A STUDY OF CORRELATION OF SERUM CHOLINESTERASE LEVEL, CLINICAL SCORE BY PERADENIYA POISONING SCALE AT PRESENTATION AND SEVERITY OF ORGANOPOISONING

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Dr. M. Harshith Kumar

List of Abbreviations

- **ACh**: Acetylcholine
- **AChE**: Acetylcholinesterase
- **APACHE**: Acute Physiology and Chronic Health Evaluation
- **ARDS**: Adult Respiratory Distress Syndrome
- **BMI**: Body Mass Index
- **BuChE**: Butyrylcholinesterase (also called plasma cholinesterase or pseudocholinesterase)
- **CBC**: Complete Blood Count
- **CNS**: Central Nervous System
- **CPK**: Creatine Phosphokinase
- **CYP**: Cytochrome P450
- **CYP450**: Cytochrome P450
- **DAMA**: Discharge Against Medical Advice
- **DFP**: Diisopropyl Fluorophosphate
- **ECG**: Electrocardiogram
- **GCS**: Glasgow Coma Scale
- **ICU**: Intensive Care Unit
- **IV**: Intravenous
- **LD50**: Median Lethal Dose
- **NN/N2**: Central (Neuronal) Nicotinic Receptors
- **NM/N1**: Peripheral (Neuromuscular) Nicotinic Receptors
- **OP**: Organophosphorus

- **OPC**: Organophosphorus Compound
- **OMPA**: Octamethyl-pyrophosphoramide
- **PAM**: Pralidoxime
- **PChE**: Pseudocholinesterase
- **PNS**: Peripheral Nervous System
- **POP**: Peradeniya Organophosphorus Poisoning (scale)
- **PSS**: Poisoning Severity Score
- **RBC AChE**: Red Blood Cell Acetylcholinesterase
- **TEPP**: Tetraetylpyrophosphate
- **TEP**: Triethylphosphate
- **TESS**: Toxic Exposure Surveillance System
- **WHO**: World Health Organization
- **2-PAM**: Pralidoxime (2-pyridine aldoxime methyl chloride)

ABSTRACT

OBJECTIVE: To evaluate the correlation between serum cholinesterase levels, clinical severity assessed by the Peradeniya Organophosphorus Poisoning (POP) scale, and outcomes in patients with organophosphorus poisoning.

BACKGROUND: Organophosphorus (OP) compounds, widely used as pesticides in agricultural communities, are a major cause of poisoning in developing countries. Their toxicity arises from acetylcholinesterase inhibition, leading to acetylcholine accumulation at synapses and subsequent cholinergic crisis. While clinical scoring systems and laboratory markers aid severity assessment, their interrelation and prognostic utility remain understudied.

METHODS: This prospective study included 100 patients with organophosphorus poisoning. Demographic data, clinical features, serum cholinesterase levels, and POP scale scores are recorded at presentation. Patients are followed up during their hospital stay to document management parameters (intubation requirement, atropine dose, and duration of hospitalization) and final outcomes.

RESULTS:

- **Demographics:** Most patients were young adults (21–40 years; 66%), with a slight male predominance (53%).
- **Severity Distribution (POP scale):** Mild (67%), moderate (31%), and severe (2%).
- **Serum Cholinesterase Levels:** Significantly correlated with severity: mild (3211.4 ± 2631.8 U/L), moderate (2028.3 ± 2102.3 U/L), and severe (726.6 ± 886.2 U/L) ($p = 0.05$).
- **Management & Outcomes:**
    - Intubation required: 20% of patients.
    - Mean atropine dose: 109.8 ± 48.7 mg, escalating with severity ($p < 0.001$).

    - Mortality: 13% overall, increasing from mild (2.9%) to moderate (29.03%) and severe (100%).
- **Prognostic Indicators:** Both POP scale severity ($p < 0.001$) and cholinesterase levels ($p = 0.004$) strongly predicted outcomes.

CONCLUSION: Serum cholinesterase levels and POP scale scores exhibit significant correlation with clinical severity and outcomes in OP poisoning. Combined use of clinical and laboratory parameters enhances risk stratification, guiding tailored management to improve survival.

KEYWORDS: Organophosphorus poisoning; Acetylcholinesterase; Peradeniya Organophosphorus Poisoning scale; Cholinergic crisis; Pesticide poisoning; Clinical severity; Prognostic indicators

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INTRODUCTION

Organophosphorus (OP) poisoning remains a critical global public health issue, particularly in developing nations where these compounds are extensively used in agriculture. According to the World Health Organization (WHO), an estimated **3 million pesticide poisoning cases** occur annually, resulting in **~250,000 deaths**, with OP compounds accounting for a significant majority of these fatalities (WHO, 2023)[1]. In agrarian communities, OP poisoning—whether accidental or intentional—has become a leading cause of toxicological morbidity and mortality[2], demanding urgent attention.

PATHOPHYSIOLOGY AND CLINICAL SPECTRUM:

The toxicity of OP agents stems from their irreversible inhibition of **acetylcholinesterase (AChE)**, leading to excessive acetylcholine accumulation at synaptic junctions. This results in **cholinergic overstimulation**, manifesting as a wide-ranging clinical syndrome affecting the autonomic, somatic, central nervous system and multiple organ systems[3]. Presentations vary from mild symptoms (e.g., nausea, miosis) to life-threatening complications (e.g., respiratory failure, seizures), necessitating prompt severity assessment and intervention.

DIAGNOSTIC AND PROGNOSTIC TOOLS:

1. **Serum Cholinesterase (Butyrylcholinesterase) Levels**
    - A well-established biomarker for OP exposure, serum cholinesterase activity declines rapidly post-exposure, often **preceding clinical symptoms**.
    - While useful for early diagnosis, its **prognostic correlation**[4] with outcomes remains debated, with studies reporting variable predictive value.
2. **Peradeniya Organophosphorus Poisoning (POP) Scale**
    - A validated **clinical scoring system** assessing six parameters:

      Pupil size, respiratory rate, heart rate, fasciculations, consciousness level, and seizures[5].
    - Demonstrated utility in **risk stratification**, particularly in resource-limited settings lacking advanced diagnostics.

- Strongly correlates with outcomes, including **ventilatory need** and **mortality risk**[6].

RATIONALE FOR THE STUDY:

Despite the individual utility of serum cholinesterase and the POP scale, their **combined prognostic significance** remains underexplored. Clarifying this relationship could:

- Enhance **early risk stratification** and guide **treatment intensity** (e.g., atropine dosing, ventilator support)[7].
- Improve **resource allocation** in high-burden settings.
- Inform **public health strategies** to mitigate OP poisoning's socioeconomic impact, including long-term neurological sequelae among survivors.

STUDY OBJECTIVES:

This study investigates the **correlation** between:

1. **Serum cholinesterase levels** at admission.
2. **Clinical severity** via the POP scale.
3. **Patient outcomes** (mortality, intubation need, hospitalization duration).

By integrating biochemical and clinical metrics, we aim to refine prognostic models and optimize management protocols for OP poisoning[8].

Long term neurological sequelae have been reported in survivors, affecting their quality of life and productivity[9].

While both serum cholinesterase levels and clinical scoring systems have shown utility in assessment, their relative value and interrelationship need further investigation. Understanding these relationships could potentially lead to the development of more refined prognostic models, combining both biochemical and clinical parameters for optimal patient assessment.[10]

AIM & OBJECTIVES

Aim:

To study the severity of organophosphate poisoning by correlating with "serum cholinesterase level and clinical score by peradeniya poisoning scale" at presentation.

Objectives:

To measure the level of serum cholinesterase and clinical score by peradeniya poisoning scale.

REVIEW OF LITERATURE

ORGANOPHOSPHORUS (OP) COMPOUNDS

HISTORICAL ASPECTS

The French scientist Philippe de Clermont was credited by Swedish pharmacologist Bo Holmstedt in a frequently cited article with synthesising the first OP (tetraethylpyrophosphate—TEPP) in 1854.[11] However, other people have suggested that some OP's might have been created even earlier. "Triethylphosphate (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.[12] Despite being the first OP cholinesterase inhibitor, TEPP was synthesised by a number of other 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. The symptoms only go away after a few hours. 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".[13]

"Gerhard Schrader, a chemist of the I.G. Farbenindustrie in Germany, is regarded as the father of contemporary OP pesticide toxicity" despite all of these earlier attempts and achievements. 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.[14] 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 OP's concurrently with Schrader; they later patented dimefox and diisopropyl fluorophosphate (DFP). Some of the OP's that Schrader synthesised during that time proved to be highly harmful to mammals. The development of OP's 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.[15] 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 organochloride pesticides were phased out or outlawed in the 1970s, their use peaked. OP's 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 OP's extensively, mostly because to their low cost in comparison to more modern pesticides.[16]

"The mechanism of action of OP's, 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 OP's. 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".[17] In fact, as early as 1939, the mechanism of action of OP's 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 OP's. Irwin Wilson of Columbia University in New York demonstrated in 1951 that hydroxylamine may restart AChE that had been blocked by OP's. 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 more general term phosphylate/phosphylation may also be used to describe the interaction of OP's with B-esterases.) This positive development in the treatment of OP poisoning was somewhat counteracted by the discovery, also in the mid-1950s, that the ability of oximes to reactivate phosphorylated Since "ageing" (the non-enzymatic removal of an alkyl chain from the phosphate) would change the inhibited enzyme into a non-reactivatable version", AChE declined over time.[18]

Since natural compounds are the source of insecticides like pyrethroids and carbamates, natural OP's have also been discovered, albeit after synthetic OP's were created. After being separated from cultures of the soil microbe Streptomyces antibioticus, two OP's (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 OP's".[19]

CHEMISTRY AND METABOLISM OF OP'S

Figure 1 depicts the overall structure of OP's, 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 OP's are also known to exist.[20] While some OP's (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 OP's 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 OP's 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 non-existent".[21]

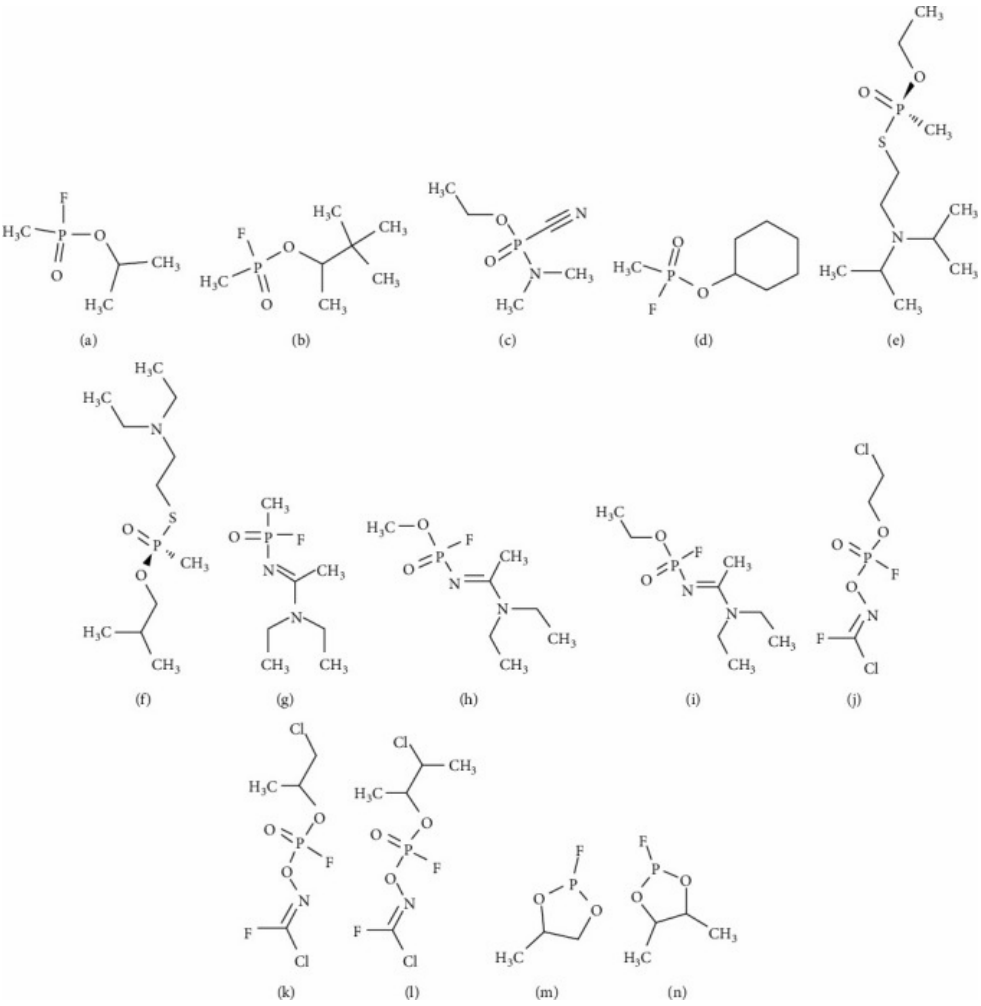
TYPES OF ORGANOPHOSPHORUS COMPOUNDS

Phosphoric acids and their derivatives are the source of organophosphorus compounds (OPC's), 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.[22] 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 OPC's are classified as phosphonothioates (S-substituted), and phosphonofluoridates include nerve poisons, also referred to as chemical warfare agents".[23]

These "nerve agents fall into four categories: (1) the German-developed G-series agents, which include cyclosarin (GF), sarin (GB), soman (GD), 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. Generally speaking, 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".[24]

Figure 1: "Chemical structure of G-series agents: (a) sarin, (b) soman, (c) tabun, and (d) cyclosarin; (e) V-series agent, VX; (f) VR (substance-33); chemical structures of A-series according to Dr. Mirzayanov: (g) A230, (h) A232, and (i) A234; chemical structures of A-series according Hoenig: (j) A230, (k) A232, and (l) A234; plausible and speculated chemical structures of Novichok: (m) Novichok-5 and (n) Novichok-7. For creating the chemical structures, ChemSketch software was used".



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 OPC's have two enantiomers, P(−) and P(+). Soman has four enantiomeric forms: C (+)P(+), C (+)P(−), C (−)P(+), and C (−)P(−)".[25] 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.[26]

Mechanism of Action

"Otto Loewi proved in 1920 that ACh functions as a chemical bridge that allows nerve impulses to travel between synapses".[27] Acetyl-coenzyme A (acetyl-CoA) is the source of the neurotransmitter sodium chloride (ACA). Choline acetyltransferase catalyses the production of acetyl-CoA from glucose and choline, which is then converted into the neurotransmitter acetylcholine (ACh). Upon stimulation, vesicles—packages of ACh held within presynaptic membranes—are released.

AChE effectively stops the neurotransmitter ACh's action on the muscarinic and nicotinic receptors by hydrolysing it into choline and acetate.[28] Organophosphates have the ability to permanently bind to AChE and stop ACh from breaking down. Muscarinic and nicotinic receptors, which are found throughout the body, are overstimulated as a result of this "liberation" of ACh.

Nicotinic Receptors

"Nicotinic receptors are of 2 types—central (neuronal) and peripheral (neuromuscular). Central nicotinic receptors, also known as NN or N2, are located in the central nervous system (CNS). They can also be found in the sympathetic and parasympathetic ganglia of the peripheral nervous system (PNS) and the adrenal medulla. Peripheral nicotinic receptors, or NM or N1, are located at the neuromuscular junctions. The N1 neuromuscular junction can cause fasciculation and muscular weakness, whereas the N2 autonomous nervous system is associated with hypertension and tachycardia".

Muscarinic Receptors

"All 5 subtypes of muscarinic receptors, M1 to M5, are distributed throughout the CNS. Postganglionic muscarinic receptors provide parasympathetic innervation to the heart, exocrine

glands, and smooth muscles of the internal organs. Sympathetic postganglionic fibers provide innervation to the sweat glands".[29]

"Stimulation of each specific receptor yields distinct clinical signs and symptoms, as mentioned below.[30]

- M2 receptors in the heart: Hypotension and bradycardia
- M2 and M3 receptors in the eyes: Miosis
- M2 and M3 receptors in the gastrointestinal system: Abdominal cramps, drooling, and salivation
- M2 and M3 receptors in the respiratory system: Bronchospasm, bronchorrhea, and rhinorrhea
- M2 and M3 receptors in the smooth muscles of internal organs: Abdominal cramps and urinary urgency
- M1 to M5 receptors in the CNS: Seizure, anxiety, and agitation."

