 are the most at risk.”[18]

Due to the extensive usage of pesticides for domestic and agricultural purposes, pesticide poisoning is very common in India. The most common cause of suicide in India for both men and women aged 15 and over is pesticide poisoning, primarily from organophosphates, which accounts for over 92,000 fatalities per year..[19]

HISTORICAL ASPECTS

The French scientist Philippe de Clermont was credited by Swedish pharmacologist Bo Holmstedt in a well cited publication with creating the first OP (tetra ethyl pyrophosphate, or TEPP) in 1854. [20] However, other people have suggested that some OPs might have been created even earlier. Triethyl phosphate (TEP) was created in 1820 by Jean Louis Lassaigne when ethanol and phosphoric acid interacted; nevertheless, Franz Anton Voegeli was later credited with this synthesis in 1848. “Jean

Pierre Boudet, another Frenchman, is thought to have created an OP from phosphoric acid and alcohol even earlier, in 1801. [21] Despite being the first OP cholinesterase inhibitor, TEPP was synthesised by a number of different chemists in addition to de Clermont (with assistance from Russian chemist Wladimir Moschnin, who was also employed at Adolphe Wurtz's laboratory in Paris). In fact, de Clermont sampled the substance and reported it as a sticky liquid with a burning taste and an odd odour. At the time, neither the toxicity nor the mode of action of TEPP were understood. Willy Lange of the University of Berlin created a few compounds with the P-F bond in 1932." He observed the harmful effects of the vapours on himself while working with graduate student Gerda von Krueger to synthesise dimethyl- and diethyl phosphofluoridate. "The vapours of these compounds have a pleasant and strongly aromatic odour, but a marked pressure develops in the larynx a few minutes after inhaling, along with breathlessness," they stated. Mild consciousness problems then appeared, along with a painful reactivity of the eyes to light and a dazzled sense. It takes a few hours for the symptoms to go down. The effects are produced in very little amounts. Although Lange appeared to be aware that OP chemicals may be used to create insecticides, he quickly departed Germany to relocate to the US, where he worked for Procter & Gamble and the University of Cincinnati before leaving the OP industry. [22]

Despite all of these prior attempts and achievements, Gerhard Schrader, a chemist at the German company I.G. Farben, is regarded as the founder of contemporary OP pesticide toxicity. One day in December 1936, Schrader was working on the synthesis of organic fluorine and sulphur compounds when he realised "that, on my way home, my visual acuity was somewhat reduced." My vision had almost fully recovered by the next day, so I went back to work. It became clear that a new synthetic drug was the cause of more visual problems. It was discovered that 0-ethyl N, N-

dimethyl-phosphoroamido-fluoridate was too poisonous to warm-blooded animals to be utilised in farming. Although it was not stable enough for plant protection, Schrader is credited with developing a novel, straightforward process for synthesising TEPP, the first OP pesticide to be sold commercially under the trade name Bladan in combination with other hexa-compounds. Schrader is credited with creating thousands of OP chemicals. [23] "Although octamethyl-pyrophosphoramide (OMPA) was synthesised in 1942, the real "breakthrough" occurred in 1944 when a novel compound with ideal stability and insecticidal action (code name E605) was created.

The Allies took over the synthesis techniques at the end of World War II, and E605 was eventually released into the agricultural market under the trade name parathion, which turned out to be the most popular insecticide in this class. British researchers McCombie and Saunders were also working on OPs concurrently with Schrader; they later patented dimefox and diisopropyl fluorophosphate (DFP). Some of the OPs that Schrader synthesised during that time proved to be highly harmful to mammals. The development of OPs followed two parallel strategies, which were declared "secret" by the German government in 1938. The first was the synthesis of chemicals that were less toxic to mammals and effective as insecticides; the second was the development of compounds with high human toxicity and high volatility, which were to be used as poison gases in place of phosgene, mustard gas, or chlorine. Although they weren't employed during World War II, compounds like Tabun, Sarin, and Soman were created during that time with the possibility of being utilised as chemical warfare weapons. [24] Hundreds of OP compounds have been produced and marketed globally as insecticides in a range of formulations since the late 1930s." When the majority of commonly used organochlorine pesticides were phased out or outlawed in the 1970s, their use peaked. OPs made up about 70% of all insecticides used in the

United States until 2000, but in the years that followed, that percentage was cut in half. Nonetheless, the majority of underdeveloped nations continue to use OPs extensively, mostly because to their low cost in comparison to more modern pesticides. [25]

"The mechanism of action of OPs, which is the inhibition of acetylcholinesterase (AChE), was also identified concurrently with their manufacture. German researchers discovered that atropine might act as an antidote to the parasympathomimetic (cholinergic) effects of OPs. These conclusions were undoubtedly made easier by the actions of physostigmine, an alkaloid that was isolated in 1864, whose mode of action as an AChE inhibitor was clarified by Loewi and Navratil in 1926, and whose miotic activity and atropine antagonism were simultaneously identified. [26] In fact, as early as 1939, the mechanism of action of OPs was proposed. Ten years later, Ken Du Bois and John Doull conclusively proved that parathion toxicity resulted from AChE inhibition. The identification of the reactivation and "ageing" of the phosphorylated AChE are two other significant turning points in the early history of OPs. Irwin Wilson of Columbia University in New York demonstrated in 1951 that hydroxylamine may restart AChE that had been blocked by OPs. Wilson (in the United States) and Albert Green and Dan Davies (in the United Kingdom) worked together over the course of the following several years to synthesise pralidoxime (2-PAM), which, when combined with atropine, is still the major treatment for OP poisoning today. The mid-1950s discovery that oximes can reactivate phosphorylated proteins somewhat counteracted this positive development in the treatment of OP poisoning. (The more general term phosphylate / phosphylation may also be used to describe the interaction of OPs with B-esterases.) Since "ageing" (the nonenzymatic removal of an alkyl chain from the phosphate) would change the inhibited enzyme into a nonreactivatable form, AChE declined over time.[27]

Since natural compounds are the source of insecticides like pyrethroids and carbamates, natural OPs have also been discovered, albeit after synthetic OPs were created. After being separated from cultures of the soil microbe Streptomyces antibioticus, two OPs (designated CGA 134735 and CGA 134736) were discovered to be strong AChE activity inhibitors. The freshwater cyanobacterium anabaena flos-aquae strain NRC-525-17 yielded another naturally occurring substance, anatoxin-a, which was discovered to be an irreversible inhibitor of AChE. Therefore, decades of chemical research have ultimately "reinvented" (and improved) what nature had already provided, even for OPs."[28]

CHEMISTRY AND METABOLISM OF OPS

"Figure 1 depicts the overall structure of OPs, which was first suggested by Schrader in 1937. Their chemistry has been extensively studied. X is the so-called "leaving group," which is eliminated when the OP phosphorylates AChE and is the most susceptible to hydrolysis. R1 and R2 are most frequently alkoxy groups (i.e., OCH3 or OC2H5), though isopropyl substitutes are also possible. The pentavalent phosphorus is double-bonded to either an oxygen or a sulphur (in this case, the compound is defined as a phosphorothioate). Phosphonothioates, phosphoramidates, phosphonates, and other chemical subclasses of OPs are also known to exist. [29] While some OPs (such as dichlorvos, methamidophos, or the nerve agents sarin or soman) have a P = O bond and do not require any bioactivation, the majority of OPs used as insecticides are phosphorothioates (i.e., they have a P = S bond) and must be bioactivated in vivo to their oxygen analogues in order to exert their toxic action. An oxidative desulfuration, this bioactivation is facilitated by a number of different cytochrome P450 enzymes. There are other bioactivation processes, such as the creation of a sulfoxide (S = O) and a sulfone (O = S = O), which are both catalysed by CYPs

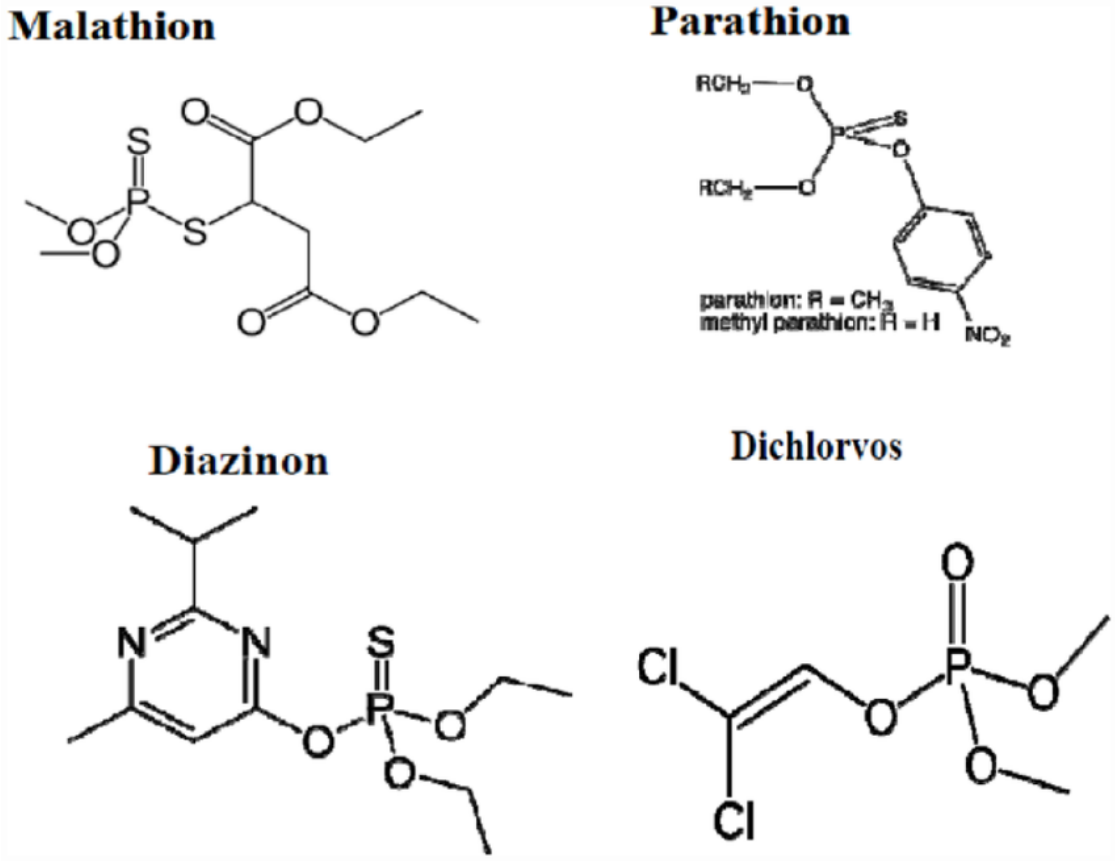
(e.g., disulfoton). The OPs are detoxified by all other biochemical reactions that are catalysed by CYPs or hydrolytic esterases (such as carboxylesterase and paraoxonase-1) and result in metabolites that are less toxic or nonexistent."[30]

TYPES OF ORGANOPHOSPHORUS COMPOUNDS

"Phosphoric acids and their derivatives are the source of organophosphorus compounds (OPCs), which are organic molecules with at least one carbon-phosphorus bond. Applications for pentavalent phosphorus-containing compounds are mostly found in industry and the environment. The toxicity of these phosphoric acid esters is mostly determined by the substituents that are joined to the phosphorus. 31 Thiols, amides, or esters of phosphonic, phosphinic, phosphoric, or thiophosphoric acids with two extra organic side chains of the phenoxy, cyanide, or thiocyanate group are known as organophosphorus insecticides. Certain OPCs are classified as phosphonothioates (S-substituted), and phosphonofluoridates include nerve poisons, also referred to as chemical warfare agents." [32] "There are four categories into which these nerve agents fall: (1) the German-developed G-series agents, which include cyclosarin (GF), soman (GD), sarin (GB), and tabun (GA). (2) V-series agents (V for venomous) include Chinese VX and Russian VX, as well as VE, VG, VM, and VX. (3) GV-series, such as GV, 2-dimethylaminoethyl-(dimethylamido)-fluorophosphate, which combine the characteristics of series G and V. In general, compounds in the G series are less harmful than those in the V series; (4) Novichok series of compounds, such as Novichok-5, Novichok-7, A230, A232, A234, and substance-33. The first individual to describe the creation of the first three compounds—substance-33, A230, and A232—at the GosNIIOKhT facility in Russia was Dr. Mirzayanov." These substances were agents that were unitary. Unitary A232 served as the basis structure for the synthesis of Novichok-5, the first binary agent, later in 1989. Novichok poisons are liquids, however

they can be made into dusty formulations by adsorbing liquid droplets onto carriers like talc, pumice, silica gel, or fuller's earth. A230, A232, and A234 were found to hydrolyse more slowly than agents from the G and V classes. In general, there is a great deal of disagreement on the structures of these compounds because of the secrecy surrounding their research; as a result, numerous structural variations have been hypothesised.[33]

Figure 1: Chemical structure of some OP Compounds



Stereogenic phosphorus atoms are found in the cyanide-releasing tabun, the fluoride-releasing volatiles soman and sarin, and the thiocholine-releasing VX. "With the exception of Soman, which has two chiral atoms—one a carbon centre and the other phosphorus—all of these OPCs have two enantiomers, P(−) and P(+). Soman has four enantiomeric forms: C (+)P(+), C (+)P(−), C (−)P(+), and C (−)P(−)." [34] Recent years have seen the compilation and careful evaluation of extensive structural data pertaining to the many types and isomers of OPC nerve agents. Stereoisomers are important when

considering the compound's range of toxicity. P(−) enantiomers are typically more hazardous.[35]

Pathophysiology

One neurotransmitter that is widely used in the neurological system is acetylcholine. "All postganglionic parasympathetic nerves, the postganglionic sympathetic nerve that innervates sweat glands, parasympathetic and sympathetic ganglia, and skeletal neuromuscular junctions contain acetylcholine. Acetylcholine is released into the synaptic cleft when an axon depolarises, activating postsynaptic receptors and causing an action potential to propagate. Acetylcholine is hydrolysed by carboxylic ester hydrolases to produce choline and acetic acid." Choline is reabsorbed into the presynaptic neurone to be used for the manufacture of more acetylcholine, and this process happens quickly. The primary enzymes in charge of this metabolism are butyrylcholinesterase (BuChE) and AChE. AChE is found on erythrocyte membranes and in skeletal and neurological tissues. Plasma and several organs, including the liver, heart, pancreas, and brain, contain BuChE. The role of BuChE is still not fully known, though. The ability of organophosphate insecticides to inhibit carboxyl ester hydrolases—with a primary focus on AChE inhibition—is their primary characteristic. By phosphorylating the enzyme's serine hydroxyl group, these pesticides render AChE inactive. Since AChE is necessary for the breakdown of acetylcholine, its inhibition causes acetylcholine to build up in the synapse, which in turn causes both nicotinic and muscarinic receptors to be overstimulated. "Myoclonic jerks and fasciculations can be caused by overstimulation of nicotinic receptors at the neuromuscular junction, which can ultimately result in depolarising blocks that cause flaccid paralysis." The adrenal glands also contain nicotinic receptors, which may be the cause of symptoms like perspiration, tachycardia, hypertension, and

left-shift leukocytosis. [36, 37] Because organophosphate poisoning acts on muscarinic receptors, it causes symptoms. Through a G-protein–coupled receptor mechanism, these effects usually manifest more slowly than nicotinic receptor actions. Both the parasympathetic and sympathetic nervous systems contain muscarinic receptors. Excessive diaphoresis is caused by the sympathetic nervous system overstimulating the sweat glands. Organophosphate poisoning can have parasympathetic effects on the heart, exocrine glands, and smooth muscles, among other systems. Breathing problems like bradycardia, bronchorrhea, and bronchospasm can result from muscarinic overstimulation, which can create serious, sometimes fatal diseases. [38]

CNS depression brought on by too much acetylcholine in the brain can result in convulsions and coma. The presence of alcohol and co-formulants is also a problem in circumstances where patients consume agricultural chemicals. Instead of being in a pure organophosphate form, pesticides are often mixed with solvents and surfactants to create an emulsifiable concentration. The degree of toxicity linked to co-formulants is still unknown. The potential of aspirating these solvents is a serious concern because organophosphate intoxication can cause coma and CNS depression. Organophosphate toxicity has been linked to reports of adult respiratory distress syndrome (ARDS) and aspiration pneumonitis. But it's still unclear if the chemical or its ambition is to blame for these illnesses.[39]

Toxicokinetics

Absorption: "The length of time the substance is in contact with the skin, its lipophilicity, and the presence of solvents (like xylene) and emulsifiers in the formulation that can aid absorption all affect how much absorption occurs." The faster and more thorough the skin absorbs a powder, the finer the powder. Additional crucial elements include the pesticide's volatility (for example, dichlorvos is far more volatile

than malathion), garment permeability, the degree of body surface coverage, and personal hygiene. The area of the skin that is impacted also affects the rate of absorption. For instance, the skin of the head and neck, axillae, and scrotum absorb parathion more easily than the skin of the hands and arms. It's likely that dermatitis or damaged skin permits OP chemicals to be absorbed more readily. Only 1.23% of the assessed potential dermal exposure was represented by the mean amount of liquid parathion absorbed dermally in one investigation.[40, 41]

Distribution and Storage: "After being absorbed, OP chemicals quickly build up in the kidneys, liver, fat, and salivary glands. The prolonged intoxication and clinical relapse that have been observed in poisoning from these OP insecticides may be explained by the fact that phosphorothioates (P=S), such as diazonin, parathion, and bromophos, are more lipophilic than phosphates (P=O), such as dichlorvos, and are thus stored extensively in fat. Since OP chemicals are often lipophilic, they typically pass through the blood-brain barrier."[40]

Biotransformation: "While phosphorothioates (P=S) require bioactivation to their phosphate counterparts (oxon) in order to become biologically active, phosphoates (P=O) are physiologically active as inhibitors of acetylcholinesterase (AChE)." Therefore, unless airborne oxidation has previously taken place to produce residues of oxon, the symptoms of poisoning following exposure to phosphorothioates (P=S) are delayed. Other than phosphates (P=O), OP compounds undergo metabolic activation to their corresponding oxon by N-oxidation, S-oxidation, flavin-containing monooxygenase enzymes, and oxidation desulfuration mediated by P450 isoforms. A-esterases, like paraoxonase, and hydrolases, such carboxylases, can deactivate the oxons that block AChE.[40]

Elimination: "Metabolites are generally eliminated in urine, with smaller amounts found in faeces and expired air. Certain OPs, such dichlorvos, which is not significantly stored in fat, can be removed in a matter of hours, whereas the inhibitory oxon of dementon-S-methyl or chlorpyrifos might linger for days due to their extensive fat storage."[40]

BIOCHEMICAL CHANGES:

Organophosphorus chemicals affect the body in a variety of toxicological ways:

Respiratory disorders: All animals experienced a central respiratory failure as a result of OPs. The main conclusions were that bradypnea progressed quickly and that the respiratory action was lost, resulting in apnoea. It has been discovered that poisoning causes a loss of central inspiratory drive. Other investigations that showed intact diaphragmatic after OP poisoning further suggest the lack of paralysis of breathing muscles.[42]

Hepatological disorders: The liver is the organ where OP chemicals are activated and detoxified. However, the kidneys are mostly responsible for their removal. After rats were intoxicated with OP, it was previously discovered that the profile of liver marker enzymes, antioxidant enzymes, and vital trace elements was negatively impacted. Congestion, glomerular necrosis, fatty alterations, alcoholic hepatitis, and sinusoidal dilatation are the histological alterations seen in the human liver in a forensic lab. Rats showed severe liver damage when given high doses of OP.[43]

Cardiovascular disorders: According to Povoa et al., OPs caused acute poisoning that resulted in myocardial necrosis. Following OP poisoning, creatinine kinase and lactate dehydrogenase levels will rise, according to Saadeh et al. [44] Heart-related Symptoms: impaired heart rate and force contraction, hypertension, hypotension, sinus bradycardia, and sinus tachycardia. ECG alterations: Low amplitude

T waves, extrasystole, elevated ST segment, prolonged QTc interval, and prolonged PR interval.[45]

Neurological disorders: In experimental rats given high acute doses of OP chemicals, neuronal necrosis has been seen in several cortical and subcortical areas. Additionally, OP causes a delay in the classification of stimuli, which is dependent on the brain's working memory system and attentional resources. This impairment seems to last for up to six months after poisoning. In individual cases or in worker cohorts, a number of chronic CNS problems brought on by acute or chronic OP agent poisoning have been documented. A cerebellar syndrome, psychiatric or more subtle cognitive dysfunction, changes in effect, libido, and memory, as well as parkinsonian and pseudobulbar symptoms, are among the many different types of syndromes.[46]

Hormonal imbalance: "Numerous experimental and epidemiological studies on hormonal imbalance, particularly sex hormones, and its effects on pesticide exposure-related developmental outcomes, such as foetal death, intrauterine growth restriction, congenital malformations, and male/female fertility, were published in the late 20th century. One risk factor for fertility is being in rural areas where a lot of pesticides are used."[47]

Oesophageal effects: Circumferential heat, oedema, and esophageal haemorrhage were discovered during an emergency esophageo-gastroscopy.[48]

Renal impairment: "Numerous research that the Ontario College analysed demonstrate a strong correlation between exposure to pesticides and solid tumours, including kidney cancer. Paternal pesticide exposure through agriculture has been linked to an increased risk of kidney cancer. Children are continuously exposed to low levels of pesticides in their food and surroundings. Additionally, prolonged pesticide exposure has been linked to kidney failure."[49]

Oxidative stress and Antioxidant status: According to studies, OP poisoning is linked to increased oxidative stress, decreased glutathione levels, improved lipid peroxidation, and raised antioxidant status.[50]

History and Physical

The precise substance involved and the period of exposure are crucial components of the patient's medical history when handling possible poisoning instances, particularly when purposeful consumption is involved. “Since the toxicity of various chemicals can vary greatly, an effort should be made to secure the pesticide container, if possible, in order to give this information to the Poison Control Centre or a medical toxicologist.” The degree of toxicity, the specific organophosphate substance involved, the exposure route, and the dosage all affect when symptoms appear. Furthermore, the compound's toxicokinetics, notably its lipophilicity, affect how long toxicity lasts. As the substance is released from fat reserves, cholinergic effects may occasionally reappear. [51] Diaphoresis, muscle fasciculations, pinpoint pupils, and unresponsiveness are characteristic symptoms of severe organophosphate exposure. Urinary incontinence, lacrimation, diarrhoea, emesis, and excessive salivation are possible further symptoms. The smell of garlic or solvent may linger when organophosphates are purposefully self-poisoned. “There are a number of useful mnemonics for remembering the symptoms of organophosphate poisoning and the receptor that causes them.”

