"A STUDY OF CLINICAL SEVERITY AND OUTCOME OF ORGANOPHOSPHATE POISONING PATIENTS USING PERADENIYA ORGANOPHOSPHOROUS POISONING (POP) SCALE WITH POISONING MORTALITY SCORE(PMS) AND PSEUDOCHOLINESTERASE LEVELS"

BY

DR. YERUVA NIKHIL REDDY

M.B.B.S.
POST GRADUATE (EMERGENCY MEDICINE)
DEPARTMENT OF EMERGENCY MEDICINE
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR-563101

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IN
EMERGENCY MEDICINE

Under the Guidance of

DR. MURALIMOHAN N. T

PROFESSOR & HOD

DEPARTMENT OF EMERGENCY MEDICINE
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR-563101

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Date:
Place:

DR. YERUVA NIKHIL REDDY
Postgraduate
Department of Emergency Medicine
Sri Devaraj Urs Medical College
Tamaka, Kolar.

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Date:
Place: Kolar

DR. MURALIMOHAN N. T.
Professor and HOD
Department of Emergency Medicine
Sri Devaraj Urs Medical College
Tamaka, Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

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Date:
Place: Kolar

DR. MURALIMOHAN N. T.
Professor & HOD
Department of Emergency Medicine
Sri Devaraj Urs Medical College
Tamaka, Kolar

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Signature of the Head of Department

DR. MURALIMOHAN N. T.
Professor
Department of Emergency Medicine
Sri Devaraj Urs Medical College
Tamaka, Kolar

Date:
Place: Kolar

Signature of the Principal

Dr. PRABHAKAR K
Principal
Sri Devaraj Urs Medical College
Tamaka, Kolar.

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"A Study of Clinical severity and outcome of Organophosphate poisoning patients using peradeniya Organophosphorus poisoning (POP) scale with poisoning mortality score (PMS) and pseudocholinesterase levels"

ABSTRACT

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"A Study of Clinical severity and outcome of Organophosphate poisoning patients using peradeniya Organophosphorus poisoning (POP) scale with poisoning mortality score (PMS) and pseudocholinesterase levels" ABSTRACT Background: Organophosphate (OP) poisoning is a significant public health issue, particularly in agricultural regions of developing countries. It inhibits acetylcholinesterase, leading to excessive acetylcholine accumulation and cholinergic crises. Accurate prognostication is critical for effective management, especially in resource-limited settings. Objectives: This study aimed to assess the clinical severity and outcomes of OP poisoning using the Peradeniya Organophosphorus Poisoning (POP) scale, Poisoning Mortality Score (PMS), and pseudocholinesterase (PChE) levels. Specific objectives included evaluating the

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Date: **DR. YERUVA NIKHIL REDDY**

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LIST OF ABBREVIATIONS

2-PAM	2-pralidoxime
AChE	Acetylcholinesterase
APACHE II	Acute Physiology and Chronic Health Evaluation II
AVPU	Alert, Verbal, Pain, Unresponsive
CPK	Creatine Phosphokinase
ED	Emergency Department
HR	Heart Rate
ICU	Intensive Care Unit
IMS	Intermediate Syndrome
NAD	Nicotinamide Adenine Dinucleotide
NTE	Neuropathy Target Esterase
OP	Organophosphate
OPIDN	Organophosphate-Induced Delayed Neuropathy
PAM	Pralidoxime
PChE	Pseudocholinesterase
PMS	Poisoning Mortality Score
POP	Peradeniya Organophosphorus Poisoning
RR	Respiratory Rate
SBP	Systolic Blood Pressure
SOFA	Sequential Organ Failure Assessment
WHO	World Health Organization

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ABSTRACT

BACKGROUND:

Organophosphate (OP) poisoning is a significant public health issue, particularly in agricultural regions of developing countries. It inhibits acetylcholinesterase, leading to excessive acetylcholine accumulation and cholinergic crises. Accurate prognostication is critical for effective management, especially in resource-limited settings.

OBJECTIVES:

This study aimed to assess the clinical severity and outcomes of OP poisoning using the Peradeniya Organophosphorus Poisoning (POP) scale, Poisoning Mortality Score (PMS), and pseudocholinesterase (PChE) levels. Specific objectives included evaluating the correlation of these tools with clinical outcomes such as ICU stay, ventilator requirement, and mortality.

METHODS:

A prospective observational study was conducted on 100 patients diagnosed with OP poisoning at Sri Devaraj Urs Medical College, Kolar, from January 2023 to December 2024. Severity was assessed using POP and PMS scores at admission. PChE levels were measured within 24 hours. Clinical outcomes including hospital stay, ICU duration, ventilator requirement, atropine and pralidoxime usage, and mortality were recorded. Statistical analysis included Pearson's correlation and ROC curve analysis using SPSS.

RESULTS:

Majority of patients were males (75%) aged 21–40 years. POP score showed strong correlation with ventilator days ($r=0.615$, $p<0.001$) and ICU stay ($r=0.472$, $p<0.001$). PMS scores were positively correlated with ventilator ($r=0.678$, $p<0.001$) and ICU stay ($r=0.319$, $p=0.001$). Lower PChE levels were significantly associated with worse outcomes ($r= -0.717$ for ICU stay, $p<0.001$). Mortality was highest among patients with POP score >7 and PMS >65, with AUC values of 0.968 and 0.987 respectively for predicting mortality.

Conclusion:

POP scale, PMS, and PChE levels are reliable predictors of severity and outcomes in OP poisoning. Combined use enhances early risk stratification, aiding timely intervention and resource allocation.

Keywords:

Organophosphate poisoning, POP scale, Poisoning Mortality Score, pseudocholinesterase, severity, prognosis, mortality prediction.

INTRODUCTION

INTRODUCTION

Organophosphate (OP) toxicity continues to pose a major public health challenge worldwide, especially in low and middle-income countries where pesticide application in agriculture is extensive. According to the World Health Organization (WHO), pesticide-related toxicities contribute to over 300,000 deaths each year, with OP compounds accounting for a significant proportion [(1)]. The fundamental pathophysiological mechanism of organophosphate poisoning is the irreversible inhibition of acetylcholinesterase, a critical enzyme that degrades acetylcholine at synaptic clefts and neuromuscular junctions. This enzymatic blockade results in the excessive accumulation of acetylcholine, leading to persistent stimulation of cholinergic receptors and the onset of a cholinergic crisis, which manifests as a constellation of muscarinic, nicotinic, and central nervous system symptoms [(2)]. Clinically, OP poisoning manifests across a broad spectrum, ranging from non-specific symptoms like nausea, vomiting, and dizziness to life-threatening complications such as convulsions, respiratory paralysis, and ultimately death [(3)].

To facilitate early risk assessment and guide clinical decision-making in organophosphate (OP) poisoning, several scoring systems have been introduced to evaluate disease severity and predict clinical outcomes. Among these, the Peradeniya Organophosphorus Poisoning (POP) scale—originating from Sri Lanka—has gained broad clinical acceptance. This validated tool employs six key clinical indicators: pupillary diameter, respiratory rate, muscle fasciculations, level of consciousness, seizure activity, and heart rate. Based on the composite score, patients are stratified into mild, moderate, or severe categories of OP poisoning, thereby aiding in timely and appropriate therapeutic interventions [(4)]. Despite its utility, the POP scale alone may not fully predict mortality, necessitating additional prognostic markers such as the Poisoning Mortality Score (PMS) and pseudocholinesterase (PChE) levels.

The Poisoning Mortality Score (PMS) is a recently developed tool designed to predict fatal outcomes by considering factors such as age, severity of poisoning, and laboratory parameters [5]. Studies have shown that incorporating PMS alongside clinical severity scores like the POP scale enhances the ability to predict mortality risk more accurately [6]. Similarly, pseudocholinesterase levels serve as an important biochemical marker of OP poisoning severity. Since OP compounds irreversibly inhibit cholinesterase, serum PChE levels often correlate with poisoning severity and recovery trends [7]. Lower PChE levels at presentation are associated with increased severity and poorer outcomes, making it a valuable adjunct for prognosis [8].

Several studies have explored the correlation between the POP scale and biochemical markers such as PChE, highlighting their role in guiding clinical management. A study by Peter et al. demonstrated that combining clinical scores with laboratory markers significantly improved outcome predictions in OP poisoning patients [9]. Another study by Eddleston et al. found that PChE levels, when used alongside clinical scales, provided a more comprehensive risk stratification [10]. However, the role of these markers in improving treatment outcomes and mortality reduction remains a topic of ongoing research. The present study is designed to assess the clinical severity and prognostic “outcomes in patients with organophosphate poisoning by employing the Peradeniya Organophosphorus Poisoning (POP) scale, the Poisoning Severity Score (PSS), and serum pseudocholinesterase (PChE)” activity as evaluative tools. By correlating these parameters, we seek to improve early risk stratification and guide clinical decision-making. The findings of this study could help refine existing prognostic models and contribute to better resource allocation in emergency settings.

AIMS & OBJECTIVES

AIM AND OBJECTIVES

AIM:

To assess the clinical severity and outcome of organophosphate poisoning patients using the Peradeniya Organophosphorus Poisoning (POP) scale, Poisoning Mortality Score (PMS), and pseudocholinesterase levels to evaluate their predictive value in determining patient prognosis and mortality risk.

OBJECTIVES:

1. To investigate the association between the "Peradeniya Organophosphorus Poisoning (POP)" scale and the clinical severity of organophosphate toxicity in affected individuals.
2. To evaluate the prognostic reliability of the "Poisoning Mortality Score (PMS)" in forecasting clinical outcomes, including morbidity and mortality, among patients with organophosphate poisoning.
3. To examine the correlation between serum pseudocholinesterase (PChE) activity and the severity of organophosphate poisoning, and to determine its predictive utility in clinical outcome assessment.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

"ORGANOPHOSPHOROUS COMPOUNDS CLASSIFICATION"

"Organophosphorus (OP) compounds represent a broad and structurally diverse class of chemicals, commonly encompassing derivatives of phosphoric, phosphonic, and phosphinic acids[11]. The term organophosphorus" broadly refers to substances characterized by organic groups bonded to a phosphorus atom. These compounds can be categorized into various subtypes such as anhydrides, aliphatic derivatives, aromatic compounds, and heterocyclic structures, based on their molecular configurations. A key feature of OP insecticides currently in use is the presence of four atoms bonded to the central phosphorus atom—typically three through single covalent bonds and one through a coordinate covalent bond. The physicochemical and toxicological behavior of these compounds is largely influenced by the immediate atomic environment surrounding the phosphorus center[12].

A significant proportion of organophosphate (OP) insecticides can be structurally traced back to phosphoric acid derivatives. Among these, the true phosphates—defined as triesters of phosphoric acid—are often considered the foundational prototypes of the broader OP compound class. These molecules typically feature four oxygen atoms bonded to the central phosphorus atom, conferring high reactivity. Such reactivity makes them particularly suitable for applications requiring minimal residual persistence, such as treatment of crops close to harvest or use around dairy livestock. However, most OP compounds in agricultural and veterinary practice are sulfur-substituted analogs, especially those containing thiophosphoryl groups, which modify their chemical stability and biological activity.

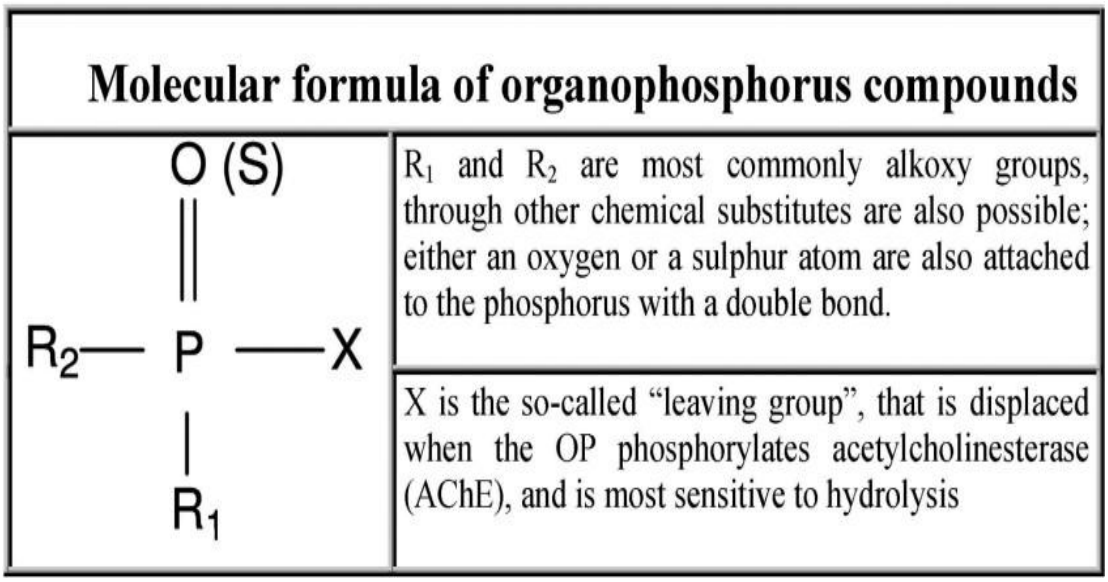

Figure 1: Structural Representation of a Generic Organophosphorus Compound

"Organophosphate (OP) molecules typically feature two alkyl groups and a third substituent—commonly referred to as the leaving group—which is more susceptible to hydrolysis than the alkyl chains. A critical distinction within this class is the presence of phosphorothioates (P=S), which exhibit minimal or no intrinsic anticholinesterase (AChE) inhibitory activity". These compounds must undergo metabolic activation through oxidative desulfuration to generate their corresponding oxon derivatives, which possess significant anti-AChE activity. Moreover, it is important to recognize that not all OP compounds are potent AChE inhibitors; some lack this bioactivity entirely and are therefore characterized by relatively low toxicity[13].

Phosphorothioate compounds encompass several widely used insecticides, including parathion, methyl parathion, diazinon, and chlorpyrifos. These agents serve as key representatives within this subclass due to their broad application in pest control. In contrast, phosphorothiolates, which exhibit significantly higher toxicity, are predominantly employed as soil-applied insecticides or systemic agents for plant protection. Another important subgroup within organophosphorus

chemistry is the phosphorothionothiolates, which primarily consist of phosphorodithioate derivatives. In these molecules, one sulfur atom is double-bonded to the phosphorus atom (P=S), while the second sulfur forms a thioester bond. Although certain organophosphates, such as sulprofos, utilize an alkyl-linked thioester, it is more common for the sulfur atom to participate in the formation of the leaving group, which plays a crucial role in the compound's biological activity and chemical reactivity.

Analogous to carboxylic acids, phosphorus-containing acids can form both esters and amides. Although the earliest organophosphate (OP) insecticides developed by Schrader were classified as phosphoramides, this subclass has remained relatively limited in commercial use. Presently, only seven phosphoramides are in active application, comprising three phosphoramidates, two phosphoramidothionates, and two phosphoramidothiolates. Another structurally distinct category includes phosphonates and related compounds, characterized by the presence of a direct phosphorus–carbon (P–C) bond. "Among these, only two compounds—trichlorfon (a phosphonate) and fonofos (a phosphonothionothiolate)—remain in use". Although phosphonothionates were previously produced, all four members of this subgroup have been phased out. Notably, EPN, a member of this class, served as a prominent insecticide for nearly four decades before being discontinued[12].

“PHARMACOLOGICAL PROPERTIES”

“The pharmacological actions of anticholinesterase (anti-ChE) agents can largely be inferred from the physiological sites where acetylcholine (ACh) is endogenously released in response to nerve impulses, the frequency of such neural activity, and the functional responses of the associated effector organs”. These agents can elicit a broad spectrum of effects, including: [14]

- Activation of muscarinic receptors at autonomic effector sites.
- An initial excitatory response at autonomic ganglia and skeletal muscles mediated through nicotinic receptors, which is subsequently followed by neuromuscular suppression or paralysis.
- Excitation of cholinergic receptors within the central nervous system (CNS), occasionally leading to CNS depression.

Lipid-soluble organophosphates are efficiently absorbed transdermally, while volatile forms readily cross the alveolar-capillary barrier in the lungs. The central and peripheral effects of anti-ChE agents—particularly those targeting muscarinic receptors in both cortical and subcortical CNS regions, as well as at peripheral autonomic sites—are effectively antagonized by atropine. In addition, atropine can mitigate certain excitatory effects of anti-ChE agents on autonomic ganglia, due to its ability to interfere with both muscarinic and nicotinic receptor-mediated neurotransmission.

“MECHANISM OF TOXICITY”

Phosphorylation of AChE

The “inhibition of acetylcholinesterase (AChE) by organophosphorus (OP) compounds is characterized by an irreversible biochemical interaction, best described as a transesterification or transphosphorylation reaction”. It is important to clarify that the term "irreversible" does not imply a permanent loss of enzymatic function per sec but rather indicates that the original OP compound is not regenerated once enzyme activity is restored. “Under physiological conditions, AChE hydrolyzes its natural substrate, acetylcholine (ACh), by facilitating acetylation of a serine hydroxyl group within its active site, thereby liberating the choline moiety. This is followed by hydrolysis of the acetylated enzyme to regenerate the active form of AChE”.

In contrast, when OP compounds inhibit AChE, they mimic the structure of ACh and covalently phosphorylate the serine hydroxyl group at the catalytic center. This phosphorylation markedly impedes enzymatic turnover, as the subsequent hydrolysis of phosphorylated AChE is extremely slow. As a result, the catalytic function of the enzyme is essentially lost for a prolonged period[16,17,18]. The effectiveness of OP compounds as anticholinesterase agents is therefore primarily determined by their capacity to phosphorylate the enzyme and the resulting resistance of the phosphorylated complex to hydrolysis.