OP POISONING

Epidemiology

Organophosphorus compound (OP) poisoning is a worldwide issue. According to estimates from the World Health Organisation, two million people are hospitalised for pesticide-related suicide attempts each year, and one million major unintentional poisonings happen annually.[31] A study from "1995 to 2004 found that the number of organophosphate exposure incidents peaked in 1997 with 20,135 cases and then decreased in subsequent years, according to the annual reports of the Toxic Exposure Surveillance System (TESS), which is kept up to date by the American Association of Poison Control Centres.[32] The National Poison Data System's 2020 annual report listed 2079 organophosphate exposure instances; no fatalities were reported.[33] The U.S. Environmental Protection Agency's decision to gradually phase out the use of

organophosphate pesticides in residential settings is largely responsible for this significant decrease in exposure to these chemicals. This project started in 2000 and ended in 2005".[32]

Accurately estimating the overall worldwide exposure rate of organophosphate and the associated toxicity is difficult. According to estimates, 371,594 people worldwide suffered from pesticide self-poisoning in 2007, which accounted for around one-third of all suicides that took place that year.[34] According to WHO estimates, there were about 20,000 fatalities and 1 million unintentional pesticide poisonings in 1990. According to a 2020 study, there were 740,000 unintended pesticide poisonings in 141 nations, which led to 7446 fatalities.[35] Due to insufficient reporting and a lack of statistical data, the true level of exposure and toxicity is probably higher.

INDIAN SCENARIO

India is primarily an agrarian nation, and farming there frequently involves the usage of pesticides. Suicidal poisoning with household agents (OP's, carbamates, pyrethrinoids, etc.) is the most frequent type of poisoning, according to statistics from the National Poison Information Centre India.[36] According to recent data from India's National Crime Bureau, in 2006 and 2007, 19.4% and 19.7% of all cases of suicidal poisoning were caused by pesticide intake.[37] 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.[39] 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.[40]

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.[41]

Etiology

Intentional self-harm exposure, accidental or occupational pesticide exposure, chemical warfare, and terrorist strikes can all result in organophosphate poisoning. More than fifty thousand chemicals have been created and tested for their ability to kill pests. There are 37 registered organophosphate insecticides in the United States, and they are all potentially hazardous. Because these substances are not as strictly regulated in the developing countries, this number is larger there. Exposure to organophosphates can happen by skin contact, ingestion, or inhalation. After ingestion and inhalation, these chemicals are easily absorbed by the body; however, systemic absorption after cutaneous exposure exhibits greater variability.

"The onset, severity, and duration of toxicity depend on the ingested amount, absorption route, and the specific pesticide's toxicokinetics. The World Health Organization (WHO) classifies these compounds into 5 groups, ranging from "Extremely hazardous" to "Active ingredients unlikely to present acute hazard in normal use." This classification is derived from the data based on the median lethal dose (LD_{50}), which represents the rat's oral lethal dose for 50% of individuals exposed to the active ingredient. However, the LD_{50} classification is limited in differentiating more toxic compounds within the same class".[42]

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 leucocytosis".[43–45]

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.[28]

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. It is still unclear, though, if the chemical or its desire is to blame for these problems.[46]

Toxicokinetics

The fastest absorption of organophosphate pesticides is through inhalation, although they can also be taken through eating, ocular contact, cutaneous exposure, and inhalation.[47] After cutaneous exposure, systemic absorption varies, but it can be accelerated by a number of conditions, including dermatitis, damaged skin, and high ambient temperatures. Both unintentional exposures in children and deliberate efforts at self-harm in adults are frequently linked to oral intake.

It is uncertain when the plasma concentration peaks following exposure to organophosphates. However, a research conducted on "human volunteers found that the time to

peak plasma concentrations was about 6 hours after relatively modest dosages of chlorpyrifos were taken orally.[48] Interestingly, these results might not hold true for other organophosphate substances, particularly when huge volumes are consumed, as occurs in deliberate efforts at self-harm. Additionally, the study used pure chlorpyrifos, which is different from agricultural pesticides and may have additives that affect the organophosphate's distribution and" absorption. In contrast to agricultural pesticides that might contain additives that could affect the organophosphate's absorption and distribution, this study also used pure chlorpyrifos.

The majority of organophosphates have a large volume of distribution and are lipophilic. They spread quickly into the liver, kidneys, and adipose tissue. They offer defence against metabolism due to their wide spread. The result following poisoning may be influenced by the patient's adipose tissue and degree of lipophilicity. A study conducted in Korea in 2014 looked at the results of 112 patients who had been acutely poisoned, 40 of whom were obese. Longer stays in the intensive care unit (ICU), longer duration of hospitalisation overall, and lengthier mechanical breathing were all encountered by patients with a body mass index (BMI) of greater than 25.[49]

Cholinergic crises can be brought on by the release of unmetabolized organophosphates from fat reserves. People with low lipophilicity and lower volumes of distribution usually do not exhibit this behaviour, which is linked to highly lipophilic substances. After absorption, organophosphates can directly block the AChE enzyme without requiring first metabolism. These direct-acting substances are known as oxons, and they are distinct from other substances termed thions, which become active only after the body's metabolism is activated.

Enzymes called cytochrome P450 (CYP450), which are mostly found in the liver and intestine, activate thion organophosphate molecules. Depending on the organophosphate's kind and quantity, different CYP450 enzymes may be involved.[50]

The enzyme AChE is rendered inactive when an organophosphate attaches to it and is cleaved, creating a stable but reversible bond. It may take hours or days to fully restore AChE function, and while a regeneration process might take place, it moves more slowly than the inhibition. "The ageing process, in which the original reversible link becomes irreversible and enzyme reproduction is no longer possible, may occur in an inactive state. various organophosphate compounds age at various rates. The antidote pralidoxime decreases the quantity of dormant enzymes available" for ageing and speeds up acetylcholine renewal. Pralidoxime only works prior to the ageing process, which is reliant on the particular organophosphate chemical and time-sensitive.[51] De novo synthesis is required for enzyme replenishment because AChE can no longer be regenerated after ageing.

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.[52]

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

"Several helpful mnemonics exist for recalling the signs and symptoms of organophosphate poisoning and the receptor responsible for them".

"To remember the nicotinic signs of AChE inhibitor toxicity, the following days of the week can be used:

- Monday = Mydriasis
- Tuesday = Tachycardia
- Wednesday = Weakness
- Thursday = Hypertension
- Friday = Fasciculations"

"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. Patients who survive acute poisoning may be at risk for additional long-term problems.

Evaluation

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)".[53]

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 scale that assesses "six common clinical features of OP poisoning, such as pupil size, heart rate, and level of consciousness. Each feature is scored on a scale of 0–2, with a score of 0–3 indicating

mild poisoning, 4–7 indicating moderate poisoning, and 8–11 indicating severe poisoning".

- **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.

Peradeniya Organophosphorus Poisoning (POP) Scale:[54]

The "POP Scale is a clinical scoring system developed to assess the severity of organophosphate poisoning quickly and easily. It was created by researchers at the University of Peradeniya in Sri Lanka, a region where OP poisoning is unfortunately common. The POP scale was developed in 1993 by N Senanayake, H J de Silva, and L Karalliedde. It assesses 14 common clinical manifestations of OP poisoning on a three-point scale. The scale is used to correlate the clinical score, serum cholinesterase level at presentation, and severity of poisoning. Here's a detailed breakdown of the scale":

1. "Pupil size:
    - 2mm: 0 points
    - <2mm: 1 point

    - Pinpoint: 2 points
2. Respiratory rate:
    - <20/min: 0 points
    - 20/min: 1 point
    - 20/min with central cyanosis: 2 points
3. Heart rate:
    - 60/min: 0 points
    - 41-60/min: 1 point
    - <40/min: 2 points
4. Fasciculation (involuntary muscle twitching):
    - None: 0 points
    - Present but not generalized or continuous: 1 point
    - Generalized and continuous: 2 points
5. Level of consciousness:
    - Conscious and rationale: 0 points
    - Impaired response to verbal commands: 1 point
    - No response to verbal commands: 2 points
6. Seizures:
    - Absent: 0 points
    - Present: 1 point

The total score ranges from 0 to 11. The severity is then classified as follows:

- 0-3 points: Mild poisoning
- 4-7 points: Moderate poisoning
- 8-11 points: Severe poisoning”

This scale is valuable because:

1. “It's quick and easy to use, requiring no complex equipment or lab tests.
2. It can be applied at the bedside or in emergency settings.
3. It helps in triaging patients and guiding initial management.
4. It has shown good correlation with clinical outcomes in several studies.

However, it's important to note that while the POP Scale is useful, it should be used in conjunction with other clinical assessments and not as the sole determinant of treatment decisions. Factors like the specific OP compound involved, time since exposure, and individual patient characteristics also play crucial roles in management”.

Comparison with other assessment methods:

1. Namba Scale:

- Simpler, classifying poisoning as latent, mild, moderate, or severe.
- Less quantitative than POP Scale.
- Doesn't account for specific symptoms like fasciculations or seizures.

2. Poisoning Severity Score (PSS):

- More general, applicable to various types of poisoning.
- Grades from 0 (no symptoms) to 4 (fatal).
- Less specific to OP poisoning mechanisms.

3. APACHE II Score:

- More comprehensive, used for various critical illnesses.
- Requires laboratory data, making it less suitable for rapid assessment.
- Not specific to OP poisoning.

4. Glasgow Coma Scale (GCS):

- Focuses solely on consciousness level.

- Often used in conjunction with other scales for OP poisoning.

Use in clinical practice:

1. Initial Assessment:
    - POP Scale is often used in emergency departments for quick triage.
    - Helps determine the need for ICU admission.
2. Treatment Guidance:
    - Assists in deciding initial atropine and oxime dosing.
    - Higher scores may indicate need for more aggressive treatment.
3. Prognosis:
    - Can help predict outcomes and length of hospital stay.
    - Useful for communicating severity to patients' families.
4. Monitoring:
    - Used to track patient progress over time.
    - Helps in deciding when to step down care.
5. Research:
    - Provides a standardized way to compare cases in clinical studies.
6. Resource Allocation:
    - In mass casualty scenarios, helps prioritize limited resources.

Advantages of POP Scale:

- Quick and easy to use
- No special equipment needed
- Good balance of simplicity and comprehensiveness

Limitations:

- Doesn't account for all possible OP poisoning symptoms

- May not reflect long-term complications
- Doesn't consider the specific OP compound involved

In practice, the POP Scale is often used in combination with other assessment tools, laboratory tests, and clinical judgment to provide comprehensive care for OP poisoning patients. It's particularly valuable in resource-limited settings or in the initial stages of assessment before more detailed information is available.

Figure 2: Peradeniya Organophosphorus Poisoning (POP) Scale

Pupil size	<2mm Pinpoint	1 2
Respiratory rate	<20 cpm ≥20 cpm ≥20 cpm with central cyanosis	0 1 2
Heart rate	>60 bpm 41-60 bpm <40 bpm	0 1 2
Fasciculations	None Present, generalised/continuous Both generalised and continuous	0 1 2
Level of consciousness	Conscious and rational Impaired response to verbal command No response to verbal command	0 1 2

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 butyrylcholinesterase 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. Otherwise, variations of just a few minutes in the cooling time of a sample would result in significant variation across multiple samples, making interpretation challenging.

"Drawbacks of cholinesterase activity assays

Plasma butyrylcholinesterase assays"

• "Inhibition of butyrylcholinesterase, also called plasma cholinesterase or pseudocholinesterase, does not give information about clinical severity of the poisoning. Many organophosphorus

pesticides are more potent inhibitors of butyrylcholinesterase than they are of acetylcholinesterase; butyrylcholinesterase inhibition might occur to a greater extent than acetylcholinesterase inhibition. Butyrylcholinesterase assays can be used to detect exposure to an organophosphorus or carbamate pesticide

• Butyrylcholinesterase is produced by the liver, and blood concentrations recover by about 7% of normal each day once the organophosphorus has been eliminated. Daily butyrylcholinesterase assays can be used to monitor when enzyme activity starts to rise again, since this recovery suggests that the organophosphorus has been eliminated.

• Variation between commercial assays can make comparisons between studies difficult. The concentration of butyrylthiocholine varies between assays. A high concentration substrate (eg, 7 mM vs 1 mM) will result in a 30% higher measured activity and a higher background.

• Measurement of butyrylthiocholine hydrolysis in the absence of plasma is needed to measure non-enzymatic hydrolysis and hence background values. Not all commercial assays provide such a control. The background amount of spontaneous butyrylthiocholine hydrolysis is affected by its concentration and pH, which both vary between assay kits.

• Temperature control is important, because butyrylcholinesterase activity increases by some 4% per 1°C increase in temperature"

"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 erythropoeisis 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 detected acetylcholinesterase activity will not accurately reflect the blood's activity at the moment of collection; assays will vary if samples are left for different times. 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 aged, an aliquot of blood should be incubated with a large amount of oxime (e.g., 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 may help determine when to cease oxime medication and enable a patient to be carefully weaned off of a ventilator when butyrylcholinesterase activity is rising.

Treatment / Management

To reduce the danger of self-contamination, all healthcare professionals must wear personal protective equipment prior to diagnosing and treating a patient with organophosphate toxicity. A low rate of contamination among healthcare professionals can be maintained by following universal measures. 56 Decontaminating the patients is the first step once healthcare personnel have been guaranteed their safety through the use of suitable protective measures. The patient's skin is then properly cleaned three times using soap and water. This method's main objective is to clean swiftly without using particular decontamination fluids. Organophosphates can be discovered in bodily secretions, such as diarrhoea and vomiting, thus care should be used when handling them. Patients should remove and discard all of their clothing. If shampooing doesn't work, long hair should be chopped because it can trap very lipophilic substances. Decontaminating the patient is necessary, but it shouldn't postpone prompt medical attention for a patient who is in serious pain. [57]

When treating individuals with organophosphate toxicity, airway control is crucial. Intubation may be necessary for certain individuals because of bronchospasm, convulsions, or bronchorrhea. It is important to remember that succinylcholine cannot be metabolised and causes persistent paralysis, hence it should be avoided during intubation. Pulse oximetry, cardiac monitoring, and intravenous access should also be provided to patients.

Because it competes with “acetylcholine at the muscarinic receptors, atropine is the main therapy for organophosphate toxicity. In order to get the adult dose, atropine is given intravenously (IV) in doses of 2 to 5 mg for adults and 0.05 mg/kg for children. The medical

staff should double the dosage every three to five minutes until respiratory secretions have cleared and there is no bronchoconstriction if the patient does not react to treatment. When the patient exhibits anticholinergic signs and symptoms, such as dry skin and mucosa, decreased bowel noises, tachycardia, no bronchospasm, reduced secretions, and mydriasis," the state of "atropinisation" is reached. [58]

Improving cardiorespiratory parameters in individuals with organophosphate toxicity is the primary goal of atropine use. Pupil size and skin wetness are less important than evaluating heart rate, blood pressure, and respiratory health. Hundreds of milligrams of atropine may need to be given to patients with severe poisoning over the period of several days to weeks, either as continuous infusion or as bolus doses, until the patient exhibits improvement. Since atropine does not lessen the effects of nicotine, patients need to be continuously watched for the emergence of respiratory failure and neuromuscular junction dysfunction. Tidal volume and negative inspiratory force are two metrics that can be monitored to help determine whether ventilatory support is required.

By attaching itself to the organophosphate, the antidote pralidoxime (2-PAM) reactivates the phosphorylated AChE. But in order for the antidote to be effective, it needs to be taken before ageing sets in, and the exact amount of time depends on the component. This substance can be used in combination with atropine and does not depress the respiratory centre. However, there is conflicting and inconclusive information about the use of oximes to treat organophosphate toxicity. According to studies, there were possible hazards and no improvement in mortality when 2-PAM was added to atropine. [59] "Therefore, it is recommended that all patients poisoned with organophosphorus chemicals be treated with an oxime until a better knowledge is obtained and other treatments are developed".