“The frequently used mnemonic that encompasses the muscarinic effects of organophosphate poisoning is DUMBELS, as mentioned below.

- D = Defecation/diaphoresis
- U = Urination
- M = Miosis

- B = Bronchospasm/bronchorrhea
- E = Emesis
- L = Lacrimation
- S = Salivation"

"Anxiety, disorientation, fatigue, emotional instability, seizures, hallucinations, migraines, insomnia, memory loss, and circulatory or respiratory depression are some other acute symptoms." The most common cause of mortality in fatal instances is respiratory failure brought on by central respiratory depression, bronchoconstriction, bronchorrhea, and respiratory muscle weakness or paralysis. It is possible for patients who survive acute poisoning to develop further long-term problems.

EVALUATION[52, 43]

Since clinical assessment is the primary method of diagnosing organophosphate poisoning, treatment must begin prior to laboratory confirmation. It is essential to have a strong clinical suspicion of organophosphate poisoning, particularly in cases where exposure or ingestion is unknown. Patients with respiratory distress, diaphoresis, and miotic pupils are the most common presentations of poisoning. Certain organophosphates have a characteristic smell, like petroleum or garlic, which might help with diagnosis.

An atropine trial may be used if organophosphate poisoning is suspected but not confirmed. Suspicion of AChE inhibitor poisoning is raised if symptoms improve after taking 0.6–1 mg of atropine. Interpreting the sensitivity and specificity of this experiment, however, might be difficult because of the paucity of data, especially in situations of severe poisoning. Therefore, more research is required to solve this problem. A tiny dose of atropine may not cause any reaction in patients with severe poisoning, which could lead to a false-negative test.

Even though certain labs are capable of measuring cholinesterase activity directly, these tests are frequently contracted out to establishments that might not deliver data quickly enough to inform treatment. Red blood cell AChE (RBC AChE) and BuChE are the two cholinesterase enzymes that are frequently tested. Compared to RBC AChE activity, BuChE activity is less selective. Iron deficiency anaemia, chronic sickness, liver disease, malnutrition, and genetic enzyme failure can all be associated with low BuChE activity. Interpreting this test is made more difficult by the fact that the degree of enzyme inhibition varies according on the particular organophosphate that caused the poisoning and that there is little information available for many of these compounds.

The clinical manifestations of organophosphate toxicity are thought to be more strongly correlated with RBC AChE activity. Although this threshold can change depending on the chemical, symptoms usually appear in clinical settings when more than 50% of this enzyme is blocked. Notably, fluoride can deactivate the enzymes, potentially producing erroneously low activity levels, hence it is crucial to collect blood samples in the proper tubes.

A variety of necessary laboratory tests, such as particular diagnostic tests for organophosphate poisoning and additional tests to evaluate the patient's general health, may be ordered by healthcare professionals. “A complete blood cell count (CBC), a basic metabolic panel test, tests for kidney and liver function, blood glucose levels, arterial blood gas analysis, and pregnancy testing are a few examples of these. Because of parasympathetic activity, sinus bradycardia is usually shown on the electrocardiogram (ECG).”

Assessment of Severity of OP Poisoning

The severity of organophosphate (OP) poisoning can be assessed using a variety of methods, including:[54]

- **Peradeniya Organophosphorus Poisoning (POP) scale**: a clinical measure that evaluates the degree of consciousness, heart rate, pupil size, and six other typical clinical signs of OP poisoning. Every aspect has a score between 0 and 2, where mild poisoning is represented by a score of 0–3, moderate poisoning by a score of 4–7, and severe poisoning by a score of 8–11.
- **Red blood cell (RBC) cholinesterase level**: A measure of cholinesterase levels in the patient's red blood cells.
- **Pseudocholinesterase (PChE)**: A prognosticator of OP poisoning, with lower levels indicating more severe poisoning.
- **Glasgow coma scale (GCS) score**: A factor that can help assess the severity of OP poisoning.
- **Acute Physiology and Chronic Health Evaluation (APACHE) II score**: A factor that can help assess the severity of OP poisoning.
- **Creatine phosphokinase**: A factor that can help assess the severity of OP poisoning.
- **Leukocyte count**: A marker for the severity of OP poisoning.

Cholinesterase assays[55]

It is optimal to use an assay to evaluate butyrylcholinesterase activity in plasma (or acetylcholinesterase in whole blood) in order to confirm the diagnosis of organophosphorus poisoning. Nevertheless, these assays' data are rarely accessible in time to influence clinical judgement. They are crucial for directing clinical research,

and interpreting studies that focus on particular chemicals and treatments requires an awareness of their limits.

Regretfully, there is a lot of misunderstanding regarding the application and interpretation of these tests. Certain pesticides are more effective in inhibiting butyrylcholinesterase than acetylcholinesterase. Although butyryl cholinesterase activity is not correlated with the degree of poisoning, it can be used to measure the body's removal of organophosphorus and as a sensitive indicator of exposure to the majority of organophosphorus chemicals or other cholinesterase-inhibiting substances.

Research indicates that red-cell acetylcholinesterase is an excellent indicator of atropine requirements and synaptic function in organophosphorus poisoning patients, making it likely a good indicator of severity. Atropine was not required in patients whose red-cell acetylcholinesterase activity was at least 30% because their muscles functioned normally. Patients who had red-cell acetylcholinesterase activity below 10%, on the other hand, required large doses of atropine due to severely disrupted muscular function. Between these ranges, acetylcholinesterase activity was linked to a moderate reduction in muscular function and an atropine requirement.

One of the main problems with acetylcholinesterase assays is that if the sample is kept at room temperature for even a short period of time, the interaction between oximes, acetylcholinesterase, and organophosphorus persists. As soon as the sample is removed from the patient, it must be cooled and diluted to stop the reaction and produce accurate results. If not, variations of just a few minutes in cooling time will result in significant variation across multiple samples, making interpretation challenging.

Drawbacks of cholinesterase activity assays

Plasma butyrylcholinesterase assays

- Butyrylcholinesterase inhibition, also known as plasma cholinesterase or pseudocholinesterase inhibition, does not reveal the clinical severity of the poisoning. Butyrylcholinesterase is more strongly inhibited by several organophosphorus pesticides than acetylcholinesterase is; in fact, butyrylcholinesterase inhibition may be more pronounced than acetylcholinesterase inhibition. Assays for butyrylcholinesterase can be used to identify pesticide exposure to carbamate or organophosphorus.
- The liver produces butyrylcholinesterase, and after the organophosphorus is removed, blood concentrations return to normal by roughly 7% every day. Since this recovery indicates that the organophosphorus has been removed, daily butyrylcholinesterase assays can be used to track when enzyme activity begins to increase once more.
- Comparisons between studies may be challenging due to variations in commercial tests. Each assay has a different butyrylthiocholine concentration. A substrate with a high concentration (e.g., 7 mM vs. 1 mM) will provide a greater background and 30% more observed activity.
- To assess non-enzymatic hydrolysis and, thus, background values, butyrylthiocholine hydrolysis must be measured without plasma. Such a control is not offered by every commercial assay. The concentration and pH of butyrylthiocholine hydrolysis, which differ amongst test kits, have an impact on the background quantity of this process.
- Temperature regulation is crucial because butyrylcholinesterase activity rises by around 4% for every degree Celsius that the temperature rises.

Red cell acetylcholinesterase assays

- Acetylcholinesterase expressed on the surface of red blood cells is measured by these techniques. One reliable indicator of this inhibition in synapses and the degree of poisoning is red-cell acetylcholinesterase inhibition. Whole blood that has had butyrylcholinesterase activity inhibited is used to assay this enzyme. Human plasma and serum contain very small amounts of acetylcholinesterase.
- Only erythropoiesis can restore red-cell acetylcholinesterase after it has aged. Therefore, regeneration at a rate of less than 1% per day is significantly slower than that of butyrylcholinesterase. Red-cell acetylcholinesterase may be a less reliable indicator of synaptic acetylcholinesterase activity while recovery is place because it is unknown how quickly spontaneous neuronal acetylcholinesterase recovers.
- If a blood sample is allowed to sit at room temperature after being drawn, reactions involving acetylcholinesterase, organophosphorus, and oximes will proceed. As a result, the measured acetylcholinesterase activity will not accurately reflect the blood's activity at the moment of collection; assays will vary if samples are left for varying periods of time. To halt the reactions, blood samples must be diluted and refrigerated right away after collection. At the bedside, we usually dilute by a factor of 20 by combining 200 µL of newly obtained blood in an EDTA tube with 4 mL of cold saline (at 4˚C). The sample is then placed in a freezer at -20˚C for five minutes.
- To reactivate any acetylcholinesterase that has not matured, an aliquot of blood should be incubated with a large amount of oxime (for example, 100 µmol/L obidoxime) for 15 minutes prior to the assay. An assay of this kind

might be used to determine whether a patient might benefit from higher dosages or from ongoing oxime therapy.

- Acetylcholinesterase tests are sensitive to pH and oxime and substrate concentrations. The background signal in the test will be reduced by assays with low substrate concentrations, pH 7•4, and therapeutic oxime concentrations; however, a blank sample devoid of plasma is required to assess the background signal.
- Ellman's reagent reacts with matrix sulfhydryl molecules found in red blood cells, primarily haemoglobin. Red-cell samples should be preincubated with the reagent during temperature equilibration in order to finish this reaction. Failure to complete this step will result in a higher background activity being recorded. It is possible to confirm significant exposure to anticholinesterase drugs by tracking a patient's cholinesterase state following organophosphate poisoning. In the future, these tests might help determine when to cease oxime medication and enable a patient to be carefully weaned off of a ventilator when butyrylcholinesterase activity is rising.

SERUM POTASSIUM AND OP POISONING

On a biological level, potassium is essential. Its primary tasks are related to nerve transmission, glomerulo-tubular renal function, and the contraction of the heart and skeletal muscles. Tripathy et al. have already shown that hypokalaemia is one of the most prevalent electrolyte abnormalities linked to organophosphorus poisoning.[56]

Pathophysiology of Hypokalemia[57]

The pathophysiology of hypokalemia in organophosphate poisoning is complex and involves multiple mechanisms. Direct Effects of Acetylcholine Excess:

- OP poisoning inhibits acetylcholinesterase, leading to acetylcholine accumulation
- Excessive acetylcholine stimulates muscarinic receptors
- This activation leads to increased cellular potassium loss through:
    - Enhanced cellular membrane permeability
    - Stimulation of Na^+/K^+-ATPase pump activity

2. Catecholamine Surge:

- OP poisoning triggers massive sympathetic activation
- Increased catecholamines (especially epinephrine) cause:
    - Activation of β2-adrenergic receptors
    - Intracellular potassium shift through enhanced Na^+/K^+-ATPase activity
    - Increased cellular uptake of potassium

3. Metabolic Effects:

- Respiratory distress and hypoxia lead to:
    - Development of metabolic acidosis
    - Compensatory respiratory alkalosis
- These pH changes affect potassium distribution:

- Initially, acidosis may cause hyperkalemia
- As compensation occurs, alkalosis promotes K^+ shift into cells

4. Gastrointestinal Losses:

- Muscarinic overstimulation causes:
  - Excessive secretion of digestive fluids
  - Severe diarrhea and vomiting
  - Direct loss of potassium through GI tract

5. Stress Response:

- Acute stress response activates:
  - Insulin release
  - Further catecholamine surge
- Both mechanisms promote cellular potassium uptake

6. Renal Effects:

- Cholinergic stimulation affects kidney function:
  - Increased renal blood flow
  - Enhanced potassium excretion
  - Altered tubular handling of electrolytes

Prognostic significance of serum potassium levels in OP poisoning:[58]

1. Admission Potassium Levels and Outcomes:

- Severe hypokalemia ($K^+ < 3.0$ mEq/L) on admission correlates with:
  - Higher mortality rates
  - Increased need for mechanical ventilation
  - Longer ICU stays
  - Greater likelihood of intermediate syndrome

2. Monitoring Disease Progression:

- Serial potassium measurements help track:
    - Treatment response
    - Development of complications
    - Need for intensive care interventions
    - Risk of cardiac arrhythmias

3. Predictive Value for Complications:

- Persistent hypokalemia suggests:
    - Greater severity of poisoning
    - Higher risk of respiratory failure
    - Increased likelihood of neuromuscular dysfunction
    - Greater probability of cardiac complications

4. Treatment Response Indicator:

- Potassium normalization often indicates:
    - Successful anticholinergic therapy
    - Improving cholinergic crisis
    - Better overall prognosis
    - Reduced risk of complications

5. Risk Stratification:

- Helps categorize patients into risk groups:
    - Mild cases (normal or mildly decreased K^+)
    - Moderate cases (moderate hypokalemia)
    - Severe cases (marked hypokalemia)

REVIEW OF RELATED ARTICLES

Basnet D et al (2024)[59] The purpose of this study was to evaluate serum potassium as a substitute predictive measure for the severity of OP poisoning. According to the POP scale, they found a strong correlation between the severity of OP poisoning and serum potassium levels in patients who presented within 12 hours after the incident. In cases of moderate to severe OP poisoning, hypokalaemia is common, and lower serum potassium levels are associated with higher POP scores.

Amrutha I D et al (2023)[60] assessed the serum potassium levels in tertiary care centres as a predictive indicator for patients with acute organophosphorus poisoning. Hypokalaemia was seen in 63.3% of patients with op poisoning. Compared to normokalemic patients, hypokalaemia patients had convulsions, respiratory distress, and the requirement for intubation. They came to the conclusion that hypokalaemia considerably raises the morbidity and fatality rates associated with organophosphorus poisoning. Hypokalaemia is therefore a valid and economical indicator of organophosphorus poisoning mortality and morbidity. In cases of OPC poisoning, prompt hospitalisation and hypokalaemia treatment can save lives.

Sonaiya S et al (2022)[61] Fifty patients with OP poisoning participated in this prospective cohort study, which examined the relationship between serum K+, Na+, creatinine, and BUN and prognostic significance. High fatality rates in patients with suicidal organophosphate poisoning were shown to be substantially associated with elevated blood creatinine and hypokalaemia. Low blood K+ levels were shown to be statistically significant (p1.21 mg/dl) in conjunction with clinical outcomes, and they are poor prognostic indications for patients who arrive with suicidal OP poisoning. In order to effectively manage OP poisoning patients, they determined that hypokalaemia

and high blood creatinine levels were associated with poor clinical outcomes. They also suggested routine monitoring of these prognostic indicators.

Difoesa B et al (2021)[57] In order to evaluate serum potassium levels in patients suffering from acute organophosphorus poisoning and ascertain whether there is a correlation between serum potassium levels and the course of acute organophosphorus poisoning, this study was conducted. According to the Peradeniya OP poisoning scale, they found a substantial correlation between the severity of acute organophosphate poisoning and the serum potassium level on the day of admission. Poor outcomes were observed in the individuals with lower serum potassium levels upon admission. The requirement for ventilator assistance was also significantly correlated with lower serum potassium levels. As a result, serum potassium can be used to forecast how severe organophosphorus poisoning will be. In addition to lowering mortality and morbidity, this can aid in early patient triage.

Desai M et al (2021)[62] A total of 24 participants in this study had hypokalaemia. Twenty individuals (51.3%) out of 39 male patients had hypokalaemia. Four individuals (36.4%) out of 11 female patients developed hypokalaemia. Of the 50 patients, 14 (28%) developed hyponatraemia (less than 135 meq/dl). 42.9% of the hyponatraemia in these six patients was caused by hypokalaemia. Six cases of hyponatraemia make up 25% of the 24 patients with hypokalaemia. In organophosphorus chemical poisoning, hypokalaemia dramatically raises morbidity and mortality. In cases of organophosphorus chemical poisoning, hypokalaemia is a valid and affordable indicator of morbidity and mortality.

Karthik M R et al (2020)[63] In order to ascertain the effect of organophosphorus compound (OPC) in prognosis, calculated the serum electrolyte levels in patients who drank it. This study came to the conclusion that hypokalaemia has a major role in the

need for a ventilator and the outcome of OPC poisoning. Serum potassium levels should be regularly measured since they can be a reliable and affordable marker that can help with prognostication and outcome prediction in cases of OPC poisoning. For certain patients, aggressive hypokalaemia treatment may be a lifesaver.

Tripathy SK et al (2018)[56] The objective was to examine the clinical characteristics of OPC poisoning and establish a correlation between electrocardiogram (ECG) alterations and electrolyte imbalances. They came to the conclusion that significant morbidity and death in OPC poisoning are linked to QTc prolongation and hypokalaemia.

Prasad DRMM et al (2014)[64] The purpose of this study was to assess the predictive value of hypokalaemia in relation to plasma cholinesterase (PChE) levels for acute OP poisoning morbidity and mortality. They came to the conclusion that serum [K+] and PChE levels are much lower in cases of severe clinical symptoms of OP poisoning. These biochemical results can therefore be suggested as prognostic markers for OP poisoning. Medical toxicologists and clinicians should view hypokalaemia linked to a lower PChE level as concerning indicators of a bad prognosis in patients who have been poisoned by OP.

MATERIAL AND METHODS

- **Study design:** Cross-sectional study
- **Study area:** Patients admitted in the medicine ICU/WARDS OF BLDE(DU) Shri BM Patil medical college and research Centre, Vijayapura.
- **Study period:** Research study was conducted from May 2023 to December 2024. Below is the work plan.

Table 1: Work plan of the study with percentage of allocation of study time and duration in months

Work plan	**% of allocation of study time**	**Duration in months**
Understanding the problem, preparation of questionnaire.	5-10%	May 2023
Pilot study, Validation of questionnaire, data collection and manipulation	Upto 80%	June 2023 to May 2024
Analysis and interpretation	5-10%	June 2024 to August 2024
Dissertation write-up and submission	5-10%	September 2024 to December 2024

- **Sample size**: Considering the confidence limit of these studies to be 97% with 3% level of significance and margin of error 0.05.The sample size computed using the following formula:

  Sample size (n) = $(Z^2*p*(1-p))/d^2$

  Where,

  z is the z score=2.17

  d is the margin of error= 0.05

n is the population size

p is the population proportion=0.04

α is the level of significance=0.03

The estimated sample size of this study is **72**

- **Inclusion criteria:**

1. Patients whose age >18years
2. Individuals with a known history of consumption or exposure to OPC poison and typical clinical symptoms of organophosphorous compound poisoning

- **Exclusion criteria:**

1. Patients who has history of serious systemic illness, malnutrition, chronic infections
2. Patients who consume alcohol while taking poison

METHODOLOGY:

Study Design and Setting:

This Cross-sectional study was conducted in the Medicine ICU and wards of BLDE (DU) Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapura. The study focused on patients presenting with acute organophosphate poisoning through both emergency department admissions and direct ICU transfers.