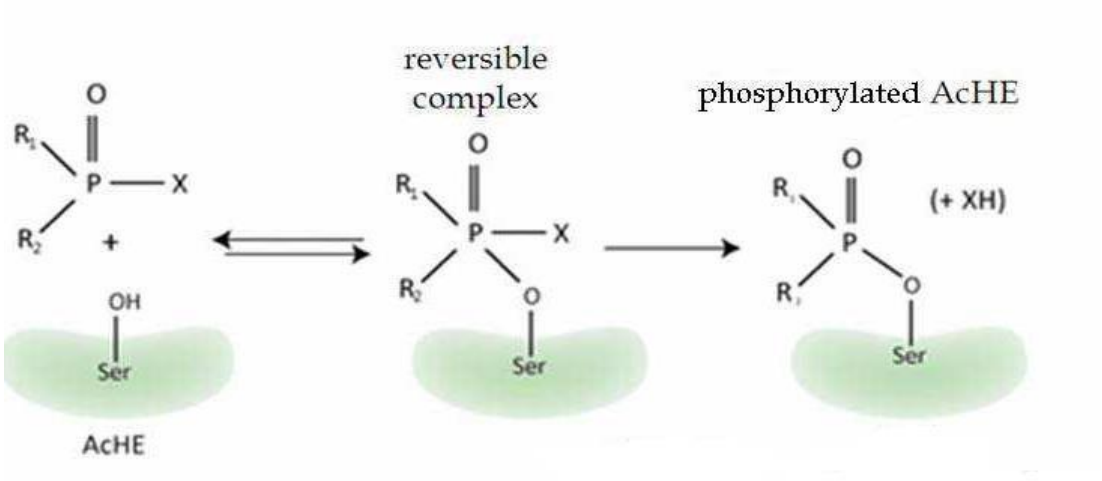

Figure 2: Phosphorylation of AChE

Organophosphate (OP) poisoning results from the pathological accumulation of acetylcholine (ACh) at cholinergic synapses and neuromuscular junctions, owing to the inhibition of acetylcholinesterase (AChE). This abnormal persistence of ACh leads to excessive stimulation of cholinergic receptors, causing hyperexcitability within affected neural circuits. Consequently, a single presynaptic stimulus may generate multiple postsynaptic responses due to sustained receptor activation. In the 'somatic nervous system', which governs 'voluntary skeletal muscle' activity, this overstimulation manifests as involuntary muscle fasciculations, tremors, tonic or clonic seizures, and in severe cases, sustained tetanic paralysis. The autonomic nervous system presents a more intricate scenario. Although both the sympathetic and parasympathetic divisions are cholinergic at their ganglionic synapses, their postganglionic neuroeffector interactions often exert antagonistic physiological effects on target tissues, complicating the clinical presentation of OP toxicity.

‘Cardiac responses to organophosphate (OP) poisoning are influenced by the dual and often opposing actions of the sympathetic and parasympathetic divisions of the autonomic nervous system’. For instance, sympathetic stimulation typically elevates heart rate, whereas parasympathetic activation exerts a bradycardic effect. As a result, the net cardiovascular outcome of OP toxicity may vary widely, manifesting as tachycardia, bradycardia, arrhythmia such as fibrillation, or even cardiac arrest, depending on the relative dominance of each autonomic branch. Adding further complexity, excessive accumulation of acetylcholine (ACh) at high concentrations can paradoxically impair cholinergic neurotransmission through a mechanism known as desensitization or depolarizing blockade. This phenomenon is especially pronounced at autonomic ganglia and at nicotinic receptors located in skeletal muscle. The interplay of these diverse and often contradictory effects contributes to the broad and variable spectrum of clinical manifestations observed in OP poisoning.

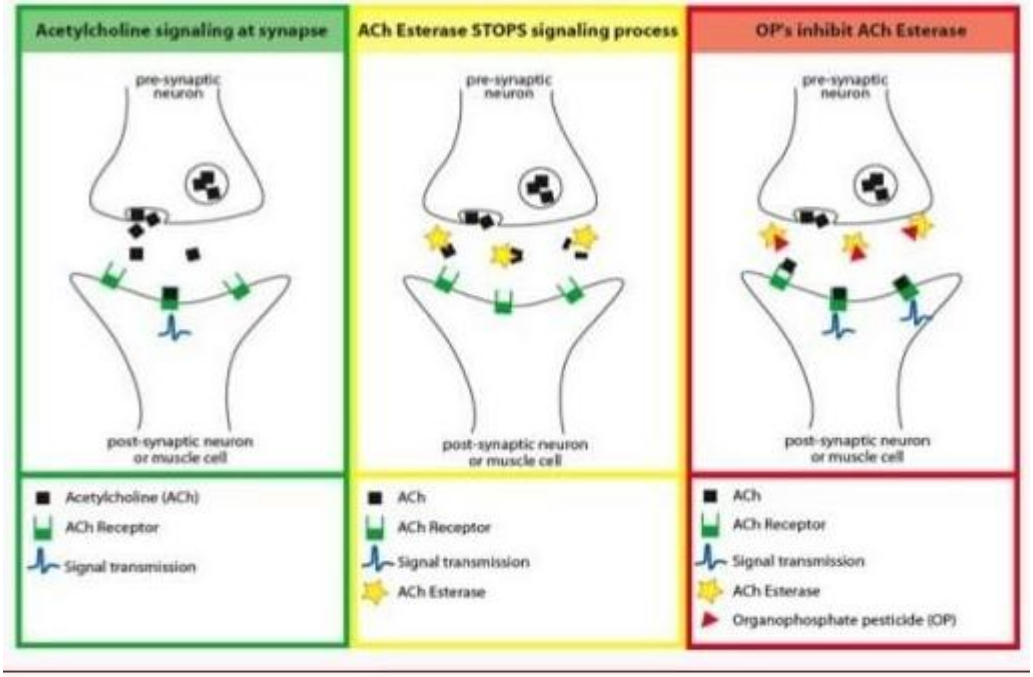

Figure 3: Mechanism of Poisoning.

Once acetylcholinesterase (AChE) is phosphorylated by organophosphate (OP) compounds, it can undergo further chemical transformations, although these processes are typically slow. Two critical post-inhibition reactions are of particular clinical interest: recovery and aging. Recovery refers to the hydrolytic cleavage of the phosphoryl group, which restores enzymatic activity. The rate of spontaneous dephosphorylation is heavily influenced by the nature of the alkyl substituents attached to the phosphorus atom. For example, dimethyl-substituted OP compounds facilitate relatively rapid recovery, with a half-life of approximately 2 hours for human erythrocyte AChE. In contrast, enzymes inhibited by diethyl phosphates show significantly slower recovery, with half-lives extending beyond 48 hours. In cases involving isopropyl-substituted OPs, enzymatic reactivation is virtually undetectable.

To accelerate the dephosphorylation process, certain chemical agents—most notably oximes—are used as nucleophilic reactivators. Among these, quaternary pyridine-2-aldoximes are considered the most effective, with 2-PAM (pralidoxime) being the most extensively studied example. These oximes act by accepting the phosphoryl group through a secondary transphosphorylation reaction, thereby liberating the active enzyme. The phenomenon known as aging presents a significant barrier to effective AChE reactivation. This process involves the loss of an alkyl side chain (dealkylation) from the phosphorylated enzyme, converting it into a monoalkyl-phosphoryl-AChE complex that is resistant to oxime-mediated reactivation. Early studies showed that AChE inhibited by OPs could be reactivated shortly after inhibition, but not after extended storage—even under refrigeration—due to this aging process. The rate of aging varies with the chemical structure of the OP compound: enzymes inhibited by methoxy-containing "OPs age more rapidly than those with ethoxy groups, while n-propoxy substitutions show minimal aging. Highly branched alkyl groups, such as isopropoxy and the 3,3-dimethyl-2-butoxy moiety found in soman, induce rapid aging". To date, no compound has proven effective at reactivating aged AChE. Thus, recovery of enzymatic function in such cases is believed to rely primarily on the synthesis of new enzyme molecules, rather than reversal of phosphorylation.

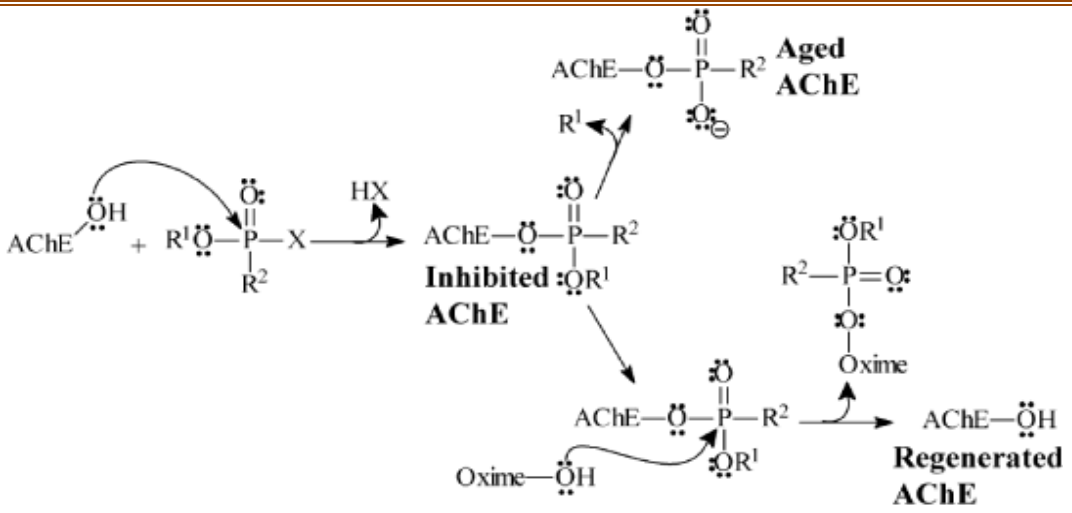

Figure 4: Reactions of Phosphorylated AChE

"TOXIC EFFECTS NOT RELATED TO AChE INHIBITION TERATOGENESIS"

Certain organophosphate (OP) compounds have been identified as teratogenic agents. In avian models, such as chick embryos, OP-induced teratogenicity has been associated with a significant reduction in nicotinamide adenine dinucleotide (NAD) levels. Interestingly, administration of NAD or its metabolic precursors has been shown to partially reverse these malformations, suggesting a critical role for cellular NAD homeostasis in embryonic development[19,20]. It has been postulated that suppression of kynurenine form amidase activity, an enzyme involved in NAD biosynthesis, may contribute to these developmental anomalies. Pharmacokinetic investigations by **Abu-Qare** and colleagues have demonstrated that the placenta provides limited protection against the transfer of methyl parathion in rat models. Their findings indicate that methyl parathion crosses the placental barrier with relative ease, raising concerns about fetal exposure during pregnancy[21,22,23].

'Among the non-cholinesterase-related toxic effects of OP compounds, organophosphate-induced delayed neuropathy (OPIDN) is the most extensively characterized. Initially described as a demyelinating syndrome', OPIDN involves a "dying-back" pattern of axonal degeneration,

predominantly affecting long myelinated nerve fibers in the peripheral and central nervous systems, such as the 'sciatic nerve and spinal cord'. Clinically, the onset of symptoms is often delayed by 10 days to several weeks post-exposure, although histopathological changes may be detectable earlier. Notably, axonal damage associated with OPIDN appears to be irreversible. The molecular target implicated in this condition is a neural enzyme referred to as 'neuropathy target esterase (NTE), also known as neurotoxic esterase', which is believed to play a central role in the pathogenesis of delayed neurotoxicity following OP exposure[24].

CLINICAL FEATURES

'The clinical manifestations of organophosphate (OP) poisoning vary based on the specific chemical agent, the dose absorbed, and the route of exposure. In most acute cases, symptoms typically emerge within the first 8 hours post-exposure, and nearly all affected individuals develop signs within 24 hours'. However, lipophilic OP compounds may undergo redistribution from adipose tissue, leading to delayed or recurrent symptomatology due to prolonged systemic exposure. Certain OP agents, such as malathion, may primarily induce localized irritation upon contact, presenting as dermatitis or bronchial irritation with symptoms such as wheezing, often in the absence of systemic cholinergic effects. There have also been isolated reports of persistent reactive airway disease that appear to occur independently of cholinesterase inhibition, suggesting alternative mechanisms of pulmonary toxicity[25,26].

Clinically, acute OP poisoning is known to progress through three distinct toxicological phases:

1. Acute cholinergic crisis – resulting from excessive accumulation of acetylcholine at synapses,
2. Intermediate Syndrome (IMS) – characterized by neuromuscular weakness developing 24–96 hours after exposure, and

3. Delayed polyneuropathy – a late-onset axonopathy that typically emerges several weeks following exposure[25,26].

Acute systemic poisoning by organophosphates (OPs) presents with a wide range of clinical features involving the 'central nervous system (CNS), muscarinic and nicotinic receptors, and somatic motor pathways'. In cases of mild to moderate toxicity, symptomatology varies in its pattern and intensity, often presenting in diverse combinations. The latency period before symptom onset is influenced by the route of exposure—inhalational exposure typically results in the most rapid onset, while transdermal absorption leads to a more delayed presentation[27]. However, compromised skin integrity, such as abrasions or dermatitis, can significantly accelerate systemic absorption through the skin. In instances of massive oral ingestion, symptoms may manifest within minutes. Central cholinergic overstimulation can produce a spectrum of neuropsychiatric and neurological signs, including anxiety, irritability, emotional instability, tremors, headache, dizziness, disorientation, delirium, hallucinations, and seizures. In severe cases, profound central nervous system depression may occur, potentially progressing to coma, accompanied by suppression of the respiratory and cardiovascular control centers [28].

Aggressive or erratic behaviour has occasionally been reported in individuals affected by organophosphate (OP) toxicity. A central mechanism underlying many clinical manifestations is the inhibition of acetylcholinesterase (AChE) within the parasympathetic division of the autonomic nervous system. This inhibition leads to excessive stimulation of muscarinic receptors by accumulated acetylcholine, producing a classic constellation of symptoms: 'salivation, lacrimation, diaphoresis, urinary incontinence, diarrhoea, gastrointestinal cramping, emesis, and bradycardia'.

In severe poisoning, both bradycardia and tachycardia may be observed, depending on the balance of parasympathetic and sympathetic nervous system responses. Excessive cholinergic

activity in the respiratory tract may result in bronchorrhea and bronchospasm, potentially leading to hypoxia and reflex tachycardia. Ocular findings such as miosis (pinpoint pupils) and blurred vision arise from acetylcholine-mediated contraction of the pupillary constrictor muscles and ciliary body. Given this profile, organophosphate poisoning should be included in the differential diagnosis of any patient presenting with 'altered mental status and bilateral pinpoint pupils', especially when accompanied by signs of cholinergic excess.

"Acetylcholine (ACh) serves as the primary neurotransmitter at nicotinic receptors within the sympathetic ganglia and the adrenal medulla". Excessive stimulation at these sites due to organophosphate (OP) toxicity can result in 'pallor, mydriasis, tachycardia, and hypertension'. Although parasympathetic activity generally dominates in OP poisoning, the clinical picture often reflects mixed autonomic responses[29,30]. At the neuromuscular junction, nicotinic receptor overstimulation produces muscle fasciculations, cramping, and generalized weakness, which can progress to flaccid paralysis and loss of reflexes. In advanced cases, this may mask seizure activity and ultimately result in 'respiratory muscle paralysis, leading to acute respiratory failure 'and potentially death. Among the clinical features, 'miosis and muscle fasciculations 'are considered reliable diagnostic indicators of OP poisoning.

A distinct neurological complication known as the Intermediate Syndrome (IMS) can develop 1 to 4 days following acute OP exposure, particularly after ingestion, and has been observed in up to 20% of cases. IMS is characterized by paralysis of the neck flexors, cranial nerve–innervated muscles, proximal limb muscles, and respiratory musculature, often necessitating mechanical ventilation. Notably, these patients usually do not exhibit ongoing signs of cholinergic excess, which can make diagnosis challenging. Electromyography (EMG) can aid in identifying this syndrome. Prompt initiation of antidotal therapy—such as atropine and oximes—combined with intensive supportive care, may reduce the severity or prevent the progression of

IMS. Most patients recover within 5 to 18 days. IMS has been most associated with exposure to specific dimethoxy OP compounds, including fenthion, dimethoate, and monocrotophos [31].

While the Intermediate Syndrome (IMS) has been frequently associated with exposure to dimethoxy organophosphates such as fenthion, dimethoate, and monocrotophos, it is not exclusive to these agents. Other compounds, including parathion, a combination of 'ethylparathion and methylparathion, methamidophos, dichlorvos, and several additional organophosphate insecticides, have also been implicated in the onset of IMS'[32-35]. Another serious neurologic complication, organophosphate-induced delayed neuropathy (OPIDN), typically emerges one to three weeks after the initial poisoning event. 'This condition is a mixed sensorimotor neuropathy attributed to the inhibition of neuropathy target esterase (NTE). Clinical presentation may begin with leg cramps and paresthesia, progressing to muscle weakness and flaccid paralysis', often mimicking the clinical pattern of Guillain-Barré syndrome. Affected individuals commonly report tingling sensations, followed by sensory loss in the extremities, difficulty walking, and abnormal deep tendon reflexes. Over time, motor nerve conduction slows, and patients may develop bilateral, symmetrical weakness, particularly affecting the distal muscles of both upper and lower limbs[36].

OPIDN may result in irreversible neurological and neurobehavioral deficits, such as persistent paralysis and cognitive or psychiatric disturbances following acute poisoning. In animal studies, progressive ataxia has been documented in several species—cats, sheep, horses, water buffalo, and ferrets—though notably, rodents do not prominently display ataxia as a symptom of OPIDN. Importantly, organophosphates with high lipid solubility may not cause immediate symptoms of toxicity. Instead, their prolonged tissue retention can lead to delayed-onset neurological sequelae. Furthermore, chronic low-level exposure to OP compounds—common among agricultural workers, pesticide manufacturing personnel, pest control professionals, and individuals using cholinergic ophthalmologic medications—can contribute to subclinical neurotoxicity and long-term health effects[37-40].

In cases of chronic or low-level organophosphate (OP) exposure, clinical manifestations may be subtle and nonspecific. Individuals may present with headache, nausea, generalized weakness, fatigue, diarrhea, or a mild cholinergic symptom complex that is often overlooked. Prolonged exposure has also been associated with neuropsychiatric consequences, including cognitive deficits, memory impairment, and mood disorders such as depression. Children are particularly vulnerable to OP toxicity due to their smaller body mass and inherently lower baseline cholinesterase levels, which heighten their susceptibility to enzyme inhibition and systemic effects[41-44].

In addition to agricultural and industrial OP compounds, several organophosphates have been weaponized as nerve agents, including sarin, soman, tabun, and VX. These agents act by irreversibly inhibiting acetylcholinesterase, leading to profound cholinergic crisis. They are extremely potent and fast-acting, with fatalities potentially occurring within minutes following inhalation or transdermal exposure. Of particular concern is soman, which undergoes rapid "aging"—a chemical transformation that renders the enzyme-inhibitor complex resistant to reactivation—thereby leaving a very narrow window for effective antidotal intervention [45,46].