Patients must get atropine before to 2-PAM in order to avoid "muscarinic-mediated

symptoms getting worse. A bolus of at least 30 mg/kg for adults and 20 to 50 mg/kg for children over 30 minutes is advised by healthcare professionals. When giving 2-PAM to patients, care should be used because giving the drug too quickly can cause cardiac arrest. Following the bolus, the drug should be continuously infused for at least 8 mg/kg/h for adults and 10 to 20 mg/kg/h for children. This may need to be done for a few days". [60]

Benzodiazepines should be given to patients who are having seizures. Benzodiazepines are generally not advised unless seizures are actively occurring, despite a solitary study that suggests diazepam may be helpful in avoiding neuropathy. Extracorporeal elimination might be advantageous for certain organophosphate chemicals with a restricted volume of distribution, including dichlorvos and dimethoate. The overall efficacy of hemoperfusion and haemodialysis in all poisoning situations, however, is not well documented. "Patients should be admitted to the hospital and closely monitored in an intensive care unit (ICU) for at least 48 hours" due to the possibility of recurrent symptoms and respiratory problems. Patients may be eligible for discharge if they show no symptoms for 12 hours. [61]

Complications Related to Organophosphate Exposure[62]

Because they are connected to the impacted systems, the problems brought on by exposure to nerve gas or organophosphate pesticides are system-specific. Both nicotinic and muscarinic receptors are overstimulated, which leads to the clinical signs of these problems. Clinical manifestations of organophosphates are mostly seen in the "cardiovascular, renal, gastrointestinal, CNS, and respiratory systems".

Respiratory System

"In the respiratory system, exposure to organophosphate pesticides may lead to complications such as aspiration pneumonia resulting from excessive salivation, progressive

respiratory failure stemming from weakened respiratory muscles, notably the diaphragm, severe bronchospasm, and noncardiogenic pulmonary edema".

Cardiovascular System

In "the respiratory system, exposure to organophosphate pesticides may lead to complications such as arrhythmias, especially ventricular tachycardia, bradycardia, hypertension, hypotension, and prolonged QTc".

Central Nervous System

Complications from organophosphate pesticide exposure in the central nervous system include hallucinations, psychosis, seizures, and changes in mental status".

Gastrointestinal and Metabolic Systems

Pancreatitis, electrolyte imbalances from fluid and electrolyte losses from the gastrointestinal tract, hyperglycemia, and decreased bicarbonate levels are just a few of the gastrointestinal and metabolic issues that can arise from exposure to organophosphate pesticides".

Renal System

Acute kidney injury can result from exposure to organophosphate insecticides in the renal system. Acute kidney damage associated with exposure to organophosphate pesticides has been reported in a small number of case reports; this condition is usually treated conservatively or by hemoperfusion.

Prognosis

Organophosphate insecticides cause between 2% and 25% of deaths worldwide. The pesticides that are most commonly linked to "deaths are trichlorfon, dichlorvos, malathion, and fenitrothion. The most common cause of death is respiratory failure".

The Peradeniya Organophosphorus Poisoning (POP) scale was created and approved in 1993 in order to evaluate the clinical severity and predict the outcome of organophosphate

poisoning cases.[55] "This scale was created in India, a country with a high toxin prevalence and few resources for very ill people. Six clinical criteria are taken into account by the POP scale: respiration, bradycardia, seizures", fasciculations, degree of consciousness, and miosis.

Before administering any medication in cases of toxicity, medical professionals assess the aforementioned factors in their patients. More adverse outcomes, such as "death, the need for ventilatory support, and the amount of atropine used in the first 24 hours, are linked to higher scores. The scale, however, is unable to forecast the probability" of developing the intermediate condition. Organophosphate toxicity was divided into three groups in another study: mild (POP score 0–3), moderate (POP score 4–7), and severe (POP score 8–11). Of the patients in this category, 21.66% had moderate poisoning, 78.33% had mild poisoning, and none had severe poisoning. Notably, the mortality rate for patients in the moderate toxicity score category was 30.8%, and all of these patients needed ventilatory assistance.[63]

REVIEW OF RELATED ARTICLES

NB Malaviya and associates (2023)[63] "This study aimed to compare the early anticipated patient prognosis assessed by the POP scale at admission with the patient outcome in order to assess the prognostic utility of the clinical parameters of the POP scale in predicting the severity of organophosphorus chemical poisoning. Acute organophosphorus chemical poisoning that manifested at the emergency room was the subject of this prospective observational study. For the trial, 60 participants were enrolled. Sixty-five percent were men, and the majority were under 20. The most often eaten OP chemical was monocrotophos. Within two to six hours of intake, the majority of patients were taken to the hospital. The main presenting symptoms were vomiting and copious secretions. The vast majority of patients (47), were classified as mild POP. Not a single patient experienced severe toxicity. Eleven of the 60 patients passed away, while 49 of them recovered. Four patients (31%) with moderate POP scale scores and seven patients (15%) with mild scores passed away. In total, mechanical breathing was required for 100% of patients with moderate POP scale scores and 61.7% of patients with mild scores. For patients with acute exposure to OP compounds, the POP scale is a useful tool for assessing severity and determining prognosis. It could be a straightforward, low-cost instrument that aids in anticipating the requirement for ventilatory support upon entry. When resources are scarce, early detection of warning indicators may aid in lowering mortality and morbidity. In contrast to utilising the POP scale alone, we discovered that combining additional clinical characteristics and biochemical markers improves prognostication".

Serum cholinesterase levels, the severity of organophosphorus poisoning, and the Peradeniya clinical score upon presentation were all correlated, according to **Shafti SS et al. (2023)**[64]. In contrast to patients with high POP scores and low Sr, Pseudocholinesterase levels, they found that patients with low POP scores and high Sr, Pseudocholinesterase levels required

less ventilator support. Patients with low Sr and pseudocholinesterase and high POP scores are more likely to die. POP score, Sr, Pseudocholinesterase level, and the need for mechanical ventilation are all significantly correlated with the eventual outcome of patients with acute organophosphorus poisoning. These factors can also be utilised as prognostic markers to determine the severity of the poisoning.

A study on "organophosphorus chemical poisoning in a tertiary care hospital and the use of the Peradeniya Organophosphorus Poisoning scale as a predictive indicator of the outcome was carried out by **Kamath SD et al. (2021)**[65]. One hundred patients with OP compound poisoning who were hospitalised to Tata Main Hospital between June 2018 and May 2020 participated in this prospective study. The length of stay in the intensive care unit, complications, death, and the need for ventilator support were all positively connected with the Peradeniya organophosphorus poisoning scale upon admission. Thus, it can be applied to risk assessment and prognostication for OPC poisoning patients".

Gurung D. et al. (2021)[66] carried out a prospective, single-centre study. The majority of the patients were female and ranged in age from 15 to 45. Miosis followed by altered sensorium was the most reliable clinical result. Methyl parathion was the most often consumed organophosphate. "Serum cholinesterase level and POP scale severity did not significantly correlate. They came to the conclusion that there is no correlation between the severity of the POP scale and that of the cholinesterase level. Because prior studies have produced contradictory outcomes, a multi-center trial with a bigger sample size is required".

Studies conducted in **2019** on 75 patients by **Chaudhary R et al. and Rehiman S et al.** showed a higher Peradeniya Score ($P < 0.05$) and a considerably greater total dose of atropine. In a study involving 75 patients, Chaudary et al. found a strong association between the serum acetylcholinesterase level and the Peradeniya poisoning scale. Higher POP scores were

associated with longer hospital stays, atropine need, PAM, and a higher degree of serum acetylcholinesterase derangement. As a result, it improves the forecast of results for patients who have acute organophosphate poisoning at index visits.[67]

A prospective investigation of 100 patients with OP poisoning was carried out in a hospital by **Honnakatti V et al. (2018)**[68]. "They came to the conclusion that there was a substantial relationship between the degree of poisoning at the time of initial presentation and the degree of derangement in the serum cholinesterase level. The degree of dysregulation in the serum cholinesterase level increased with a higher POP scale score".

Vernekar PV et al. (2017)[69]This study aims to evaluate the Peradeniya organophosphorus poisoning (POP) scale, a suggested grading system based on clinical criteria, on Indian patients in a tertiary care setting. Fifty patients with varying degrees of OP poisoning who were admitted to a tertiary care facility were included in the study after the inclusion and exclusion criteria were applied. Following admission, the patients were intensively monitored to track their morbidity and mortality rates using the Peradeniya scoring system. A significant component of the study population was younger (less than 30 years old), with males making up the majority (66%) compared to females (34%). Only 6% of patients were in the severe poisoning category upon admission, whereas the majority (50%) and 44% of patients were in the light or moderate poisoning groups. The moderate and mild groups gradually experienced a decrease in the severe group's incidence of respiratory failure (100%) and death (66%), which resulted in a longer length of stay in the intensive care unit. As a predictor of respiratory failure, length of intensive care unit stays, and mortality, the Peradeniya score for the severity of poisoning was found to have a strong link with outcome.

Dubey, T. N. et al. (2016)[70] In order to determine the severity of organophosphorus chemical poisoning as determined by Peradeniya score, ventilator demand, and death, this study

estimated the serum amylase and CPK levels at the time of admission. They came to the conclusion that the severity of poisoning and the Peradeniya poisoning scale were positively correlated.

Patients who had pinpoint pupils and higher fasciculation ratings were significantly more likely to require ventilatory support ($P < 0.001$), according to **Rajeev H et al.'s (2013)**[71] study. He has determined that the need for assisted ventilation in cases of OP poisoning was strongly predicted by clinical and biochemical parameters, including a longer time lag between consuming OP poison and receiving specific treatment, a lower GCS score, generalized fasciculations, low pseudocholinesterase levels, and a larger initial dose of atropine needed for atropinisation.

S., Rehiman and associates (2008)[72] **"**The study's objective was to establish a correlation between the severity of poisoning, the serum cholinesterase level upon presentation, and the clinical score as defined by the Peradeniya Organophosphorus Poisoning (POP) scale. Fifty patients met the requirements for inclusion. There was a direct correlation between the level of serum cholinesterase and the degree of poisoning ($P<0.001$). There were significant differences ($P<0.05$) in the average length of hospital stay, the total amount of atropine required to treat, and the mean requirements of atropine on the first day of admission. Although only 4% of patients had severe poisoning and 26% of patients had moderate poisoning, 14% of patients died overall, suggesting that even patients with moderate poisoning had passed away. The dysregulation in serum cholinesterase levels at initial presentation and the severity of the POP scale did not correspond with death. This could be because of other co-morbidities or because the atropine infusion was accidentally stopped, especially at night in the wards. Serum cholinesterase at presentation and the POP scale seemed to be helpful in determining the severity

of poisoning, especially when considering higher atropine levels and longer hospital stays.

Patients who exhibit signs of moderate to severe poisoning require careful observation".

MATERIAL AND METHODS

STUDY DESIGN AND SETTING

- **Type**: Hospital-based cross-sectional observational study.
- **Location**: Department of General Medicine, Shri B.M. Patil Medical College and Research Centre, Vijayapura, Karnataka, India.
- **Duration**: May 2023 to March 2025.

STUDY TIMELINE

Phase	Time Allocation	Duration
Problem identification, questionnaire preparation	5–10%	May 2023
Pilot study, data collection	80%	June 2023 – September 2025
Data analysis & interpretation	5–10%	October 2024 – January 2025
Dissertation write-up & submission	5–10%	February 2025 – March 2025

SAMPLE SIZE CALCULATION

- **Basis**: Anticipated correlation coefficient (r = 0.474) between POP scale and atropine dose.
- **Parameters**:
    - 95% confidence level (Zα = 1.96), 99% power (Zβ = 3.09).
    - Formula:

      $N=(CZ\alpha+Z\beta)2+3$where$C=0.5\ln(1-r1+r)=0.5152$

- **Result**: **100 participants** enrolled.

INCLUSION CRITERIA

1. Age ≥18 years with:

- History of OP exposure (self/attendant-reported or container verification).
- Clinical features of OP poisoning (e.g., salivation, diarrhea, fasciculations, miosis).

2. Characteristic odor of OP in gastric aspirate/vomitus.

EXCLUSION CRITERIA

1. Pre-existing major systemic illness (e.g., cardiac, renal, hepatic failure).
2. Pre-hospital atropine administration.
3. Co-ingestion of alcohol or other toxins.

Methodology

CLINICAL ASSESSMENT

- **POP Scale Scoring**: Evaluated on admission using 6 parameters:

Parameter	Criteria	Score
Pupil size	>2 mm / <2 mm / Pinpoint	0–2
Respiratory rate	<20/min / >20/min / >20/min + cyanosis	0–2
Heart rate	>60/min / 41–60/min / <40/min	0–2
Fasciculations	Absent / Present / Generalized	0–2
Consciousness	Normal / Impaired / Unresponsive	0–2
Seizures	Absent / Present	0–1

-

- **Severity Grading**:
    - **Mild**: 0–3
    - **Moderate**: 4–7
    - **Severe**: 8–11

LABORATORY INVESTIGATIONS

- **Routine Tests**: CBC, urine analysis, RBS, renal/liver function tests, electrolytes.

- **Specialized Tests**:
    - **Serum cholinesterase** (key biomarker).
    - **Toxicology screening** of gastric aspirate.

ADDITIONAL DIAGNOSTICS

- ECG (cardiac monitoring).
- Chest X-ray (aspiration pneumonia evaluation).

PATIENT MONITORING & OUTCOMES

- Tracked until discharge/death:
    - Complications, interventions (e.g., intubation).
    - Atropine dose, hospital stay duration.

STATISTICAL ANALYSIS

- **Software**: SPSS v21.
- **Quantitative Data**: Mean ± SD, median, range.
- **Qualitative Data**: Frequencies, percentages.
- **Tests**: Student's t-test ($p < 0.05$ significant).

-

RESULTS

This hospital-based cross-sectional study evaluated 100 patients with organophosphorus (OP) poisoning at Shri B.M. Patil Medical College, correlating **serum cholinesterase levels** with **Peradeniya Poisoning Scale (PPS)** scores to assess severity and outcomes.

Table 1: Distribution of patients according to age

Age (in years)	Frequency	Percentage
15-20	25	25%
21-40	66	66%
41-60	3	3%
61-80	6	6%

Key Finding: Most patients were young adults (21–40 years; 66%).

Graph 1: Distribution of patients according to age

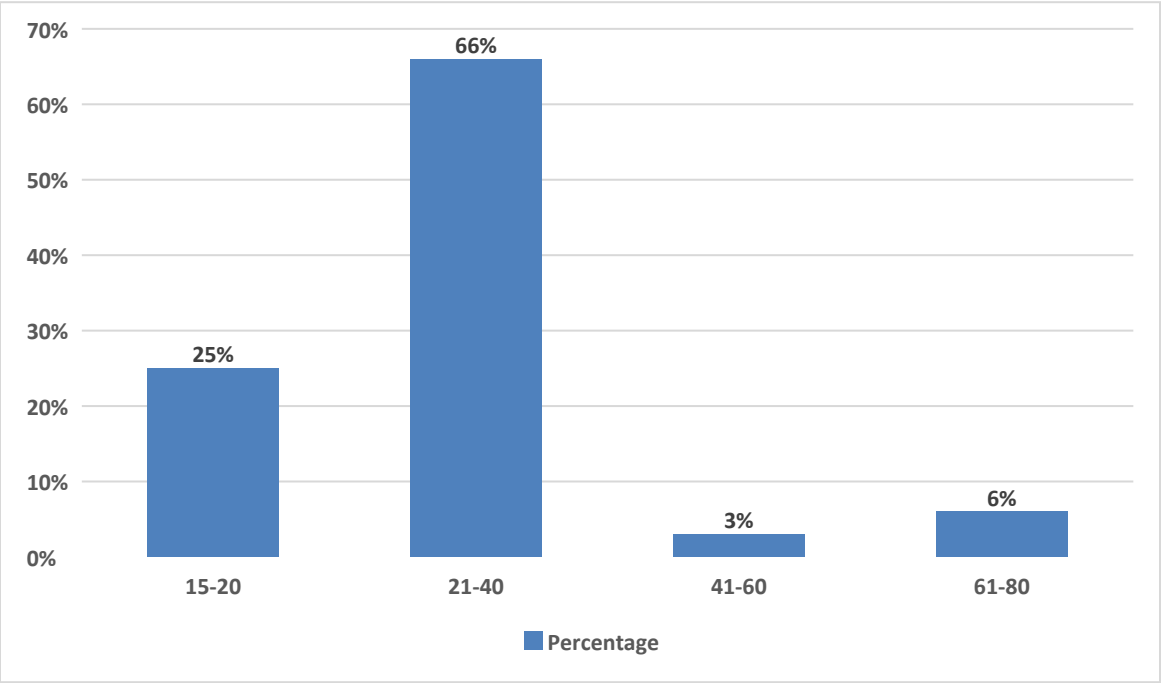


Table 2: Distribution of patients according to gender

Gender	Frequency	Percentage
Female	47	47%
Male	53	53%

Key Finding: Slight male predominance (53% vs. 47%).