Patient Selection and Recruitment

Patients were screened upon admission to the casualty medicine department or ICU for eligibility based on predetermined inclusion and exclusion criteria. Written informed consent was obtained from either the patients or their legal representatives before enrollment in the study. The diagnosis of organophosphate poisoning was established based on history of exposure, characteristic clinical features, and confirmation through poison detection center reports.

Data Collection:

A comprehensive assessment of each patient was conducted using a standardized proforma. This included detailed documentation of demographic information, circumstances of poisoning, time elapsed since exposure, quantity and type of organophosphate compound (if known), pre-hospital treatment received, and mode of poisoning. A thorough clinical examination was performed, with particular attention to vital parameters, level of consciousness, pupillary signs, respiratory status, and presence of cholinergic symptoms.

Laboratory Investigations:

Blood samples were collected from all participants at the time of admission, prior to the initiation of specific treatment. The primary parameters of interest - serum potassium and plasma cholinesterase levels - were measured using standardized laboratory techniques. Serum potassium was analysed using ion-selective electrode method, while plasma cholinesterase activity was determined using spectrophotometric analysis.

Additional laboratory investigations included complete blood count with ESR, comprehensive metabolic panel including liver function tests (LFT) and renal function tests (RFT), random blood sugar estimation, and complete urinalysis. All laboratory tests were performed in the hospital's central laboratory following standard operating procedures and quality control measures.

Diagnostic Imaging and Other Investigations:

Chest radiographs were obtained for all patients to assess for pulmonary complications. Twelve-lead electrocardiograms were recorded to evaluate for cardiac manifestations of organophosphate toxicity and electrolyte disturbances. Arterial blood gas analysis was performed to assess acid-base status and oxygenation.

Severity Assessment:

Patients were classified into two groups - severe and non-severe cases - based on specific clinical manifestations at the time of presentation. The severe category included patients presenting with any of the following: convulsions, significant muscle weakness/fasciculations, or respiratory distress requiring ventilatory support. Mortality outcomes were also recorded for each patient during the course of hospitalization. This classification system allowed for comparative analysis of laboratory parameters, particularly serum potassium and plasma cholinesterase levels, between the severe and non-severe groups.

The presence of these specific manifestations was chosen as classification criteria because they represent significant cholinergic toxicity and are associated with poorer outcomes in organophosphate poisoning. This classification helped in evaluating whether serum potassium levels could serve as an early predictive marker for severity and mortality in acute organophosphate poisoning cases.

All patients were followed throughout their hospital stay to document the development of complications, need for intensive care management, ventilatory support duration (if required), and final outcome (survival or mortality). This prospective tracking allowed for comprehensive assessment of the prognostic value of initial serum potassium levels in relation to clinical course and outcome.

Documentation and Monitoring:

Patients were monitored throughout their hospital stay with regular assessment of clinical parameters and repeated laboratory investigations as clinically indicated. All therapeutic interventions, including administration of atropine and oximes, were documented in detail. Complications, clinical course, and outcomes were recorded systematically.

Quality Control Measures:

Laboratory quality control procedures were strictly followed for all biochemical analyses. Regular calibration of equipment and standardization of measurement techniques were ensured. The poison detection centre reports were obtained following standardized protocols for toxicological analysis.

Safety Considerations:

All investigations performed were part of routine clinical care for organophosphate poisoning patients. No experimental procedures or interventions were conducted. Universal precautions were observed during sample collection and handling. Patient confidentiality was maintained throughout the study period.

Data Management:

All collected data was systematically recorded in individual case record forms. Regular auditing of data collection was performed to ensure completeness and accuracy. The data was subsequently transferred to a secure electronic database for analysis, with appropriate backup measures in place.

STATISTICAL ANALYSIS

Data was entered in excel sheet and analyzed using SPSS version 21. Results were presented in tabular and graphical forms Mean, median, standard deviation and ranges were calculated for quantitative data. Qualitative data were expressed in terms of frequency and percentages. Student t test (Two Tailed) was used to test the significance of mean and P value <0.05 was considered significant.

RESULTS

The present study was conducted in the department of General medicine at Shri B.M.Patil medical college, Hospital and research centre, Vijayapura from May2023toDecember 2024 to correlate serum potassium and plasma cholinesterase in assessing the severity of acute organophosphate poisoning. Total of 83 patients were included in the study.

Following were the results of the study:

Table 2: Distribution of patients according to age

Age (in years)	Frequency	Percentage
<20	18	21.7%
20-40	56	67.5%
41-60	3	3.6%
61-80	6	7.2%
Total	**83**	**100%**

Table 1 and graph1 shows the age distribution of patients with organophosphate poisoning, with the majority (67.5%) falling in the 20-40 years age group, followed by those below 20 years (21.7%), while fewer patients were in the older age groups of 61-80 years (7.2%) and 41-60 years (3.6%).

Figure 2: Distribution of patients according to age

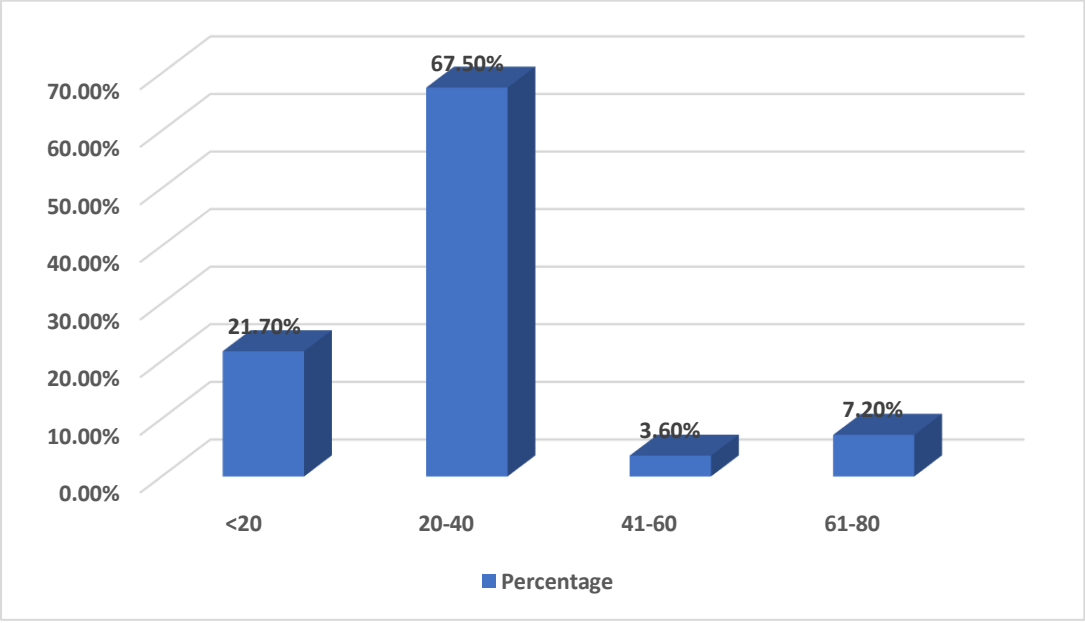


Table 3: Distribution of patients according to gender

Gender	Frequency	Percentage
Female	40	48.2%
Male	43	51.8%
Total	**83**	**100%**

Table 2 and graph 2 demonstrates a relatively balanced gender distribution among the 83 patients studied, with males slightly predominating at 51.8% compared to females at 48.2%.

Figure 3: Distribution of patients according to gender

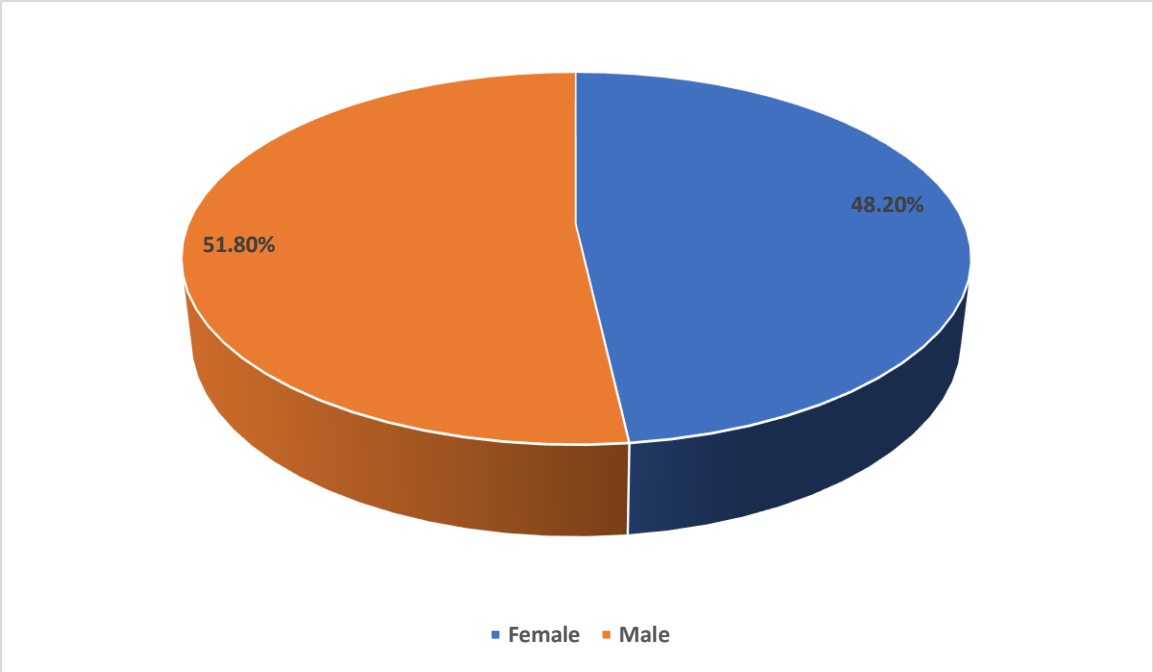


Table 4: Distribution of patients according to clinical features

Clinical features	Frequency	Percentage
Vomiting	62	74.7%
Abdominal pain	8	9.6%
Diarrhoea	18	21.7%
Salivation	16	19.3%
Lacrimation	1	1.2%
Bronchospasm	25	30.1%
Muscle weakness	32	62.6%
Convulsions	5	6%

Table 3 and graph 3 reveals that vomiting was the most common clinical feature observed in 74.7% of patients, followed by muscle weakness (62.6%) and bronchospasm (30.1%), while less common symptoms included diarrhea (21.7%), salivation (19.3%), abdominal pain (9.6%), convulsions (6%), and lacrimation (1.2%).

Figure 4: Distribution of patients according to clinical features

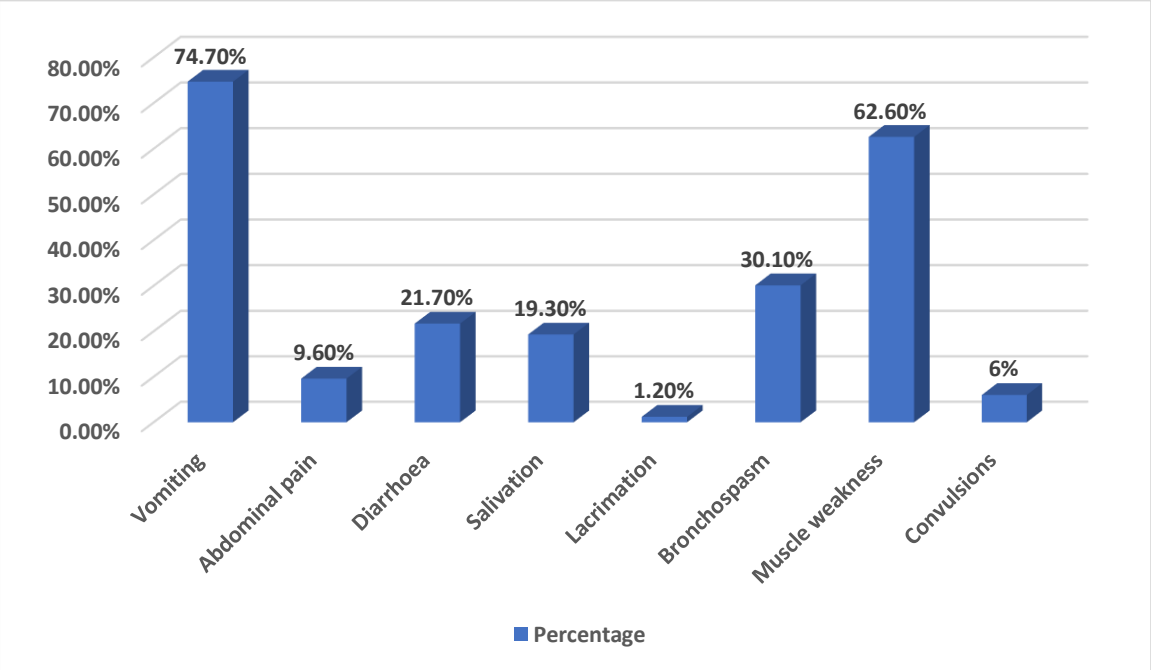


Table 5: Distribution of patients according to GCS

GCS	Frequency	Percentage
3-8	17	20.5%
9-12	11	13.3%
13-15	55	66.3%
Total	**83**	**100%**

Table 4 and graph 4 indicates that the majority of patients (66.3%) maintained a good level of consciousness with Glasgow Coma Scale (GCS) scores of 13-15, while 13.3% had moderate impairment (GCS 9-12), and 20.5% presented with severe impairment (GCS 3-8).

Figure 5: Distribution of patients according to GCS

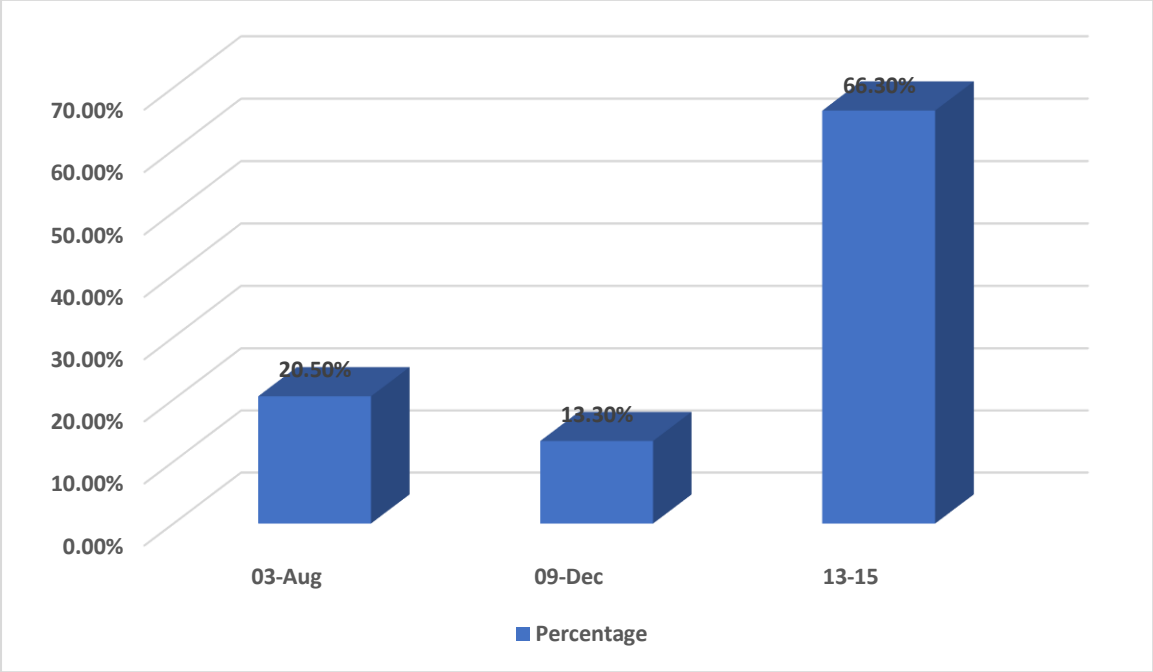


Table 6: Distribution of patients according to requirement of mechanical ventilation

mechanical ventilation	Frequency	Percentage
Required	22	26.5%
Not required	61	73.5%
Total	**83**	**100%**

Table 5 and graph 5 shows that mechanical ventilation was required in approximately one-quarter (26.5%) of the patients, while the majority (73.5%) did not require this intervention.

Figure 6: Distribution of patients according to requirement of mechanical ventilation

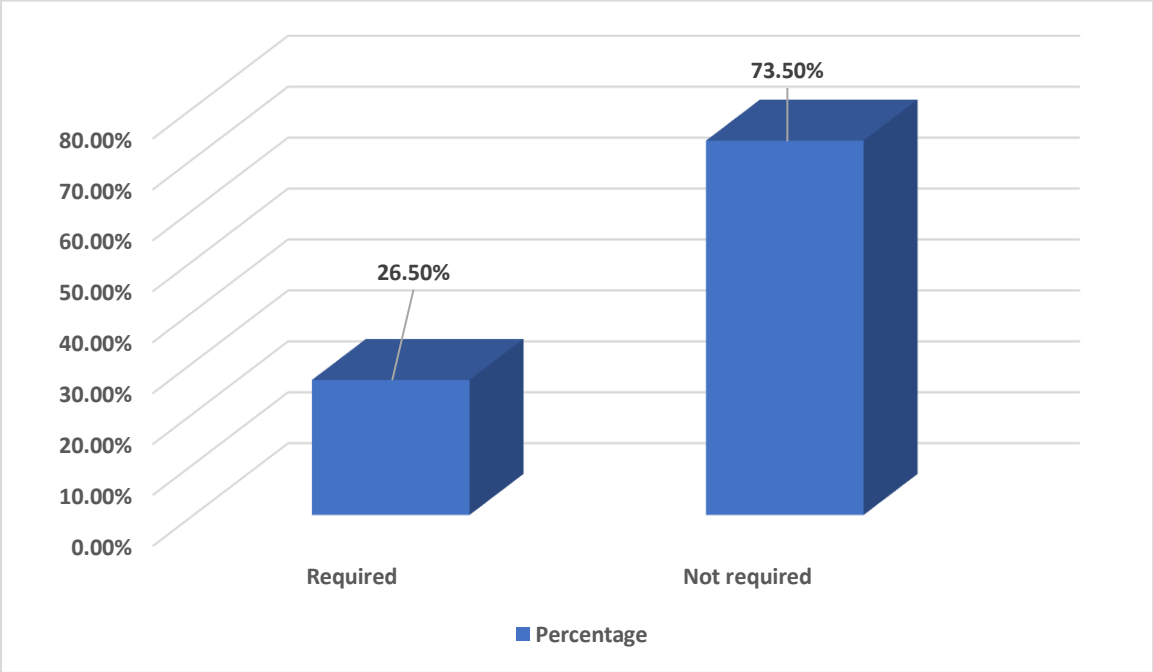


Table 7: Distribution of patients according to investigations

Investigations (mean±SD)	Initial	24 hours
Serum potassium (mEq/L)	3.8±0.63	3.82±0.48
Plasma ChE	2712.2±2785.7	2803.4±2864.9

Table 6 and graph 6 presents the mean values of key investigations, showing initial serum potassium levels of 3.8±0.63 mEq/L with minimal change after 24 hours (3.82±0.48 mEq/L), and plasma cholinesterase (ChE) levels of 2712.2±2785.7 initially with a slight increase to 2803.4±2864.9 after 24 hours.