DIAGNOSIS

‘The diagnosis of ‘organophosphate (OP)’ poisoning relies on a combination of clinical history, recognition of a characteristic toxidrome, and laboratory confirmation’. A suggestive exposure history, coupled with clinical signs consistent with cholinergic excess, is central to initial identification. The presence of a distinctive odor—often described as hydrocarbon-like or garlic-like—can also aid in early suspicion of OP exposure. ‘The cholinergic toxidrome may present with a spectrum of symptoms depending on the relative predominance of muscarinic, nicotinic, or central nervous system involvement, as well as the severity and route of intoxication’. Laboratory evaluation includes functional assays of cholinesterase activity, particularly in plasma

(pseudocholinesterase) and red blood cells (true acetylcholinesterase). While red blood cell AChE activity more accurately reflects synaptic enzyme inhibition and correlates with clinical severity, plasma cholinesterase measurements are more readily available and commonly used in clinical settings[45,46]. The extent of enzyme inhibition required to induce symptoms is not fixed, but clinical manifestations typically emerge when cholinesterase activity drops below 50% of the individual's baseline level. 'Reference laboratory testing for specific OP compounds may provide confirmatory evidence but is often reserved for specialized settings due to limited availability' [47].

Although cholinesterase activity is theoretically expected to correlate with the severity of organophosphate (OP) toxicity, discordance between enzyme levels and clinical presentation is not uncommon. Some symptomatic individuals may exhibit cholinesterase levels within normal limits, leading to false-negative results. Following OP exposure, recovery of enzyme function is a prolonged process. In the absence of early administration of pralidoxime—prior to the "aging" of the phosphorylated enzyme—plasma cholinesterase levels may require '4 to 6 weeks, and red blood cell acetylcholinesterase (AChE) activity may take 90 to 120 days to return to baseline'[48,49]. The prognostic utility of plasma cholinesterase levels in acute OP poisoning is debated. While several studies report no significant correlation with atropine requirements, mechanical ventilation needs, or mortality, other investigations suggest that plasma cholinesterase may still hold prognostic value under certain conditions. In cases of gradual cholinesterase inhibition, such as during chronic exposure, clinical symptoms are often mild or nonspecific, making diagnosis more challenging [50-53].

It is important to consider that plasma cholinesterase levels can be reduced in a variety of non-toxicological conditions, including "genetic polymorphisms, chronic diseases, hepatic dysfunction, cirrhosis, malnutrition, low serum albumin states, infections, neoplasms, and pregnancy". Conversely, red blood cell AChE activity may be influenced by disorders affecting erythrocyte turnover, such as hemoglobinopathies. While routine laboratory findings in OP

poisoning are not diagnostic, they may reveal ‘pancreatitis, hypo- or hyperglycemia, leukocytosis’, or abnormal liver function tests. In severe cases, chest radiographs may show pulmonary edema, and electrocardiographic abnormalities may reflect both toxicity and prognosis. ‘Common ECG findings include ventricular arrhythmias, torsades de pointes, idioventricular rhythms, atrioventricular conduction blocks, and QT interval prolongation. A prolonged QTc interval is especially associated with increased severity and mortality in OP poisoning’. Additionally, electromyography (EMG) may assist in identifying and quantifying AChE inhibition at the neuromuscular junction[54].

TREATMENT

Initial management of a patient with suspected organophosphate (OP) poisoning should involve prompt initiation of supportive care, beginning with placement on supplemental oxygen, cardiac monitoring, and pulse oximetry. Administration of oxygen via a 100% non-rebreather mask is particularly beneficial in individuals with bronchospasm and excessive airway secretions, as it enhances oxygenation and may reduce the risk of ventricular arrhythmias during antidotal treatment.

Airway clearance is crucial and can be facilitated by gentle suctioning to manage hypersalivation, bronchorrhea, or emesis. In cases of coma, seizure activity, respiratory failure, or severe bronchospasm, endotracheal intubation may be warranted to secure the airway and ensure adequate ventilation. When neuromuscular blockade is required, a non-depolarizing neuromuscular blocking agent is preferred. Succinylcholine should be avoided due to its metabolism by plasma cholinesterase; in OP poisoning, inhibited enzyme activity may result in prolonged neuromuscular paralysis. Establishing an intravenous (IV) line is essential for fluid administration and medication delivery, along with obtaining baseline blood samples for laboratory analysis, including cholinesterase levels. If hypotension is present, initial management should include fluid resuscitation with isotonic crystalloids to restore circulatory stability[55-57].

Gastric decontamination remains a commonly practiced intervention following organophosphate (OP) ingestion, particularly in parts of Asia, despite limited evidence supporting its efficacy in improving clinical outcomes. Due to the rapid gastrointestinal absorption of many OP compounds, gastric lavage is unlikely to be beneficial unless performed within two hours of a substantial ingestion[58]. Additionally, airway protection is essential prior to the procedure to mitigate the risk of aspiration, especially when hydrocarbons are involved as the vehicle for ingestion. Activated charcoal is occasionally recommended based on its demonstrated ability to bind organophosphates in vitro [59]. However, clinical studies have not confirmed a definitive benefit from either single or multiple doses in improving patient prognosis. Cathartics are generally avoided in patients with significant diarrhea resulting from cholinergic hyperactivity. Therapies such as urinary alkalinization, commonly practiced in countries like Brazil and Iran, lack robust controlled evidence to support their use. Similarly, hemodialysis, hemofiltration, and hemoperfusion have not shown proven benefit in the management of OP toxicity.

'Definitive treatment relies on antidotal therapy, specifically with atropine and pralidoxime. Atropine, a competitive muscarinic receptor antagonist, counteracts the central and peripheral effects of excess acetylcholine due to parasympathetic overactivity'. The dose is titrated to clinical effect, particularly until tracheobronchial secretions diminish—a key therapeutic endpoint. "Mydriasis (pupil dilation) is not considered a reliable marker of atropine efficacy. Importantly, atropine administration should not be withheld in the presence of tachycardia, which may result from hypoxia, respiratory muscle paralysis, or autonomic ganglionic stimulation rather than the drug itself". The initial adult dose is typically 1 mg or more IV, while in children, a dose of 0.01 to 0.04 mg/kg (with a minimum of 0.1 mg) is recommended. If intravenous access is not immediately available, intramuscular administration of up to 6 mg is appropriate. In general, if no antimuscarinic effects are observed after a trial dose of atropine, this may serve as a diagnostic indicator of OP poisoning.

Atropine dosing may be repeated at 5-minute intervals until muscarinic symptoms—particularly excessive bronchial secretions and bradycardia—are adequately controlled. In cases of severe organophosphate poisoning, especially involving large-volume ingestion, cumulative atropine doses may reach several hundred milligrams, and prolonged administration may be required. Inadequate atropinization can result in treatment failure, underscoring the importance of titrating to effective clinical endpoints. To further manage pulmonary cholinergic symptoms, nebulized anticholinergics such as atropine or ipratropium may be employed. 'However, agents like ipratropium and glycopyrrolate, which do not cross the blood–brain barrier, are ineffective in treating central neurological manifestations of OP toxicity. Notably, atropine does not reverse nicotinic symptoms, such as skeletal muscle weakness or paralysis'.

For this purpose, oxime compounds, such as pralidoxime, are utilized to reactivate acetylcholinesterase by removing the phosphoryl group bound to the enzyme's active site. Pralidoxime acts by restoring AChE function and may also assist in detoxifying unbound organophosphate molecules, thereby reducing further systemic toxicity. Clinically, pralidoxime can alleviate muscarinic, nicotinic, and central nervous system symptoms, including reversal of neuromuscular paralysis if administered before the aging process occurs—a biochemical transformation that renders enzyme reactivation impossible. Although its efficacy is highest when given early, pralidoxime is still recommended even after 24 to 48 hours post-exposure, particularly in moderate to severe poisoning. The recommended dose for adults is 1 to 2 grams, while in pediatric patients, 20 to 40 mg/kg (up to 1 gram) is advised. 'The drug should be diluted in normal saline and administered via intravenous infusion over 5 to 10 minutes'.

Clinical response to pralidoxime therapy is typically observed within 10 to 40 minutes and is characterized by a reduction in muscle fasciculations and weakness, alongside improvement in muscarinic symptoms when administered concurrently with atropine. While intravenous administration is preferred, intramuscular (IM) delivery is an alternative when IV access is limited.

In cases where neuromuscular paralysis persists or recurs, a continuous infusion of 500 mg/hour in adults or 5 to 10 mg/kg/hour in children is considered more effective than repeated bolus doses. The 'World Health Organization (WHO) recommends an alternative regimen consisting of a 30 mg/kg IV bolus, followed by a continuous infusion at 8 mg/kg/hour'. Pralidoxime therapy is generally continued for 24 to 48 hours, with regular monitoring of cholinesterase activity to assess therapeutic efficacy. While some clinical observations and animal studies have demonstrated promising outcomes, including enhanced survival and acetylcholinesterase regeneration, high-quality evidence supporting oxime efficacy in acute OP poisoning remains limited. Despite widespread use, the clinical benefit of oximes, including pralidoxime, remains a topic of ongoing debate. Management of seizures in OP poisoning includes airway protection, supplemental oxygen, benzodiazepines, and antidotal therapy with 'atropine and pralidoxime'. Atropine may also serve to prevent or mitigate early-onset seizures caused by cholinergic overstimulation. Additionally, diazepam has been shown in animal models of nerve agent toxicity to attenuate central nervous system effects and improve survival outcomes.

Management of pulmonary complications in organophosphate poisoning, including pulmonary edema and bronchospasm, requires a comprehensive approach involving supplemental oxygen, endotracheal intubation, positive pressure ventilation, and administration of antidotes such as atropine and pralidoxime. Cardiac rhythm disturbances, including both bradycardia and tachycardia, are addressed with supportive care and anticholinergic therapy. Importantly, the presence of tachycardia does not contraindicate atropine use, as it may reflect compensatory responses to hypoxia secondary to 'bronchospasm or bronchorrhea, both of which can be alleviated by atropine. Certain medications, including succinylcholine, ester-based anesthetics, and β-adrenergic blockers, may exacerbate the toxic effects of organophosphates and should therefore be avoided during management'. These agents may either prolong neuromuscular paralysis or worsen cholinergic symptoms, further complicating clinical outcomes.

"Peradeniya Organophosphorus Poisoning (POP) Scale"

'The Peradeniya Organophosphorus Poisoning (POP) Scale is a clinically validated scoring tool developed to evaluate the severity of organophosphate poisoning upon patient admission'. Introduced in 1993 by **Senanayake et al.** [60] colleagues at the University of Peradeniya in Sri Lanka, the scale was specifically designed to offer a straightforward and practical method for assessing poisoning severity, particularly in resource-constrained healthcare settings. It provides a structured approach to clinical grading, enabling timely and effective triage and management of OP poisoning cases.

Development and Purpose

The POP Scale was introduced to provide an objective and uniform method to classify the severity of OP poisoning without requiring laboratory investigations, which may not always be available in developing countries. It assesses clinical parameters reflecting muscarinic, nicotinic, and central nervous system effects of OP poisoning [61].

Components of the POP Scale

The POP Scale consists of six clinical parameters, each scored from 0 to 2, leading to a maximum score of 11. Patients are classified into mild, moderate, or severe poisoning categories based on their total score [62].

Table 1: Components of the POP Scale classification

Parameter	**'Score 0'**	**'Score 1'**	**'Score 2'**
"Pupil Size"	Normal	Pinpoint	Mid-dilated
"Respiratory Rate"	< 20/min	20-30/min	> 30/min
"Heart Rate"	> 60/min	50-60/min	< 50/min
"Fasciculations"	None	Localized	Generalized
Level of Consciousness	Conscious	Drowsy	Unconscious
Seizures	Absent	-	Present

Classification Based on Total Score

- **Mild Poisoning (Score: 0-3)** → Patients with minimal symptoms, usually do not require ICU admission.
- **Moderate Poisoning (Score: 4-7)** → Increased risk of complications, often requiring close monitoring.
- **Severe Poisoning (Score: 8-11)** → High risk of ventilatory support, ICU admission, and mortality.

Clinical Application and Prognostic Value

- The POP Scale is widely used in emergency settings to predict the need for ventilatory support**,** ICU admission, and mortality risk.
- Studies have shown a strong correlation between higher POP scores and clinical severity, including increased atropine requirements, hospital stay, and ventilatory support needs.
- Several studies have validated the POP scale as a reliable and cost-effective tool for early risk stratification in OP poisoning patients[3].

Limitations of the POP Scale

- The POP Scale does not include laboratory markers such as serum cholinesterase levels, which can provide additional prognostic insights.
- Variability in clinical assessment can lead to differences in scoring among physicians.
- 'Seizures are not always a reliable early indicator in OP poisoning cases, which may lead to underestimation of severity'.

Poisoning Mortality Score (PMS)

Introduction to the Poisoning Mortality Score (PMS)

The Poisoning Mortality Score (PMS) is a clinical scoring system developed to predict mortality risk in patients with acute poisoning, particularly organophosphate (OP) poisoning. It is

designed to be a comprehensive, objective, and quantitative tool to aid in clinical decision-making, stratification of risk, and early intervention. 'Traditional severity scores, such as the Peradeniya Organophosphorus Poisoning (POP) Scale, are limited to clinical assessments, while the PMS incorporates a broader range of physiological parameters to improve prognostication in poisoned patients' [63].

"Components of the Poisoning Mortality Score (PMS)"

The PMS score integrates ten predictors; each assigned a specific score based on statistical significance and clinical impact. These include vital signs, neurological status, poisoning intent, and systemic effects. Each patient's PMS score is calculated by summing the individual parameter scores.

Table 2: Components of the Poisoning Mortality Score (PMS)

Parameter	**Scoring Criteria**	**Points Assigned**
Age	< 30 years / ≥ 30 years	0 / 1
Sex	Male / Female	0 / 1
Type of Poison	Non-toxic / Toxic substances	0 / 2
Intent of Poisoning	Accidental / Suicidal	0 / 3
Route of Poisoning	Oral / Other	0 / 1
Systolic Blood Pressure (SBP)	>90 mmHg / ≤90 mmHg	0 / 2
Heart Rate	<100 bpm / ≥100 bpm	0 / 1
Respiratory Rate	<24 breaths/min / ≥24 breaths/min	0 / 2
Temperature	<38°C / ≥38°C	0 / 1
AVPU Scale (Neurological Status)	Alert, Voice, Pain, Unresponsive (AVPU Score)	0-3

Classification of PMS Scores and Mortality Risk

The total PMS score is used to categorize patients into four risk groups, with higher scores indicating increased mortality risk.

Table 3: Classification of PMS Scores and Mortality Risk

PMS Score	Risk Level	Mortality Risk (%)
0 - 30	**Very Low Risk**	<5%
31 - 50	**Low Risk**	5 - 15%
51 - 65	**Intermediate Risk**	16 - 30%
> 65	**High Risk**	>30%

Patients with PMS scores above 65 have a significantly higher likelihood of requiring ICU admission, mechanical ventilation, and experiencing mortality compared to those with lower scores.

Clinical Applications of PMS

1. **Early Identification of High-Risk Patients:** Helps in prioritizing critical care and timely interventions.
2. **ICU Admission Criteria:** Patients with PMS > 50 may require ICU admission and close monitoring.
3. **Guiding Antidote Therapy:** Helps in determining the need for aggressive atropine and pralidoxime therapy.
4. **Resource Allocation:** Useful in developing countries where ICU beds and ventilators are limited.

Table 4: Comparison of PMS with Other Scoring Systems

Scoring System	Parameters Used	Predictive Accuracy for Mortality
POP Scale	Clinical signs only (e.g., pupil size, fasciculations)	Moderate
SOFA Score	Organ failure parameters	High
APACHE II Score	Physiological & laboratory markers	Very High
PMS Score	Clinical, physiological & systemic markers	High

Studies comparing PMS, SOFA, and APACHE II scores found that PMS had a strong correlation with mortality prediction, but SOFA and APACHE II had slightly higher predictive accuracy in ICU settings [3]

Limitations of the PMS Score

1. **Does not include laboratory markers** like serum **cholinesterase levels**, which can further refine prognosis.
2. **Subjective assessment** (e.g., AVPU scale) may vary between observers.
3. **Limited external validation**—requires further studies in different poisoning types beyond organophosphates.

The Poisoning Mortality Score (PMS) is a useful, cost-effective, and easy-to-use tool in emergency medicine for assessing mortality risk in poisoned patients, especially those with organophosphate poisoning. It is more comprehensive than the POP Scale and has been shown to be comparable to 'SOFA and APACHE II scores in predicting mortality risk'. However, further external validation is needed to refine its application across different types of poisoning cases [64].

Pseudocholinesterase Levels and Severity in Organophosphorus Poisoning

Introduction to Pseudocholinesterase

Pseudocholinesterase (PChE), also known as butyrylcholinesterase (BChE), is an enzyme synthesized in the liver that plays a crucial role in metabolizing choline esters such as succinylcholine and various toxins, including organophosphates (OPs). 'Organophosphorus poisoning occurs due to irreversible inhibition of cholinesterases, leading to an accumulation of acetylcholine at cholinergic synapses, which results in cholinergic overstimulation'. PChE levels are used as a diagnostic and prognostic biomarker to assess the severity of OP poisoning [65].

Role of Pseudocholinesterase in Organophosphorus Poisoning

1. **Diagnosis:** A decrease in PChE activity is an important biochemical marker of OP poisoning.
2. **Severity Assessment:** Lower PChE levels correlate with more severe poisoning, increased risk of respiratory failure, and higher mortality.
3. **Prognosis:** Monitoring PChE activity during treatment helps assess response to therapy and recovery.

Classification of PChE Levels and Poisoning Severity

Organophosphate poisoning severity can be graded based on serum PChE levels, with lower values indicating more severe intoxication.