Graph 2: Distribution of patients according to gender

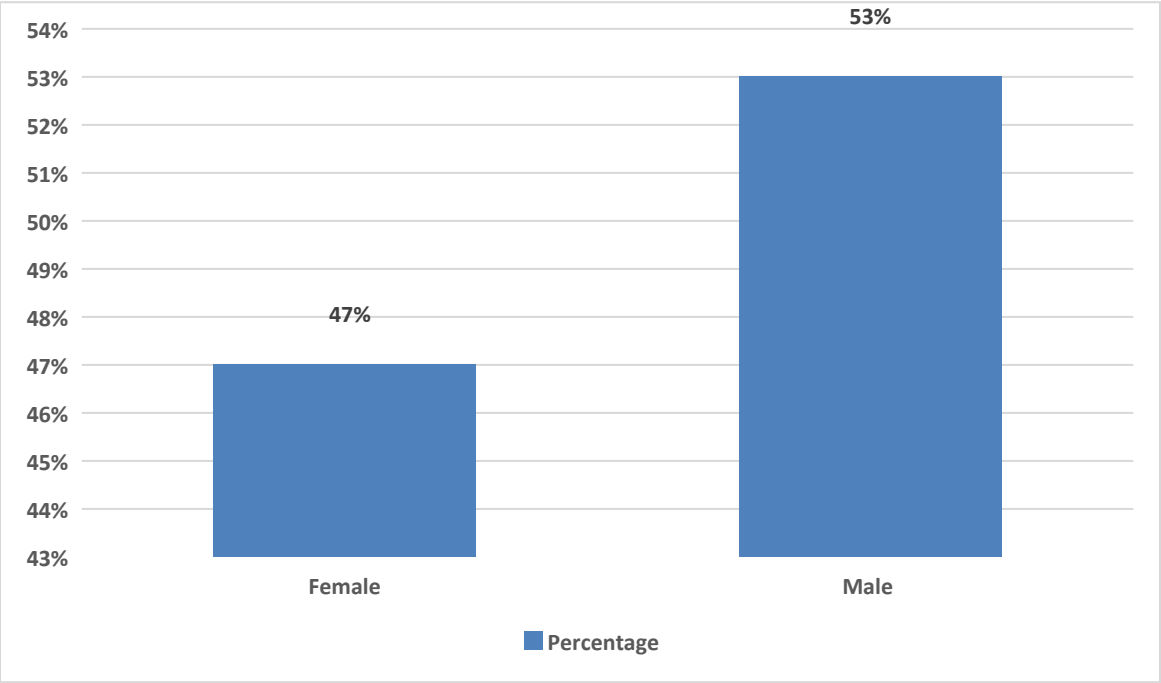


Table 3: Distribution of patients according to acetyl cholinesterase enzyme levels

acetyl cholinesterase enzyme levels	Frequency	Percentage
<500	37	37%
500-1000	5	5%
1000-2499	11	11%

2500-5319	17	17%
>5320	30	30%

Key Finding: 37% had severe enzyme depression (<500 U/L)

Graph 3: Distribution of patients according to acetyl cholinesterase enzyme levels

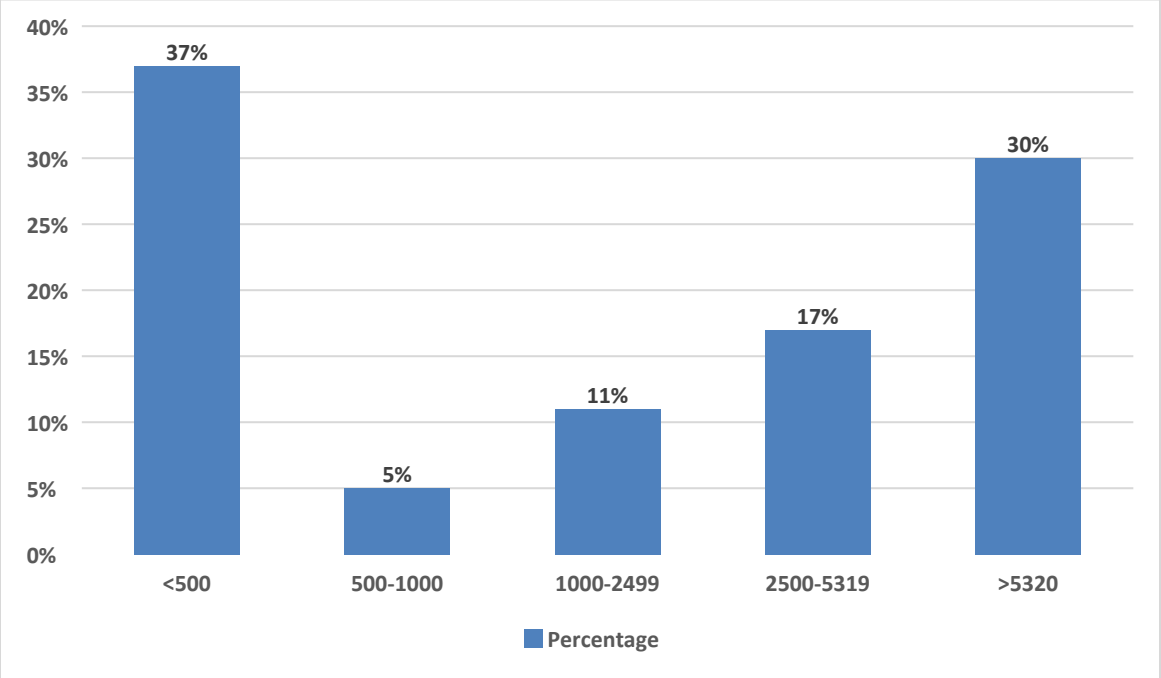


Table 4: Distribution of patients according to time since exposure

Time since exposure (hrs)	
Mean	4.77
SD	3.27

Table 4 and graph 4 indicates that patients arrived for medical attention relatively quickly after exposure, with a mean time since exposure of 4.77 hours (standard deviation 3.27 hours).

Graph 4: Distribution of patients according to time since exposure

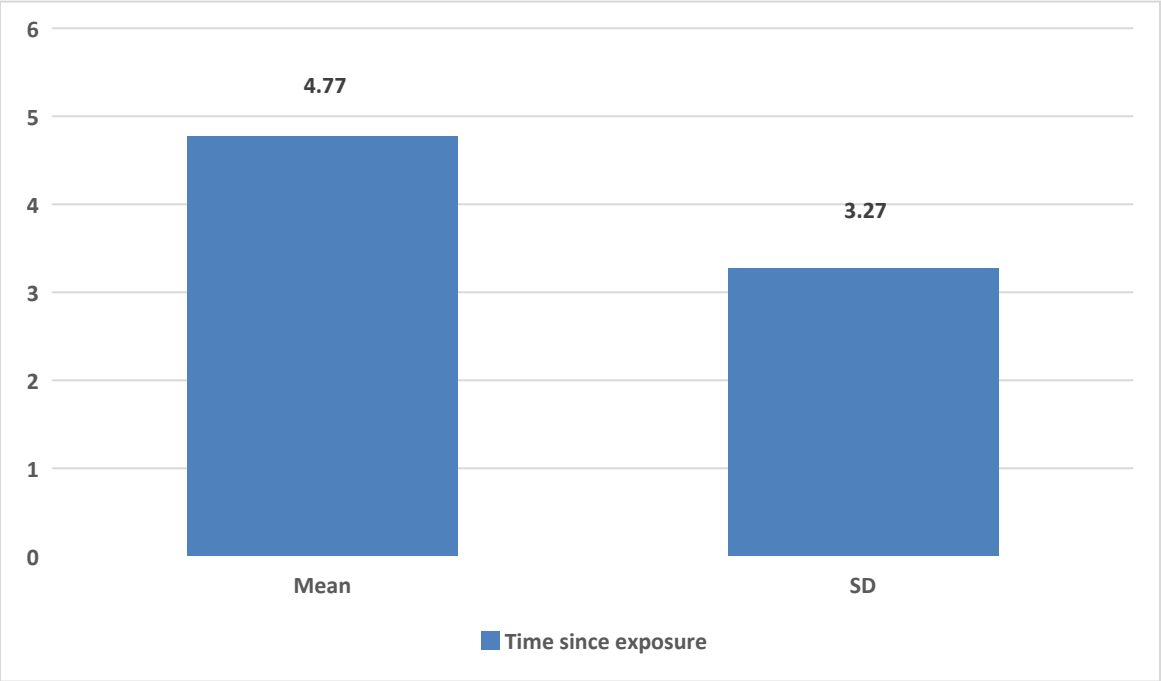


Table 5: Distribution of patients according to severity grading by Peradeniya Organophosphorus Poisoning scale

PPS severity grading	Frequency	Percentage
Mild	67	67%
Moderate	31	31%
Severe	2	2%

Key Finding: Majority had mild poisoning (67%).

Graph 5: Distribution of patients according to severity grading by Peradeniya Organophosphorus Poisoning scale

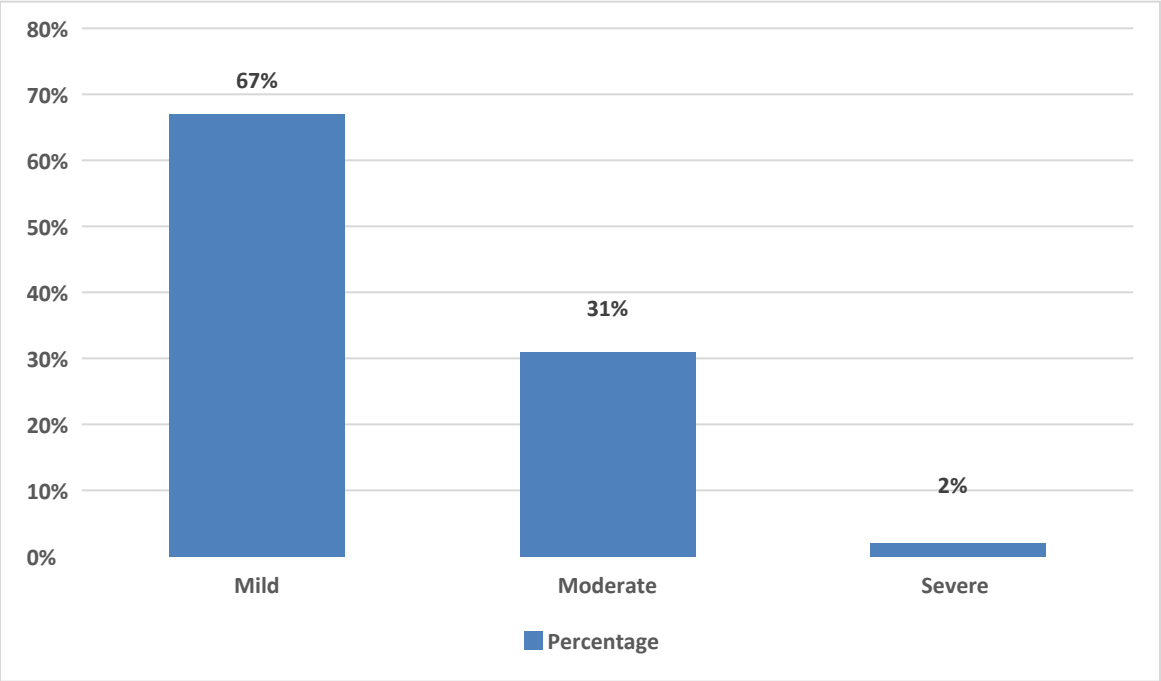


Table 6: Distribution of patients according to Intubation

Intubation	Frequency	Percentage
Yes	20	20%
No	80	80%

Table 6 and graph 6 shows that only 20% of patients required intubation for respiratory support, while the majority (80%) did not need this intervention.

Graph 6: Distribution of patients according to Intubation

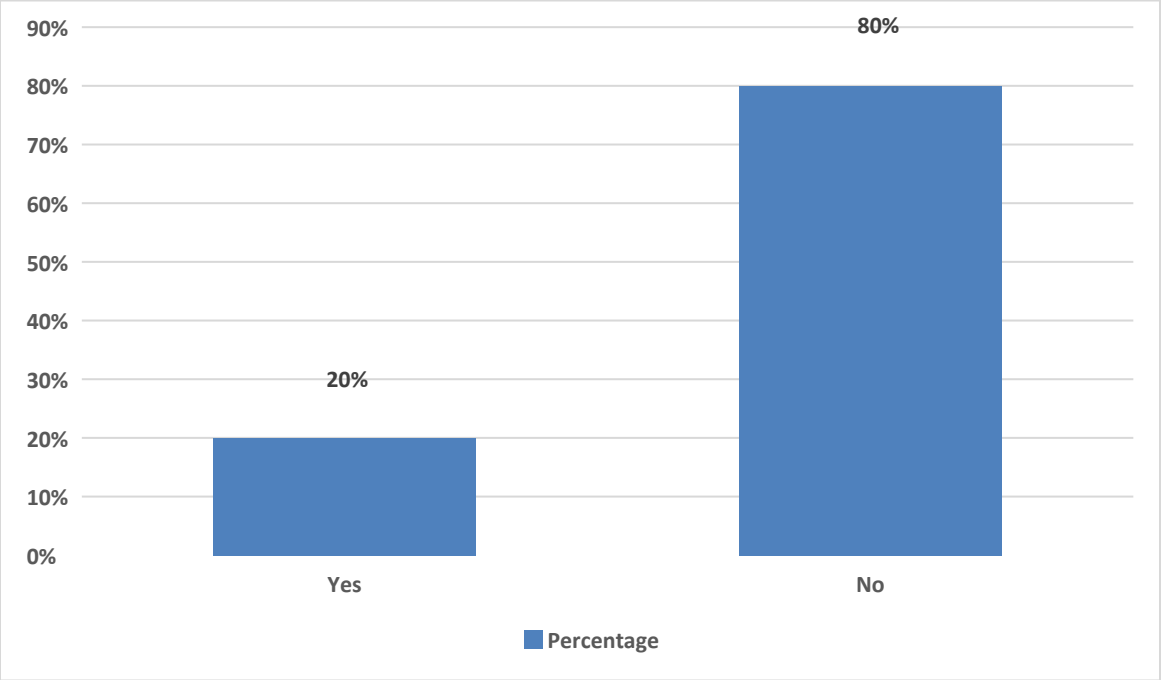


Table 7: Distribution of patients according to different parameters

Parameters	duration of hospital stay (days)	Atropine dose (mg)
Mean	6.13	109.8
SD	3.76	48.7

Table 7 and graph 7 presents key treatment parameters, showing that patients stayed in the hospital for an average of 6.13 days (SD 3.76) and received a mean atropine dose of 109.8 mg (SD 48.7) during their treatment.

Graph 7: Distribution of patients according to different parameters

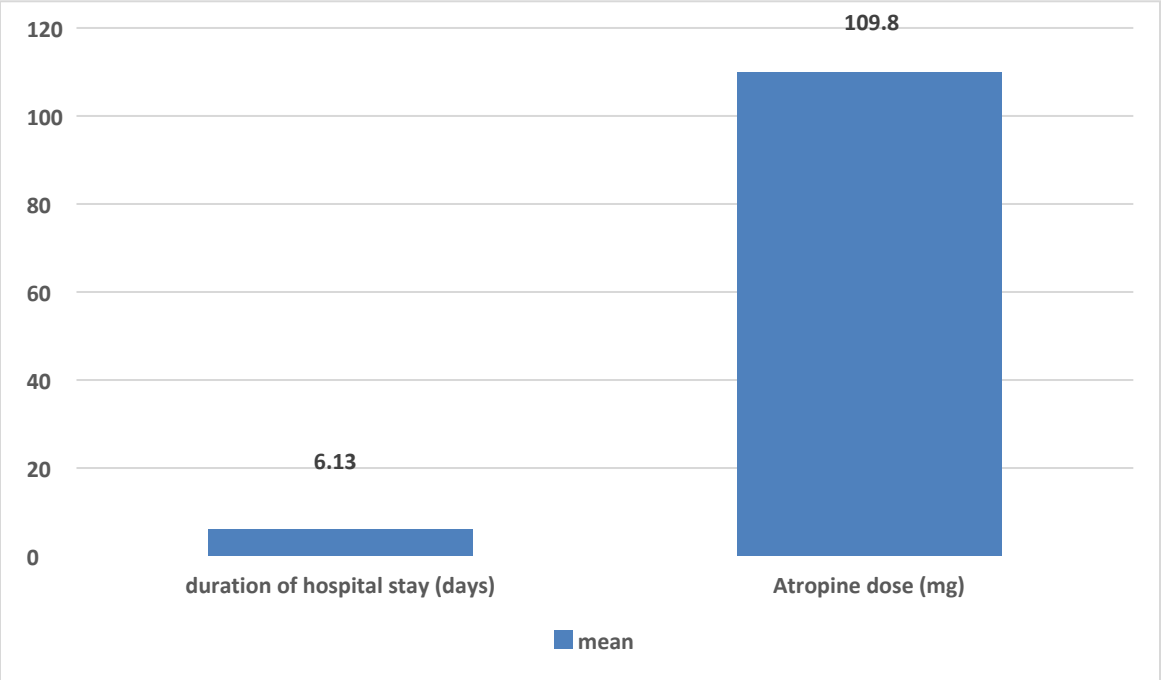


Table 8: Distribution of patients according to outcome

Outcome	Frequency	Percentage
Alive	75	75%
DAMA	12	12%
Death	13	13%

Table 8 and graph 8 reveals the outcomes of poisoning cases, with majority(75%) of the patients surviving.