Figure 7: Distribution of patients according to investigations

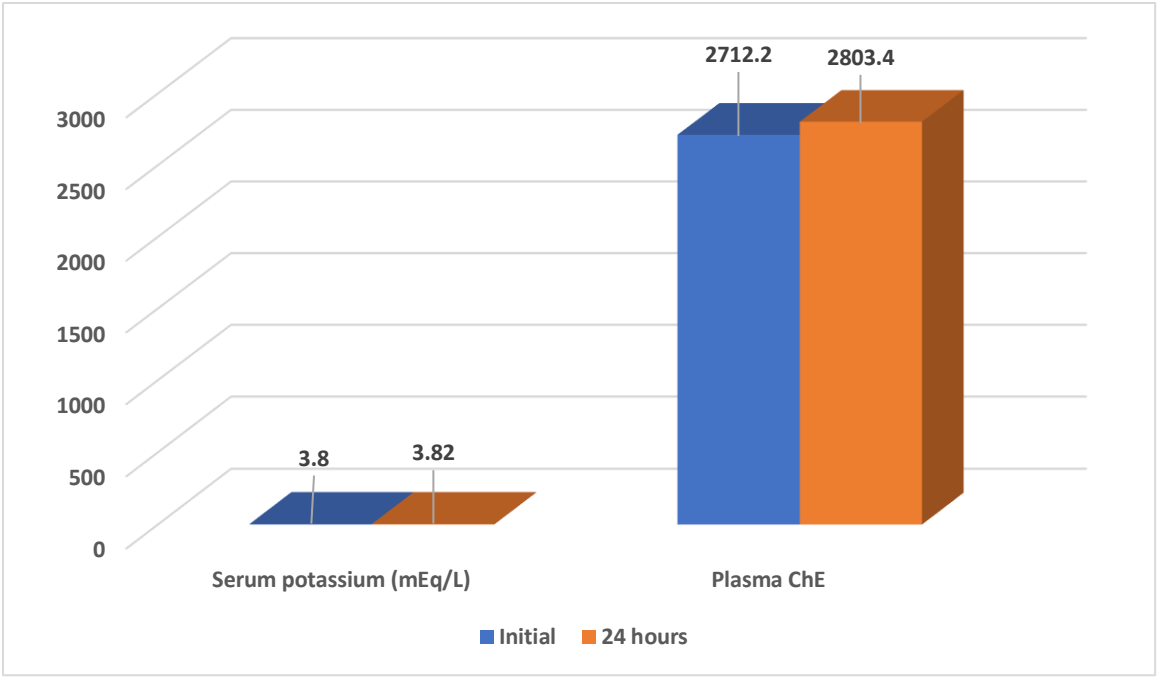


Table 8: Distribution of patients according to vital signs

Vitals	RR	HR
Mean±SD	24.16±6.1	99.3±21.1

Table 7 and graph 7 provides information on vital signs, with patients having a mean respiratory rate of 24.16±6.1 breaths per minute and a mean heart rate of 99.3±21.1 beats per minute.

Figure 8: Distribution of patients according to vital signs

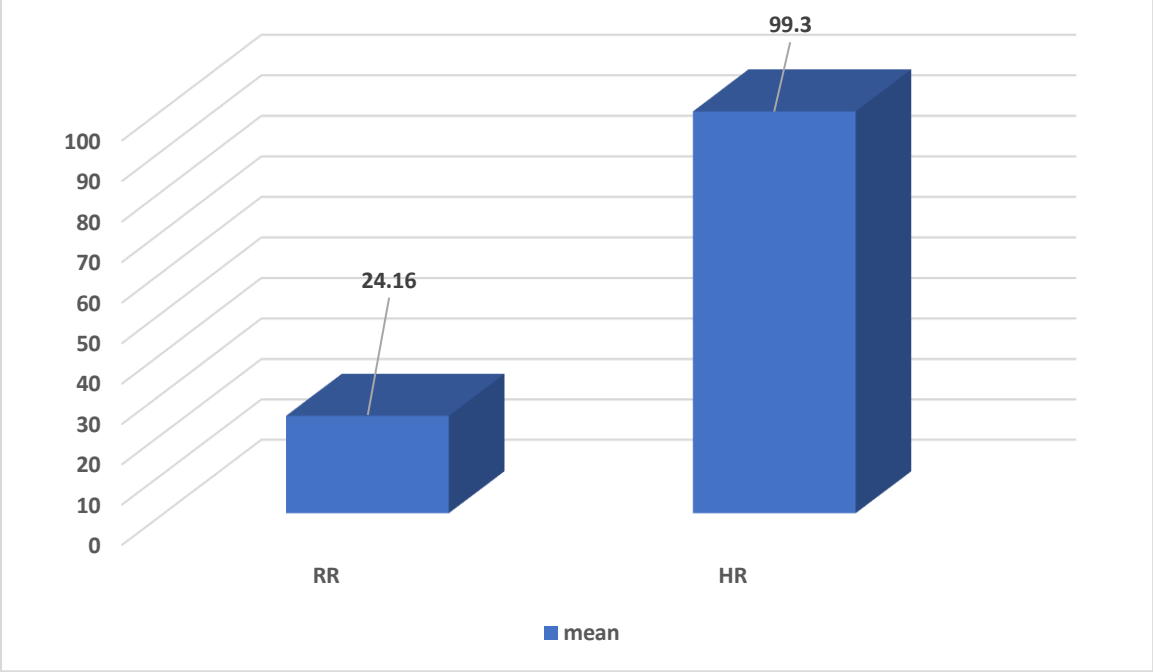


Table 9: Distribution of patients according to pupil size

Pupil size	
Mean±SD	2.04±0.83

Table 8 and graph 8 shows that the mean pupil size among all patients was 2.04±0.83 mm, indicating the miotic effect typical of organophosphate poisoning.

Figure 9: Distribution of patients according to pupil size

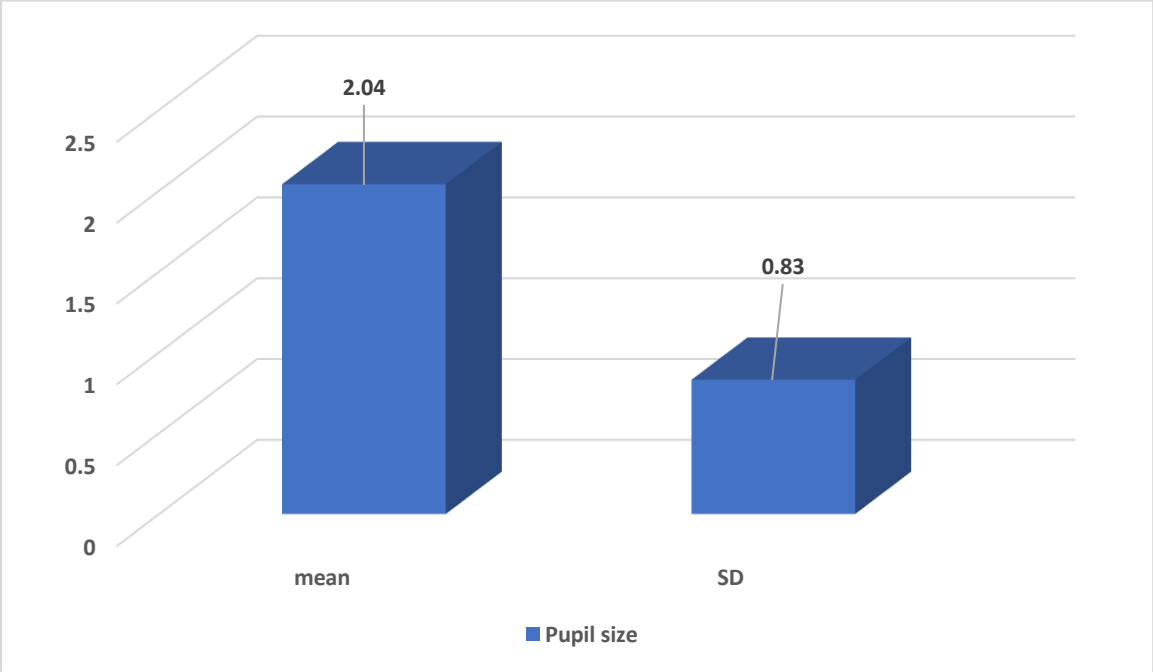


Table 10: Distribution of patients according to severity of OP poisoning

Severity of OP poisoning	Frequency	Percentage
Severe	31	37.3%
Not severe	52	62.6%
Total	**83**	**100%**

Table 9 and graph 9 classifies patients according to poisoning severity, with 37.3% categorized as having severe organophosphate poisoning while 62.6% were classified as not severe.

Figure 10: Distribution of patients according to severity of OP poisoning

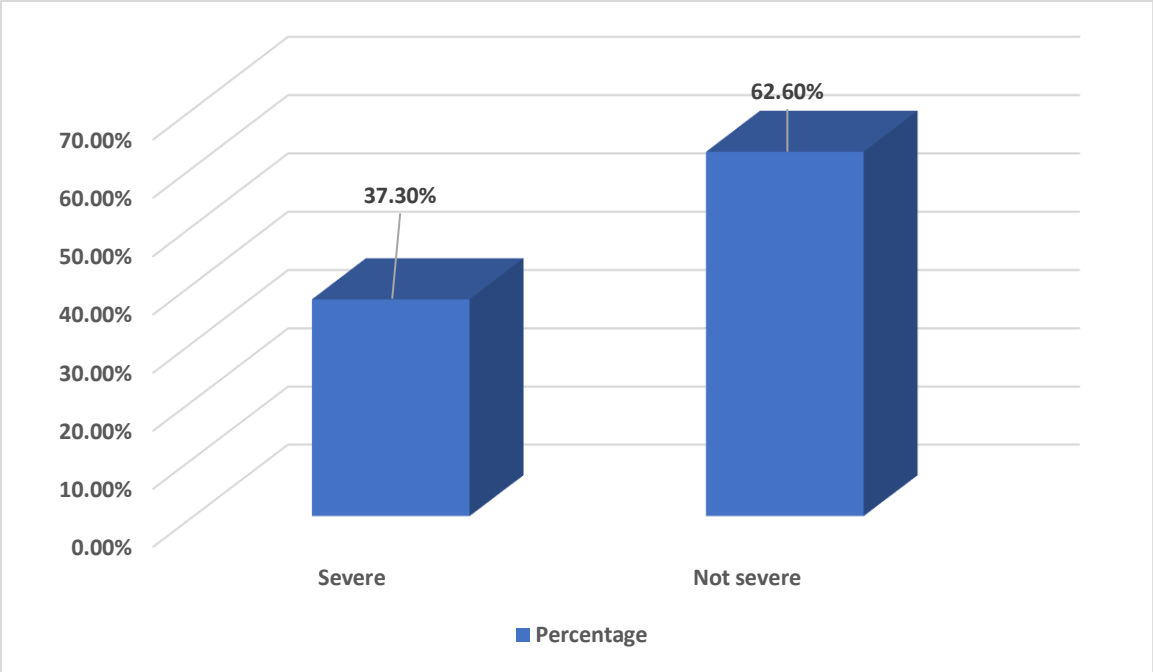


Table 11: Association of severity of OP poisoning with investigations

Investigations (mean±SD)		Severity of OP poisoning		p-value
		Not Severe	Severe	
Serum potassium	**Initial**	4.08±0.47	3.32±0.59	**<0.001**
	24 hours	4±0.34	3.6±0.55	**0.001**
Plasma ChE	**Initial**	3455.4±2785.4	1465.4±2336.2	**0.001**
	24 hours	3550.9±2794.6	1633.5±2620.9	**0.01**

Table 10 and graph 10 demonstrates significant associations between severity of poisoning and biochemical parameters, with severely poisoned patients showing significantly lower initial serum potassium (3.32±0.59 vs 4.08±0.47 mEq/L, p<0.001), lower 24-hour potassium (3.6±0.55 vs 4±0.34 mEq/L, p=0.001), lower initial plasma cholinesterase (1465.4±2336.2 vs 3455.4±2785.4, p=0.001), and lower 24-hour plasma cholinesterase levels (1633.5±2620.9 vs 3550.9±2794.6, p=0.01).

Figure 11A: Association of severity of OP poisoning with serum potassium

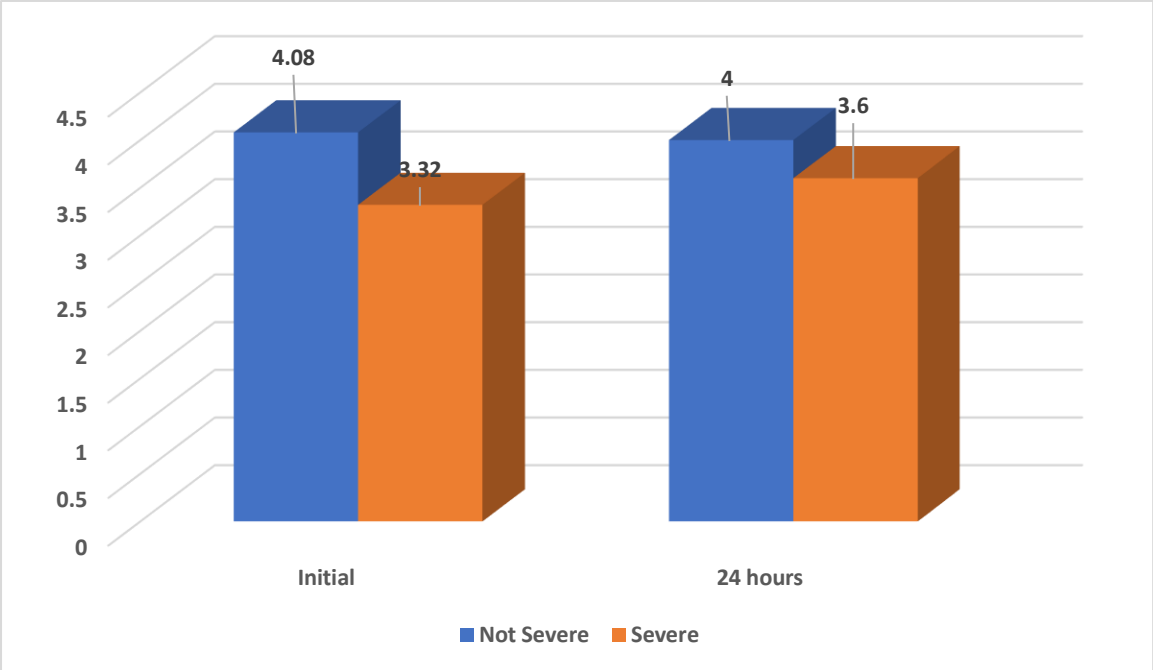


Figure 11B: Association of severity of OP poisoning with Plasma ChE

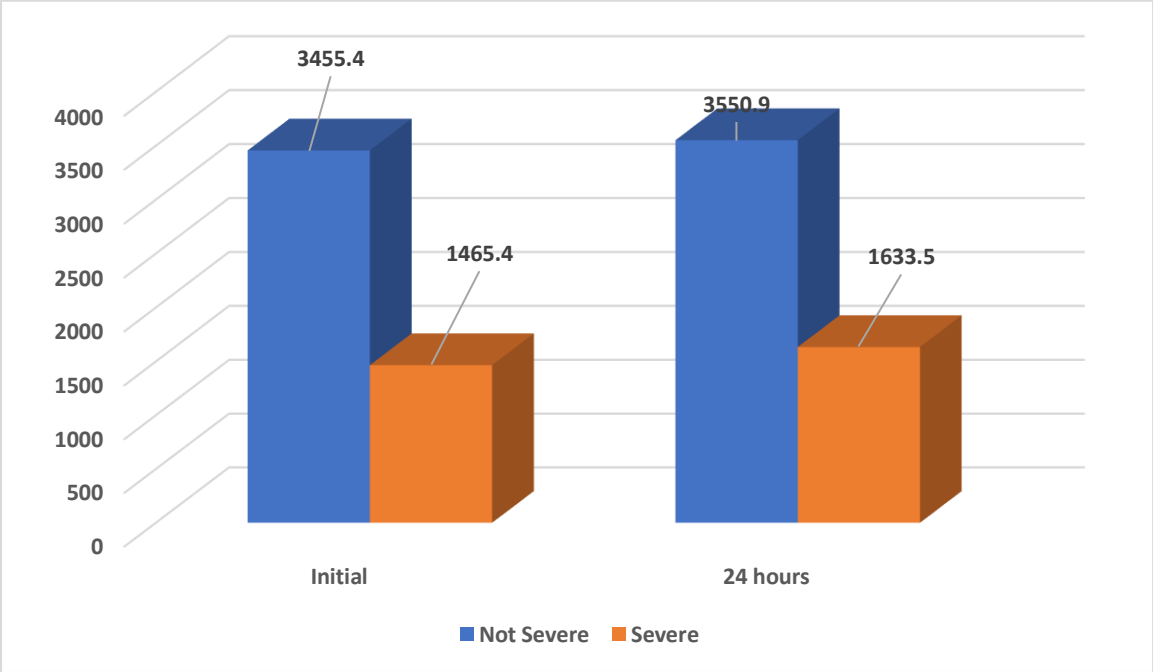


Table 12: Association of severity of OP poisoning with pupil size

Pupil size (mm)	Severity of OP poisoning		p-value
	Not Severe	Severe	
mean±SD	2.37±0.74	1.48±0.67	**<0.001**

Table 11 and graph 11 reveals that patients with severe organophosphate poisoning had significantly smaller pupil sizes compared to non-severe cases (1.48±0.67 mm vs 2.37±0.74 mm, p<0.001), reflecting more pronounced cholinergic effects.

Figure 12: Association of severity of OP poisoning with pupil size

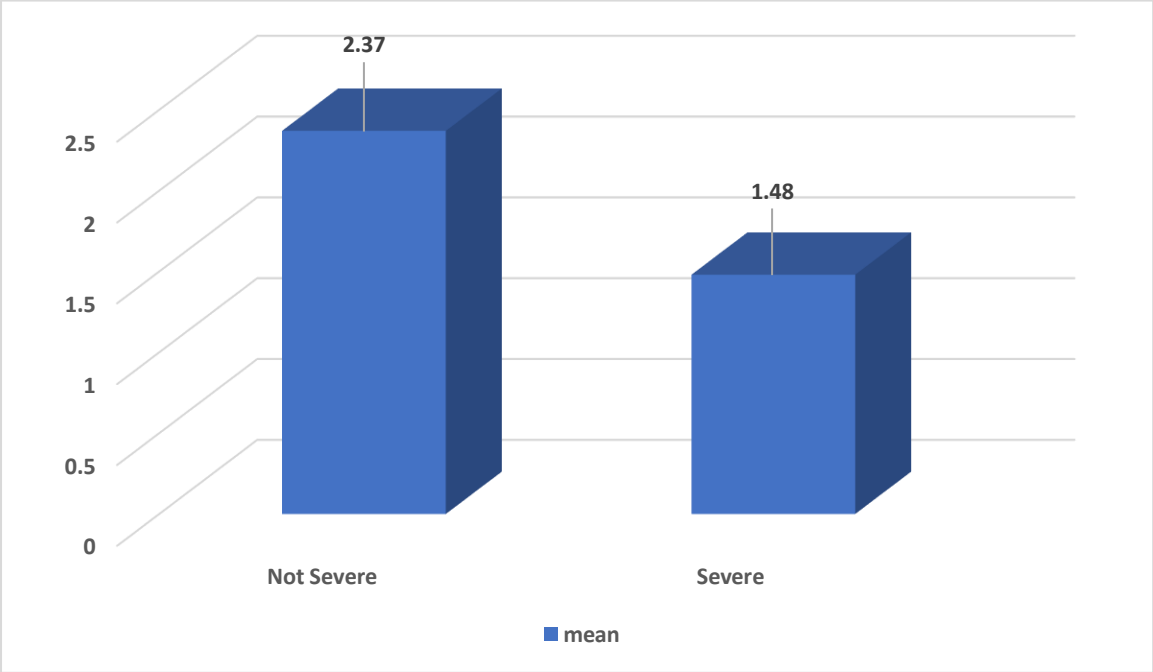


Table 13: Association of severity of OP poisoning with GCS

GCS	Severity of OP poisoning		p-value
	Not Severe	Severe	
3-8	1 (1.9%)	16 (51.6%)	**<0.001**
9-12	3 (5.8%)	8 (25.8%)	
13-15	48 (92.3%)	7 (22.6%)	
Total	**52 (100%)**	**31 (100%)**	

Table 12 and graph 12 shows a strong association between GCS scores and poisoning severity, with 51.6% of severe cases having GCS scores of 3-8 compared to only 1.9% of non-severe cases, while 92.3% of non-severe cases had GCS scores of 13-15 compared to only 22.6% of severe cases (p<0.001).