Table 5: Classification of PChE Levels and Poisoning Severity

Pseudocholinesterase (U/L)	Severity of Poisoning	Clinical Implication
>8000 U/L	Normal	No poisoning or mild exposure
4000 – 8000 U/L	Mild Poisoning	Symptoms may be mild; no ventilation needed
1000 – 4000 U/L	Moderate Poisoning	Increased risk of respiratory failure
<1000 U/L	Severe Poisoning	High likelihood of requiring mechanical ventilation, ICU admission, and increased mortality risk

Clinical Correlation of PChE with Severity and Outcome

1. Severely poisoned patients (PChE <1000 U/L) → High risk of mechanical ventilation and mortality.
2. Moderately poisoned patients (PChE 1000-4000 U/L) → Require close monitoring and atropine therapy.
3. Mildly affected patients (PChE >4000 U/L) → Usually recover with minimal intervention.
4. Recovery of PChE levels is correlated with clinical improvement, but it may take weeks to months due to slow hepatic regeneration.

Comparison of Pseudocholinesterase with Other Biomarkers **w**hile PChE is a widely used marker for OP poisoning, other biochemical markers such as red blood cell acetylcholinesterase (RBC AChE), serum amylase, and creatine phosphokinase (CPK) may provide additional prognostic insights. Studies show that PChE levels correlate well with both POP Scale scores and ventilatory requirements. However, combining clinical scores (e.g., Peradeniya OP Scale, PMS) with biochemical markers like PChE improves prognostic accuracy .[65]

Table 6: Comparison of Pseudocholinesterase with Other Biomarkers

Biomarker	Diagnostic Utility	Limitations
PChE	Quick, widely available, cost-effective	High variability, liver disease can affect levels
RBC AChE	More specific to OP poisoning severity	Expensive, requires specialized labs
Serum Amylase	Indicates pancreatic involvement	Not specific for OP poisoning
CPK	Correlates with muscle damage	Elevated in multiple conditions

Limitations of Pseudocholinesterase as a Marker

1. Inter-Individual Variability: PChE levels can vary due to genetic polymorphisms, liver disease, malnutrition, or chronic illnesses.
2. Slow Recovery: PChE activity may take weeks to months to normalize, making it less useful for short-term monitoring.
3. Not Organophosphate-Specific: PChE inhibition occurs in both OP and carbamate poisoning, making differential diagnosis difficult.

Conclusion

Pseudocholinesterase (PChE) levels are valuable biochemical markers in diagnosing and predicting severity of organophosphorus poisoning. While low PChE levels correlate with increased mortality, mechanical ventilation, and ICU admission, their variability and slow regeneration limit their use in real-time monitoring. Combining PChE with clinical severity scores (POP Scale, PMS) enhances prognosis and risk stratification in OP poisoning management [63].

LITERATURE PUBLISHED:

1. **Dubey et al. (2016)** 'conducted a prospective observational study' at Hamidia Hospital, Bhopal, India, on 100 patients with organophosphorus poisoning. The study aimed to assess the correlation between the severity of poisoning using the Peradeniya Organophosphorus Poisoning (POP) scale and serum amylase and creatine phosphokinase (CPK) levels. Results showed a significant correlation ($p < 0.0001$) between POP scores and elevated serum amylase and CPK levels. Serum amylase was found to be a better predictor of ventilator requirement and mortality compared to CPK levels (Coefficient=0.44 vs. 0.17). The study concluded that serum amylase and CPK levels correlate well with the severity of poisoning and can serve as useful biomarkers in clinical settings[66].
2. **Malaviya et al. (2023)** 'conducted a prospective observational study' at Sir Sayajirao General Hospital, Baroda, India, on 60 patients with acute organophosphorus poisoning. The study evaluated the prognostic utility of the POP scale in predicting severity and the need for ventilatory support. The results showed that 61.7% of patients with mild POP scores and 100% of those with moderate scores required mechanical ventilation. Mortality was 15% in mild cases and 31% in moderate cases. The study concluded that the POP scale is an effective prognostic tool, but its accuracy improves when combined with other clinical and biochemical markers [67].
3. **Chaudhary et al. (2019)** conducted a cross-sectional study at BP Koirala Institute of Health Sciences, Nepal, correlating clinical scores and serum acetylcholinesterase (AChE) levels in organophosphate poisoning cases. The study found that a higher POP score correlated with lower AChE levels, increased need for atropine, longer hospital stays, and poorer outcomes. A significant inverse correlation was observed between POP scores and AChE levels

(coefficient -0.356; P=0.001). The study concluded that the POP score and AChE levels are reliable indicators of poisoning severity and clinical outcomes [65].

4. **Ahmed et al. (2014)** conducted a retrospective study in the ICU of a tertiary hospital in India, analyzing 117 patients with organophosphorus poisoning requiring mechanical ventilation. The study found that mortality was directly related to the duration of mechanical ventilation, with a 33.3% death rate in patients ventilated for more than seven days. Delayed administration of pralidoxime (PAM) increased mortality significantly. The study concluded that early administration of PAM and timely mechanical ventilation significantly reduce mortality in organophosphate poisoning cases [68].

5. **Krishna Moorthy et al. (2023)** performed a retrospective study on 236 patients with organophosphate poisoning at a tertiary hospital in India. The study compared the predictive accuracy of the newly developed Poisoning Mortality Score (PMS) against the Sequential Organ Failure Assessment (SOFA) and APACHE II scores. Results showed that PMS had a high predictive value for mortality, with a sensitivity of 77.27% and specificity of 96.26%. The study concluded that PMS is a reliable tool for predicting mortality and guiding clinical decision-making in organophosphate poisoning cases [63].

6. **Kamath et al. (2021)** conducted a prospective study on 100 patients with organophosphate poisoning at Tata Main Hospital, Jamshedpur, India. The study aimed to evaluate the prognostic value of the POP scale. Results showed that 100% of patients with a POP score of 8-11 required ventilator support, and higher POP scores were significantly associated with increased atropine requirements, ICU stays, complications, and mortality. The study concluded that the POP scale is a useful prognostic tool in organophosphate poisoning cases [62].

7. **Chintale et al. (2016)** 'performed a cross-sectional study at a rural tertiary care center in India' on acute pesticide poisoning cases. The study found that pesticide poisoning was more

common in males (74.26%) and that excessive salivation was the most frequent symptom (72.05%). The study concluded that the severity of poisoning and mortality depend on the amount of exposure, time to treatment, and availability of medical facilities [69].

8. **Eddleston et al. (2008)** reviewed the management of acute organophosphorus poisoning, highlighting the lack of standardized treatment protocols. The study emphasized early resuscitation with atropine, oxygen, respiratory support, and fluids. The role of oximes was found to be unclear, with potential benefits only in moderate poisoning. The study recommended banning highly toxic organophosphorus pesticides as a primary method to reduce mortality [3].

Arvind Kumar et al. (2024). The study concluded that serum pseudocholinesterase (PChE) levels significantly decreased with increasing severity of organophosphorus (OP) poisoning, as classified by the Peradeniya OP Poisoning (POP) scale. Lower PChE levels correlated with more severe poisoning and longer ICU stays. No direct cardiac abnormalities were found via echocardiography. Additionally, a low Glasgow Coma Scale score and elevated respiratory rate at admission were linked to poorer clinical outcomes, highlighting their prognostic value[70].

MATERIALS & METHODS

MATERIAL AND METHODS

Source of Data: The study was conducted in the Department of Emergency Medicine of 'Sri Devaraj Urs Medical College Tamaka Kolar' involving patients diagnosed with organophosphate poisoning.

Study Population: 'Patients presenting with organophosphate poisoning to the emergency department or admitted to the ICU of Sri Devaraj Urs Medical College Tamaka, Kolar during the study period'.

Inclusion Criteria:

1. Patients above 18 years of age diagnosed with organophosphate poisoning.
2. Patients with a history of organophosphate exposure confirmed by clinical features and history.
3. Patients with pseudocholinesterase level reports available within 24 hours of hospital admission.
4. Patients or their guardians providing informed consent to participate in the study.

Exclusion Criteria:

1. Patients with mixed poisoning (multiple substances ingested).
2. Patients with pre-existing neuromuscular disorders or chronic liver disease affecting pseudocholinesterase levels.
3. Patients with unknown poisoning substance exposure.
4. Patients who left against medical advice or could not be followed up during the study period.

Duration of Study: The study was conducted over a period of 18 months from May **2023 to October 2024**.

Study Design: A prospective observational study.

. **Sampling Technique:** Convenience sampling method, where all eligible patients presenting with organophosphate poisoning during the study period were included.

Sample Size: 'The sample size estimation for the present study was based on a previously published investigation evaluating the Peradeniya Organophosphorus Poisoning (POP) 'Scale as a prognostic indicator of clinical outcomes in organophosphate poisoning **Kamath SD et al.**[62] reported correlation coefficient of 0.4903, considering alpha error of 1%, at a power of 90% with 99% CI, 55 cases of organophosphate poisoning were required, expecting a dropout rate of 20% the final sample size will be 66 cases of organophosphorus poisoning [80].

- 'The standard normal deviate for $\alpha = Z_\alpha = 2.5758$'
- 'The standard normal deviate for $\beta = Z_\beta = 1.2816$'
- '$C = 0.5 * \ln[(1+r)/(1-r)] = 0.5365$'

'Total sample size = $N = [(Z_\alpha + Z_\beta)/C]^2 + 3 = 55$'

Method of Data Collection:

A structured proforma was employed to systematically document each patient's demographic information, clinical symptoms, vital signs, laboratory parameters—including pseudocholinesterase levels—along with details of treatment interventions and clinical outcomes. The severity of organophosphate poisoning was evaluated at the time of admission using the 'Peradeniya Organophosphorus Poisoning (POP) scale', while the Poisoning Mortality Score (PMS) was computed for each case to assess prognostic implications. Patients were monitored for a range of in-hospital outcomes, including the development of complications, requirement for ventilatory support, duration of ICU stay, and mortality.

1. Data Collection Proforma

A structured proforma was designed to systematically collect and record all relevant patient data. The following parameters were included:

1. **Demographic Details**: Age, gender, residence, socioeconomic status, occupation, and history of prior poisoning attempts.
2. **Clinical Presentation**: Time since exposure, mode of poisoning (accidental, suicidal, or occupational), symptoms at admission (nausea, vomiting, salivation, bronchorrhea, fasciculations, seizures, altered consciousness, respiratory distress, etc.), 'Glasgow Coma Scale (GCS) score, and hemodynamic parameters (pulse rate, blood pressure, respiratory rate, and oxygen saturation)'.
3. **Laboratory Investigations**: 'Baseline blood tests including pseudocholinesterase levels, arterial blood gas (ABG) analysis, complete blood count, renal and liver function tests, and serum electrolytes'.
4. **Treatment Provided**: Details of initial resuscitation, decontamination measures (gastric lavage if indicated), administration of antidotes (atropine and pralidoxime), need for mechanical ventilation, ICU admission, and duration of hospital stay.
5. **Outcome Assessment**: Complications such as respiratory failure, aspiration pneumonia, seizures, requirement for inotropic support, length of ICU stay, total duration of hospitalization, and outcome (recovery or mortality).

2. Assessment of Poisoning Severity Using Peradeniya Organophosphorus Poisoning (POP) Scale[71]

'The POP scale was applied to each patient at the time of admission to classify poisoning severity. The scale includes six clinical parameters':

1. **Pupil size** (normal, pinpoint, or mid-dilated)
2. **Respiratory rate** (normal or tachypnea)
3. **Heart rate** (bradycardia or normal)
4. **Fasciculations** (absent, localized, or generalized)
5. **Level of consciousness** (alert, drowsy, or unconscious)
6. **Seizures** (present or absent)

Based on the total score (0-11), patients were categorized into **mild (0-3), moderate (4-7), or severe (8-11) poisoning**.

This classification helped in predicting prognosis and guiding treatment decisions.

3. Calculation of Poisoning Mortality Score (PMS)[63]

The Poisoning Mortality Score (PMS) was calculated for each patient to assess the probability of mortality.

PMS includes multiple parameters such as:

1. Glasgow Coma Scale (GCS) score
2. Oxygen saturation levels
3. Need for ventilatory support
4. Blood pH and serum bicarbonate levels
5. Hemodynamic instability (shock, need for inotropes)
6. Organ failure involvement (renal, hepatic, or neurological)
7. A higher PMS score was correlated with increased mortality risk, and this data was used for predictive analysis.

4. Follow-up and Outcome Assessment

1. Daily monitoring was done for each patient throughout the hospital stay, focusing on clinical progression, laboratory parameters, and response to treatment.

2. Complications such as aspiration pneumonia, respiratory failure, seizures, acute kidney injury, and multiorgan dysfunction syndrome (MODS) were documented.
3. Ventilatory support was recorded, including duration and the need for tracheostomy if prolonged respiratory failure occurred.
4. 'Length of ICU stay, and total hospital stay were noted'.
5. Outcome was documented as recovery, discharge, referral, or death.
6. Patients were followed up post-discharge (if applicable) to assess residual neurological deficits or any late complications related to poisoning.

Statistical Analysis:[72,73,74]

'Data entry was performed using Microsoft Excel, and statistical analysis was conducted with IBM SPSS Statistics version 22 (Somers, NY, USA)'. 'Categorical variables were summarized as frequencies and proportions, and comparisons between qualitative variables were made using the Chi-square test. Continuous variables were expressed as means ±standard deviation (SD)'. Visual representations of data, including bar diagrams and pie charts, were created using MS Excel and MS Word. 'A p-value less than 0.05 was considered to indicate statistical significance'.

Ethical Considerations:

1. Institutional Ethical Clearance was obtained before initiating the study.
2. 'Informed consent was taken from all patients or their legally authorized representatives before recruitment'.
3. Standard of Care was provided to all patients throughout the study period, including appropriate treatment, monitoring, and follow-up.

RESULTS

RESULTS

Table 7: Age distribution

		Count	%
Age	18-20 years	13	13.0%
	21 to 30 years	29	29.0%
	31 to 40 years	29	29.0%
	41 to 50 years	16	16.0%
	51 to 60 years	8	8.0%
	>60 years	5	5.0%
	Total	100	100.0%

'In the present study, most subjects were in the age group of 21-30 years and 31-40 years, each comprising 29.0% of the total sample. The mean age of the subjects was 35.99 ±13.687 years'.

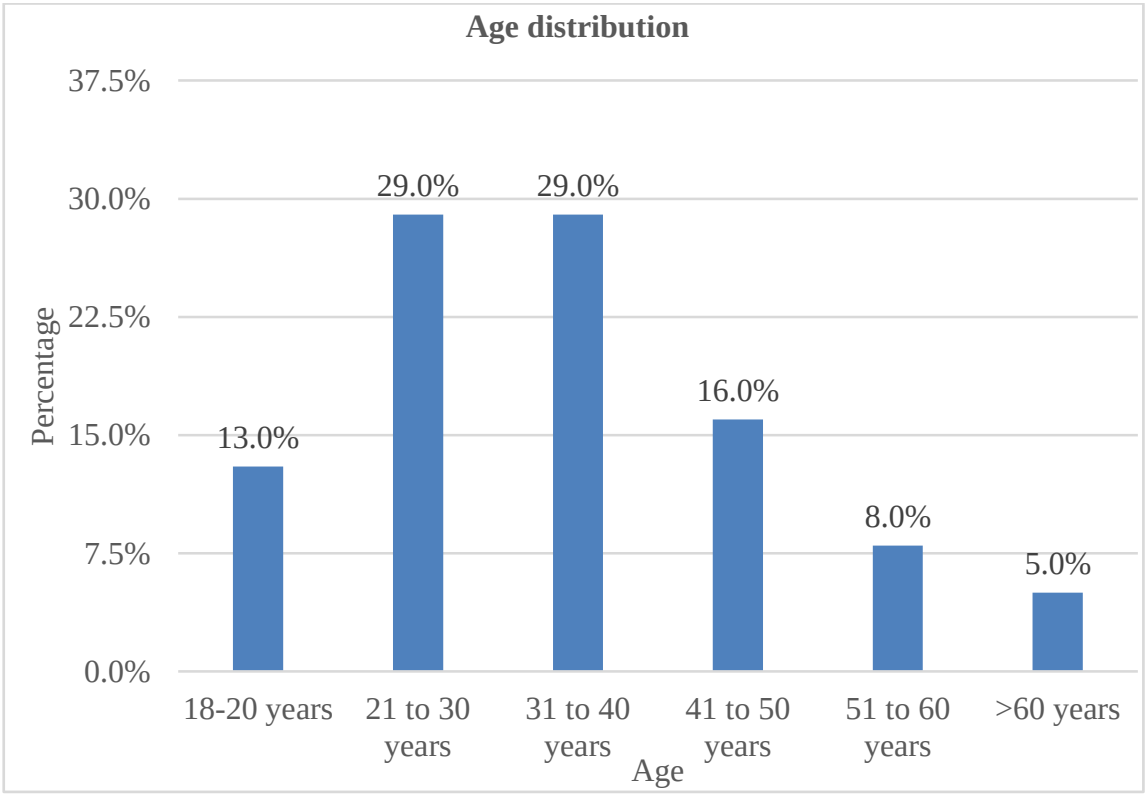

Figure 5: Bar Diagram Showing Age distribution.

Table 8: Gender

		Count	%
Gender	Female	25	25.0%
	Male	75	75.0%

In the present study, the majority of the subjects were male, constituting 75.0% of the total sample, while females accounted for 25.0%.

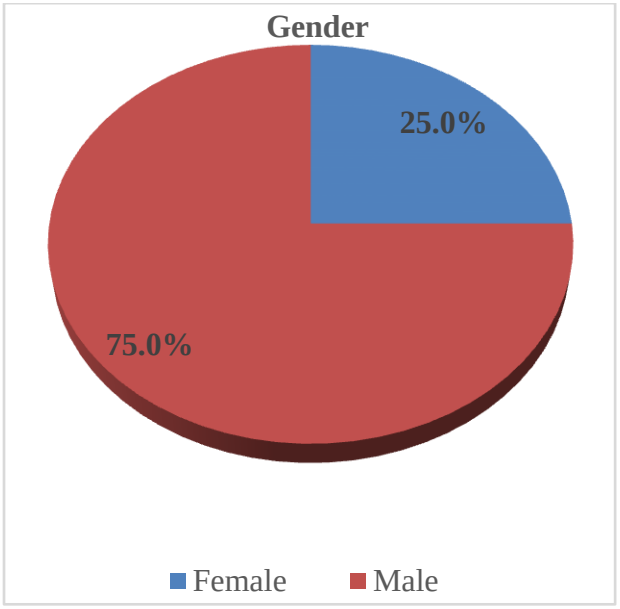

Figure 6: Bar Diagram Showing Gender.