Graph 8: Distribution of patients according to outcome

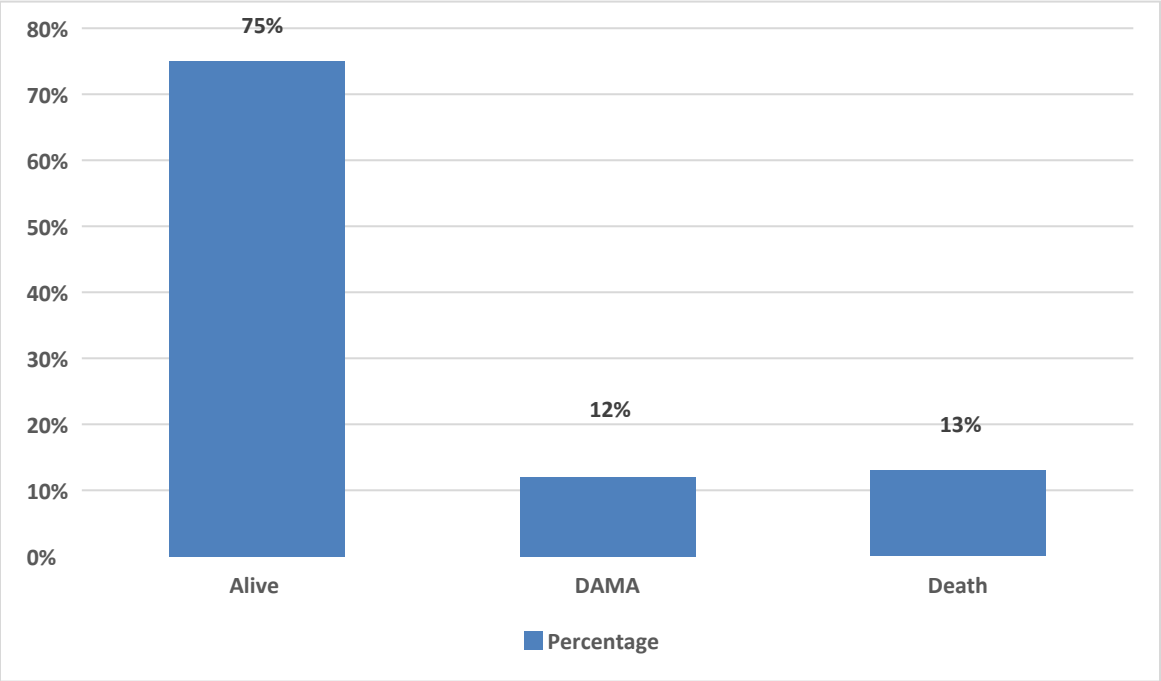


Table 9: Association of severity of OP poisoning with serum cholinesterase

serum cholinesterase	severity of OP poisoning			p-value
	Mild	Moderate	Severe	
Mean±SD	3211.4±2631.8	2028.3±2102.3	726.6±886.2	**0.05**

Key Finding: Enzyme levels decreased significantly with severity ($p = 0.05$).

Graph 9: Association of severity of OP poisoning with serum cholinesterase

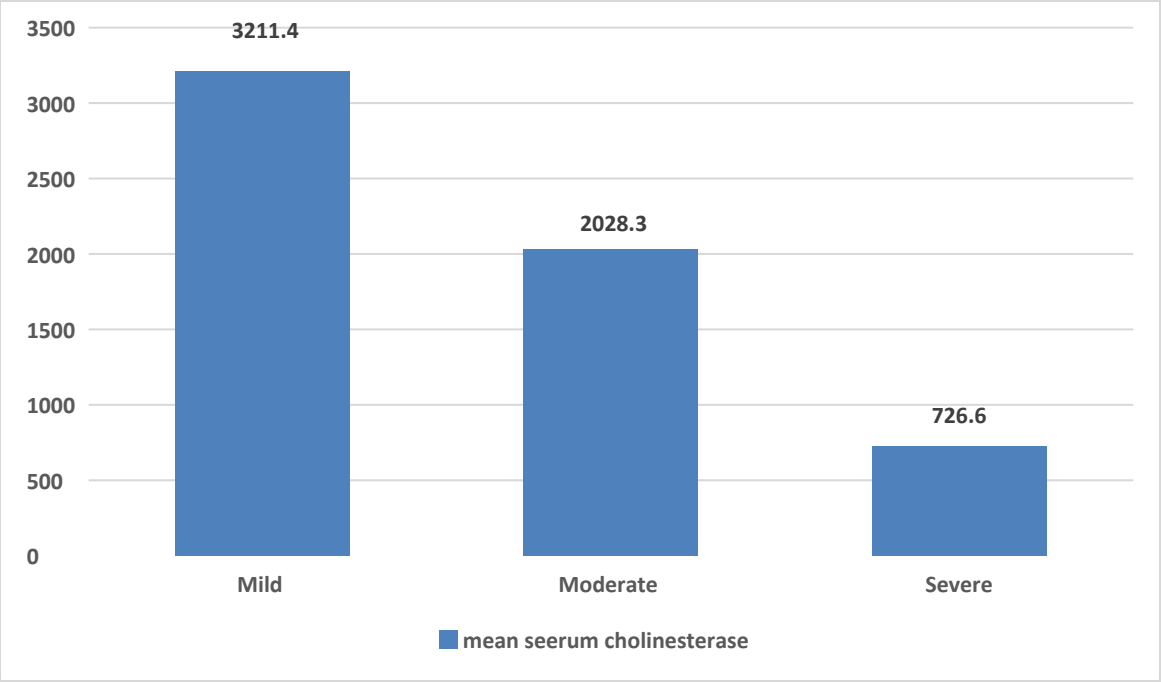


Table 10: Association of severity of OP poisoning with outcome

Outcome	severity of OP poisoning			p-value
	Mild	Moderate	Severe	
Alive	55 (82.1%)	20 (64.5%)	0	**<0.001**
DAMA	10 (14.9%)	2 (6.4%)	0	

Death	2 (2.9%)	9 (29.03%)	2 (100%)	
Total	**67 (100%)**	**31 (100%)**	**2 (100%)**	

Key Finding: Mortality escalated with severity (2.9% mild vs. 100% severe).

Graph 10: Association of severity of OP poisoning with outcome

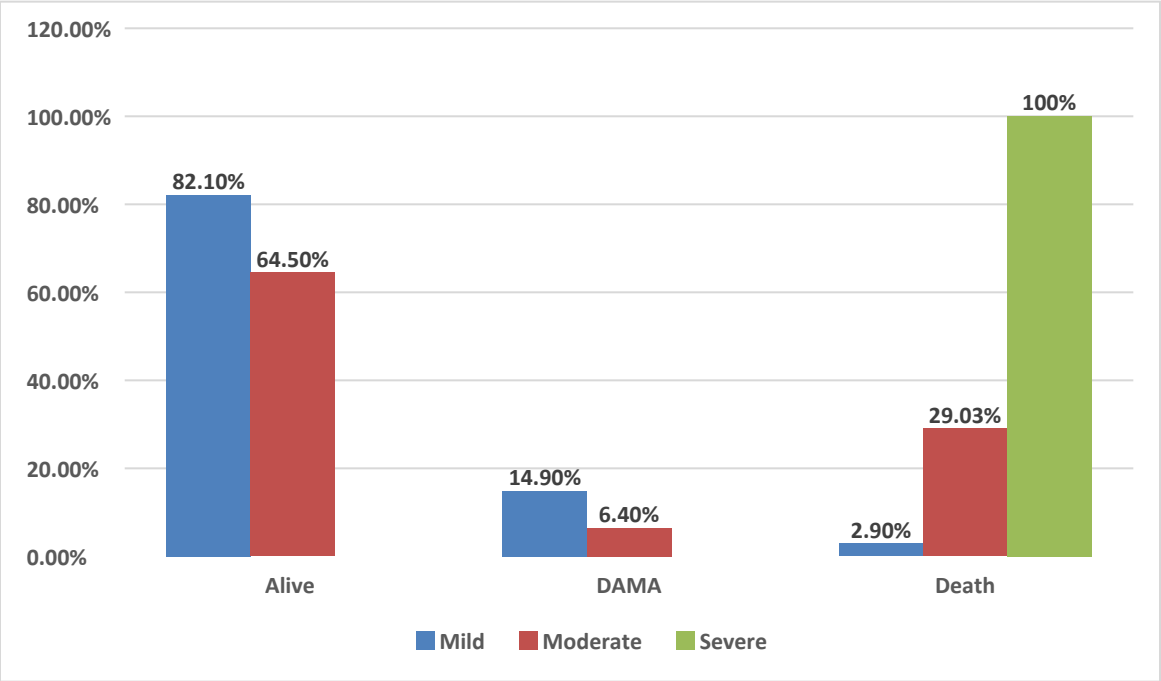


Table 11: Association of serum acetylcholinesterase levels with outcome

Outcome	serum acetylcholinesterase levels		p-value
	<5320	**>5320**	
Alive	46 (65.7%)	29 (96.7%)	**0.004**
DAMA	12 (17.1%)	0	

Death	12 (17.1%)	1 (3.3%)	
Total	**70 (100%)**	**30 (100%)**	

Key Finding: Normal enzyme levels (>5320 U/L) predicted better survival (96.7%).

Graph 11: Association of serum acetylcholinesterase levels with outcome

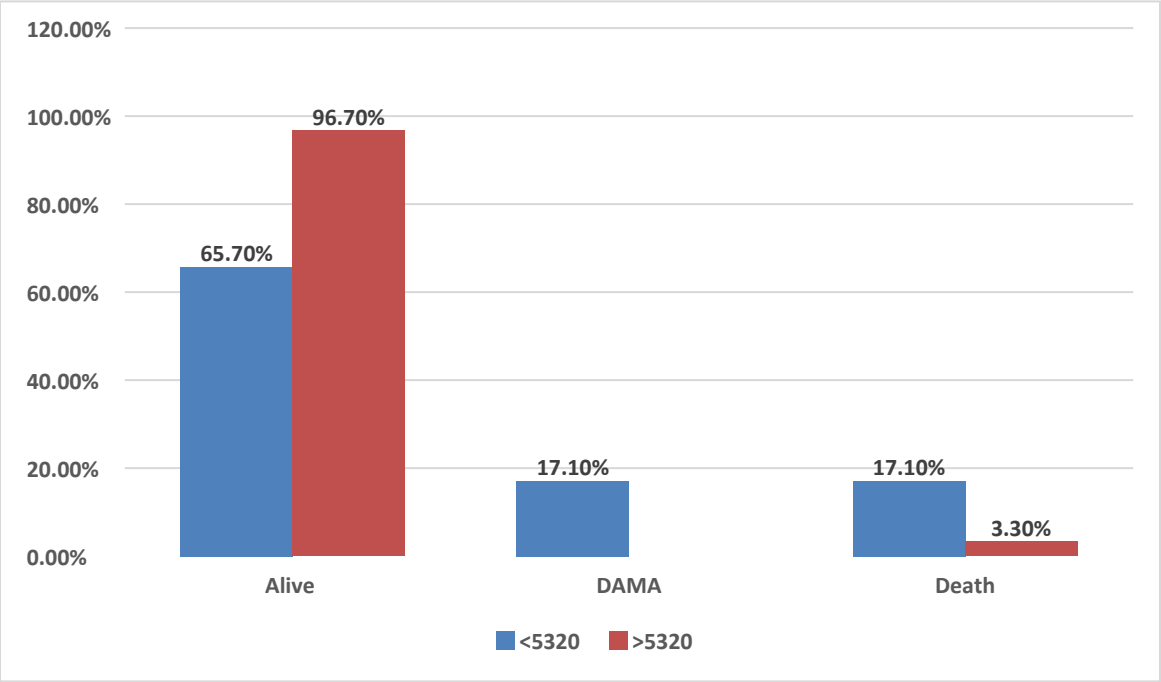


Table 12: Association of severity of OP poisoning with atropine dose

atropine dose	severity of OP poisoning			p-value
	Mild	**Moderate**	**Severe**	
Mean±SD	83.1±28.2	162.5±33.6	190.1±42.5	**<0.001**

Table 12 and graph 12 demonstrates increasing requirement of atropine dose with increasing severity.

Graph 12: Association of severity of OP poisoning with atropine dose

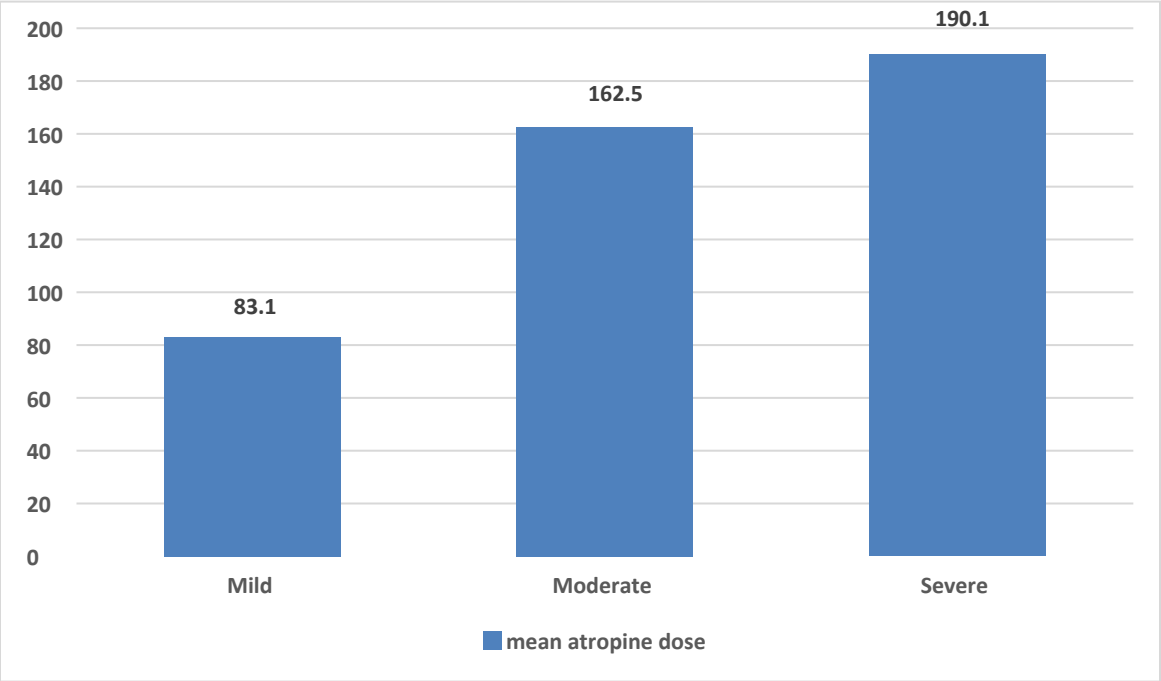
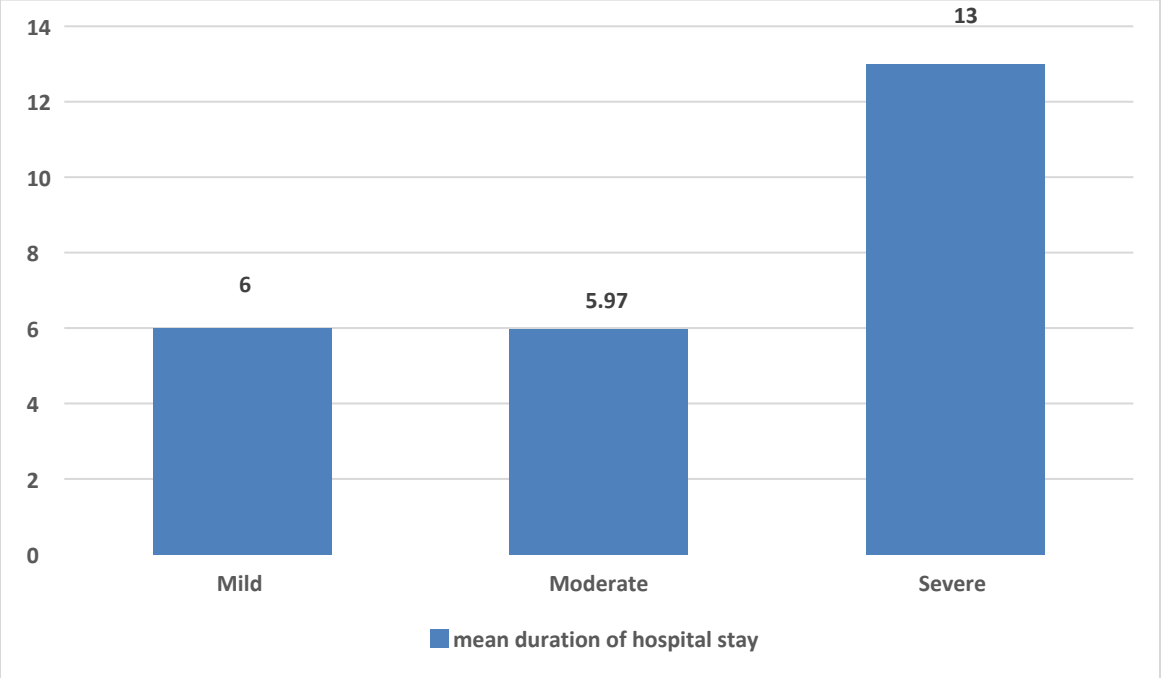