Figure 13: Association of severity of OP poisoning with GCS

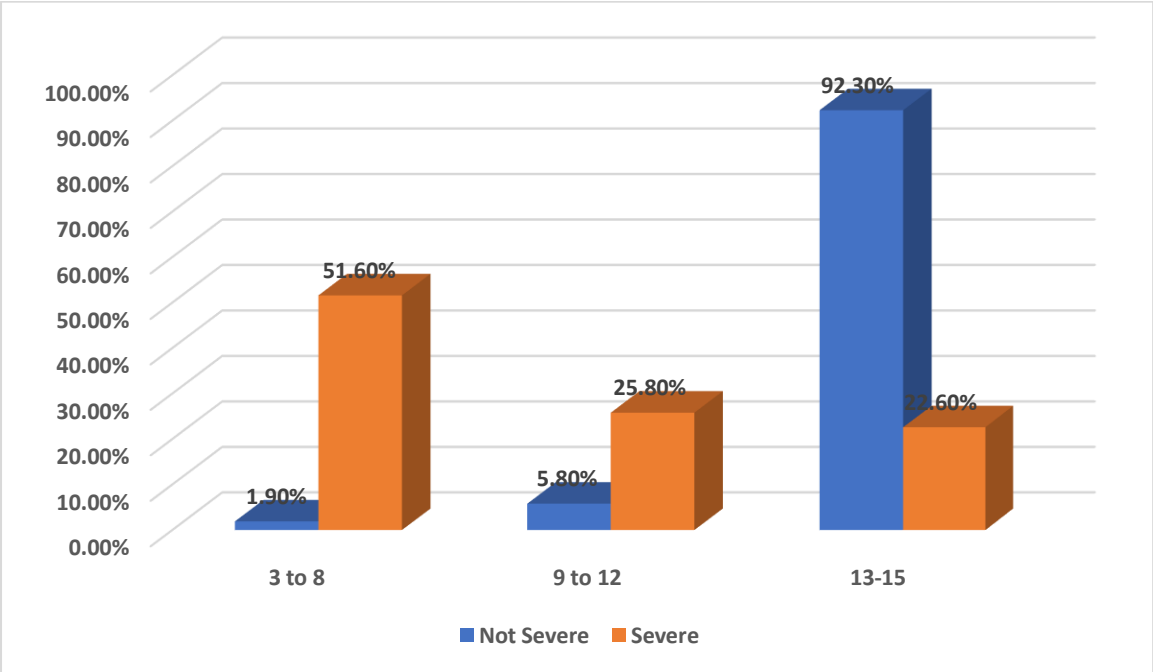


Table 14: Correlation of potassium and acetyl cholinesterase according to severity

Serum Potassium	Severity of OP poisoning	
	Not Severe	**Severe**
Acetyl cholinesterase	r=0.582 **p=0.003**	r=0.675 **p=0.01**

Table 13 demonstrates a significant positive correlation between serum potassium levels and acetylcholinesterase activity in both severe and non-severe organophosphate poisoning cases. The correlation coefficient (r=0.582) for non-severe cases indicates a moderate positive correlation, while the stronger correlation (r=0.675) in severe cases suggests that this relationship becomes more pronounced with increasing poisoning severity. The statistically significant p-values (p=0.003 and p=0.001 respectively) confirm that these correlations are unlikely to be due to chance.

Table 15: Association of mechanical ventilation with serum potassium

serum potassium (mean±SD)	Mechanical ventilation		p-value
	Not required	Required	
Initial	3.97±0.54	3.12±0.48	**<0.001**
At 24 hours	3.91±0.41	3.48±0.52	**0.001**

Table 14 and graph 14 shows a more pronounced difference in serum potassium levels between patients requiring mechanical ventilation and those who don't. Patients requiring ventilatory support had significantly lower initial potassium levels (3.12±0.48 mEq/L vs. 3.97±0.54 mEq/L, p<0.001) and this difference persisted, though slightly less marked, at 24 hours (3.48±0.52 mEq/L vs. 3.91±0.41 mEq/L, p=0.001).

Figure 14: Association of mechanical ventilation with serum potassium

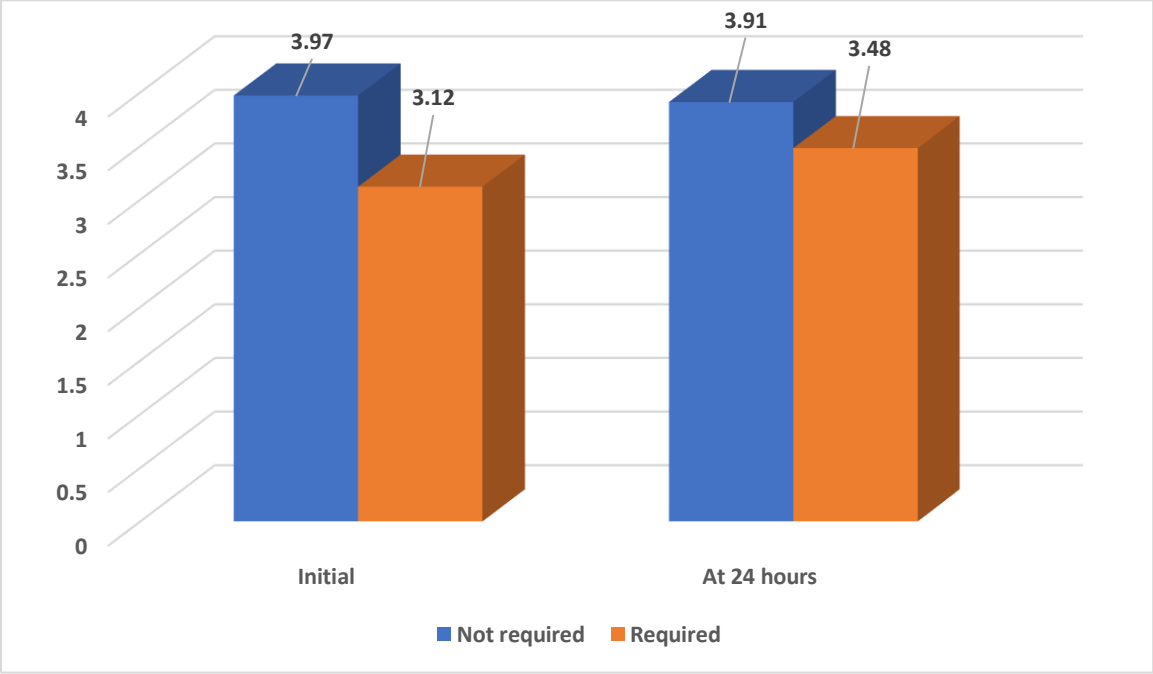


Table 16: Association of mechanical ventilation with serum acetyl cholinesterase levels

serum acetyl cholinesterase levels (mean±SD)	Mechanical ventilation		p-value
	Not required	Required	
Initial	3348.2±2812.4	948.5±1808.1	**<0.001**
At 24 hours	3426.6±29.9.1	1128.7±1983.9	**0.005**

Table 15 and graph 15 shows that acetylcholinesterase levels were significantly lower in patients requiring mechanical ventilation both initially (948.5±1808.1 vs 3348.2±2812.4) and at 24 hours (1128.7±1983.9 vs 3426.6±29.9.1) compared to those not requiring ventilation, with p-values of <0.001 and 0.005 respectively.

Figure 15: Association of mechanical ventilation with serum acetyl cholinesterase levels

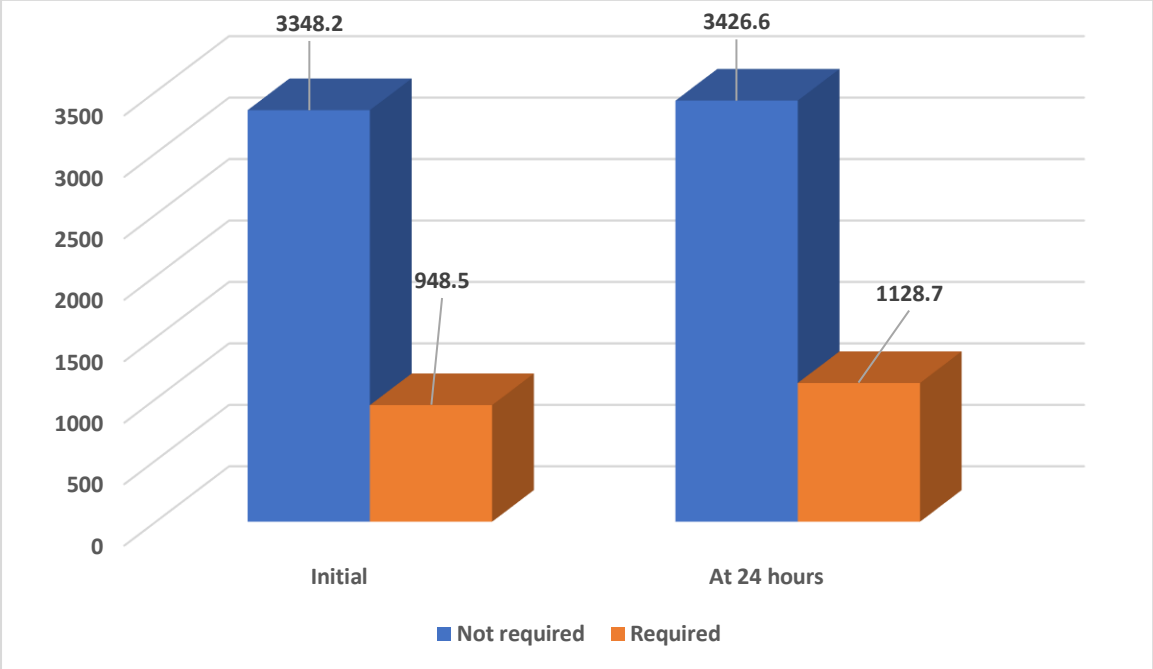


Table 17: Association of outcome with serum potassium levels

serum potassium (mean±SD)	Outcome			p-value
	Death	AMA	Discharge	
Initial	2.86±0.44	3.62±0.58	3.94±0.51	**<0.001**
At 24 hours	3.28±0.39	3.57±0.48	3.92±0.37	**0.002**

Table 16 and graph 16 reveals a striking relationship between serum potassium levels and mortality outcomes. Patients who died had markedly lower initial potassium levels (2.86±0.44 mEq/L) compared to those discharged (3.94±0.51 mEq/L), with patients who left against medical advice (AMA) having intermediate values (3.62±0.58 mEq/L). This gradient across outcome groups became highly statistically significant (p<0.001).

Figure 16: Association of outcome with serum potassium

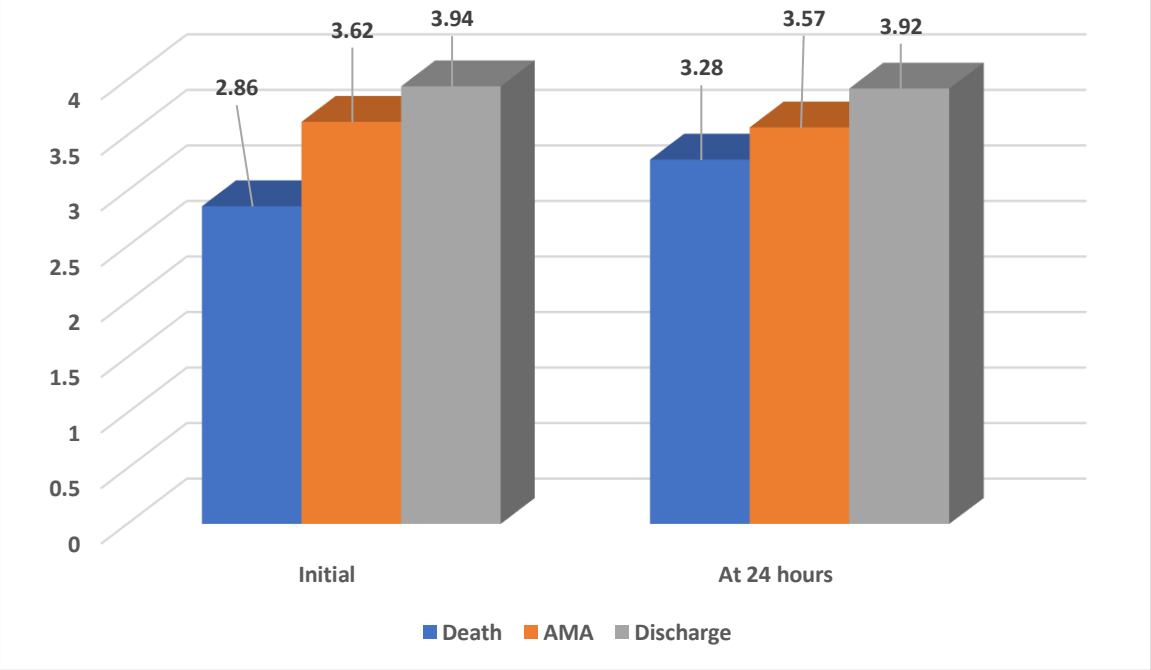
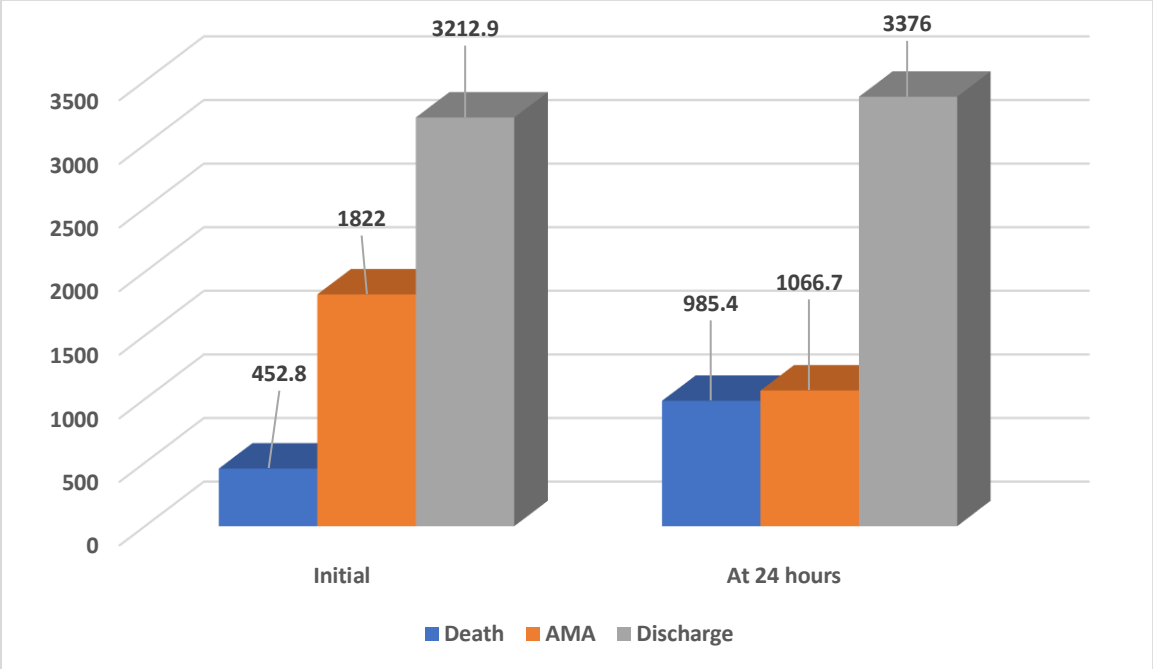


Table 18: Association of outcome with serum acetyl cholinesterase levels

serum acetyl cholinesterase levels (mean±SD)	Outcome			p-value
	Death	AMA	Discharge	
Initial	452.8±198.6	1822±2494.4	3212.9±2824.8	**<0.001**
At 24 hours	985.4±324.7	1066.7±2035.4	3376±2900.9	**<0.001**

Table 17 and graph 17 shows dramatically lower initial acetylcholinesterase levels in patients who died (452.8±198.6) compared to those discharged (3212.9±2824.8), with a highly significant p-value (<0.001). This profound depression of enzyme activity in fatal cases reflects severe organophosphate toxicity. At 24 hours, while there was some increase in enzyme levels among deceased patients (985.4±324.7), they remained substantially lower than in discharged patients (3376±2900.9).

Figure 17: Association of outcome with serum acetyl cholinesterase levels



DISCUSSION

Organophosphate (OP) compounds are widely used as insecticides, pesticides, and chemical warfare agents, making them a significant cause of poisoning worldwide, particularly in agricultural regions. The easy accessibility, low cost, and high toxicity of these compounds contribute to their frequent use in intentional self-harm, especially in developing countries. Acute organophosphate poisoning represents a major public health challenge, characterized by complex pathophysiology primarily involving the inhibition of acetylcholinesterase (AChE) enzyme, leading to an accumulation of acetylcholine at cholinergic synapses and resulting in a characteristic toxidrome. This toxidrome manifests as muscarinic (increased secretions, bronchospasm, miosis, bradycardia), nicotinic (muscle fasciculations, weakness, tachycardia), and central nervous system effects (altered consciousness, seizures, respiratory depression). Early recognition of severity and appropriate management are crucial determinants of patient outcomes. This study sought to explore the correlation between serum potassium levels and plasma cholinesterase activity in assessing the severity of acute organophosphate poisoning, aiming to identify reliable and accessible prognostic markers that could guide clinical decision-making and improve patient outcomes.

Demographic Characteristics

In our study conducted at Shri B.M. Patil Medical College, Hospital and Research Centre, Vijayapura from May 2023 to December 2024, we observed that the majority of organophosphate poisoning cases (67.5%) occurred in the 20-40 years age group, followed by those below 20 years (21.7%). This age distribution aligns with findings from numerous studies worldwide, highlighting the vulnerability of young adults to OP poisoning. Banday et al. reported similar findings in their study from Kashmir, where 62.8% of OP poisoning cases were in the 21-40 years age group, attributing this to the

high levels of stress, emotional instability, and socioeconomic challenges faced by this productive age group.[65] Similarly, Adinew et al. in their Ethiopian study found that 76.8% of poisoning cases were among those aged 21-30 years.[66]

The gender distribution in our study showed a slight male predominance (51.8%) compared to females (48.2%), which differs somewhat from many other studies that report a more pronounced male predominance. For instance, Chaudhary et al. reported a male-to-female ratio of 2.7:1 in their study from Nepal.[67] This difference might reflect regional variations in occupational exposure patterns, sociocultural factors, and changing gender-related stressors. However, our finding is consistent with recent observations by Patil et al. who noted a narrowing gender gap in OP poisoning cases in parts of India, suggesting evolving social dynamics.[68]

The relatively balanced gender distribution in our study contrasts with earlier literature that predominantly showed male preponderance. Panda et al. reported 72.3% male victims in their study, while Karki et al. observed 62% male predominance.[69,70] This shifting pattern warrants attention as it may indicate changing sociocultural dynamics and exposure patterns. Nigam et al. hypothesized that increasing financial and societal pressures on women in developing regions might be contributing to this changing epidemiological profile.[71]

Clinical Manifestations

Our study recorded vomiting as the most prevalent clinical feature (74.7%), followed by muscle weakness (62.6%) and bronchospasm (30.1%). These findings are consistent with the established cholinergic toxidrome of OP poisoning but show some variation in frequency compared to other studies. Vomiting as the predominant symptom aligns with findings by Ahmed et al., who reported vomiting in 81.3% of their patients.[72] This high prevalence of gastrointestinal symptoms can be attributed to the muscarinic

effects of acetylcholine accumulation on the gastrointestinal tract.

The significant proportion of patients presenting with muscle weakness (62.6%) in our study reflects the nicotinic effects of OP poisoning. Yurumez et al. observed similar findings with muscle weakness in 58% of their patients.[73] However, Kang et al. reported a lower incidence (34.7%) of neuromuscular manifestations, suggesting potential variations based on the specific OP compound involved, route of exposure, and time elapsed before medical intervention.[74]

Bronchospasm, observed in 30.1% of our patients, represents a critical respiratory complication of OP poisoning. Comparatively, Eddleston et al. reported respiratory manifestations in 39.7% of cases in their large cohort study from Sri Lanka.[75] This variation might be attributed to differences in the potency of the OP compounds, exposure severity, and pre-existing respiratory conditions. The respiratory complications are of particular clinical significance as they often necessitate aggressive airway management and mechanical ventilation.