Table 9: Compound

		Count	%
Compound	Chlorpyrifos	62	62.0%
	Dichlorvos	5	5.0%
	Dimethoate	3	3.0%
	Diazinon	1	1.0%
	Ethion	2	2.0%
	Monocrotophors	2	2.0%
	Propofenos	20	20.0%
	Phentoate	5	5.0%

Regarding the distribution of compounds, compound Chlorpyrifos was the most common, found in 62.0% of cases, followed by compound Propofenos in 20.0% of subjects.

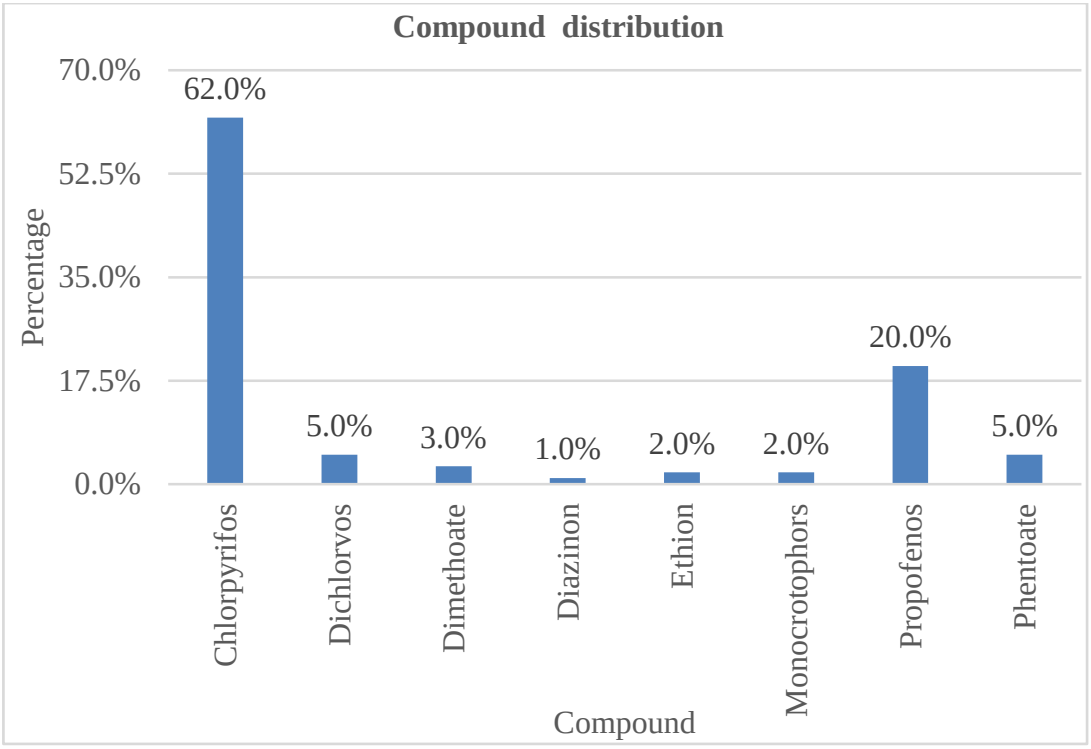

Figure 7: Bar Diagram Showing Compound.

Table 10: Peradeniya Organophosphorus Poisoning Severity

		Count	%
Peradeniya Organophosphorus Poisoning Severity	Mild (0-3)	39	39.0%
	Moderate (4-7)	41	41.0%
	Severe (8-11)	20	20.0%
	Total	100	100.0%

In the present study, 41.0% of subjects had moderate Peredeniya Organophosphorous Poisoning severity (scores of 4-7), followed by 39.0% with mild severity (scores of 0-3), while 20.0% had severe Peredeniya Organophosphorous Poisoning severity (scores of 8-11).

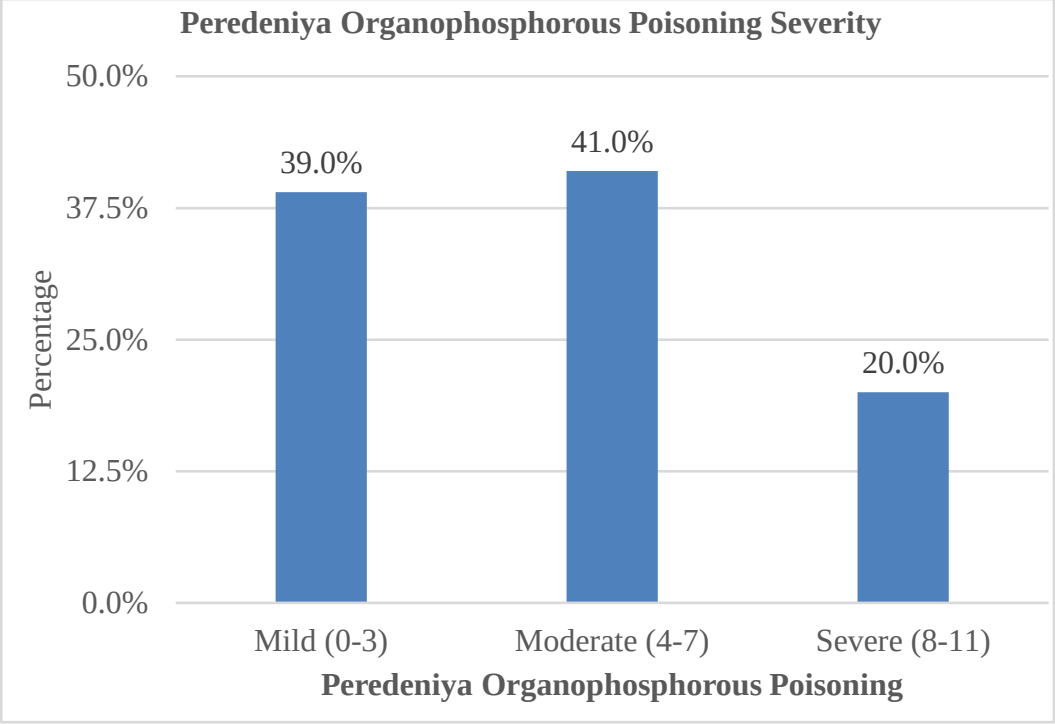

Figure 8: Bar Diagram Showing Peredeniya Organophosphorous Poisoning Severity.

Table 11: AVPU

		Count	%
AVPU	Alert	68	68.0%
	Verbal	15	15.0%
	Pain	10	10.0%
	Unresponsive	7	7.0%
	Total	100	100.0%

- **A**: Alert, **V**: Verbal, **P**: Pain, **U**: Unresponsive

The AVPU scale assessment showed that most subjects (68.0%) were alert, while 15.0% responded to verbal stimuli, 10.0% responded to pain, and 7.0% were unresponsive.

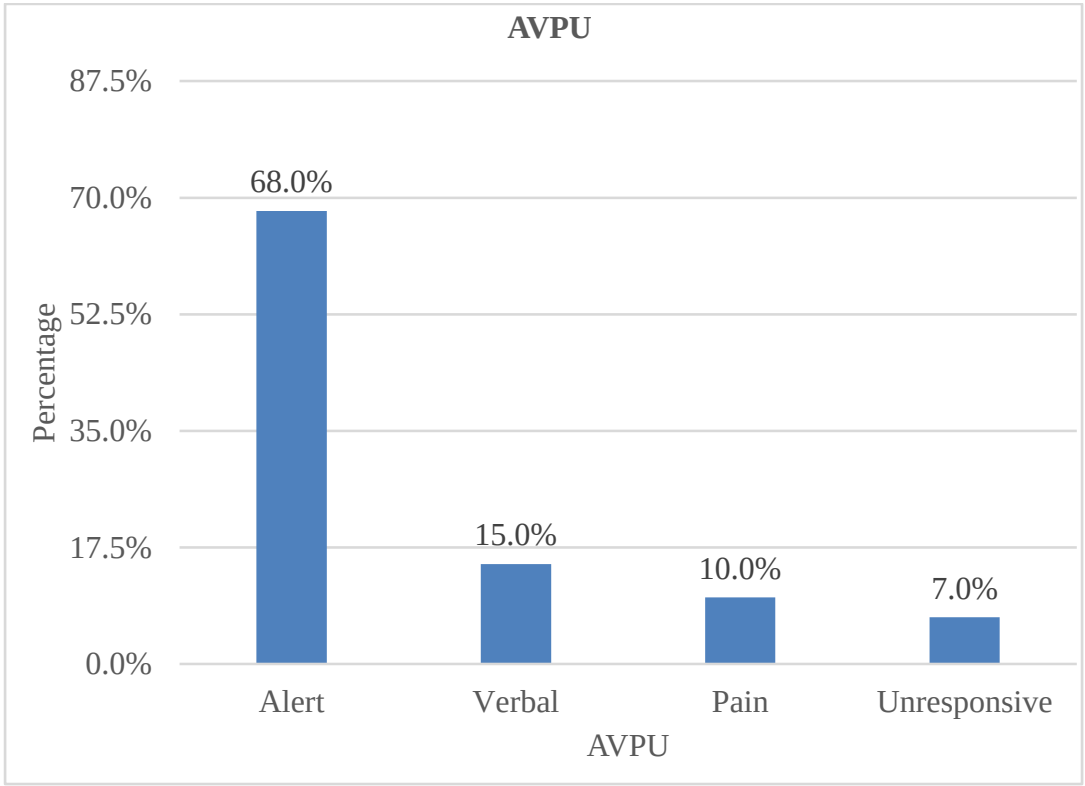

Figure 9: Bar Diagram Showing AVPU.

Table 12: Poisoning mortality score Severity Risk Level

		Count	%
Poisoning mortality score Severity **Risk Level**	0-30 (Very Low Risk)	0	0.0%
	31 to 50 (Low Risk)	55	55.0%
	51 to 65 (Intermediate Risk)	32	32.0%
	>65 (High Risk)	13	13.0%
	Total	100	100.0%

In terms of Poisoning mortality score severity risk levels, 55.0% of subjects were categorized as low risk (31-50), while 32.0% had an intermediate risk (51-65), and 13.0% were at high risk (>65). No subjects fell into the very low-risk category.

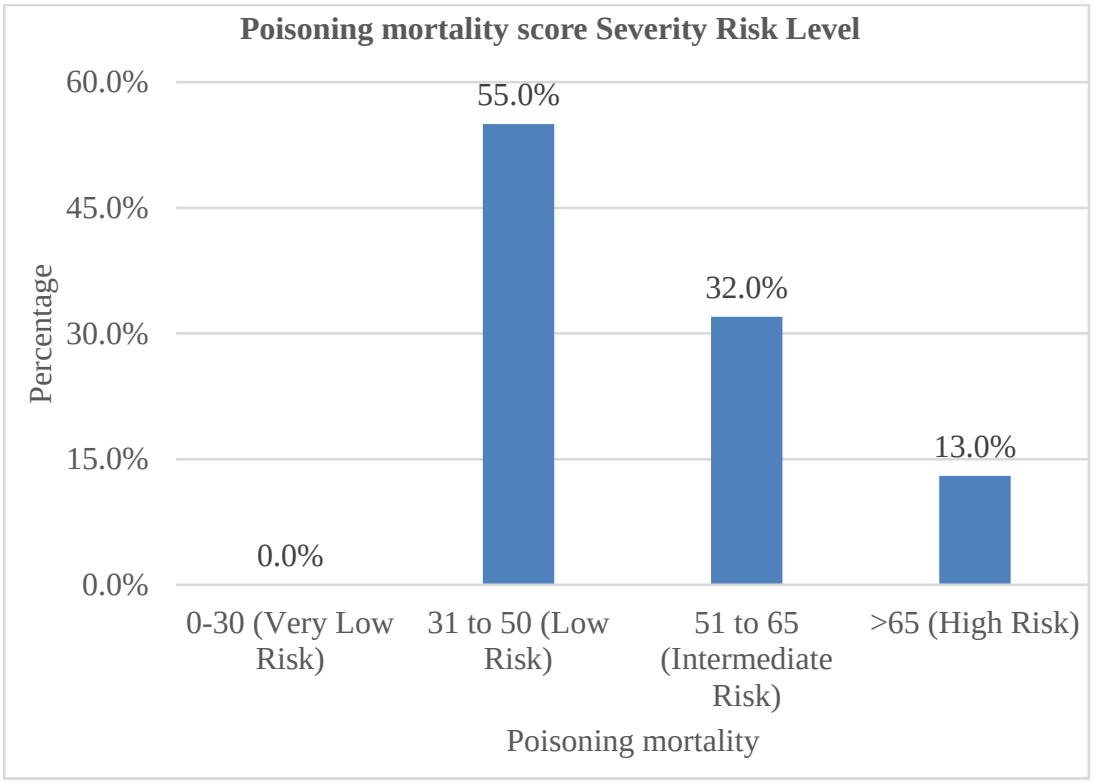

Figure 10: Bar Diagram Showing Poisoning mortality score Severity Risk Level.

Table 13: Proud foot classification

		Count	%
Proud foot classification	Mild	25	25.0%
	Moderate	28	28.0%
	Severe	47	47.0%
	Total	100	100.0%

Based on Proud Foot classification, 47.0% of subjects were classified as severe, 28.0% as moderate, and 25.0% as mild.

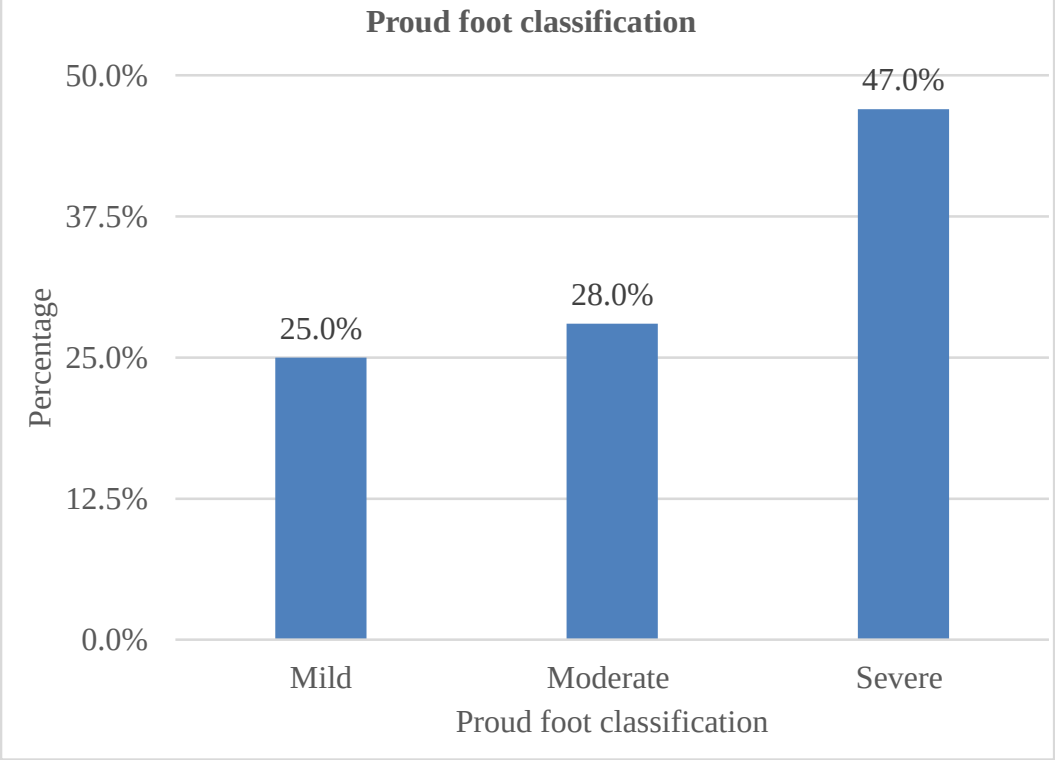

Figure 11: Bar Diagram Showing Proud foot classification.

Table 14: Correlation between POP score and Outcome parameters

		Ventilators stay days	ICU stay days	Hospital stays days
POP score	Pearson Correlation	0.615**	0.472**	0.294**
	P value	<0.001*	<0.001*	0.003*
	N	100	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

“A statistically significant positive correlation was observed between Peradeniya Organophosphorus Poisoning (POP) scores and several clinical outcomes parameters”. Specifically, higher POP scores were associated with prolonged duration of ventilator support ‘($r = 0.615$, $p < 0.001$), extended ICU stay ($r = 0.472$, $p < 0.001$), and increased total hospital stay ($r = 0.294$, $p = 0.003$)’. These correlations, significant at the 0.01 level (two-tailed), suggest that greater severity of poisoning as reflected by the POP score correlates with poorer clinical outcomes and increased resource utilization.

Table 15: Correlation between PMS score and Outcome parameters

		Ventilators stay days	ICU stay days	Hospital stays days
PMS SCORE	Pearson Correlation	0.678**	0.319**	-0.107
	P value	<0.001*	0.001*	0.290
	N	100	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

Similarly, PMS scores were positively correlated with ventilator stay days ($r = 0.678$, $p < 0.001$) and ICU stay days ($r = 0.319$, $p = 0.001$), but no significant correlation was found with hospital stay days ($p = 0.290$).

Table 16: Correlation between Pseudocholinesterase level and Outcome parameters

		Ventilators stay days	ICU stay days	Hospital stays days
Pseudocholinesterase level	Pearson Correlation	-.566**	-.717**	-.485**
	P value	.000	.000	.000
	N	100	100	100

**. 'Correlation is significant at the 0.01 level (2-tailed)'.

Pseudocholinesterase levels showed a significant negative correlation with ventilator stay days ($r = -0.566$, $p < 0.001$), ICU stay days ($r = -0.717$, $p < 0.001$), and hospital stay days ($r = -0.485$, $p < 0.001$), indicating that lower levels were associated with worse outcomes.

Table 17: Discharge or mortality

		Count	%
Outcome	Discharge	88	88.0%
	Mortality	12	12.0%

The overall outcome of the study showed that 88.0% of subjects were discharged, while 12.0% experienced mortality.