Table 13: Association of severity of OP poisoning with duration of hospital stay

duration of hospital stay	severity of OP poisoning			p-value
	Mild	Moderate	Severe	
Mean±SD	6±3.6	5.97±3.2	13±11.3	**0.03**

Table 13 and graph 13 shows a significant association (p=0.03) between poisoning severity and hospital stay duration, with severe cases requiring notably longer hospitalization (13±11.3 days)

Graph 13: Association of severity of OP poisoning with duration of hospital stay



DISCUSSION

Organophosphorus (OP) poisoning remains a critical public health challenge, particularly in agrarian communities where these compounds are widely accessible. Our study provides compelling evidence for the combined use of **serum cholinesterase levels** and the **Peradeniya Organophosphorus Poisoning (POP) scale** in assessing severity and predicting outcomes in OP poisoning.

DEMOGRAPHIC PATTERNS AND RISK FACTORS

Our findings align with global epidemiological trends:

- **Age Distribution**: 66% of cases were young adults (21–40 years)[73], consistent with studies by Banday et al. (69%) and Thunga et al. (41.2% in 21–30 years) [74]. This likely reflects psychosocial stressors (financial, emotional) in this age group.
- **Gender**: The near-equal distribution (53% male, 47% female) contrasts with studies showing male predominance[75] (e.g., Chaudhary et al.: 73.5%) [76], suggesting evolving socioeconomic dynamics in our region.

BIOCHEMICAL AND CLINICAL CORRELATIONS

1. **Serum Cholinesterase as a Biomarker**:
    - **Severe depression (<500 U/L)** was observed in 37% of patients, correlating with worse outcomes.
    - **Inverse relationship with severity**: Mean levels decreased significantly from mild (3211.4 ± 2631.8 U/L) to severe poisoning (726.6 ± 886.2 U/L; $p = 0.05$), echoing Rehiman et al.'s findings [77].
    - **Limitations**: Baseline variability due to genetics, nutrition, and liver function [78,79] necessitates cautious interpretation.
2. **POP Scale Utility**:
    - **Severity distribution**: Mild (67%), moderate (31%), severe (2%).
    - **Prognostic value**: Mortality escalated with POP scores—2.9% (mild) vs. 100% (severe) ($p < 0.001$)—validating its use for rapid triage, as shown by Senanayake et al. [80,81] and Sharma et al. [82].

MANAGEMENT AND OUTCOMES

- **Ventilatory Support**: 20% required intubation, primarily moderate/severe cases, aligning with Acikalin et al. (24.7%) [83] and Manu et al[84].
- **Hospital Stay**: Longer stays for severe cases (13 ± 11.3 days vs. ~6 days for mild/moderate; $p = 0.03$), likely due to complications like intermediate syndrome [85].
- **Mortality**: Overall 13%, with cholinesterase >5320 U/L predicting survival (96.7% vs. 65.7%; $p = 0.004$).

INTEGRATED ASSESSMENT: A PRAGMATIC APPROACH

Our study highlights the synergy between clinical (POP scale) and laboratory (cholinesterase) tools:

- **POP scale**: Rapid, resource-efficient severity grading.
- **Cholinesterase**: Objective confirmation and monitoring.

  This dual approach is especially valuable in resource-limited settings where advanced diagnostics are scarce.

LIMITATIONS AND FUTURE DIRECTIONS

- **Constraints**: Small severe cases subgroup (n = 2), lack of OP compound stratification, and unaccounted confounders (comorbidities, genetics).
- **Future research**: Larger cohorts, compound-specific toxicity profiles, and novel biomarkers (e.g., paraoxonase-1, oxidative stress markers) could refine prognostication.

CONCLUSION

The integration of **POP scale** and **serum cholinesterase** levels enhances risk stratification, guides treatment intensity, and improves outcomes in OP poisoning. While the POP scale enables swift bedside assessment, cholinesterase levels offer biochemical validation. Together, they form a robust framework for managing this life-threatening condition, particularly in resource-constrained environments.

SUMMARY

INTRODUCTION:

Organophosphorus (OP) pesticide poisoning remains a critical public health issue in agricultural communities. This study evaluated the correlation between clinical assessment (Peradeniya Organophosphorus Poisoning/POP scale) and laboratory parameters (serum cholinesterase levels) in predicting outcomes of OP poisoning.

METHODS:

- Prospective study of 100 OP poisoning patients
- Data collected: demographic characteristics, POP scores, serum cholinesterase levels
- Outcomes measured: mortality, intubation needs, atropine requirements, hospital stay duration

KEY FINDINGS:

1. **Patient Characteristics:**
    - Predominantly young adults (66% aged 21-40 years)
    - Slight male predominance (53%)
    - Mean presentation time: 4.77±3.27 hours post-exposure
2. **Severity Distribution:**
    - Mild: 67%
    - Moderate: 31%
    - Severe: 2%
3. **Serum Cholinesterase Levels:**
    - Strong inverse correlation with severity ($p=0.05$)
    - Mean levels: Mild=3211 U/L, Moderate=2028 U/L, Severe=727 U/L
    - 37% had levels <500 U/L (higher mortality risk)
4. **Clinical Outcomes:**
    - Intubation required: 20%
    - Mean atropine dose: 109.8±48.7 mg (higher in severe cases)
    - Mean hospital stay: 6.13 days (severe cases: 13 days)
    - Mortality: 13% overall (100% in severe cases)
5. **Predictive Value:**
    - Both POP scale and cholinesterase levels significantly predicted mortality ($p<0.001$)
    - Combined use improved severity assessment and outcome prediction

CONCLUSION:

The study demonstrates that integrating the POP clinical scoring system with serum cholinesterase measurement provides the most comprehensive approach for:

- Early severity assessment
- Prognostication
- Guiding treatment decisions
- Improving patient outcomes in OP poisoning

This dual-method approach is particularly valuable in resource-limited settings where rapid clinical decisions are crucial for managing this life-threatening condition.

REFERENCES

1. World Health Organization. The impact of pesticides on health. Geneva: WHO; 2022.
2. Kumar SV, Fareedullah M, Sudhakar Y, et al. Current review on organophosphorus poisoning. Arch Appl Sci Res. 2020;2(4):199-215.
3. Eddleston M, Phillips MR. Self poisoning with pesticides. BMJ. 2021;328(7430):42-44.
4. Agarwal SB, Bhatnagar VK, Agarwal A, et al. Impairment in clinical indices in acute organophosphate insecticide poisoning patients in India. Internet J Toxicol. 2019;4(1):1-6.
5. Senanayake N, de Silva HJ, Karalliedde L. A scale to assess severity in organophosphorus intoxication: POP scale. Hum Exp Toxicol. 2023;12:297-299.
6. Prasad DR, Jirli PS, Mahesh M, et al. Relevance of plasma cholinesterase to clinical findings in acute organophosphorous poisoning. Asia Pac J Med Toxicol. 2021;2(1):23-27.
7. Thunga G, Sam KG, Khera K, et al. Evaluation of incidence, clinical characteristics and management in organophosphorus poisoning patients in a tertiary care hospital. J Toxicol Environ Health Sci. 2020;2(5):73-76.
8. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet. 2021;371:597-607.
9. Peter JV, Sudarsan TI, Moran JL. Clinical features of organophosphate poisoning: A review of different classification systems and approaches. Indian J Crit Care Med. 2022;18(11):735-745.

10. Brahmi N, Mokline A, Kouraichi N, et al. Prognostic value of human erythrocyte acetylcholinesterase in acute organophosphate poisoning. Am J Emerg Med. 2020;24(7):822-827.
11. 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.
12. Petroianu G. A. (2010a). History of organophosphate synthesis: The very early days. Pharmazie 65, 306–311.
13. Petroianu G. A. (2010b). Toxicity of phosphor esters; Willy Lange (1900–1976) and Gerda von Krueger (1907-after 1970). Pharmazie 65, 776–780.
14. *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.*
15. *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.*
16. Atwood D., Paisley-Jones C. (2017). Pesticides Industry Sales and Usage: 2008–2012 Market Estimates, pp. 32 US Environmental Protection Agency, Washington, DC.
17. Casida J. E. (1964). Esterase inhibitors as pesticides. Science 146, 1011–1017.
18. Hobbiger F. (1963). Reactivation of phosphorylated acetylcholinesterase In: Cholinesterases and Anticholinesterase Agents, (Koelle G. B., Ed.), pp. 922–988. Springer-Verlag, Berlin.

19. 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.
20. 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.
21. 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.
22. Balali-Mood B. Basic and Clinical Toxicology of Organophosphorus Compounds. London, UK: Springer; 2014. Chemistry and classification of OP compounds; pp. 1–23.
23. Gupta R. C. Toxicology of Organophosphate & Carbamate Compounds. Amsterdam, Netherlands: Elsevier; 2006. Classification and uses of organophosphates and carbamates; pp. 5–24.
24. Mukherjee S, Gupta RD. Organophosphorus Nerve Agents: Types, Toxicity, and Treatments. J Toxicol. 2020 Sep 22;2020:3007984.
25. Benschop H. P., De Jong L. P. A. Nerve agent stereoisomers: analysis, isolation and toxicology. Accounts of Chemical Research. 1988;21(10):368–374.
26. 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.

27. Borges R, García AG. One hundred years from Otto Loewi experiment, a dream that revolutionized our view of neurotransmission. Pflugers Arch. 2021 Jun;473(6):977-981.
28. Rusyniak DE, Nañagas KA. Organophosphate poisoning. Semin Neurol. 2004 Jun;24(2):197-204.
29. Sellin AK, Shad M, Tamminga C. Muscarinic agonists for the treatment of cognition in schizophrenia. CNS Spectr. 2008 Nov;13(11):985-96.
30. Abrams P, Andersson KE, Buccafusco JJ, Chapple C, de Groat WC, Fryer AD, Kay G, Laties A, Nathanson NM, Pasricha PJ, Wein AJ. Muscarinic receptors: their distribution and function in body systems, and the implications for treating overactive bladder. Br J Pharmacol. 2006 Jul;148(5):565-78.
31. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: Systematic review. BMC Public Health. 2007;7:357.
32. Sudakin DL, Power LE. Organophosphate exposures in the United States: a longitudinal analysis of incidents reported to poison centers. J Toxicol Environ Health A. 2007 Jan 15;70(2):141-7.
33. Gummin DD, Mowry JB, Beuhler MC, Spyker DA, Bronstein AC, Rivers LJ, Pham NPT, Weber J. 2020 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 38th Annual Report. Clin Toxicol (Phila). 2021 Dec;59(12):1282-1501.
34. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: systematic review. BMC Public Health. 2007 Dec 21;7:357.

35. 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.
36. Srivastava A, Peshin SS. An epidemiological study of poisoning cases reported to the National Poisons Information centre, All India Institute of Medical Sciences, New Delhi. Hum Exp Toxicol. 2005;24:279–85.
37. Accidental deaths and suicides in India, National Crime Records Bureau, Ministry of Home affairs, Government of India. [Accessed April 6, 2010]. at: http://ncrb.nic.in/adsi2008/suicides-08.pdf
38. World Health Statistics 2016: Monitoring health for the SDGs. (2022). Accessed: February 22, 2023: https://reliefweb.int/report/world/world-health-statistics-2016-monitoring-health-sdgs.
39. Taruni N, Bijoy TK, Momonchand A: A profile of poisoning cases admitted in Rims Hospital, Imphal. Journal of Forensic Medicine. 2001, 18:31-3.
40. Suicide- World Head Organization. (2022). Accessed: February 22, 2023: https://www.who.int/news-room/fact-sheets/detail/suicide.
41. 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.
42. Kamanyire R, Karalliedde L. Organophosphate toxicity and occupational exposure. Occup Med (Lond). 2004 Mar;54(2):69-75.
43. Sikary AK. Homicidal poisoning in India: A short review. J Forensic Leg Med. 2019 Feb;61:13-16.

44. Jokanović M. Neurotoxic effects of organophosphorus pesticides and possible association with neurodegenerative diseases in man: A review. Toxicology. 2018 Dec 01;410:125-131.
45. 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.
46. Eddleston M. The pathophysiology of organophosphorus pesticide self-poisoning is not so simple. Neth J Med. 2008 Apr;66(4):146-8.
47. Kwong TC. Organophosphate pesticides: biochemistry and clinical toxicology. Ther Drug Monit. 2002 Feb;24(1):144-9.
48. Nolan RJ, Rick DL, Freshour NL, Saunders JH. Chlorpyrifos: pharmacokinetics in human volunteers. Toxicol Appl Pharmacol. 1984 Mar 30;73(1):8-15.
49. Lee DH, Jung KY, Choi YH, Cheon YJ. Body mass index as a prognostic factor in organophosphate-poisoned patients. Am J Emerg Med. 2014 Jul;32(7):693-6.
50. Buratti FM, Volpe MT, Meneguz A, Vittozzi L, Testai E. CYP-specific bioactivation of four organophosphorothioate pesticides by human liver microsomes. Toxicol Appl Pharmacol. 2003 Feb 01;186(3):143-54.
51. Eyer P. The role of oximes in the management of organophosphorus pesticide poisoning. Toxicol Rev. 2003;22(3):165-90.
52. Merrill DG, Mihm FG. Prolonged toxicity of organophosphate poisoning. Crit Care Med. 1982 Aug;10(8):550-1.
53. 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.

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. Roberts D, Senarathna L. Secondary contamination in organophosphate poisoning. QJM. 2004 Oct;97(10):697-8.

57. Little M, Murray L., Poison Information Centres of New South Wales, Western Australia, Queensland, New Zealand, and the Australian Capital Territory. Consensus statement: risk of nosocomial organophosphate poisoning in emergency departments. Emerg Med Australas. 2004 Oct-Dec;16(5-6):456-8.

58. Eddleston M, Buckley NA, Checketts H, Senarathna L, Mohamed F, Sheriff MH, Dawson A. Speed of initial atropinisation in significant organophosphorus pesticide poisoning--a systematic comparison of recommended regimens. J Toxicol Clin Toxicol. 2004;42(6):865-75.

59. Syed S, Gurcoo SA, Farooqui AK, Nisa W, Sofi K, Wani TM. Is the World Health Organization-recommended dose of pralidoxime effective in the treatment of organophosphorus poisoning? A randomized, double-blinded and placebo-controlled trial. Saudi J Anaesth. 2015 Jan;9(1):49-54.

60. Walton EL. Pralidoxime and pesticide poisoning: A question of severity? Biomed J. 2016 Dec;39(6):373-375.

61. Dickson EW, Bird SB, Gaspari RJ, Boyer EW, Ferris CF. Diazepam inhibits organophosphate-induced central respiratory depression. Acad Emerg Med. 2003 Dec;10(12):1303-6.