Interestingly, our observation of salivation in 19.3% of cases is notably lower than that reported by Banerjee et al., who found excessive salivation in 63.2% of their patients.[76] This disparity might be attributed to variations in the timing of clinical evaluation, pre-hospital management, or specific agent characteristics. The relatively low incidence of convulsions (6%) in our study is comparable to findings by Muley et al., who reported seizures in 5.8% of cases.[77]

Glasgow Coma Scale and Consciousness Level

The Glasgow Coma Scale (GCS) distribution in our study revealed that 66.3% of patients maintained good consciousness levels (GCS 13-15), while 20.5% presented with severe impairment (GCS 3-8). The GCS serves as a valuable clinical tool in assessing the central nervous system effects of OP poisoning and has been consistently associated with

prognosis in various studies.

The significant association between GCS scores and poisoning severity demonstrated in our study ($p<0.001$) corroborates the findings of numerous researchers. Akdur et al. reported that lower admission GCS scores were significantly associated with higher mortality rates and need for intensive care.[78] Similarly, Davies et al. identified GCS as one of the strongest predictors of outcome in their prospective study of 2,209 OP poisoning cases.[79]

The observation that 51.6% of severe OP poisoning cases in our study had GCS scores of 3-8, compared to only 1.9% of non-severe cases, underscores the importance of early neurological assessment in risk stratification. Hiremath et al. demonstrated similar findings and suggested that GCS should be incorporated into severity scoring systems for OP poisoning.[80] The strong correlation between GCS and poisoning severity can be attributed to the central nervous system effects of OP compounds, particularly their ability to cross the blood-brain barrier and affect neurotransmission.

Mechanical Ventilation Requirements

In our cohort, mechanical ventilation was required in 26.5% of patients, which is comparable to rates reported in other studies. Sungur and Güven reported a mechanical ventilation rate of 21.6% in their retrospective analysis of 47 OP poisoning cases.[81] The need for mechanical ventilation in OP poisoning stems from several factors, including respiratory muscle paralysis, bronchospasm, excessive secretions, and central respiratory depression.

The requirement for mechanical ventilation has been established as a marker of severe poisoning and is associated with increased mortality. Eddleston et al. reported a mechanical ventilation rate of 23.9% with an associated mortality rate of 35.6% among ventilated patients.[82] Similarly, Peter et al. observed that 31.5% of OP poisoning patients

required mechanical ventilation, with a mortality rate of 28.3% in this subgroup.[83]

The determination of factors predicting mechanical ventilation needs has been a focus of several studies. Kang et al. identified low plasma cholinesterase levels, reduced GCS scores, and higher APACHE II scores as significant predictors of mechanical ventilation requirements.[84] Our findings indirectly support this association, as patients classified as having severe poisoning based on clinical and biochemical parameters were more likely to require ventilatory support.

Biochemical Parameters and Their Correlation with Severity

Serum Potassium Levels

One of the most significant findings of our study was the strong association between serum potassium levels and OP poisoning severity. We observed that patients with severe poisoning had significantly lower initial serum potassium levels (3.32±0.59 mEq/L) compared to non-severe cases (4.08±0.47 mEq/L), with a highly significant p-value of <0.001. This observation aligns with several recent studies that have highlighted hypokalemia as a potential prognostic marker in OP poisoning.

Bhattarai et al. reported similar findings in their prospective study, noting that patients with severe OP poisoning had mean serum potassium levels of 3.21±0.48 mEq/L compared to 3.86±0.52 mEq/L in milder cases.[85] They attributed this hypokalemia to increased sympathetic activity, respiratory alkalosis from hyperventilation, and vomiting-induced losses. The persistence of this difference at 24 hours in our study (3.6±0.55 vs 4±0.34 mEq/L, p=0.001) suggests that potassium dynamics might reflect ongoing toxicity rather than just initial physiological stress.

Yang et al. demonstrated in their study that hypokalemia was not only common in acute OP poisoning but also correlated significantly with mortality.[86]They reported that

patients with serum potassium levels below 3.5 mEq/L had a mortality rate of 22.9% compared to 7.8% in those with normal levels. This association remained significant even after adjusting for confounding factors like age, gender, and comorbidities.

The pathophysiological basis for hypokalemia in OP poisoning is multifactorial. Shah et al. proposed that increased adrenergic activity leads to potassium shift into cells through enhanced Na^+-K^+ ATPase activity.[87]Additionally, Vijayakumar et al. suggested that acetylcholine-induced insulin secretion might contribute to intracellular potassium shift.[88] Our findings add to this growing body of evidence suggesting that serum potassium could serve as a simple, cost-effective prognostic marker in resource-limited settings.

The clinical implications of hypokalemia in OP poisoning extend beyond its role as a severity marker. Hypokalemia can potentiate cardiac arrhythmias, exacerbate muscle weakness, and complicate respiratory management. Karki et al. reported increased incidence of cardiac complications in OP poisoning patients with hypokalemia.[89]Therefore, monitoring and correcting potassium levels might have therapeutic value beyond its prognostic significance.

Plasma Cholinesterase Levels

Our study demonstrated that plasma cholinesterase levels were significantly lower in patients with severe OP poisoning compared to non-severe cases, both initially (1465.4±2336.2 vs 3455.4±2785.4, p=0.001) and at 24 hours (1633.5±2620.9 vs 3550.9±2794.6, p=0.01). This finding is consistent with the established role of cholinesterase inhibition in OP toxicity and supports its utility as a severity marker.

Plasma cholinesterase (butyrylcholinesterase) has been widely used as a biomarker of OP exposure and severity assessment. Nouira et al. reported that plasma cholinesterase levels below 1000 IU/L were associated with increased mortality and need

for mechanical ventilation.[90] In our study, this association was even more dramatic, with non-survivors showing profoundly depressed initial enzyme activity (452.8±198.6) compared to survivors (3212.9±2824.8, $p<0.001$). This extreme reduction in cholinesterase activity in fatal cases represents a nearly 86% depression from normal values and indicates severe enzyme inhibition incompatible with normal physiological function. The slight increase in mean plasma cholinesterase levels after 24 hours observed in our study (from 2712.2±2785.7 to 2803.4±2864.9) likely reflects the effects of therapeutic interventions, particularly oxime therapy. This pattern aligns with observations by Pawar et al., who reported gradual recovery of enzyme activity following appropriate treatment.[91] However, the persistent significant difference between severe and non-severe cases at 24 hours suggests that initial severity continues to influence biochemical recovery. Notably, even after 24 hours of treatment, cholinesterase levels in patients who ultimately died (985.4±324.7) remained drastically lower than in those who survived (3376±2900.9, $p<0.001$), indicating limited enzyme reactivation despite therapeutic interventions.

While plasma cholinesterase serves as an established biomarker, its utility has certain limitations. Eddleston et al. noted that the correlation between plasma cholinesterase inhibition and clinical severity varies depending on the specific OP compound involved.[92] Compounds that are poor inhibitors of plasma cholinesterase but potent inhibitors of acetylcholinesterase in neural tissue might cause severe poisoning despite relatively preserved plasma enzyme activity.

Correlation Between Potassium and Cholinesterase

One of the most novel and significant findings in our study was the strong positive correlation between serum potassium levels and acetylcholinesterase activity in both severe (r=0.675, p=0.001) and non-severe (r=0.582, p=0.003) OP poisoning cases. This correlation has not been extensively investigated in previous studies and provides important insights into the interrelated pathophysiological mechanisms in OP poisoning.

The stronger correlation observed in severe cases (r=0.675) compared to non-severe cases (r=0.582) suggests that as poisoning severity increases, the relationship between cholinesterase inhibition and potassium dysregulation becomes more pronounced. This finding has significant clinical implications, as it indicates that serum potassium could potentially serve as a surrogate marker for cholinesterase activity, particularly in settings where specialized enzyme assays may not be readily available.

The mechanistic basis for this correlation likely involves multiple pathways. First, both parameters are directly affected by the degree of OP exposure and subsequent toxicity. Second, the acetylcholine excess resulting from cholinesterase inhibition leads to autonomic disturbances that can alter potassium homeostasis through changes in cellular membrane permeability and ion transport mechanisms. Third, shared pathophysiological processes such as oxidative stress and cellular dysfunction may impact both parameters simultaneously.

Sumathi et al.[93] examined various biochemical parameters in OP poisoning and suggested that combined assessment of multiple markers provides superior predictive value compared to single parameter evaluation. Our finding of a significant correlation between potassium and cholinesterase levels supports this integrated approach to severity assessment and risk stratification. This correlation could be particularly valuable in developing streamlined clinical algorithms for rapid assessment of poisoning severity in

emergency settings, combining the specificity of cholinesterase measurement with the accessibility and rapid turnaround time of potassium testing.

Pupillary Changes and Their Significance

Our study found that patients with severe OP poisoning had significantly smaller pupil sizes compared to non-severe cases (1.48±0.67 mm vs 2.37±0.74 mm, $p<0.001$). This miotic effect, resulting from unopposed cholinergic stimulation of the pupillary sphincter, serves as a visible clinical marker of cholinergic excess.

The correlation between pupil size and poisoning severity has been reported by several researchers. Abedin et al. described progressive miosis with increasing severity of poisoning and proposed pupillary diameter as a simple bedside parameter for initial assessment.[94] Similarly, Senanayake et al. included pupillary constriction as a major component in their Peradeniya Organophosphorus Poisoning (POP) scale for severity assessment.[95]

Interestingly, the degree of miosis does not always correlate perfectly with plasma cholinesterase levels. Jayawardane et al. observed that pupillary changes often persist despite improving cholinesterase activity, suggesting complex neuro-pharmacological interactions.[96] This might explain why some patients with moderate enzyme inhibition still exhibit pronounced miosis. The autonomic effects of OP compounds appear to have variable temporal dynamics, with ocular manifestations often persisting longer than other systemic effects.

The practical value of pupillary assessment lies in its simplicity and non-invasive nature. In resource-limited settings, where biochemical testing might be delayed or unavailable, pupillary examination can provide immediate information about potential poisoning severity. Panda et al. demonstrated that incorporation of pupillary assessment

into clinical scoring systems improved their predictive accuracy for outcomes in OP poisoning.[97]

Vital Signs and Their Clinical Implications

The mean respiratory rate (24.16±6.1 breaths per minute) and heart rate (99.3±21.1 beats per minute) observed in our study reflect the complex autonomic effects of OP poisoning. These values represent the net effect of opposing muscarinic (parasympathetic) and nicotinic (sympathetic and neuromuscular) actions of accumulated acetylcholine.

Respiratory patterns in OP poisoning can be influenced by bronchospasm, excessive secretions, and respiratory muscle weakness. Eddleston et al. noted that tachypnoea often precedes respiratory failure and should prompt close monitoring.[98] The relatively elevated mean respiratory rate in our cohort suggests compensatory mechanisms against developing hypoxemia or acidosis.

The mean heart rate of 99.3±21.1 beats per minute in our patient population. Karki et al. reported similar findings, with tachycardia being more common than bradycardia in their series of OP poisoning cases.[99]The cardiac effects of OP poisoning can be particularly challenging to manage due to their dynamic nature, with potential transitions between bradycardia and tachycardia based on the relative dominance of muscarinic versus nicotinic effects.

The variability in vital signs across different OP poisoning cases highlights the complexity of the toxidrome and the need for individualized assessment. Liu et al. demonstrated that vital sign instability, particularly alternating tachycardia and bradycardia, was associated with poorer outcomes.[100] This underscores the importance of continuous monitoring rather than relying on single measurements.

Study Strengths and Limitations

The strengths of our study include its prospective design, comprehensive assessment of both clinical and biochemical parameters, and serial evaluation of key markers. The inclusion of 83 consecutive cases provides adequate statistical power for the primary associations examined. Moreover, the study setting in an agricultural region with high OP exposure rates enhances the external validity of our findings.

However, certain limitations should be acknowledged. First, we did not specifically identify the type of OP compound involved in each case, which might influence the pattern and severity of clinical manifestations. Different OP compounds have varying potencies, lipid solubilities, and aging rates that can affect their toxicodynamic profiles. Eddleston et al. demonstrated substantial differences in clinical course and mortality based on the specific OP agent involved.[101]

Second, our follow-up was limited to 24 hours, which precludes assessment of intermediate and long-term outcomes. Some complications of OP poisoning, such as intermediate syndrome and delayed polyneuropathy, typically manifest beyond this timeframe. Senanayake and Karalliedde reported that intermediate syndrome typically develops 24-96 hours after acute poisoning and can cause significant morbidity.[102]

Third, we did not measure red blood cell acetylcholinesterase activity, which more accurately reflects neural acetylcholinesterase inhibition compared to plasma cholinesterase. Worek et al. demonstrated that red blood cell acetylcholinesterase correlates better with severity of central nervous system effects.[103] However, plasma cholinesterase measurement is more widely available in clinical settings and serves as a reasonable proxy.

Finally, our severity classification was based on a composite assessment rather than a single validated scoring system. While this approach captures multiple dimensions

of poisoning severity, it might limit direct comparability with studies using standardized scales like the Peradeniya Organophosphorus Poisoning (POP) scale or the Poisoning Severity Score (PSS).

Clinical Implications and Future Directions

The findings of our study have several important clinical implications. First, they support the utility of serum potassium as an accessible biomarker for severity assessment in OP poisoning. In resource-limited settings where specialized tests may be unavailable, potassium measurement can provide valuable prognostic information. Healthcare facilities managing OP poisoning should consider incorporating routine potassium monitoring into their protocols.

Second, the strong association between GCS scores and poisoning severity reinforces the importance of neurological assessment in risk stratification. Early identification of patients with depressed consciousness can facilitate timely intervention, including mechanical ventilation, which might improve outcomes. Training healthcare providers in standardized GCS assessment could enhance the consistency of severity classification.

Third, the combination of clinical parameters (GCS, pupil size) with biochemical markers (serum potassium, plasma cholinesterase) appears to provide complementary information about poisoning severity. Developing integrated scoring systems that incorporate these multiple dimensions could enhance risk stratification and guide treatment intensity.

For future research, several directions appear promising. Longitudinal studies with extended follow-up periods could better characterize the relationship between early markers like hypokalemia and long-term outcomes, including delayed neurological sequelae. Additionally, investigating the mechanisms underlying hypokalemia in OP

poisoning might reveal novel therapeutic targets.

Comparative studies of different OP compounds and their specific effects on potassium homeostasis and cholinesterase inhibition patterns would enhance our understanding of toxin-specific responses. Furthermore, evaluating the impact of early potassium correction on clinical outcomes could determine whether this represents a therapeutic opportunity beyond its prognostic value.

Implementation studies examining the feasibility and impact of incorporating these findings into clinical algorithms would help translate research insights into practical care improvements. Finally, exploring genetic factors that might influence individual susceptibility to OP toxicity and biomarker expression could advance personalized approaches to poisoning management.

Conclusion

Our study demonstrates significant correlations between serum potassium levels, plasma cholinesterase activity, and the clinical severity of acute organophosphate poisoning. Lower serum potassium levels were strongly associated with increased poisoning severity, suggesting its potential utility as a simple prognostic marker. This association persisted at 24 hours, indicating that potassium dynamics reflect ongoing toxicity rather than just initial physiological stress.

The demographic pattern observed in our study, with predominance in the young adult population and a relatively balanced gender distribution, highlights the evolving epidemiological profile of OP poisoning. The clinical manifestations were consistent with the established cholinergic toxidrome, with vomiting, muscle weakness, and bronchospasm being the most frequent presentations.

The strong correlations between clinical parameters (GCS, pupil size) and biochemical markers (serum potassium, plasma cholinesterase) support an integrated

approach to severity assessment. This multi-dimensional evaluation provides complementary information that enhances risk stratification and could guide treatment decisions.

In conclusion, our findings contribute to the growing evidence base on prognostic markers in OP poisoning and highlight the potential value of readily available tests like serum potassium in resource-limited settings. Future research should focus on elucidating the mechanisms underlying these associations and evaluating their impact on clinical outcomes when incorporated into management protocols.

CONCLUSION

The present study provides valuable insights into the utility of serum potassium and plasma cholinesterase levels as biomarkers for assessing the severity of acute organophosphate poisoning. Our findings demonstrate that patients with severe organophosphate poisoning consistently present with significantly lower serum potassium levels compared to those with non-severe poisoning, both at admission and after 24 hours of treatment. This correlation between hypokalemia and poisoning severity suggests that serum potassium can serve as a simple, cost-effective, and readily available prognostic marker, particularly in resource-limited settings where specialized toxicology tests may not be immediately accessible.

Similarly, plasma cholinesterase activity showed significant depression in severe cases compared to non-severe ones, confirming its established role in reflecting the degree of organophosphate toxicity. The persistence of this difference at 24 hours post-admission indicates that both initial poisoning severity and response to treatment can be monitored through these biochemical parameters. The strong association observed between these laboratory markers and clinical indicators such as GCS scores and pupillary size provides a comprehensive approach to severity assessment that combines objective biochemical data with bedside clinical evaluation.

Our demographic analysis revealed that young adults between 20-40 years constitute the most vulnerable population for organophosphate poisoning, with a nearly equal gender distribution. This finding reflects changing socioeconomic pressures and exposure patterns that differ from historical trends. The predominance of gastrointestinal and neuromuscular manifestations in our cohort aligns with the established cholinergic toxidrome but highlights the variability in symptom expression that can complicate clinical assessment.

The significant correlation between reduced GCS scores and poisoning severity underscores the importance of early neurological assessment in risk stratification. The finding that over half of the severe poisoning cases required mechanical ventilation emphasizes the need for prompt identification of high-risk patients to ensure timely respiratory support. The integration of clinical parameters (GCS, pupil size, requirement for mechanical ventilation) with biochemical markers (serum potassium, plasma cholinesterase) provides a more comprehensive assessment of poisoning severity than either approach alone.

In conclusion, our study supports the use of serum potassium levels, alongside plasma cholinesterase activity and clinical parameters, in the assessment and prognostication of acute organophosphate poisoning. This multi-dimensional approach enhances the early identification of high-risk patients and facilitates appropriate triage and management decisions. Future research should focus on validating these findings in larger, multi-center studies and exploring the therapeutic implications of early potassium supplementation in patients with organophosphate-induced hypokalemia.

SUMMARY

INTRODUCTION

Organophosphate (OP) compounds are widely used as pesticides, particularly in agricultural regions, and constitute a significant cause of poisoning worldwide. The toxicity primarily results from inhibition of acetylcholinesterase enzyme, leading to accumulation of acetylcholine at synapses and manifesting as a characteristic cholinergic toxidrome. While plasma cholinesterase levels have been traditionally used to confirm exposure and assess severity, their correlation with clinical outcomes is not always consistent. Recent studies have suggested alterations in serum electrolytes, particularly potassium, as potential markers of poisoning severity. This study aimed to correlate serum potassium and plasma cholinesterase levels with the clinical severity of acute organophosphate poisoning.

AIMS AND OBJECTIVES

Objectives:

3. To evaluate the levels of serum potassium and plasma cholinesterase
4. To correlate the min assessing the severity of acute organophosphorous poisoning

MATERIAL AND METHODS

This prospective study was conducted at Shri B.M. Patil Medical College, Hospital and Research Centre, Vijayapura, from May 2023 to December 2024. A total of 83 patients with acute organophosphate poisoning were included. Detailed clinical assessment, including Glasgow Coma Scale (GCS) scoring and pupillary examination, was performed at admission. Serum potassium and plasma cholinesterase levels were measured at admission and after 24 hours. Patients were classified as having severe or non-severe poisoning based on clinical parameters and requirement for mechanical

ventilation. Statistical analysis was performed to assess the correlation between biochemical markers and poisoning severity.