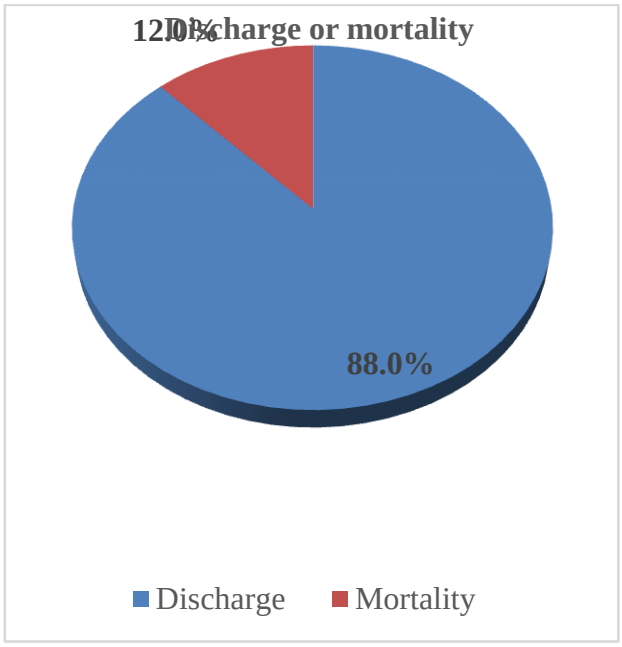

Figure 12: Pie Diagram Showing Discharge or mortality.

Table 18: Association between POP Score, PMS Score, Pseudocholinesterase level and Proud foot classification with Outcome

		Outcome				P value
		Discharge		Mortality		
		Count	Row N %	Count	Row N %	
POP Severity	Mild (0-3)	39	100.0 %	0	0.0%	<0.001*
	Moderate (4-7)	41	100.0 %	0	0.0%	
	Severe (8-11)	8	40.0 %	12	60.0 %	
PMS Severity	0-30 (Very Low Risk)	0	0.0%	0	0.0%	<0-.001*
	31 to 50 (Low Risk)	55	100.0 %	0	0.0%	
	51 to 65 (Intermediate Risk)	31	96.9 %	1	3.1%	
	>65 (High Risk)	2	15.4 %	11	84.6 %	
Pseudocholinesterase level	Mild Poisoning (4000 - 8000)	33	100.0 %	0	0.0%	<0.001*
	Moderate Poisoning (1000-4000)	45	100.0 %	0	0.0%	
	Severe Poisoning (<1000)	10	45.5 %	12	54.5 %	
Proud foot classification	Mild	25	100.0 %	0	0.0%	<0.001*
	Moderate	28	100.0 %	0	0.0%	
	Severe	35	74.5 %	12	25.5 %	

Pearson Chi-Square Tests

A significant association was observed between POP severity and outcome ($p < 0.001$). All patients with mild or moderate POP severity survived, whereas 60.0% of those with severe POP severity succumbed.

PMS severity was also significantly associated with outcome ($p < 0.001$). While all low-risk patients survived, 84.6% of high-risk patients experienced mortality.

Pseudocholinesterase levels were significantly associated with outcomes ($p < 0.001$), with all patients in the mild and moderate poisoning categories surviving, whereas 54.5% of those with severe poisoning did not survive.

Proud Foot classification was significantly associated with outcomes ($p < 0.001$), where all mild and moderate cases survived, while 25.5% of severe cases experienced mortality.

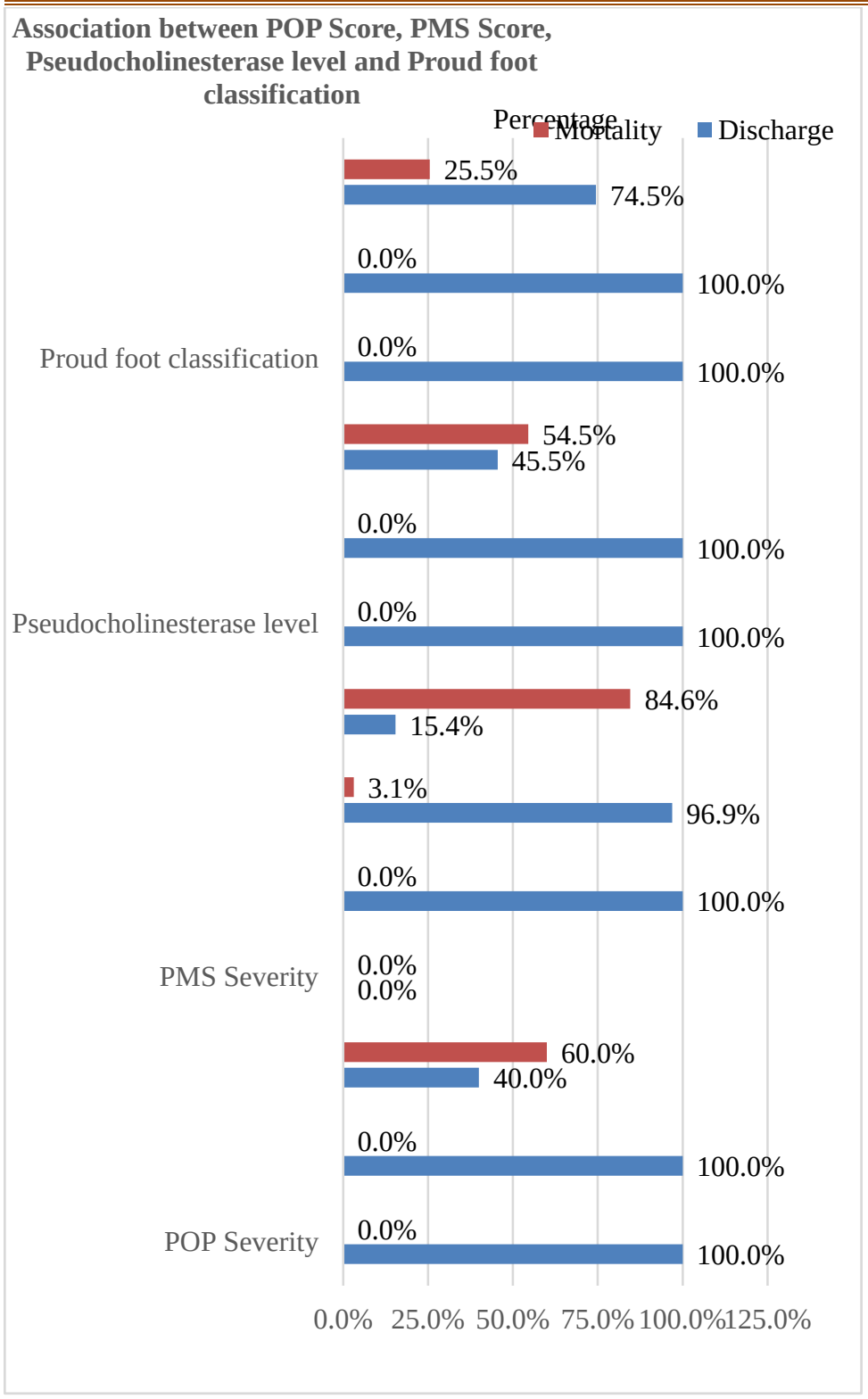

Figure 13: Bar Diagram Showing Association between POP Score, PMS Score, Pseudocholinesterase level and Proud foot classification with Outcome

Table 19: Comparison of Outcome parameters with respect to POP Severity

	POP Severity									P value
	Mild (0-3)			Moderate (4-7)			Severe (8-11)			
	Mean	SD	Median	Mean	SD	Median	Mean	SD	Median	
Total Dose of Atropine (mg)	26.59	17.51	25	56.02	19.33	57	84.45	33.03	87	<0.001*
Total PAM (g)	7.85	2.46	8	16.02	11.78	14	17.00	6.10	18	<0.001*
Ventilators stay days	.00	.00	0	.76	1.43	0	4.35	3.28	4	<0.001*
ICU stay days	2.69	1.61	2	6.29	3.08	6	6.40	3.45	6	<0.001*
Hospital stays days	7.15	4.91	6	14.00	5.85	14	10.85	7.62	11	<0.001*

ANOVA Test

Patients with severe POP severity required significantly higher total doses of atropine (84.45 ± 33.03 mg, $p < 0.001$) and PAM (17.00 ±6.10 g, $p < 0.001$) compared to those with mild and moderate severity. They also had significantly prolonged ventilator stay (4.35 ±3.28 days, $p < 0.001$), ICU stay (6.40 ±3.45 days, $p < 0.001$), and hospital stay (10.85 ±7.62 days, $p < 0.001$).

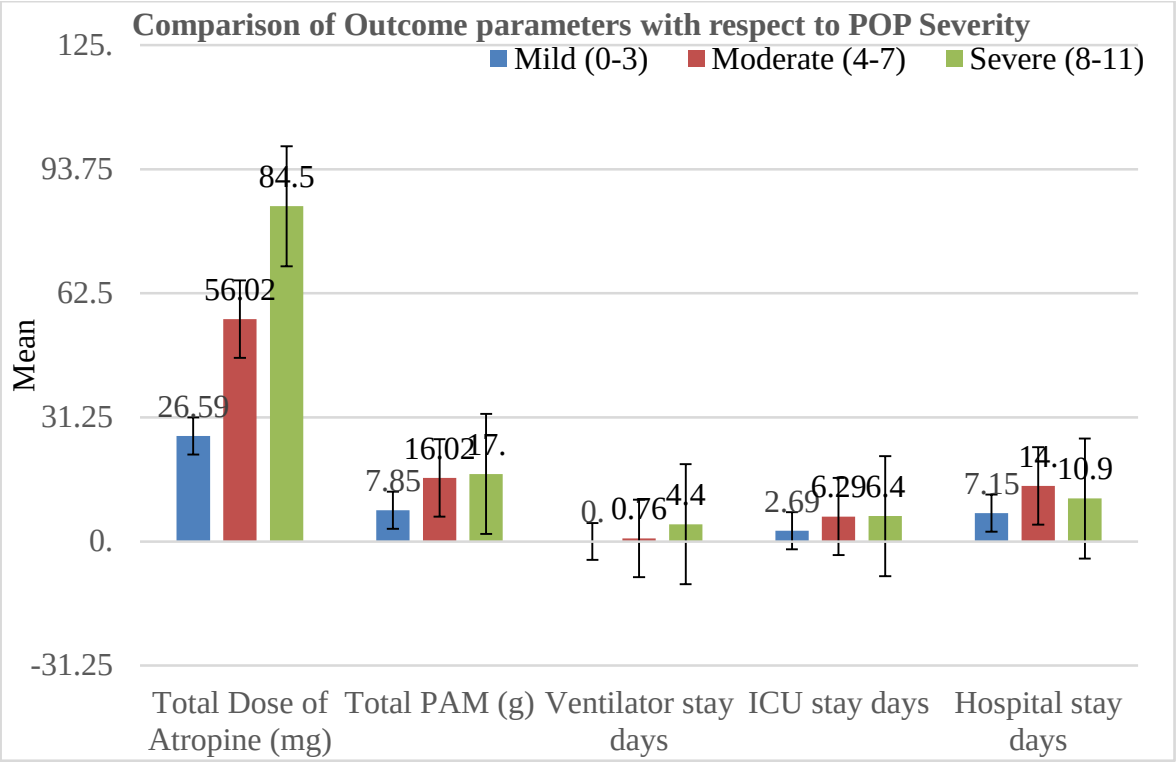

Figure 14: Bar Diagram Showing Comparison of Outcome parameters with respect to POP Severity.

Table 20: Comparison of Outcome parameters with respect to PMS Severity

	PMS Severity									P value
	31 to 50 (Low Risk)			51 to 65 (Intermediate Risk)			>65 (High Risk)			
	Mean	SD	Median	Mean	SD	Median	Mean	SD	Median	
Total Dose of Atropine (mg)	41.11	23.54	42	46.84	25.07	46	97.15	30.19	90	<0.001*
Total PAM (g)	11.47	5.27	12	13.31	13.54	11	18.92	6.09	22	0.027*
Ventilators stay days	.51	1.30	0	.59	1.32	0	5.46	3.28	6	<0.001*
ICU stay days	4.27	2.99	4	5.28	3.21	5	6.69	3.54	6	0.035*
Hospital stays days	11.44	7.36	10	10.56	5.78	10	7.92	4.42	8	0.226

ANOVA Test

PMS severity had a significant impact on total atropine dosage ($p < 0.001$) and total PAM usage ($p = 0.027$). 'High-risk patients had significantly prolonged ventilator stay (5.46 ±3.28 days, $p < 0.001$) and ICU stay (6.69 ±3.54 days, $p = 0.035$), but no significant difference was noted in hospital stay duration ($p = 0.226$)'.

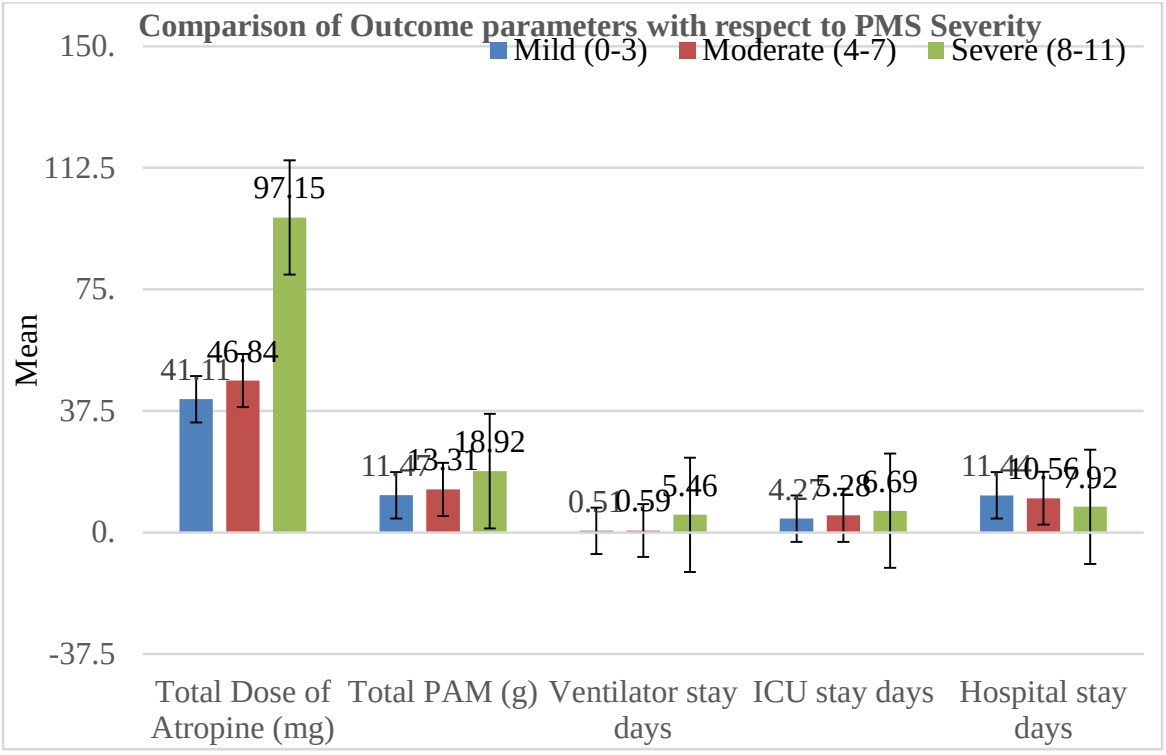

Figure 15: Bar Diagram Showing Comparison of Outcome parameters with respect to PMS Severity

Table 21: Comparison of Outcome parameters with respect to Pseudocholinesterase level

	Pseudocholinesterase level									P value
	Mild Poisoning (4000 - 8000)			Moderate Poisoning (1000-4000)			Severe Poisoning (<1000)			
	Mean	SD	Median	Mean	SD	Median	Mean	SD	Median	
Total Dose of Atropine (mg)	23.73	16.82	20	51.60	18.01	52	87.18	28.69	87	<0.001*
Total PAM (g)	7.82	2.53	8	13.22	4.60	12	20.45	15.39	19	<0.001*
Ventilators stay days	.00	.00	0	.56	1.18	0	4.23	3.24	4	<0.001*
ICU stay days	2.09	1.13	2	5.49	2.36	5	7.95	3.47	8	<0.001*
Hospital stays days	6.06	4.17	5	13.07	5.59	12	12.82	7.96	12	<0.001*

ANOVA Test

Patients with severe pseudocholinesterase poisoning required significantly higher doses of atropine (87.18 ±28.69 mg, $p < 0.001$) and PAM (20.45 ±15.39 g, $p < 0.001$). They also had significantly prolonged ventilator stay (4.23 ±3.24 days, $p < 0.001$), ICU stay (7.95 ±3.47 days, $p < 0.001$), and hospital stay (12.82 ±7.96 days, $p < 0.001$).

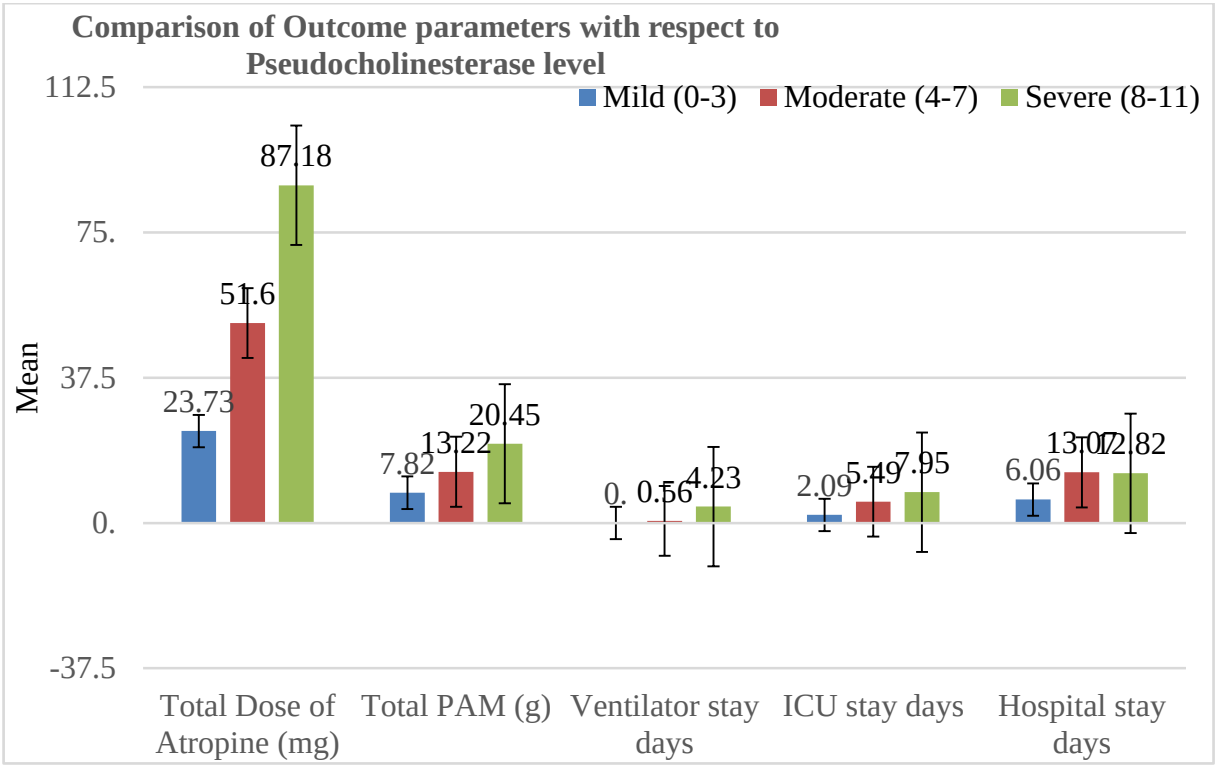

Figure 16: Bar Diagram Showing Comparison of Outcome parameters with respect to Pseudocholinesterase level.