62. Adeyinka A, Muco E, Regina AC, et al. Organophosphates. [Updated 2023 Nov 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK499860/

63. Malaviya NB, Parikh R, Pancholi K, Belim OB. Assessment of the Peradeniya Organophosphorus Poisoning Scale as a Severity and Prognostic Marker in Patients With Acute Organophosphorus Poisoning Presenting to an Emergency Medicine Department. Cureus. 2023 Jun;15(6):e40277.

64. Shafti SS, Singh J. Suicidal behavior among Iranian psychiatric patients. International Journal of Advanced Research in Medicine. 2023;5(2):78-84

65. Kamath SD, Gautam VK. Study of organophosphorus compound poisoning in a tertiary care hospital and the role of Peradeniya Organophosphorus Poisoning scale as a prognostic marker of the outcome. J Family Med Prim Care. 2021 Nov;10(11):4160-4167.

66. Gurung D. Study of Correlation of Serum Cholinesterase Level with Peradeniya Organophosphorus Poisoning Scale in Organophosphorus Poisoning. Journal of Karnali Academy of Health Sciences. 2021; 4(3)

67. Chaudhary R, Bhandari R, Malla G, Poudel M, Lamsal M. Correlation of clinical score and serum acetylcholinesterase in the emergency ward of a tertiary hospital. J BP Koirala Inst Health Sci 2019;2:19-27.

68. Honnakatti V, Nimbal N, Doddapattar P. A study on serum cholinesterase level in organophosphorus poisoning and its correlation with severity of organophosphorus poisoning. Int J Adv Med. 2018;5:1021-5.
69. Vernekar PV, Shivraj K. Peradeniya organophosphorus poisoning scale (POP) as a predictor of respiratory failure and mortality in organophosphorus poisoning. Sch J Appl Med Sci. 2017;5:1841–4.
70. T. N. Dubey, Sudhanshu Yadav, K K. Kawre. Correlation of severity of organophoshorus poisoning as assessed by peradeniya organophosphorus poisoning scale with serum amylase and CPK level. International Journal of Contemporary Medical Research 2016;3(9):2534-2537.
71. Rajeev H, Arvind MN. Study of clinical and biochemical parameters in predicting the need for ventilator support in organophosphorus compound poisoning. J Evol Med Dent Sci 2013;12:955570.
72. S, Rehiman & Lohani, Shyam & Bhattarai, Madhur. (2008). Correlation of Serum Cholinesterase Level, Clinical Score at Presentation and Severity of Organophosphorous Poisoning. JNMA; journal of the Nepal Medical Association. 47. 47-52. 10.31729/jnma.306.
73. 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.
74. Thunga G, Sam KG, Khera K, Pandey S, Sood S. Evaluation of incidence, clinical characteristics and management in organophosphorus poisoning patients in a tertiary care hospital. J Toxicol Environ Health Sci. 2010;2(5):73-76.

75. 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).
76. 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.
77. Rehiman S, Lohani SP, Bhattarai MD. Correlation of serum cholinesterase level, clinical score at presentation and severity of organophosphorus poisoning. JNMA J Nepal Med Assoc. 2008;47(170):47-52.
78. Agarwal SB, Bhatnagar VK, Agarwal A, Agarwal U, Venkaiah K, Nigam SK, et al. Impairment in clinical indices in acute organophosphate insecticide poisoning patients in India. Internet J Toxicol. 2007;4(1):1-6.
79. Aygun D, Doganay Z, Altintop L, Guven H, Onar M, Deniz T, et al. Serum acetylcholinesterase and prognosis of acute organophosphate poisoning. J Toxicol Clin Toxicol. 2002;40(7):903-910.
80. 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.
81. Raveendra K R, Mohan C N, Nandan Kodur. A study to assess the utility of peradeniya organophosphorous poisoning (POP) scale, poisoning severity score (PSS) and glasgow coma scale (GCS) in predicting severity and treatment outcome in acute organophosphorous poisoning. International Journal of Contemporary Medical Research 2020;7(2):B20-B24.

82. Sharma B, Harish D, Sharma A, Bangar S, Gupta M. The epidemiology of poisoning: An Indian view point. J Forensic Med Toxicol. 2002;19(2):5-11.

83. Acikalin A, Disel NR, Matyar S, Sebe A, Kekec Z, Gokel Y, et al. Prognostic factors determining morbidity and mortality in organophosphate poisoning. Pak J Med Sci. 2017;33(3):534-539.

84. Manu MS, Prashant V, Akila P, Suma MN, Basavanagowdappa H. A retrospective analysis of serial measurement of serum cholinesterase in acute poisoning with organophosphate compounds. Toxicol Int. 2012;19(3):255-259.

85. 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.

ANNEXURE- I

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

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/ 927/2023-24 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,** scrutinized 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: "A STUDY OF CORRELATION OF SERUM CHOLINESTERASE LEVEL, CLINICAL SCORE BY PERADENIYA POISONING SCALE AT PRESENTATION AND SEVERITY OF ORGANOPHOSPHOROUS POISONING".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.M.HARSHITH KUMAR

NAME OF THE GUIDE: DR.MALLANNA MULIMANI, 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- 2

INFORMED CONSENT FORM

BLDE (DEEMED TO BE UNIVERSITY)'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE,

VIJAYAPURA- 586103

TITLE OF THE PROJECT

" A STUDY OF CORRELATION OF SERUM CHOLINESTERASE LEVEL, CLINICAL SCORE BY PERADENIYA POISONING SCALE AT PRESENTATION AND SEVERITY OF ORGANOPHOSPHORUS POISONING**"**

PRINCIPAL INVESTIGATOR - Dr. M. HARSHITH KUMAR

P.G.GUIDE NAME - Dr. MALLANNA S MULIMANI, PROFESSOR OF MEDICINE

CHAIRMAN ETHICAL COMMITTEE

All aspects of this consent form are explained to the patient in the language they understand.

1) PURPOSE OF RESEARCH:

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

2) PROCEDURE:

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

3) RISK AND DISCOMFORTS:

I understand that I may experience pain and discomfort during the examination or my treatment. This is mainly the result of my condition, and the procedure of this study is not expected to exaggerate these feelings associated with the usual course of treatment.

4) BENEFITS:

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

5) CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records. Still, it will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate location. Suppose the data are used for publication in the medical literature or teaching purposes; in that case, no name will be used. Other identifiers, such as photographs and audio or videotapes, will be used only with my special written permission. I understand that I may see the pictures and videos and hear the audiotapes before giving this permission.

6) REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr M.HARSHITH KUMAR is available to answer my questions or concerns. I know that I will be informed of any significant new findings discovered during the study, which might influence my continued participation. If, during the study or later, I wish to discuss my involvement or concerns regarding this study with a person not directly involved, I am aware that the hospital's social

I understand that I may experience pain and discomfort during the examination or my treatment. This is mainly the result of my condition, and the procedure of this study is not expected to exaggerate these feelings associated with the usual course of treatment.

7) CONFIDENTIALITY:

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9) REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr M.HARSHITH KUMAR 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 physician or physical therapist if this is appropriate.

10) INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study if such damage were reported promptly, the appropriate treatment would be available, but no further compensation would be provided. I understand that my agreement to participate in this study is not waiving my legal rights. In the patient's language, I have explained the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability.

I understand that I may experience pain and discomfort during the examination or my treatment. This is mainly the result of my condition, and the procedure of this study is not expected to exaggerate these feelings associated with the usual course of treatment.

11) CONFIDENTIALITY:

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

medical literature or teaching purposes; in that case, no name will be used. Other identifiers, such as photographs and audio or videotapes, will be used only with my special written permission. I understand that I may see the pictures and videos and hear the audiotapes before giving this permission.

12) REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr M.HARSHITH KUMAR is available to answer my questions or concerns. I know that I will be informed of any significant new findings discovered during the study, which might influence my continued participation. If, during the study or later, I wish to discuss my involvement or concerns regarding this study with a person not directly involved, I am aware that the hospital's social worker is available to talk with me. I will be given a copy of this consent form to keep for careful reading.

13) REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr M.HARSHITH KUMAR 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 physician or physical therapist if this is appropriate.

_____ _____

DR. M.HARSHITH KUMAR
(Investigator)

Date :

II)STUDY SUBJECT CONSENT STATEMENT

I confirm that DR. M.HARSHITH KUMAR has explained the research's purpose, the study procedures I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my language. I have read and understand this consent form. Therefore, I agree to consent to participate as a subject in this research project.

_____

_____

Participant / Guardian Date:

_____

_____

Witness to signature Date:

BLDE (DEEMED TO BE UNIVERSITY) ಶ್ರೀ ಬಿ.ಎಂ.ಪಟ್ಟೀಲ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜು, ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ವಿಜಯಪುರ-586103

ಪ್ರಬಂಧ/ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಮಾಹಿತಿ ಪಡೆದ ಸಮ್ಮತಿ

ನಾನು, ಕೆಳಗಿನವರು_____ ಸಹಿಯಿಟ್ಟವರು, ಮಗ/ಮಗಳು/ಪತ್ನಿಯ _____ ವಯಸ್ಸು _____ವರ್ಷಗಳು, ಸಾಮಾನ್ಯವಾಗಿ ನಿವಾಸಿಸುವ ಸ್ಥಳದ ಹೆಸರು_____, ಇಲ್ಲಿ ಹೇಳಿದ್ದೇನೆ/ಘೋಷಿಸುತ್ತೇನೆ ಡಾಕ್ಟರ್ ಹೆಸರು_____ ಅವರು ಆಸ್ಪತ್ರೆ ಹೆಸರು_____ ಅವರು ನನ್ನನ್ನು ಪೂರ್ಣವಾಗಿ ಪರೀಕ್ಷಿಸಿದರು ದಿನಾಂಕದಲ್ಲಿ_____ ಸ್ಥಳ ಹೆಸರು_____ ಮತ್ತು ನನಗೆ ನನ್ನ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ ನಾನು ಒಂದು ರೋಗ (ಸ್ಥಿತಿ) ಅನುಭವಿಸುತ್ತಿದ್ದೇನೆ. ಮುಂದುವರಿದು ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ಅವರು ಒಂದು ಪದ್ದತಿ/ಸಂಶೋಧನೆ ನಡೆಸುತ್ತಿದ್ದಾರೆ ಶೀರ್ಷಿಕೆಯುಳ್ಳ_____ ಡಾಕ್ಟರ್_____ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯನ್ನು ಕೇಳಿದ್ದಾರೆ ಅಧ್ಯಯನದಲ್ಲಿ.

ಡಾಕ್ಟರ್ ನನಗೆ ಇದನ್ನು ಕೂಡಾ ತಿಳಿಸಿದ್ದಾರೆ ಈ ಕ್ರಮದ ನಡೆವಲ್ಲಿ ಪ್ರತಿಕೂಲ ಫಲಿತಾಂಶಗಳನ್ನು ಎದುರಿಸಬಹುದು. ಮೇಲೆ ಹೇಳಿದ ಪ್ರಕಟಣೆಗಳಲ್ಲಿ, ಅಧಿಕಾಂಶವು ಚಿಕಿತ್ಸಿಸಬಹುದಾದರೂ ಅದನ್ನು ನಿರೀಕ್ಷಿಸಲಾಗುತ್ತಿಲ್ಲ ಆದ್ದರಿಂದ ನನ್ನ ಸ್ಥಿತಿಯ ಹಿರಿದಾಗುವ ಅವಕಾಶವಿದೆ ಮತ್ತು ಅಪರೂಪದ ಸಂದರ್ಭಗಳಲ್ಲಿ ಅದು ಮರಣಕಾರಕವಾಗಿ ಪರಿಣಮಿಸಬಹುದು ಹೊಂದಿದ ರೋಗನಿರ್ಧಾರ ಮತ್ತು ಯಥಾಶಕ್ತಿ ಚಿಕಿತ್ಸೆ ಮಾಡಲು ಹೊಂದಿದರೂ. ಮುಂದುವರಿದು ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ ಈ ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳ ಮೌಲ್ಯಮಾಪನದಲ್ಲಿ ಸಹಾಯಕವಾಗುತತ್ತದೆ ಇತರ ಸಮಾನ ಪ್ರಕರಣಗಳ ಚಿಕಿತ್ಸೆಗೆ ಉಪಯುಕ್ತ ಉಲ್ಲೇಖವಾಗಿದೆ, ಮತ್ತು ನಾನು ಅನುಭವಿಸುವ ರೋಗದಿಂದ ವಿಮುಕ್ತಿ ಅಥವಾ ಗುಣಮುಖಗೊಳ್ಳುವಲ್ಲಿ ನನಗೆ ಪ್ರಯೋಜನವಾಗಬಹುದು.

ಡಾಕ್ಟರ್ ನನಗೆ ಇದನ್ನು ಕೂಡಾ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನಿಂದ ನೀಡಿದ ಮಾಹಿತಿ, ಮಾಡಿದ ಪರಿಶೀಲನೆಗಳು / ಫೋಟೋಗ್ರಾಫ್ ಗಳು / ವೀಡಿಯೋ ಗ್ರಾಫ್ ಗಳು ನನ್ನ ಮೇಲೆ ತೆಗೆದುಕೊಳ್ಳಲಾಗುವ ಅನ್ವೇಷಕರು ರಹಸ್ಯವಾಗಿ ಇಡುವರು ಮತ್ತು ನಾನು ಅಥವಾ ನನಗೆ ಕಾನೂನು ದೃಷ್ಟಿಯಲ್ಲಿ ಸಂಬಂಧಿತ ಹೊರತುಪಡಿಸಿ ಇತರ ವ್ಯಕ್ತಿಯಿಂದ ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದಿಲ್ಲ. ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ ಶುದ್ಧವಾಗಿ ಸ್ವೇಚ್ಛಾಯಿತ, ನನ್ನಿಂದ ನೀಡಿದ ಮಾಹಿತಿಯ ಆಧಾರದ ಮೇಲೆ, ಚಿಕಿತ್ಸೆ / ಅಧ್ಯಯನದ ಸಂಬಂಧದಲ್ಲಿ ರೋಗನಿರ್ಧಾರ, ಚಿಕಿತ್ಸೆಯ ವಿಧಾನ, ಚಿಕಿತ್ಸೆಯ ಫಲಿತಾಂಶ ಅಥವ ಆ ಭವಿಷ್ಯದ ಪ್ರವೃತ್ತಿಗಳು ಬಗ್ಗೆ ಯಾವುದೇ ಸ್ಪಷ್ಟತೆ ಕೇಳಬಹುದು. ಅದೇ ಸಮಯದಲ್ಲಿ ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯನ್ನು

ನಿಲ್ಲಿಸಬಹುದು ನಾನು ಬಯಸಿದರೆ ಅಥವಾ ಅನ್ವೇಷಕರು ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನನ್ನನ್ನು ನಿಲ್ಲಿಸಬಹುದು.

ಪ್ರಬಂಧ ಅಥವಾ ಸಂಶೋಧನೆಯ ಸ್ವಭಾವ, ಮಾಡಿದ ರೋಗನಿರ್ಧಾರ ಮತ್ತು ಚಿಕಿತ್ಸೆಯ ವಿಧಾನವನ್ನು ಅರ್ಥಮಾಡಿಕೊಂಡು, ನಾನು ಕೆಳಗಿನ ಶ್ರೀ / ಶ್ರೀಮತಿ _____ ನನ್ನ ಪೂರ್ಣವಾದ ಪ್ರಜ್ಞೆಯ ಸ್ಥಿತಿಯಲ್ಲಿ ಹೇಳಿದ ಸಂಶೋಧನೆ / ಪ್ರಬಂಧದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪುತ್ತೇನೆ.

ರೋಗಿಯ ಸಹಿ

ಡಾಕ್ಟರನ ಸಹಿ

ಸಾಕ್ಷಿಗಳು

1)

2)

ANNEXURE-3

BLDE (DEEMED TO BE UNIVERSITY)

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

SCHEME OF CASE TAKING

Informant :

Name: CASE NO:

Age: IP NO:

Sex: DOA:

Religion: DOD:

Past Occupation:

Present Occupation:

Residence:

Chief complaints:

History of present illness:

Past History:

Personal History:

Family History:

Treatment History:

General Physical Examination

Height:

Weight:

Body Mass Index:

Vitals

PR:

BP:

RR:

Temp:

Head-to-toe examination:

SYSTEMIC EXAMINATION.