RESULTS

- The majority of patients (67.5%) were between 20-40 years of age, with a slight male predominance (51.8%). Common clinical features included vomiting (74.7%), muscle weakness (62.6%), and bronchospasm (30.1%). Most patients (66.3%) maintained good consciousness levels (GCS 13-15), while 20.5% presented with severe impairment (GCS 3-8). Mechanical ventilation was required in 26.5% of patients.
- Severe poisoning was observed in 37.3% of patients. These patients had significantly lower serum potassium levels both initially (3.32±0.59 vs 4.08±0.47 mEq/L, $p<0.001$) and at 24 hours (3.6±0.55 vs 4±0.34 mEq/L, $p=0.001$) compared to non-severe cases. Similarly, plasma cholinesterase levels were significantly lower in severe cases, both initially (1465.4±2336.2 vs 3455.4±2785.4, $p=0.001$) and at 24 hours (1633.5±2620.9 vs 3550.9±2794.6, $p=0.01$).
- A significant positive correlation was observed between serum potassium and acetylcholinesterase levels in both severe ($r=0.675$, $p=0.001$) and non-severe ($r=0.582$, $p=0.003$) poisoning cases. Patients requiring mechanical ventilation had markedly lower initial potassium levels (3.12±0.48 mEq/L vs. 3.97±0.54 mEq/L, $p<0.001$) and lower acetylcholinesterase levels (948.5±1808.1 vs 3348.2±2812.4, $p<0.001$).
- Patients who died had dramatically lower initial potassium levels (2.86±0.44 mEq/L) compared to survivors (3.94±0.51 mEq/L, $p<0.001$). Similarly, patients who died had profoundly reduced initial acetylcholinesterase levels

(452.8±198.6) compared to those who were discharged (3212.9±2824.8, $p<0.001$). Severe poisoning was also associated with lower GCS scores ($p<0.001$) and smaller pupil sizes (1.48±0.67 vs 2.37±0.74 mm, $p<0.001$).

CONCLUSION:

Serum potassium levels show a significant correlation with the severity of acute organophosphate poisoning, comparable to the established marker plasma cholinesterase. The combination of these biochemical parameters with clinical indicators like GCS scores and pupillary changes provides a comprehensive approach to severity assessment. Serum potassium measurement can serve as a simple, cost-effective, and readily available tool for early risk stratification, particularly in resource-limited settings.

REFERENCES

1. Eddleston M, Phillips MR. Self poisoning with pesticides. BMJ. 2019;328(7430):42-4.
2. World Health Organization. The WHO recommended classification of pesticides by hazard and guidelines to classification 2019-2020. Geneva: WHO; 2020.
3. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: systematic review. BMC Public Health. 2018;7:357.
4. Thiermann H, Szinicz L, Eyer P, Worek F. Modern strategies in therapy of organophosphate poisoning. Toxicol Lett. 2019;107:233-9.
5. Roberts DM, Aaron CK. Managing acute organophosphorus pesticide poisoning. BMJ. 2017;334:629-34.
6. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet. 2018;371:597-607.
7. Peter JV, Jerobin J, Nair A, Bennett A. Clinical profile and outcome of patients hospitalized with organophosphate poisoning. JAPI. 2019;58:11-4.
8. Senanayake N, de Silva HJ, Karalliedde L. A scale to assess severity in organophosphorus intoxication: POP scale. Hum Exp Toxicol. 2018;12:297-9.
9. Konickx LA, Bingham K, Eddleston M. Is oxygen required before atropine administration in organophosphorus or carbamate pesticide poisoning? Clin Toxicol. 2019;52:531-7.

10. Eddleston M, Chowdhury FR. Pharmacological treatment of organophosphorus insecticide poisoning: current status and future direction. Expert Rev Clin Pharmacol. 2020;9:1363-73.
11. L Haddad, J Winchester. Clinical management of poisoning and overdose. Philedelphia, WB Saunders, 1983, 575-586.
12. T Namba, C Nolte, J Jackrel. Am J Med, 1971, 50, 475-492.
13. Environmental protection agency: National study of hospital admitted pesticide poisonings for 1974 to 1976. Washington DC, US Government Printing Office, 1979.
14. J Veltri, T Litovitz: 1983 Annual report of the American association of poison control centers national data collection system. Am J Emerg Med, 1984, 2, 420-443.
15. 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.
16. Health implications from monocrotophos use: a review of the evidence in India – WHO 2009, 1-6.
17. Taruni N, Bijoy TK, Momonchand A: A profile of poisoning cases admitted in Rims Hospital, Imphal. Journal of Forensic Medicine. 2001, 18:31-3.
18. Suicide- World Head Organization. (2022). Accessed: February 22, 2023: https://www.who.int/news-room/fact-sheets/detail/suicide.
19. Samaria S, Pandit V, Akhade S, et al. (January 16, 2024) Clinical and Epidemiological Study of Poisoning Cases Presenting to the Emergency Department of a Tertiary Care Center in Central India. Cureus 16(1): e52368.

20. Holmstedt B. (1963). Structure-activity relathionships of the organophosphorus anticholinesterase agents In Cholinesterases and Anticholinesterase Agents, (Koelle G. B., Ed.), pp. 428–485. Springer-Verlag, Berlin.
21. Petroianu G. A. (2010a). History of organophosphate synthesis: The very early days. Pharmazie 65, 306–311.
22. Petroianu G. A. (2010b). Toxicity of phosphor esters; Willy Lange (1900–1976) and Gerda von Krueger (1907-after 1970). Pharmazie 65, 776–780.
23. *Costa L. G. (1987). Toxicology of pesticides: A brief history In Toxicology of Pesticides: Experimental, Clinical, and Regulatory Perspectives (Costa L. G., Galli C. L., Murphy S. D., Eds.), NATO ASI Series, Vol. 113, pp. 1–9. Springer-Verlag, Berlin.*
24. *Delfino R. T., Ribeiro T. S., Figueroa-Villar J. D. (2009). Organophosphorus compounds as chemical warfare agents: A review. J. Braz. Chem. Soc. 20, 407–428.*
25. Atwood D., Paisley-Jones C. (2017). Pesticides Industry Sales and Usage: 2008–2012 Market Estimates, pp. 32 US Environmental Protection Agency, Washington, DC.
26. Casida J. E. (1964). Esterase inhibitors as pesticides. Science 146, 1011–1017.
27. Hobbiger F. (1963). Reactivation of phosphorylated acetylcholinesterase In: Cholinesterases and Anticholinesterase Agents, (Koelle G. B., Ed.), pp. 922–988. Springer-Verlag, Berlin.

28. Mahmood N. A., Carmichael W. W. (1987). Anatoxin-a(s), an anticholinesterase from the cyanobacterium *anabaena flos-aquae* NRC-525-17. Toxicon 25, 1221–1227.
29. Costa L. G. (1988). Organophosphorus compounds. In Recent Advances in Nervous System Toxicology (Galli C. L., Manzo L., Spencer P. S., Eds.), pp. 203–246.
30. Chambers J. E., Meek E. C., Chambers H. W. (2010b). The metabolism of organophosphorus insecticides, In Hayes' Handbook of Pesticide Toxicology (Krieger R., Ed.), pp 1399–1407.
31. Balali-Mood B. Basic and Clinical Toxicology of Organophosphorus Compounds. London, UK: Springer; 2014. Chemistry and classification of OP compounds; pp. 1–23.
32. Gupta R. C. Toxicology of Organophosphate & Carbamate Compounds. Amsterdam, Netherlands: Elsevier; 2006. Classification and uses of organophosphates and carbamates; pp. 5–24.
33. Mukherjee S, Gupta RD. Organophosphorus Nerve Agents: Types, Toxicity, and Treatments. J Toxicol. 2020 Sep 22;2020:3007984.
34. Benschop H. P., De Jong L. P. A. Nerve agent stereoisomers: analysis, isolation and toxicology. Accounts of Chemical Research. 1988;21(10):368–374.
35. Mousavi M., Hellström-Lindahl E., Guan Z.-Z., Bednar I., Nordberg A. Expression of nicotinic acetylcholine receptors in human and rat adrenal medulla. Life Sciences. 2001;70(5):577–590.
36. Sikary AK. Homicidal poisoning in India: A short review. J Forensic Leg Med. 2019 Feb;61:13-16.

37. 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.
38. Rusyniak DE, Nañagas KA. Organophosphate poisoning. Semin Neurol. 2004 Jun;24(2):197-204.
39. Eddleston M. The pathophysiology of organophosphorus pesticide self-poisoning is not so simple. Neth J Med. 2008 Apr;66(4):146-8.
40. JA Vale. Toxicology letters, 1998, 102-103, 649-652.
41. WF Durham, HR Wolfe, JW Elliott. Absorption and excretion of Parathion by spraymen. Arch Environ Health, 1972, 24, 381-387.
42. DL Rickett, JF Glenn, ET Beers. Neurotoxicology, 1986, 7, 225-236.
43. Sarojini Tripathi, Ajai Kumar Srivastav. Pesticide Biochemistry and Physiology, 2010;97:60-65.
44. AM Saadeh, NA Farsakh, MK Al-Ali. Heart, 1997, 77, 461-464.
45. Y Yusuf, Y Yucel, S Hayrettin, D Polat, O Seda, A Okhan, et al. J Emerg Med, 2009, 36, 39- 42.
46. T Dassanayake, V Weerasinghe, U Dangahadeniya, K Kularatne, A Dawson, L Karalliedde, et al. Clinical Neurophysiology, 2008, 119, 144-150.
47. C Maurizio, MT Gian, C Roberto, LR Cinzia, M Francesca, R Francesco, et al. Reproductive Toxicology, 2008, 26, 13-18.
48. K Hideki, Y Mayumi, A Kunihiko, Y Hiroshi, L Mitsuo, F Masatoshi. Gastrointestinal Endoscopy, 1999, 49, 642-643.
49. J Vidyasagar, N Karunakar, MS Reddy, K Rajnarayana, T Surender, DR Krishna. Indian J Pharmacol, 2004, 36, 76-79.

50. SK Rastogi, PVV Satyanarayan, D Ravishankar, Sachin Tripathi. Indian J Occup Environ Med, 2009, 13, 131-134.
51. Merrill DG, Mihm FG. Prolonged toxicity of organophosphate poisoning. Crit Care Med. 1982 Aug;10(8):550-1.
52. Namba T, Nolte CT, Jackrel J, Grob D. Poisoning due to organophosphate insecticides. Acute and chronic manifestations. Am J Med. 1971 Apr;50(4):475-92.
53. Robb EL, Regina AC, Baker MB. Organophosphate Toxicity. [Updated 2023 Nov 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470430/
54. Senanayake N, de Silva HJ, Karalliedde L. A scale to assess severity in organophosphorus intoxication: POP scale. Hum Exp Toxicol. 1993 Jul;12(4):297-9.
55. Eddleston, M., Buckley, N. A., Eyer, P., & Dawson, A. H. (2007). Management of acute organophosphorus pesticide poisoning. *The Lancet*, *371*(9612), 597–607. https://doi.org/10.1016/s0140-6736(07)61202.
56. 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.
57. 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.

58. Thunga G, Sam KG, Khera K, et al. "Evaluation of incidence, clinical characteristics and management in organophosphorus poisoning patients in a tertiary care hospital." Journal of Toxicology and Environmental Health Sciences. 2010.
59. Basnet D, Shakya YL, Gupta S, Shah NA, Gupta B, Yadav M. Serum Potassium Level in Acute Organophosphorus Poisoning and its Correlation with Severity in Patients Presenting to Emergency Room in a Tertiary Hospital. Journal of Karnali Academy of Health Sciences [Internet]. 2024Aug.31 [cited 2025Jan.17];7(2).
60. Amrutha I D, Gandhi M G K, Mishra C P. 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;22(12):40-42.
61. Sonaiya S, Dosi R. Biochemical abnormalities in patients presenting with acute organo-phosphorus poisoning in a tertiary care hospital, and the prognostic significance. Int J Adv Med 2022;9:714-7.
62. Desai, Mohit & kumar, Brajendra. (2021). A study of hypokalemia in organophosphorus poisoning in dahod gujarat. Paripex indian journal of research. 37-38. 10.36106/paripex/6001242.
63. MR, K. (2020). Electrolyte derangements as a prognostic marker in acute organophosphorus poisoning. *Journal of Medical Science and Clinical Research*, *08*(11). https://doi.org/10.18535/jmscr/v8i11.79
64. Prasad DRMM. Reduced Levels of Serum Potassium and Plasma Cholinesterase in Acute Organophosphate Poisoning: Possible Predictive Markers. Asia Pac J Med Toxicol 2014;3:68-72.

65. 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;7(6):259-265.
66. Adinew GM, Asrie AB, Birru EM. Pattern of acute organophosphorus poisoning at University of Gondar Teaching Hospital, Northwest Ethiopia. BMC Res Notes. 2017;10(1):149.
67. Chaudhary SC, Singh K, Sawlani KK, Jain N, Vaish AK, Atam V, et al. Prognostic significance of estimation of pseudocholinesterase activity and role of pralidoxime therapy in organophosphorous poisoning. Toxicol Int. 2013;20(3):214-217.
68. 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;10(12):UC18-UC20.
69. Panda S, Nanda R, Mangaraj M, Rathod PK, Mishra PK. Glycemic status in organophosphorus poisoning. J Nepal Health Res Counc. 2015;13(31):214-219.
70. Karki P, Ansari JA, Bhandary S, Koirala S. Cardiac and electrocardiographical manifestations of acute organophosphate poisoning. Singapore Med J. 2004;45(8):385-389.
71. Nigam M, Jain AK, Dubey BP, Sharma VK. Trends of organophosphorus poisoning in Bhopal region: An autopsy based study. J Indian Acad Forensic Med. 2004;26(2):62-65.
72. 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;58(1):11-17.

73. Yurumez Y, Durukan P, Yavuz Y, Ikizceli I, Avsarogullari L, Ozkan S, et al. Acute organophosphate poisoning in university hospital emergency room patients. Intern Med. 2007;46(13):965-969.
74. Kang EJ, Seok SJ, Lee KH, Gil HW, Yang JO, Lee EY, et al. Factors for determining survival in acute organophosphate poisoning. Korean J Intern Med. 2009;24(4):362-367.
75. Eddleston M, Eyer P, Worek F, Mohamed F, Senarathna L, von Meyer L, et al. Differences between organophosphorus insecticides in human self-poisoning: A prospective cohort study. Lancet. 2005;366(9495):1452-1459.
76. Banerjee I, Tripathi SK, Roy AS. Clinico-epidemiological characteristics of patients presenting with organophosphorus poisoning. N Am J Med Sci. 2012;4(3):147-150.
77. Muley A, Shah C, Lakhani J, Bapna M, Mehta J. To identify morbidity and mortality predictors in acute organophosphate poisoning. Indian J Crit Care Med. 2014;18(5):297-300.
78. Akdur O, Durukan P, Ozkan S, Avsarogullari L, Vardar A, Kavalci C, et al. Poisoning severity score, Glasgow coma scale, corrected QT interval in acute organophosphate poisoning. Hum Exp Toxicol. 2010;29(5):419-425.
79. Davies JOJ, Eddleston M, Buckley NA. Predicting outcome in acute organophosphorus poisoning with a poison severity score or the Glasgow coma scale. QJM. 2008;101(5):371-379.

80. Hiremath P, Rangappa P, Jacob I, Rao K. Pseudocholinesterase as a predictor of mortality and morbidity in organophosphorus poisoning. Indian J Crit Care Med. 2016;20(10):601-604.
81. Sungur M, Güven M. Intensive care management of organophosphate insecticide poisoning. Crit Care. 2001;5(4):211-215.
82. Eddleston M, Mohamed F, Davies JOJ, Eyer P, Worek F, Sheriff MHR, et al. Respiratory failure in acute organophosphorus pesticide self-poisoning. QJM. 2006;99(8):513-522.
83. Peter JV, Jerobin J, Nair A, Bennett A, Samuel P, Chrispal A, et al. Clinical profile and outcome of patients hospitalized with dimethyl and diethyl organophosphate poisoning. Clin Toxicol. 2010;48(9):916-923.
84. Kang EJ, Seok SJ, Lee KH, Gil HW, Yang JO, Lee EY, et al. Factors for determining survival in acute organophosphate poisoning. Korean J Intern Med. 2009;24(4):362-367.
85. Bhattarai MD, Singh DL, Chalise BS, Koirala P. A case control study of hypokalemia in acute organophosphate compounds poisoning and its correlation with mortality and morbidity. JNMA J Nepal Med Assoc. 2018;56(210):637-642.
86. Yang CC, Deng JF. Intermediate syndrome following organophosphate insecticide poisoning. J Chin Med Assoc. 2007;70(11):467-472.
87. Shah SD, Rajiva DF, Apte SS, Nehete SP. Study of clinical profile of organophosphate compound poisoning patients and their outcome in relation to pseudocholinesterase levels at a tertiary care center. Med Pulse Int J Med. 2018;5(2):79-82.

88. Vijayakumar S, Fareedullah M, Ashok Kumar E, Mohan Rao K. A prospective study on electrocardiographic findings of patients with organophosphorus poisoning. Cardiovasc Toxicol. 2011;11(2):113-117.
89. Karki P, Hansdak SG, Koirala S, Shrestha A, Khanal K. A clinico-epidemiological study of organophosphorus poisoning at a rural-based teaching hospital in eastern Nepal. Trop Doct. 2001;31(1):32-34.
90. Nouira S, Abroug F, Elatrous S, Boujdaria R, Bouchoucha S. Prognostic value of serum cholinesterase in organophosphate poisoning. Chest. 1994;106(6):1811-1814.
91. 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(9553):2136-2141.
92. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet. 2008;371(9612):597-607.
93. Sumathi ME, Kumar SH, Shashidhar KN, Takkalaki N. Prognostic significance of various biochemical parameters in acute organophosphorus poisoning. Toxicol Int. 2014;21(2):167-171.
94. Abedin MJ, Sayeed AA, Basher A, Maude RJ, Hoque G, Faiz MA. Open-label randomized clinical trial of atropine bolus injection versus incremental boluses plus infusion for organophosphate poisoning in Bangladesh. J Med Toxicol. 2012;8(2):108-117.
95. Senanayake N, de Silva HJ, Karalliedde L. A scale to assess severity in organophosphorus intoxication: POP scale. Hum Exp Toxicol. 1993;12(4):297-299.

96. Jayawardane P, Dawson AH, Weerasinghe V, Karalliedde L, Buckley NA, Senanayake N. The spectrum of intermediate syndrome following acute organophosphate poisoning: A prospective cohort study from Sri Lanka. PLoS Med. 2008;5(7):e147.
97. Panda S, Nanda R, Mangaraj M, Rathod PK, Mishra PK. Glycemic status in organophosphorus poisoning. J Nepal Health Res Counc. 2015;13(31):214-219.
98. Eddleston M, Gunnell D, Karunaratne A, de Silva D, Sheriff MH, Buckley NA. Epidemiology of intentional self-poisoning in rural Sri Lanka. Br J Psychiatry. 2005;187:583-584.
99. Karki P, Ansari JA, Bhandary S, Koirala S. Cardiac and electrocardiographical manifestations of acute organophosphate poisoning. Singapore Med J. 2004;45(8):385-389.
100. Liu JH, Chou CY, Liu YL, Liao PY, Lin PW, Lin HH, et al. Acid-base interpretation can be the predictor of outcome among patients with acute organophosphate poisoning before hospitalization. Am J Emerg Med. 2008;26(1):24-30.
101. Eddleston M, Eyer P, Worek F, Juszczak E, Alder N, Mohamed F, et al. Pralidoxime in acute organophosphorus insecticide poisoning—a randomised controlled trial. PLoS Med. 2009;6(6):e1000104.
102. Senanayake N, Karalliedde L. Neurotoxic effects of organophosphorus insecticides. An intermediate syndrome. N Engl J Med. 1987;316(13):761-763.
103. Worek F, Koller M, Thiermann H, Szinicz L. Diagnostic aspects of organophosphate poisoning. Toxicology. 2005;214(3):182-189.

ANNEXURE I

Azadi Ka Amrit Mahotsav

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 905/2023-24 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "CORRELATION OF SERUM POTASSIUM AND PLASMA CHOLINESTERASE IN ASSESSING THE SEVERITY OF ACUTE ORGANOPHOSPHATE POISONING".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.SONTINENI ESWARASAI

NAME OF THE GUIDE: DR.PRAKASH G. MANTUR, PROFESSOR, DEPT. OF MEDICINE.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103, Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE II

CONSENT FORM

BLDEDU'S SHRI B. M. PATIL MEDICAL COLLEGEHOSPITAL

AND RESEARCH CENTRE, VIJAYAPUR- 586103

TITLE OF THE PROJECT - **"CORRELATION OF SERUM POTASSIUM AND PLASMA CHOLINESTERASE IN ASSESSING THE SEVERITY OF ACUTE ORGANOPHOSPHATE POISONING"**

PRINCIPAL INVESTIGATOR - **Dr. ESWARA SAI SONTINENI**

+91 9491194368

P.G. GUIDE NAME - Dr. PRAKASH.G.MANTUR

PROFFESSOR

DEPARTMENT OF MEDICINE.

All aspects of this consent form are explained to the patient in thelanguage understood by him/her.

INFORMED PART PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a freechoice of participation in this study.

PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. Ihave been asked to undergo the necessary investigations and treatment, which will help the investigator inthis study.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the

procedure of this study is not expected toexaggerate these feelings that are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in this study will help to patient's survival and better outcome.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records andwill be subject to the confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by code number. The code-key connecting name to numbers will be kept in a separate location.

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

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. **Dr. ESWARA SAI SONTINENI** is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me.A copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consentand discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr. ESWARA SAI SONTINENI may terminate my participation in the study after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

INJURY STATEMENT:

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

I have explained to the purpose of the research, the procedures required and the possible risks and benefitsto the best of my ability in patient's own language.

_____ _____

Dr. ESWARA SAI SONTINENI Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that **Dr. ESWARA SAI SONTINENI** has explained to me the purpose of research, the study procedures that I willundergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and I understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

_____ _____

Participant / Guardian Date

_____ _____

Witness to signature Date

ANNEXURE III

OP POISONING CASE PROFORMA

NAME:

AGE/SEX:

OCCUPATION:

ADDRESS:

RELIGION:

DATE OF ADMISSION:

IPNO:

DATE OF DISCHARGE:

CASE NO.:

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

PAST HISTORY:

FAMILYHISTORY:

PERSONALHISTORY:

1. DIET:
2. APPETITE:
3. SLEEP:
4. BOWEL/BLADDER HABITS:
5. HABITS:

GENERALPHYSICALEXAMINATION:

- LEVELOFCONSCIOUSNESS-

CONSCIOUS	
ORIENTED	
DROWSY	
STUPOR	
COMATOSE	

- PUPILSIZE - mm
- FASCICULATION – YES/NO
- PALLOR - YES/NO
- ICTERUS - YES/NO
- CLUBBING AND CYANOSIS - YES/NO
- LYMPHADENOPATHY - YES/NO
- EDEMA - YES/NO

VITALS:

PULSERATE-

BLOOD PRESSURE –

SPO2 –

TEMPERATURE –

RESPIRATORYRATE-

SYSTEMICEXAMINATION:

1. PERABDOMEN :
2. CARDIOVASCULARSYSTEM :
3. RESPIRATORY SYSTEM:
4. CENTRAL NERVOUS SYSTEM:

- FASCICULATION - YES / NO
- PUPILSIZE - mm

INVESTIGATIONS: 1. COMPLETE BLOOD COUNT:

TOTALCOUNT	CELLS/CMM
HAEMOGLOBIN	GM/DL
PLATELETCOUNT	LAKHS/CMM
NEUTROPHLIS	%
LYMPHOCYTES	%
MONOCYTES	%
BASOPHILS	%
RBC	LAKHS/CMM
ESR	MM/HR

2.RANDOM BLOOD SUGAR - mg/dl

3.SERUMCHOLINESTERASE - U/ Ml

4.LIVERFUNCTIONTEST :

TOTALBILIRUBIN	MG/DL
DIRECTBILIRUBIN	MG/DL
INDIRECTBILIRUBIN	MG/DL
ALBUMIN	MG/DL
SGOT	UNITS/Lt
SGPT	UNITS/Lt
ALP	UNITS/Lt

RENALFUNCTIONTEST:

CREATININE	MG/DL
UREA	MG/DL
SODIUM	MEQ/Lt
POTASSIUM	MEQ/Lt

6 . ARTERIAL BLOOD GROUP:

BLOODPH	
PO2	mmHg
PCO2	mmHg
HCO3	mmol/L
LACTATE	mmol/L

7 . ELECTRO CARDIO GRAPHY:

Standardisation :

Rate :

Rhythm :

PWave :

PRInterval :

QRSComplex :

STSegment :

TWave :

Axis :

8 .**POISON DETECTION CENTRE REPORT:**

Final diagnosis:

Dr. PRAKASH.G. MANTUR

ANNEXURE IV

MASTER CHART

CLINICAL PRESENTATION																							
sl.no	**age**	**gender**	**time since exposure (hrs)**	**serum K initial (mEq/L)**	**S.K+ FOLLOW UP**	**Plasma ChE initial**	**Plasma ChE 24 hrs**	**vomiting**	**abd pain**	**diarrohea**	**salivation**	**lacrimation**	**bronchospasm**	**muscle weakness**	**convulsions**	**GCS score**	**RR**	**HR**	**pupil size (mm)**	**mechanical ventilation**	**length of ICU stay (days)**	**outcome**	**NAME**	**IP NO**	**Severity**
1	27	M	51/2 hours	3.5	2.6,2.6,2.4	<200	480.2	+	-	-	-	-	+	+	-	3	18	92	2 mm	+	6	DEAD	laxman waddar	160654	+
2	19	F	11/2 hours	3.7	3.7	4382	5635.7	+	-	-	-	-	-	-	-	15	18	98	3 mm	-	2	discharged	roopa shrishail banikol	169791	-
3	65	M	3hours	3.5	3.6,3.8	206.8	308.8	-	-	+	-	-	-	-	-	14	18	78	3-4 mm	-	5	AMA	ishwar pandu bandagar	217010	+
4	30	F	31/2hours	3.9	3.9	5145.4	5368.4	+	-	-	-	-	-	-	-	15	16	78	2-3 mm	-	3	discharged	savithri prashant biradar	216953	-
5	22	F	unknown	3.7	3.3,3.4,3.8	287.7	1793.7	+	-	-	-	-	-	+	-	15	16	76	2 mm	-	6	discharged	ashwini irayya mathapati	222088	-
6	26	F	>24 hours	3.9	3.9	367.6	-	+	-	-	-	-	+	-	-	13	20	144	1 -2 mm	+	2	dead	siddappa parasappa talawar	234925	+
7	16	M	7 hours	4.9	4.4,3.9	<200	436.8	+	+	-	-	-	-	-	-	15	16	120	3-4mm	-	6	AMA	rukmini danasingh pujari	245066	-
8	28	F	4 hours	4	3.8	4570.4	7329.2	+	+	-	-	-	-	-	-	15	16	74	2-3mm	-	4	discharged	shanubhai yuvaraj naik	247449	-
9	23	F	11/2 hours	3.2	3.7,3.4	<200	260.8	-	-	-	+	-	-	+	-	15	18	120	2 mm	-	5	discharged	geeta nana jadhav	250421	+
10	40	F	30 mins	3.9	3.5,3.3	5943	4444.3	-	-	-	+	-	-	-	-	11	16	88	2-3mm	-	7	discharged	mahadevi parushuram rathod	250918	-
11	60	M	11/2 hours	3.5	3.6,3.3	269.1	-	+	-	+	-	-	-	+	-	15	16	68	3-4 mm	-	4	AMA	vachu meghu rathod	252360	-
12	26	M	8 hours	4.6	4.5	4223	4171.4	+	-	+	-	-	+	-	-	3	36	140	1mm	+	1	DEAD	prahalad shivappa kattimani	255424	+
13	29	M	31/2hours	3.3	3.2,3.6	7898.2	6741	+	-	-	-	-	-	+	-	12	19	84	2-3mm	+	4	DEAD	manjunath siddaray kadimani	259855	+
14	24	M	2 hours	4.7	4.0,3.9	255.1	253.2	-	-	+	-	-	-	-	-	15	20	96	2mm	-	4	discharged	parashuram raju kattimani	259948	-
15	21	F	4hours	4.1	-	5888.4	5678.9	+	-	-	-	-	-	-	-	15	18	98	4mm	-	2	AMA	roopa tiretappa hirekurabar	260663	-
16	18	M	1hour	4	4.3,4.2	351.3	311.3	+	-	-	-	-	-	-	-	15	18	96	3-4mm	-	4	AMA	mallikarjun c mathapati	261749	-
17	22	F	21/2 hours	4.2	-	3727.6	-	-	-	-	+	-	-	-	-	15	16	98	3mm	-	1	discharged	aisha davalasab kalegar	261648	-
18	17	M	3 hours	4	3.9	236.4	271.9	+	-	-	-	-	-	-	-	15	16	102	3mm	-	5	discharged	sachin b naykodi	265336	-
19	38	M	30 mins	4.1	-	<200	350.1	-	-	-	-	-	-	-	-	15	18	96	2mm	-	4	discharged	paramanand basappa hadapad	278468	-

20	26	F	2hrs	4.6	3.5	4006.6	2733	+	-	-	-	-	-	-	-	15	16	112	3mm	-	4	discharged	kiratiashok bistagond	285163	-
21	22	M	31/2 hours	3.8	3.6,3.5	<200	837.4	+	-	-	-	-	+	+	-	13	18	72	1-2 mm	-	4	discharged	basavaraj malkappa ilajeri	292982	-
22	30	M	3 hours	3.4	3.0,3.3,3.3	2195.5	1283.2	+	-	-	-	-	-	+	-	15	24	112	2mm	-	5	discharged	muttappa ashok biradar	299267	+
23	23	F	5hours	3.2	3.2,3.4,2.9	<200	332.6	+	-	-	-	-	+	+	+	4	36	108	1mm	+	7	AMA	savithri jagadevappa biradar	310434	+
24	45	M	10 hours	2.8	-	593.9	382.4	+	-	-	+	-	+	+	-	9	30	118	2mm	+	2	dead	yallappa l madar	311540	+
25	35	F	30 mins	2.8	3.8	2221.2	-	-	-	-	-	-	-	+	-	15	16	120	3mm	-	3	discharged	manjula mallikarjun babaleshwar	333652	+
26	80	M	4hours	4.3	-	7911.4	-	+	-		-	-	-	-	-	15	16	88	3mm	-	2	discharged	mallikarjun kallappa kudari	334976	-
27	25	M	51/2 hours	4.9	4.6	7569.4	-	-	+	-	-	-	-	-	-	15	16	88	3mm	-	3	discharged	mallappa s hittanalli	347372	-
28	65	M	51/2 hours	3.2	2.3	<200	<200	+	-	+	-	-	+	+	-	7	22	102	2	+	3	AMA	kallappa vittal naikodi	347905	+
29	24	M	7 hours	3.4	3.3,3.3,3.4	1031.5	1497.5	+	-	-	-	-	+	+	-	15	22	98	1mm	-	8	discharged	saibanna jateppa kambar	348268	+
30	80	F	21/2 hours	4.6	4	458.7	-	-	-	-	+	-	+	-	-	7	24	120	1mm	+	2	dead	rakmaji laxman lokhande	349160	+
31	35	M	7hours	4.3	4.2	<200	<200	-	-	-	-	-	-	-	-	15	20	70	2mm	-	3	discharged	mallappa lagamappa naikodi	350411	-
32	25	M	5 hours	4.3	3.7,3.6	<200	<200	+	-	+	-	-	+	-	-	3	36	106	1-2mm	+	14	discharged	akshay kumar shivaray dalawai	369470	+
33	17	M	11 hours	3.7	3.9	2762.2	5465	+	-	-	-	-	+	+	-	11	36	96	1-2 mm	-	4	discharged	basavantrayagouda s patil	379227	-
34	28	M	30 mins	3.7	3.8,3.6	7898.2	10356.8	+	-	-	-	-	-	-	+	12	18	88	3mm	-	5	discharged	Raghavendra sadashiva balochi	388252	+
35	29	F	6hours	4.6	-	7248	8778.8	+	-	-	-	-	-	-	-	15	18	114	3-4mm	-	5	discharged	kavitha rajendra bagali	390778	-
36	18	F	41/2 hours	3.6	4.3	990.9	-	-	-	+	+	-	-	-	-	15	20	96	2mm	-	5	discharged	megha hanamanth mashyalkar	3874	-
37	25	F	5 hours	3.5	3.8,3.6	<200	1333.8	+	-	+	+	-	-	+	+	11	18	126	1mm	-	8	discharged	roopa shivarudra shivanagi	6535	+
38	25	F	5 hours	3.4	3.5,3.6,3.6,3.7	975.4	3963.1	+	-	-	-	-	+	+	-	3	18	100	1mm	+	9	discharged	karishma irappa yaranal	9106	+
39	28	F	31/2 hours	4.1	-	962.3	2356.2	-	+	-	-	-	-	-	-	15	18	102	3mm	-	6	discharged	reshma dastagiri dhaded	15013	-
40	24	M	10 hours	3.4	3.5,4.1	<200	<200	+	-	-	-	-	-	-	-	15	16	60	2-3mm	-	6	AMA	haleppa husenappa kolinal	20208	-
41	65	F	31/2 hours	3	3.6,3.7,3.8	1453.3	-	-	-	-	+	-	+	+	-	3	39	42	1mm	+	5	AMA	chandubaisomalu chavan	21493	+
42	22	M	4 hours	3.3	3.6,3.8,4.0	<200	<200	-	-	+	+	-	+	+	+	10	28	132	1mm	+	18	discharged	irappa g belavaddagi	34785	+
43	30	M	unknown	3.8	3.0,3.6,3.6	6234.3	6591.8	-	+	-	-	-	-	-	-	3	28	58	1-2mm	-	8	discharged	anand rajshekar pujari	33745	-
44	19	F	4hours	5.1	-	4045	4000.6	+	-	-	-	-	-	-	-	15	18	102	2-3mm	-	4	discharged	ashwini channu chavan	49032	-
45	21	F	2 hours	4.2	-	6584.6	5748.7	+	-	-	-	-	-	-	-	15	18	120	4mm	-	4	discharged	bhagyashree shivaraj alamatti	55284	-
46	16	F	unknown	3.9	-	7112.2	-	+	-	-	-	-	-	-	-	15	18	120	3mm	-	1	AMA	archana basavaraj hosamani	57293	-
47	26	M	17hours	3.4	3.9,3.5	1064	-	+	-	-	-	-	+	+	-	15	36	136	1mm	-	5	discharged	kashinath laxman betagoudar	100240	-

48	34	F	3 hours	3.4	3.5,3.4	4110.5	-	+	-	-	-	-	-	+	-	15	20	62	1-2 mm	-	5	discharged	deepa mallikarjun dolli	112229	-
49	38	F	4 hours	4.1	-	4267.7	-	+	-	-	-	-	-	-	-	15	18	120	2mm	-	2	AMA	ambika mahantesh chavan	11223	-
50	18	F	2 hours	3.3	3.4	7781.3	-	+	-	+	-	-	-	+	-	15	18	120	2mm	-	5	discharged	tanuja karan logavi	120577	+
51	20	M	6 hours	4.3	4.2	4456.5	-	-	-	-	+	-	+	-	-	10	28	130	2-3mm	-	3	discharged	bharat kumar p meti	138722	-
52	25	F	2hours	4.2	3.8	359	<200	+	-	-	-	-	-	-	-	15	18	90	2-3 mm	-	6	discharged	savithri revansidda managuli	140405	-
53	28	M	3 hours	2.8	3.4	1961	-	+	-	-	-	-	+	+	-	7	28	102	1mm	+	2	dead	girish shankareppa masuti	143084	+
54	19	F	11/2 hours	3.6	3.7,3.8	5176	6309	-	-	-	-	-	-	-	-	15	16	96	2-3mm	-	5	discharged	sanjana suresh rathod	224626	-
55	52	M	12 hours	5.1	3.9	1520	-	+	-	-	-	-	-	-	-	15	18	94	2-3mm	-	4	AMA	allisab md sab mulla	155117	-
56	35	M	3 hours	3.3	3.6,3.7	<200	<200	+	-	+	-	-	+	+	-	8	24	130	1mm	+	8	DEAD	shrikant dharma raj hasanapur	162460	+
57	25	F	6 hours	4	3.7	<200	<200	+	-	-	-	-	-	-	-	15	18	124	2-3 mm	-	6	discharged	renuka pujari	175303	-
58	32	M	6 hours	3.3	3.7,3.3,3.4,3.4,3.4	351	-	+	-	+	-	-	+	+	-	10	20	116	2mm	+	10	discharged	malingray m yaladagi	175292	+
59	24	M	91/2 hours	3.4	3.3,3.2,3.1,3.1	2827	4469	+	-	+	-	-	-	+	-	12	24	130	2mm	-	8	discharged	laxman guranna	189796	+
60	20	M	3 hours	3.9	4	6002	6198	+	+	-	-	-	-	-	-	15	20	86	3 mm	-	6	discharged	praveen anil rathod	230759	-
61	25	M	8 hours	4.4	3.9	5210	6277	+	-	-	-	-	-	-	-	15	16	98	2-3 mm	-	3	discharged	akshay bharat salunke	241312	-
62	29	M	4 hours	4.2	3.6	<200	<200	+	-	-	-	-	-	-	-	15	18	72	3mm	-	9	discharged	nana jadhav	258356	-
63	19	F	11/2 hours	3.8	3.2,3.3,3.7,3.6,3.8	<200	<200	+	+	-	-	-	-	-	-	15	16	130	1-2 mm	-	18	discharged	kaveri bhimaray pujari	269269	-
64	32	F	16 hours	3.9	-	1000	1054	+	-	-	-	-	-	-	-	15	18	86	3	-	6	discharged	mallamma suresh kenganal	272364	-
65	24	F	4 hours	3.5	-	1674	5115	+	-	+	-	-	-	-		15	18	84	2-3mm	-	5	discharged	deepa namadev shinge	276797	-
66	18	F	8 hours	4.8	-	4992	4265	+	+	-	-	-	-	-	-	15	18	98	3mm	-	5	discharged	vidya shree s mamadapur	291951	-
67	65	M	3 hours	3.4	3.2,3.4,3.2	<200	<200	+	-	-	-	-	+	+	-	3	34	66	1-2 mm	+	8	discharged	amasiddh dhondappa gheradi	1026	+
68	28	F	2 hours	3.4	3.3	6989	6066	-	-	-	-	-	-	-	-	15	18	116	2-3 mm	-	9	discharged	ashwini sambaji pawar	2048	-
69	26	M	5 hours	3.2	4.1,3.7,3.3	<200	<200	+	-	+	+	-	+	+	-	9	28	120	1mm	+	9	DEAD	arun gangayya hiremath	3216	+
70	35	M	4 hours	2.8	-	<200	<200	+	-	-	-	-	+	+	-	3	36	98	1mm	+	3	DEAD	sharanappa bhimanna agasar	3910	+
71	20	F	4 hours	3.9	4.6	2338	2872	+	-	-	+	-	-	-	-	15	24	106	2mm	-	9	discharged	preethi iranna hatti	5944	-
72	18	F	3 hours	2.8	3.6,3.0	<200	<200	+	-	-	+	-	+	+	-	3	24	86	1mm	+	5	DEAD	aishwarya anand badiger	10714	+
73	25	F	2 hours	3.4	3.3	<200	<200	-	-	-	+	-	+	+	-	7	24	80	1mm	+	7	DEAD	lakshmibai bhirappa jambagi	11346	+
74	21	F	2 hours	3.8	-	<200	-	+	-	+		-	-	-	-	15	18	102	2mm	-	6	discharged	supiya sadiq mulla	11339	-
75	40	M	7 hours	4.3	-	5955.3	-	+	-	-	-	-	-	-	-	13	20	70	3mm	-	5	discharged	ravi mannur rathod	11733	-

76	28	M	3 hours	4.3	-	6966	-	+	-	-	-	-	-	-	-	15	18	80	3mm	-	6	discharged	prakash mallappa methi	12147	-
77	24	M	6 hours	3.8	3.6	9299	-	+	-	-	+	-	-	-	-	15	18	78	3mm	-	7	discharged	ravi kumar galave	12312	-
78	32	F	7 hours	3.2	4.2	<200	<200	+	-	+	+	+	+	+	-	3	24	100	1mm	+	7	discharged	baby santhosh chavan	14661	+
79	18	F	41/2 hours	3.9	-	2195.7	-	+	-	-	-	-	-	-	-	15	16	102	3mm	-	5	discharged	sanika rathod	15511	-
80	20	F	11/2 hours	4.4	-	4099.1	7119.3	-	-	-	-	-	-	-	-	15	20	92	2-3mm	-	5	discharged	rohini yallappa bajantri	16801	-
81	24	M	5 hours	3.7	3.5	7221.4	7110	+	-	-	-	-	-	-	-	15	18	120	3mm	-	4	discharged	irfan alisab nagadev	17424	-
82	31	M	5 hours	3.8	3.1,3,3	6516.8	5869.2	+	-	-	-	-	-	+	-	15	18	82	2-3 mm	-	6	discharged	prakash shrikant ukkli	82353	-
83	30	M	12 hours	2.9	-	<200	-	-	-	+	-	-	-	+	+	7	32	130	1mm	+	1	DEAD	Arun ashok pilaranakar	152300	+