Table 22: Comparison of Outcome parameters with respect to Proud foot classification

	Proud foot classification									P value
	Mild			Moderate			Severe			
	Mean	SD	Median	Mean	SD	Median	Mean	SD	Median	
Total Dose of Atropine (mg)	22.28	17.02	12	43.25	19.69	46	69.26	28.96	66	<0.001*
Total PAM (g)	7.52	2.49	6	10.25	3.85	10	17.62	11.14	16	<0.001*
Ventilator stay days	.00	.00	0	.18	.67	0	2.40	2.96	2	<0.001*
ICU stay days	1.92	.95	2	3.68	1.56	4	7.23	2.99	7	<0.001*
Hospital stay days	6.00	4.15	5	8.82	5.10	8	14.32	6.50	14	<0.001*

ANOVA Test

Patients with severe Proud Foot classification required significantly higher atropine doses (69.26 ± 28.96 mg, $p < 0.001$) and PAM doses (17.62 ±11.14 g, $p < 0.001$). They also had significantly prolonged ventilator stay (2.40 ±2.96 days, $p < 0.001$), ICU stay (7.23 ±2.99 days, $p < 0.001$), and hospital stay (14.32 ±6.50 days, $p < 0.001$).

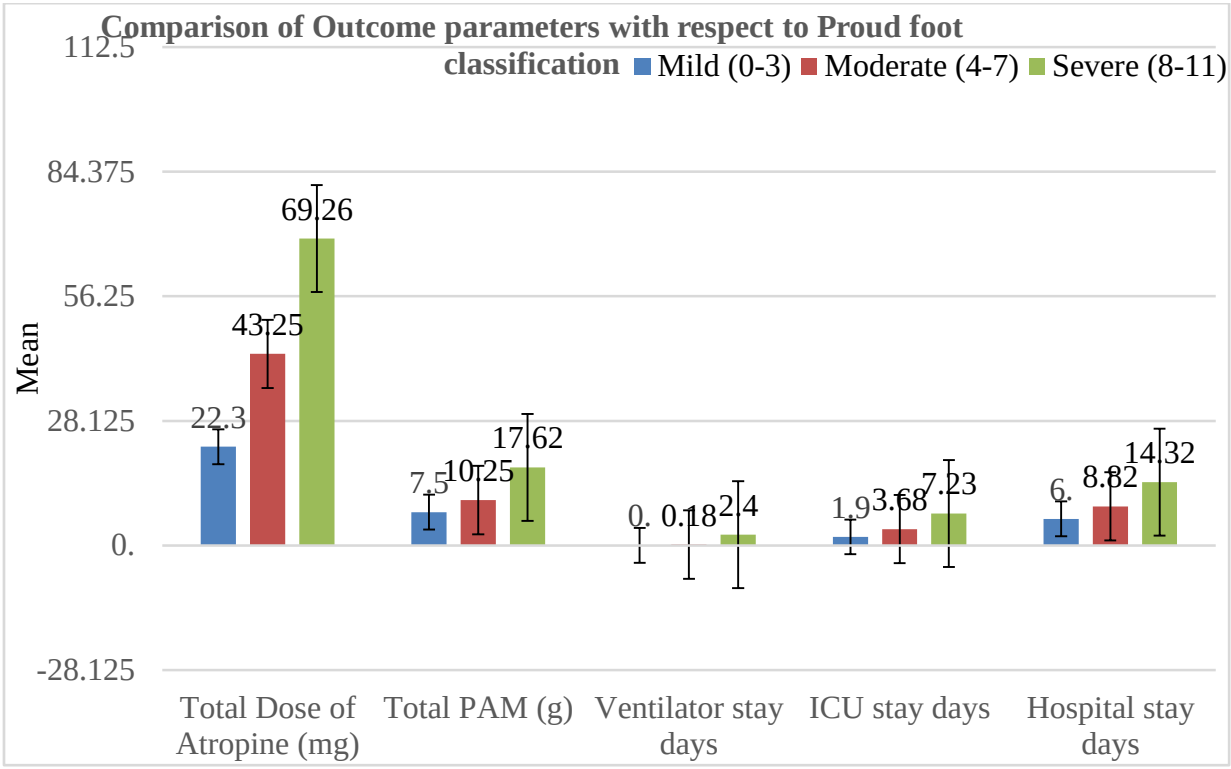

Figure 17: Bar Diagram Showing Comparison of Outcome parameters with respect to Proud foot classification

Table 23: 'Area under the ROC curve (AUC) for POP score in predicting mortality'

'Area under the ROC curve (AUC)'	0.968
'Standard Error'	0.0152
'95% Confidence interval '	0.912 to 0.993
'z statistic'	30.850
'Significance level P (Area=0.5)'	<0.0001*

‘Youden index’

‘Youden index J’	0.9091
‘Associated criterion’	>7
‘Sensitivity’	100.00
‘Specificity’	90.91

The ROC analysis showed that POP score had an AUC of 0.968 ($p < 0.0001$) in predicting mortality, with an optimal cut-off score of >7, achieving a sensitivity of 100% and specificity of 90.91%.

‘Criterion values and coordinates of the ROC curve ’

Criterion	Sensitivity	95% CI	Specificity	95% CI	+PV	95% CI	-PV	95% CI
≥0	100.00	73.5 - 100.0	0.00	0.0 - 4.1	12.0	12.0 - 12.0		
>0	100.00	73.5 - 100.0	1.14	0.03 - 6.2	12.1	11.9 - 12.4	100.0	
>1	100.00	73.5 - 100.0	10.23	4.8 - 18.5	13.2	12.4 - 14.0	100.0	
>2	100.00	73.5 - 100.0	25.00	16.4 - 35.4	15.4	13.9 - 17.0	100.0	
>3	100.00	73.5 - 100.0	44.32	33.7 - 55.3	19.7	16.9 - 22.8	100.0	
>4	100.00	73.5 - 100.0	65.91	55.0 - 75.7	28.6	23.0 - 34.8	100.0	
>5	100.00	73.5 - 100.0	71.59	61.0 - 80.7	32.4	25.6 - 40.1	100.0	
>6	100.00	73.5 - 100.0	84.09	74.8 - 91.0	46.2	34.6 - 58.1	100.0	
>7	100.00	73.5 - 100.0	90.91	82.9 - 96.0	60.0	43.7 - 74.4	100.0	
>8	66.67	34.9 - 90.1	96.59	90.4 - 99.3	72.7	45.0 - 89.7	95.5	90.5 - 97.9
>9	25.00	5.5 - 57.2	98.86	93.8 - 100.0	75.0	25.3 - 96.4	90.6	87.4 - 93.1
>10	0.00	0.0 - 26.5	100.00	95.9 - 100.0			88.0	88.0 - 88.0

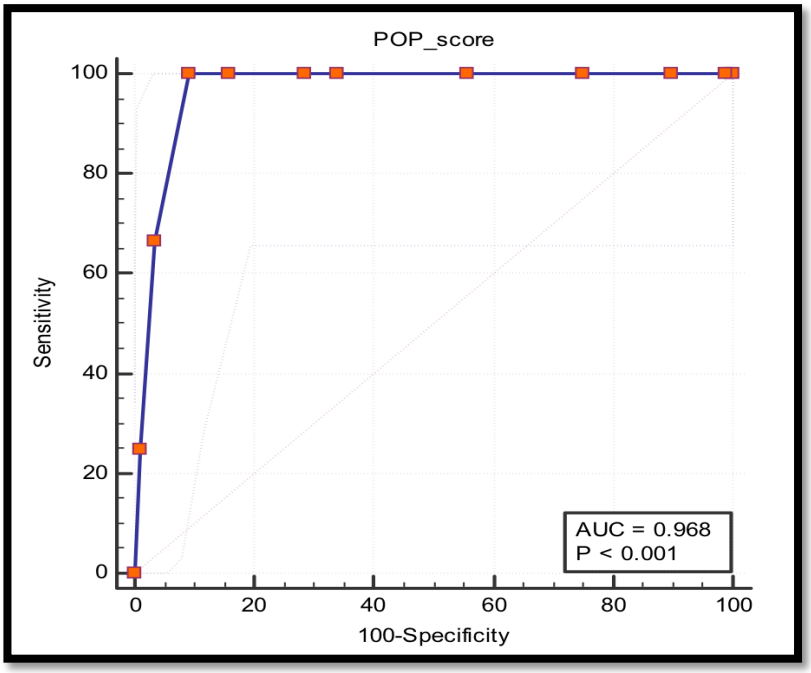

Figure 18:ROC Curve showing Area under the ROC curve (AUC) for POP score in predicting mortality.

Table 24: 'Area under the ROC curve (AUC) for PMS score in predicting mortality'

'Area under the ROC curve (AUC)'	0.987
'Standard Error'	0.00880
'95% Confidence interval'	0.941 to 0.999
'z statistic'	55.345
'Significance level P (Area=0.5)'	<0.0001*

'Youden index'

'Youden index J'	0.8977
'Associated criterion'	>57
'Sensitivity'	100.00
'Specificity'	89.77

Similarly, the PMS score had an AUC of 0.987 ($p < 0.0001$) with an optimal cut-off of >57, showing 100% sensitivity and 89.77% specificity in predicting mortality.

'Criterion values and coordinates of the ROC curve '

Criterion	Sensitivity	95% CI	Specificity	95% CI	+PV	95% CI	-PV
≥43	100.00	73.5 - 100.0	0.00	0.0 - 4.1	12.0	12.0 - 12.0	
>43	100.00	73.5 - 100.0	19.32	11.7 - 29.1	14.5	13.2 - 15.8	100.0
>44	100.00	73.5 - 100.0	26.14	17.3 - 36.6	15.6	14.0 - 17.3	100.0
>47	100.00	73.5 - 100.0	51.14	40.2 - 61.9	21.8	18.4 - 25.7	100.0
>49	100.00	73.5 - 100.0	53.41	42.5 - 64.1	22.6	19.0 - 26.8	100.0
>50	100.00	73.5 - 100.0	62.50	51.5 - 72.6	26.7	21.7 - 32.3	100.0
>51	100.00	73.5 - 100.0	79.55	69.6 - 87.4	40.0	30.6 - 50.2	100.0
>52	100.00	73.5 - 100.0	80.68	70.9 - 88.3	41.4	31.5 - 52.0	100.0
>54	100.00	73.5 - 100.0	81.82	72.2 - 89.2	42.9	32.5 - 53.9	100.0
>56	100.00	73.5 - 100.0	88.64	80.1 - 94.4	54.5	40.1 - 68.3	100.0
>57	100.00	73.5 - 100.0	89.77	81.5 - 95.2	57.1	41.8 - 71.2	100.0
>60	91.67	61.5 - 99.8	93.18	85.7 - 97.5	64.7	45.4 - 80.2	98.8
>62	91.67	61.5 - 99.8	96.59	90.4 - 99.3	78.6	54.3 - 91.9	98.8
>65	91.67	61.5 - 99.8	97.73	92.0 - 99.7	84.6	58.0 - 95.6	98.9
>82	41.67	15.2 - 72.3	100.00	95.9 - 100.0	100.0		92.6
>84	33.33	9.9 - 65.1	100.00	95.9 - 100.0	100.0		91.7
>87	8.33	0.2 - 38.5	100.00	95.9 - 100.0	100.0		88.9
>89	0.00	0.0 - 26.5	100.00	95.9 - 100.0			88.0

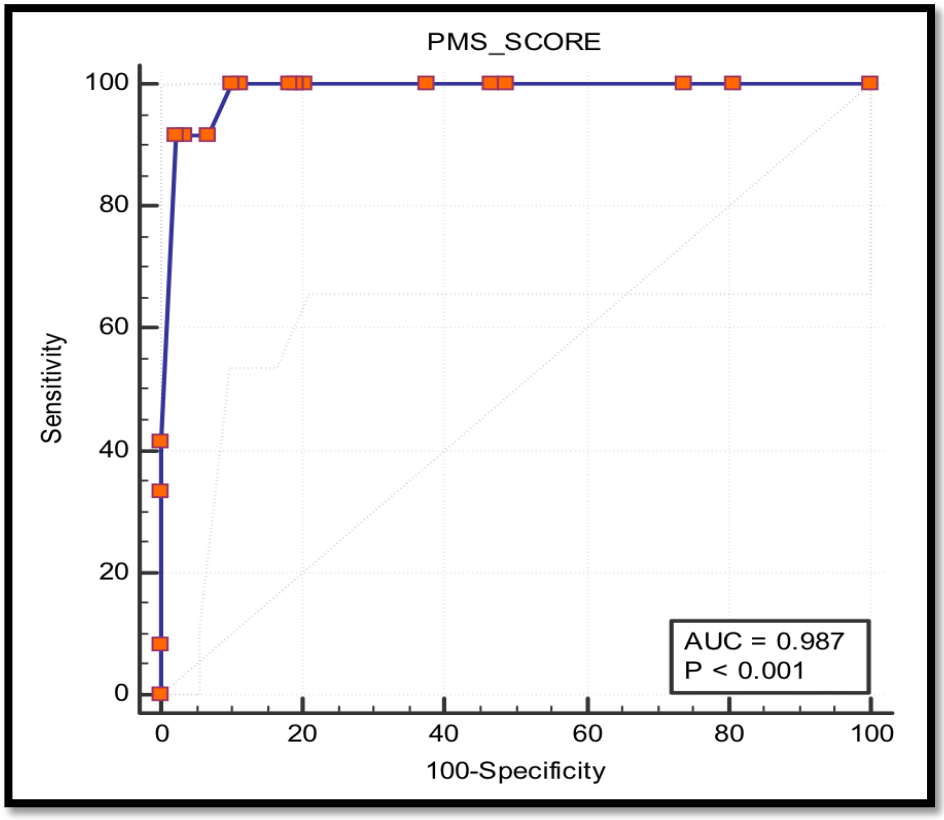

Figure 19: ROC Curve Area under the ROC curve (AUC) for PMS score in predicting mortality.

Table 25: 'Area under the ROC curve (AUC) for Pseudocholinesterase levels in predicting mortality'

'Area under the ROC curve (AUC)'	0.965
'Standard Error'	0.0173
'95% Confidence interval'	0.908 to 0.992
'z statistic'	26.856
'Significance level P (Area=0.5)'	<0.0001*

Youden index

'Youden index J'	0.8977
'Associated criterion'	≤900
'Sensitivity'	100.00
'Specificity'	89.77

Pseudocholinesterase levels had an AUC of 0.965 ($p < 0.0001$) with an optimal cut-off of ≤900, achieving 100% sensitivity and 89.77% specificity in predicting mortality.

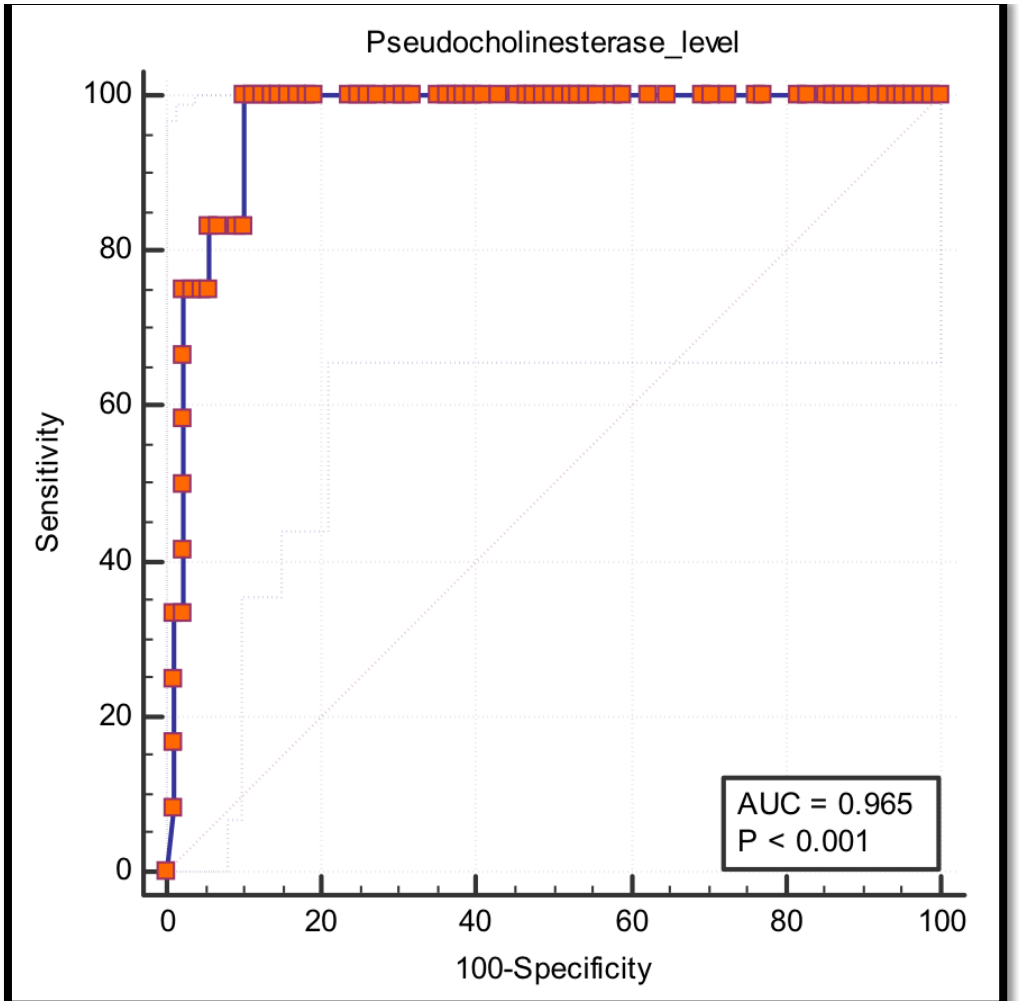

Figure 20: 'ROC Curve showing Area under the ROC curve (AUC) for Pseudocholinesterase levels in predicting mortality'.