CENTRAL NERVOUS SYSTEM:

RESPIRATORY SYSTEM:

CARDIOVASCULAR SYSTEM:

PER ABDOMEN:

INVESTIGATIONS

1. HAEMATOLOGY –

1)Hemoglobin	gm. %
2)Total WBC counts	Cells/mm^3
3)Differential counts -	
Neutrophils	%
Lymphocytes	%
Eosinophils	%
Monocytes	%
Basophils	%
Platelet count	
ESR	At the end of 1st hour

- URINE ROUTINE

- ABSOLUTE EOSINOPHIL COUNT

2. BIOCHEMISTRY:

- RANDOM BLOOD SUGAR
- LIVER FUNCTION TEST
- RENAL FUNCTION TEST
- SERUM ACETYLCHOLINESTERASE

3. RADIOLOGY:

- CHEST X-RAY PA VIEW:

4. ELECTROCARDIOGRAPHY:

5. TOXICOLOGY REPORT FROM GASTRIC ASPIRATE:

CONCLUSION:

Date:-

Signature:-

CURRICULUM-VITAE

P. G. GUIDE:

NAME :	Dr. MALLANNA S MULIMANI
DESIGNATION:	PROFESSOR OF MEDICINE SHRI B M PATIL MEDICAL COLLEGE
CONTACT:	08352-EXT-2148
DATE OF BIRTH:	01/06/1963
EDUCATION:	
MBBS-	COLLEGE: Vijayanagara Institute of Medical Sciences, Bellary (VIMS) Gulbarga University.
MD (GENERAL MEDICINE)-	COLLEGE: Mahadevappa Rampure Medical College, Gulbarga (MRMC).
PRESENT DESIGNATION:	Professor of Medicine Department of Medicine BLDE (DU) Shri B. M. Patil Medical College, Vijayapura, Karnataka.
REGISTRATION NO:	Karnataka Medical Council 26326
PREVIOUS EXPERIENCE:	24 years as PG Guide
ADDRESS:	Dr. MALLANNA S MULIMANI Vijayapura
CONTACT NO:	08352-EXT-2148

BIO-DATA

INVESTIGATOR NAME: Dr M.HARSHITH KUMAR

QUALIFICATION: M.B.B.S

YEAR OF COMPLETION OF

INTERNSHIP: 2021

KARNATAKA MEDICAL

COUNCIL NUMBER: KMC-156142

ADDRESS: 402, GANDIKOTA APARTMENTS, HYD.

						Peradeniya poisoning scale											
Name	**IP no.**	**Age**	**Gender**	**Time since exposure (hrs)**	**Serum cholinestaerase (U/L)**	**Consciousness score**	**Seizure score**	**Fasciculation score**	**Heart rate**	**Respiraory rate**	**Miosis score**	**PPS score**	**Severity grade**	**Total Atropine dose(mg)**	**Duration of hospital stay (days)**	**Intubated (Y/N)**	**Outcome**
Laxman waddar	160654	27	M	5.50	200	2	0	1	0	1	1	5	moderate	205.2	5	Y	Death
Roopa shrisail banikol	169791	19	F	1.50	4382	0	0	0	0	0	0	0	mild	90	7	N	Alive
Ishwar pandu bandagar	217010	65	M	3.00	206.8	1	1	1	0	0	0	3	mild	95.4	5	N	DAMA
Savithri prashanth biradar	216953	30	F	3.50	5145.4	0	0	0	0	0	0	0	mild	74	7	N	Alive
Ashwini irayya mathapati	222088	22	F	0.00	287.7	0	0	0	0	0	2	2	mild	88.2	13	N	Alive
Sidappa parasappa talawar	234925	26	F	2.50	367.6	1	0	0	0	2	2	5	moderate	160	3	Y	Death
Rukmini danasingh pujari	245066	16	F	7.00	200	1	0	0	0	0	0	1	mild	75	12	N	DAMA
Shanubai yuvraj naik	247449	28	F	4.00	4570.4	0	0	0	0	0	1	1	mild	44	7	N	Alive
Geeta jadhav	250421	23	F	1.50	200	1	0	1	0	1	2	5	moderate	126	7	N	Alive
Mahadevi parashuram rathod	250918	40	F	0.50	5943	1	0	0	0	0	0	1	mild	67.8	7	N	Alive
Vachu meghu rathod	252360	60	F	1.50	269.1	0	0	1	0	1	0	2	mild	90	4	N	DAMA
Prahalad shivappa kattimani	255424	26	M	8.00	4223	2	0	0	0	1	1	4	moderate	120	1	Y	Death
Manjunath siddaray kadimani	259855	29	M	3.50	7898.2	1	0	0	0	1	1	3	mild	105	4	Y	Death
Parashuram raju kattimani	259948	24	M	2.00	255.1	0	0	1	0	1	2	4	moderate	155.4	7	N	Alive
Roopa tiratappa hire kurabar	260663	21	F	4.00	5888.4	0	0	0	0	0	0	0	mild	60	3	N	DAMA
Mallikarjun C mathapati	261749	18	M	1.00	351.3	0	0	0	0	0	0	0	mild	64	7	N	DAMA
Aisha davalsab kalegar	261648	15	F	2.50	3727.6	0	0	1	0	0	1	2	mild	55.2	3	N	Alive
Sachin B nayakodi	265336	17	M	3.00	236.4	0	0	0	0	0	1	1	mild	58	6	N	Alive
Paramanand basppa hadapad	278468	38	M	0.50	200	0	0	0	0	0	1	1	mild	82.8	7	N	Alive
Kirati ashok bistagoud	285163	26	M	2.00	4006.6	0	0	0	0	0	0	0	mild	49.2	6	N	Alive
Basavaraj malkappa ilajeri	292982	22	M	3.50	200	0	0	0	0	0	0	0	mild	53.4	4	N	Alive
Muttappa ashok biradar	299267	30	M	3.00	2195.5	1	0	0	0	2	1	3	mild	88.2	8	N	Alive
Savitri jagadevappa	310434	23	F	5.00	200	1	1	0	0	0	2	4	moderat	137.4	8	Y	DAMA

biradar													e				
Yallappa L madar	311540	45	M	10.00	593.9	1	0	1	0	1	1	4	moderate	143.4	2	Y	Death
Manjula mallikarjun babaleshwar	333652	35	F	0.50	2221.2	0	0	1	0	0	0	1	mild	83.4	4	N	Alive
Mallikarjun kallappa kudari	334976	80	M	4.00	7911.4	0	0	0	0	0	0	0	mild	60	2	N	Alive
Mallappa S hittanahalli	347372	25	M	5.50	7569.4	0	0	0	0	0	0	0	mild	64.8	4	N	Alive
Kallappa vittal naikodi	347905	65	M	5.50	200	2	0	1	0	1	0	4	moderate	124.4	2	N	DAMA
Saibanna jateppa kambar	348268	24	M	7.00	1031.5	0	0	1	0	1	2	4	moderate	140.4	10	N	Alive
Rakmaji laxman lokhande	349160	80	M	2.50	458.7	1	0	1	0	1	1	4	moderate	121.8	1	Y	Death
Mallappa lagnappa naikodi	350411	35	M	7.00	200	1	0	1	1	1	1	5	moderate	207.6	5	N	Alive
Akshay kumar shivaray dalawai	369470	25	M	5.00	200	2	0	1	0	1	1	5	moderate	181.8	17	Y	Alive
Basavantraya gouda sidaramappa patil	379227	17	M	11.00	2762.2	0	0	1	0	1	1	3	mild	129	3	N	Alive
Ragavendra sadashiva balochi	388252	28	M	0.50	7898.2	0	1	0	0	0	0	1	mild	88.8	6	N	Alive
Kavitha rajendra bagali	390778	29	F	6.00	7248	0	0	0	0	0	0	0	mild	40.2	4	N	Alive
Megha hanamanth masyalkar	3874	18	F	4.50	990.9	0	0	1	0	1	1	3	mild	135	5	N	Alive
Roopa shivarudra shivanagi	6535	25	F	5.00	200	0	1	1	0	0	1	3	mild	137.4	9	N	Alive
Karishma irappa yaranal	9106	25	F	5.00	975.4	2	0	1	0	1	2	6	moderate	260.4	9	Y	Alive
Reshma dastagir dhadad	15013	28	F	3.50	962.3	0	0	0	0	1	0	1	mild	75	7	N	Alive
Haleppa husenappa kolinal	20208	24	M	10.00	200	0	1	1	0	0	1	3	mild	104.4	6	N	DAMA
Chandubai somalu chavan	21493	65	F	3.50	1453.3	2	0	1	2	1	2	8	severe	160	5	Y	DAMA
Irappa bela vaddagi	34785	22	M	4.00	200	2	0	1	0	1	2	6	moderate	220.2	21	Y	Alive
Anand rajshekar pujari	33745	30	M	2.00	6234.3	1	0	0	0	0	0	1	mild	128.4	8	N	Alive
Ashwini channu chavan	49032	19	F	4.00	4045	0	0	0	0	0	1	1	mild	105	4	N	Alive
Bhagyashree shivaraj alamatti	55284	21	F	2.00	6584.6	1	0	1	0	1	2	5	moderate	172.8	4	N	Alive
Archana basavaraj hosamani	57293	16	F	3.00	7112.2	1	0	0	0	0	0	1	mild	49.2	1	N	DAMA
Kashinath laxman	100240	26	M	17.00	1064	0	0	1	0	1	0	2	mild	114.6	5	N	Alive

betagoudar																	
Deepa mallikarjun dolli	112229	34	F	3.00	4110.5	2	0	0	0	1	1	4	moderate	121.2	5	N	Alive
Ambika mahantesh chavan	11223	38	F	4.00	4267.7	1	0	0	0	1	1	3	mild	90.4	2	N	DAMA
Tanuja karan logavi	120577	18	F	2.00	7781.3	0	0	1	0	1	2	4	moderate	130.8	5	N	Alive
Bharat kumar p meti	138722	20	M	6.00	4456.5	0	0	0	0	0	0	0	mild	59.4	3	N	Alive
Savithri revansidda managuli	140405	25	F	2.00	359	0	0	0	0	0	0	0	mild	54.6	6	N	Alive
Girish shankareppa masuti	143084	28	M	3.00	1961	0	0	1	0	0	1	2	mild	91.2	2	Y	Death
Sanjana suresh rathod	224626	19	F	1.50	5176	0	0	0	0	0	1	1	mild	73.2	5	N	Alive
Allisab md sab mulla	155117	52	M	12.00	1520	0	0	0	0	0	1	1	mild	106.8	4	N	DAMA
Shrikant dharma raj hasanapur	162460	35	M	3.00	200	0	0	1	0	1	0	2	mild	99	8	Y	Death
Renuka pujari	175303	25	F	6.00	200	2	0	0	0	1	1	4	moderate	123	6	N	Alive
Malingray m yaladagi	175292	32	M	6.00	351	1	0	0	0	1	1	3	mild	120.6	10	N	Alive
Laxman guranna	189796	24	M	9.50	2827	0	0	1	0	1	2	4	moderate	121.8	8	N	Alive
Praveen anil rathod	230759	20	M	3.00	6002	0	0	0	0	0	0	0	mild	49.2	6	N	Alive
Akshay bharat salunke	241312	25	M	8.00	5210	0	0	0	0	0	0	0	mild	71.4	3	N	Alive
Nana jadhav	258356	29	M	4.00	200	0	0	1	0	0	1	2	mild	91.8	9	N	Alive
Kaveri bhimaray pujari	269269	19	F	1.50	200	0	0	0	0	0	1	1	mild	72.6	18	N	Alive
Mallamma suresh kenganal	272364	32	F	16.00	1000	0	0	0	0	0	1	1	mild	85.2	6	N	Alive
Deepa namadev shinge	276797	24	F	4.00	1674	0	0	0	0	0	0	0	mild	36	5	N	Alive
Vidya shree s mamadapur	291951	18	F	8.00	4992	0	0	0	0	0	0	0	mild	49.2	5	N	Alive
Amasiddh dhondappa gheradi	1026	65	M	3.00	200	1	0	0	0	2	1	3	mild	122.4	8	N	Alive
Ashwini sambaji pawar	2048	28	F	2.00	6989	1	1	0	0	0	2	4	moderate	169.2	9	N	Alive
Arun gangayya hiremath	3216	26	M	5.00	200	1	0	1	0	1	1	4	moderate	180	9	Y	Death
Sharanappa bhimanna agasar	3910	35	M	4.00	200	0	0	1	0	0	0	1	mild	91.8	3	Y	Death
Preethi iranna hatti	5944	20	F	4.00	2338	0	0	0	0	0	0	0	mild	83.4	9	N	Alive
Aishwarya anand badiger	10714	18	F	3.00	200	0	0	1	0	0	0	1	mild	107.4	5	Y	Death

Lakshmibai bhirappa jambagi	11346	25	F	2.00	200	0	0	1	0	1	2	4	moderate	186	7	Y	Death
Supiya sadiq mulla	11339	21	F	2.00	200	1	0	1	0	1	1	4	moderate	192	6	N	Alive
Ravi mannur rathod	11733	40	M	7.00	5955.3	1	0	1	1	1	1	5	moderate	182.4	5	N	Alive
Prakash mallappa methi	12147	28	M	3.00	6966	2	0	1	0	1	1	5	moderate	201.6	6	N	Alive
Ravi kumar galave	12312	24	M	6.00	9299	0	0	1	0	1	1	3	mild	126	7	N	Alive
Baby Santhosh chavan	14661	32	F	7.00	200	0	1	0	0	0	0	1	mild	107.4	7	N	Alive
Sanika rathod	15511	18	F	4.50	2195.7	0	0	0	0	0	0	0	mild	52.2	5	N	Alive
Rohini yallappa bajantri	16801	20	F	1.50	4099.1	0	0	1	0	1	1	3	mild	106.8	5	N	Alive
Irfan alisab nagadev	17424	24	M	5.00	7221.4	0	1	1	0	0	1	3	mild	100.8	4	N	Alive
Prakash shrikant ukkli	82353	31	M	5.00	6516.8	2	0	1	0	1	2	6	moderate	207	6	N	Alive
Arun ashok pilaranakar	152300	30	M	12.00	200	0	0	0	0	1	0	1	mild	123.6	1	Y	Death
Malappa parappa meti	12616	18	M	2.50	9024.3	2	0	1	0	1	1	5	moderate	156.6	4	N	Alive
Prasad G hipparagi	13532	28	M	5.50	7784	0	0	0	0	0	0	0	mild	76.2	5	N	Alive
Yallappa sarubai banikol	15740	38	M	5.00	4055	1	1	1	0	0	0	3	mild	122.4	6	N	Alive
Sudha	15820	19	F	5.00	326	0	0	0	0	0	0	0	mild	39	5	N	Alive
Anita umesh rathod	20405	27	F	12.00	5949	0	0	0	0	0	2	2	mild	76.2	3	N	Alive
Mallanna chinnappa mudalageri	204	24	M	8.50	7476	1	0	0	0	2	2	5	moderate	165.6	3	N	Alive
Yamanappa manageri	250109147	21	M	11.50	402	1	0	0	0	0	0	1	mild	61.2	7	N	Alive
Rekha dhumagond	2501140034	23	F	5.00	6354	0	0	0	0	0	1	1	mild	57	4	N	Alive
Keerati ramesh vaddaer	2501231407	18	F	5.00	1243	1	0	1	0	1	2	5	moderate	168.6	5	N	Alive
Laxmi siddappa kokatanur	2502011456	20	F	2.00	200	1	0	0	0	0	0	1	mild	63.6	24	Y	Alive
Prakash arjun achigara	2502111464	24	M	4.00	5573	0	0	1	0	1	0	2	mild	123.6	7	N	Alive
Aishwarya B goundi	2502181457	17	F	5.50	5751	2	0	0	0	1	1	4	moderate	160.2	5	N	Alive
Shiva hiremath	2502101574	40	M	2.50	5929	1	0	0	0	1	1	3	mild	139.2	5	N	Alive
Shrusti mendegar	2502230600	19	F	1.50	6107	1	0	1	0	1	1	4	moderate	141	9	Y	Alive

Madev kotalagi	2503010003	31	M	4.50	6285	0	0	1	0	0	0	1	mild	44	8	N	Alive
Manjunath basappa kumbar	2503070478	38	M	7.50	6463	0	0	0	0	0	0	0	mild	40.2	5	N	Alive
Basavaraj sharanappa yankanchi	2503051311	30	M	10.50	6641	1	0	1	0	1	2	5	moderate	174.6	5	N	Alive