Table 26: 'Comparison of Area under the ROC curve (AUC) for POP Score, PMS Score and Pseudocholinesterase levels in predicting mortality'

Variable	AUC	SE	95% CI
POP score	0.968	0.0152	0.912 to 0.993
PMS Score	0.987	0.00880	0.941 to 0.999
Pseudocholinesterase level	0.965	0.0173	0.908 to 0.992

A comparison of these predictive models showed that the PMS score had the highest AUC (0.987), followed by POP score (0.968) and pseudocholinesterase levels (0.965), indicating that PMS score was the most reliable predictor of mortality in the present study.

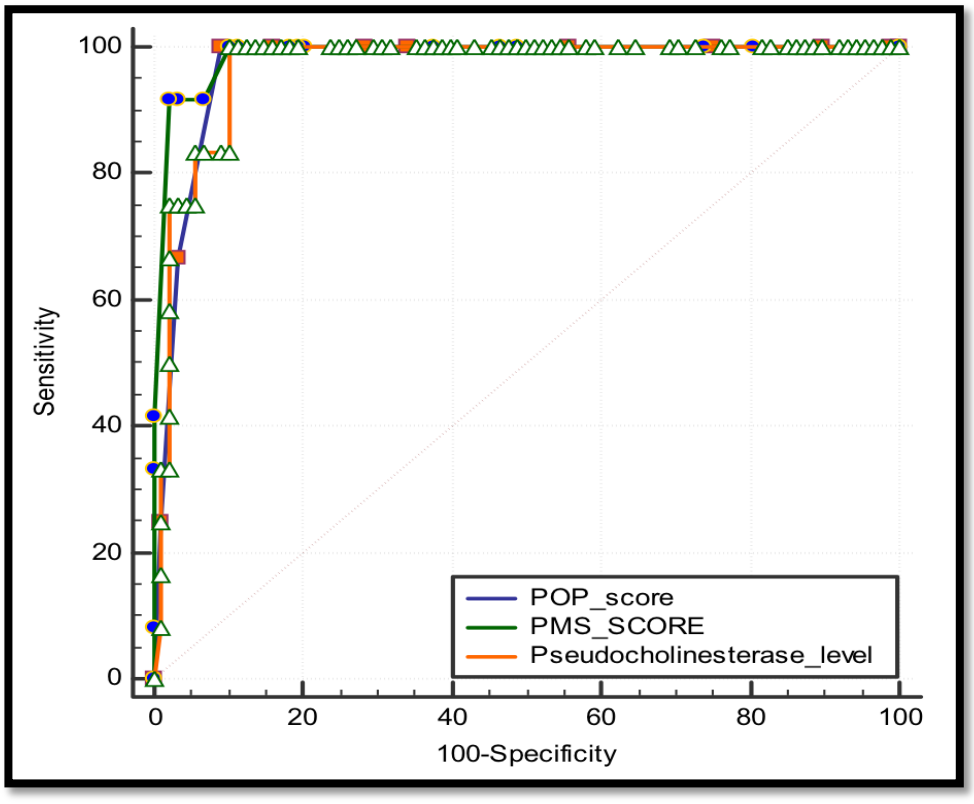

Figure 21: ROC Curve showing Comparison of Area under the ROC curve (AUC) for POP Score, PMS Score and Pseudocholinesterase levels in predicting mortality.

DISCUSSION

DISCUSSION

This prospective observational study, conducted over 18 months in the Department of Emergency Medicine at Sri Devaraj Urs Medical College, aimed to explore critical prognostic indicators in patients with organophosphate (OP) poisoning. A total of 100 patients aged above 18 years, all with a confirmed history of OP compound exposure and characteristic clinical features, were included after obtaining informed consent. The availability of serum pseudocholinesterase levels within the first 24 hours of hospital admission was a key inclusion criterion, ensuring timely biochemical correlation with clinical findings.

The study was designed around three primary objectives: first, to evaluate the correlation between the 'Peradeniya Organophosphorus Poisoning (POP) scale and clinical severity; second, to assess the predictive accuracy of the Poisoning Mortality Score (PMS) in determining outcomes such as morbidity and mortality; and third, to analyze the relationship between serum pseudocholinesterase levels and the severity of poisoning, particularly their utility in predicting patient prognosis'.

The comprehensive approach of combining clinical scoring systems with biochemical markers provided a robust framework for assessing the severity of OP poisoning. By correlating these parameters with treatment needs and outcomes, the study aimed to enhance clinical decision-making and facilitate early risk stratification in emergency and ICU settings. This discussion underscores the importance of integrating standardized clinical tools and timely laboratory investigations for the effective management of OP poisoning, while also laying the groundwork for future research involving larger, multi-centric populations to validate and expand upon these findings.

Profile of subjects

The dominance of patients in the 21–40-year age group, with a male preponderance (75%), aligns with previous studies, such as **Chintale et al.**[69], who observed a similar demographic distribution, likely due to higher occupational exposure and risk-taking behavior among males in agricultural settings. The predominance of chlorpyrifos (62%) among poisoning agents in this study is consistent with **Dubey et al.**[66], who also reported chlorpyrifos as the most common OP compound, likely reflecting regional pesticide usage trends. The POP scale in our study showed strong correlation with clinical severity, reaffirming its prognostic validity, as established by **Kamath and Gautam**[62], who highlighted its utility in early triaging and management. Variations in POP score severity across studies may be attributed to differences in the time of presentation and dosage of compound ingested. PMS was found to be an effective mortality predictor, in concordance with **Krishna Moorthy et al.**[63], who validated its comparative performance with SOFA and APACHE II scores. Our results further demonstrated that lower pseudocholinesterase levels correlated with higher severity and worse outcomes, echoing findings by **Chaudhary et al.**[65], who reported significant associations between enzyme levels and clinical deterioration. Discrepancies in enzyme levels across studies can result from inter-individual variation, genetic polymorphisms, or delays in sampling.

Severity of OP Poisoning:

The current study revealed that a majority of organophosphate (OP) poisoning cases fell under moderate severity (41%) on the Peradeniya Organophosphorus Poisoning (POP) scale, aligning closely with findings by **Kamath and Gautam**, who reported 42% moderate cases and emphasized the scale's prognostic utility in clinical settings[62]. However, the proportion of severe cases in our study (20%) was slightly lower than the 27.5% reported by **Dubey et al.**[66], possibly

due to earlier hospital presentations or regional differences in compound toxicity. The AVPU findings, showing 68% of patients as alert, suggest relatively prompt clinical intervention, which may explain the lower severe case proportion, similar to outcomes in Ahmed et al.'s ICU study where early intubation correlated with survival90. Regarding the Poisoning Mortality Score (PMS), our study's categorization of 55% low-risk and 13% high-risk contrasts with Krishna Moorthy et al., who found higher intermediate-risk prevalence, possibly reflecting sample differences in exposure levels or healthcare accessibility[89].

Furthermore, our pseudocholinesterase level interpretation, although not detailed numerically, aligns with **Chaudhary et al.,** who established a significant inverse relationship between enzyme levels and severity, suggesting enzyme activity as a reliable biochemical marker when combined with clinical scales[65]. Notably, Proudfoot classification in our study showed 47% severe cases, which is higher than **Chintale et al.** findings (36.7%), potentially due to regional usage patterns of high-potency organophosphates or delay in antidote administration[69]. Differences across studies likely stem from variations in compound type, healthcare infrastructure, and timing of clinical management, underscoring the importance of multimodal assessment using POP, PMS, and biochemical indices for accurate prognosis and tailored care.

Correlation between POP score, PMS score, Pseudocholinesterase level and Outcome parameters:

The study's findings underscore the prognostic relevance of the 'Peradeniya Organophosphorus Poisoning (POP) 'scale, Poisoning Mortality Score (PMS), and pseudocholinesterase levels in determining the clinical severity and outcomes of organophosphate poisoning. The strong positive correlation between POP scores and both ventilator and ICU stay days aligns with previous studies, such as **Kamath and Gautam (2021)**, which confirmed the POP scale's utility in gauging poisoning severity and predicting prolonged hospitalization and

respiratory failure outcomes[62]. Similarly, **Dubey et al. (2016)** emphasized the association of higher POP scores with elevated enzyme markers and clinical deterioration[66]. The moderate correlation with hospital stays in the present study may reflect early intervention protocols or variability in supportive care efficiency.

Regarding PMS, the robust correlation with ventilator and ICU stay mirrors findings by **Krishna Moorthy et al. (2023)**, who noted PMS's comparable efficacy with advanced scoring systems like 'SOFA and APACHE II 'in predicting organ dysfunction and mortality[63]. However, the lack of correlation with hospital stay days in this study could be attributed to confounding discharge criteria or the heterogeneity in poison types and doses consumed.

The significant negative correlation between pseudocholinesterase levels and clinical outcomes is consistent with **Chaudhary et al. (2019)**, who observed that lower enzyme levels were linked to increased morbidity and mechanical ventilation needs[65]. This finding is further corroborated by **Eddleston et al. (2008)**, who highlighted pseudocholinesterase depression as a marker of poisoning severity and a potential therapeutic guide[3]. Variations across studies may stem from differences in population demographics, hospital resources, and time to treatment initiation. Collectively, these correlations affirm the combined use of POP, PMS, and pseudocholinesterase as a multi-pronged approach to early risk stratification in organophosphate poisoning.

Comparison of Outcome parameters with respect to POP Severity, PMS Severity and Pseudocholinesterase level

The present study underscores a significant correlation between 'the Peradeniya Organophosphorus Poisoning (POP) scale and clinical severity', as patients with severe POP scores necessitated substantially higher atropine and PAM doses alongside prolonged ventilator, ICU, and hospital stays, aligning with findings by **Kamath and Gautam (2021)** who

demonstrated the POP scale's prognostic utility in stratifying clinical outcomes[62]. Similarly, **Chintale et al. (2016)** observed that higher POP grades corresponded with increased morbidity, reinforcing its relevance in initial triage[69].

The Poisoning Mortality Score (PMS) also proved to be an effective predictor of outcomes, as high-risk patients required greater therapeutic intervention and prolonged ICU stay, comparable to **Krishna Moorthy et al. (2023)** who validated the PMS against SOFA and APACHE II, highlighting its specificity in acute organophosphate poisoning settings[63]. However, unlike our study, they observed a stronger predictive value of PMS on overall hospital stay, possibly due to differences in hospital discharge policies or sample demographics. Pseudocholinesterase levels displayed a strong inverse relationship with severity and resource utilization, supporting observations by **Chaudhary et al. (2019)**, who found low enzyme levels to be consistent with poor outcomes and prolonged hospitalization[65].

While our study found pseudocholinesterase levels predictive across all clinical parameters, **Dubey et al. (2016)** reported a weaker correlation, potentially due to delays in blood sampling post-exposure or enzyme variability in chronic exposures[66]. Additionally, analysis by **Ahmed et al. (2014)** indicated that mechanical ventilation needs were best predicted by enzyme levels and clinical scoring, validating our findings on ventilator and ICU stay duration[68]. Variations across studies may stem from regional differences in compound exposure, treatment latency, and supportive care quality, as highlighted by **Eddleston et al. (2008)**, who emphasized contextual management nuances[3].

Severity Scale	Atropine Dose (mg)	PAM Dose (g)	Ventilator Stay (days)	ICU Stay (days)	Hospital Stay (days)
POP Scale	84.45 ± 33.03	17.00 ± 6.10	4.35 ± 3.28	6.40 ± 3.45	10.85 ± 7.62
PMS	Higher in high-risk ($p < 0.001$)	Higher in high-risk ($p = 0.027$)	5.46 ± 3.28	6.69 ± 3.54	No significant difference ($p = 0.226$)
Pseudocholinesterase	87.18 ± 28.69	20.45 ± 15.39	4.23 ± 3.24	7.95 ± 3.47	12.82 ± 7.96
Proud Foot	69.26 ± 28.96	17.62 ± 11.14	2.40 ± 2.96	7.23 ± 2.99	14.32 ± 6.50

POP Scale, PMS Score, and 'Pseudocholinesterase Levels 'in Predicting Mortality in Organophosphate Poisoning

The study demonstrates high predictive accuracy of the 'Peradeniya Organophosphorus Poisoning (POP) 'scale, Poisoning Mortality Score (PMS), and pseudocholinesterase levels in assessing clinical severity and mortality among organophosphate poisoning patients, with PMS showing the highest area under the curve (AUC = 0.987).

Parameter	AUC (Present Study)	Sensitivity (%)	Specificity (%)	Optimal Cut-off
POP Scale	0.968	100	90.91	>7
PMS Score	0.987	100	89.77	>57
Pseudocholinesterase Levels	0.965	100	89.77	≤900

These findings are consistent with existing literature. **Kamath and Gautam (2021)** reported the POP scale to be a reliable prognostic marker, showing significant correlation with patient outcomes and mortality risk, though their reported AUC was slightly lower, likely due to differences in sample size and inclusion criteria[62]. Similarly, **Chintale et al. (2016)** observed that higher POP scores aligned with increased clinical severity and mortality, though their study lacked ROC-based validation[69]. **Dubey et al. (2016)** also emphasized POP scale effectiveness, correlating it with biochemical markers such as serum amylase and CPK, but reported moderate specificity, possibly due to demographic and regional toxin variations[66].

The PMS score's superior performance in the current study echoes findings by **Krishna Moorthy et al. (2023)**, who demonstrated its enhanced sensitivity over traditional scores like SOFA and APACHE II, attributing its reliability to incorporation of multiple physiological and clinical parameters[63].

In evaluating pseudocholinesterase levels, **Chaudhary et al. (2019)** and **Ahmed et al. (2014)** found strong inverse relationships with clinical severity, aligning closely with the present study's AUC of 0.965, although variations in lab techniques and population enzyme baselines might explain minor differences in specificity[65,68].

Collectively, these results reinforce the utility of multimodal assessment in organophosphate poisoning, with PMS emerging as the most robust predictor, while POP scale and pseudocholinesterase levels remain valuable adjuncts, especially in resource-limited settings.

CONCLUSION

CONCLUSION

The findings from the study demonstrated a strong positive correlation between POP scores and critical outcome parameters such as ventilator duration, ICU stay, and overall hospitalization, underscoring the scale's value in assessing clinical severity. Similarly, PMS scores exhibited significant associations with the need for ventilator support and ICU admission, highlighting its predictive strength for morbidity and mortality. Serum pseudocholinesterase levels, on the other hand, showed strong inverse relationships with clinical outcomes, with lower levels corresponding to more severe disease presentations and poorer prognoses. Comparative analysis across these parameters revealed that all three tools — POP, PMS, and pseudocholinesterase levels — were significantly linked with therapeutic requirements, including atropine and pralidoxime doses, and the length of critical care interventions.

Furthermore, receiver operating characteristic (ROC) curve analysis affirmed the high predictive accuracy of all three models in determining mortality risk, with PMS scoring slightly higher in diagnostic precision. Overall, the study highlights that POP and PMS scoring systems, along with biochemical markers like pseudocholinesterase, can be reliably used for early risk stratification, guiding treatment intensity, and anticipating outcomes in organophosphate poisoning cases. These tools serve as essential components for clinical decision-making in emergency and intensive care settings, enabling timely and targeted interventions that can potentially reduce morbidity and improve survival rates among affected individuals.

RECOMMENDATIONS

RECOMMENDATIONS

1. **Integration of POP and PMS Scoring in Initial Assessment:** It is recommended that healthcare providers in emergency and intensive care settings systematically implement the Peradeniya Organophosphorus Poisoning (POP) scale and Poisoning Mortality Score (PMS) during the initial evaluation of patients presenting with organophosphate poisoning. These scoring systems offer rapid, clinically relevant insights into the severity of poisoning and expected outcomes, enabling effective triage and prioritization of care. Utilizing these tools at the point of admission can facilitate early identification of high-risk patients who may require intensive monitoring and aggressive therapeutic interventions, ultimately improving patient outcomes and optimizing resource allocation.

2. **Routine Monitoring of Serum Pseudocholinesterase Levels:** Given the significant inverse relationship between pseudocholinesterase levels and clinical severity, it is advisable to incorporate serum pseudocholinesterase estimation as a routine biochemical marker within the first 24 hours of hospital admission for suspected organophosphate poisoning. This biomarker not only supports the clinical diagnosis but also serves as a reliable predictor of morbidity and mortality. Establishing standardized protocols for its timely measurement can enhance the prognostic accuracy of clinical assessments and aid in tailoring treatment regimens based on biochemical severity.

Development of Comprehensive Treatment Protocols Based on Severity Scores: Institutions managing organophosphate poisoning cases should develop and implement evidence-based treatment algorithms that integrate POP scores, PMS scores, and pseudocholinesterase levels. These protocols should guide therapeutic decisions, including the administration of atropine and pralidoxime, ventilator support, and ICU admission. Such stratified care pathways can ensure that

treatment intensity aligns with clinical need, improving survival rates, reducing hospital stay durations, and promoting efficient use of healthcare resource

LIMITATIONS

LIMITATIONS

This study was limited by its single-center design and small sample size, which may restrict the generalizability of the findings to broader populations. It also lacked long-term follow-up, focusing only on immediate clinical outcomes without assessing delayed neurological effects or quality of life post-discharge. Additionally, potential confounding variables—such as comorbidities, compound type and dose, time to hospital presentation, and variations in initial management—were not fully controlled, which may have influenced the observed correlations and overall conclusions.

SUMMARY

SUMMARY

This prospective observational study was conducted over a 18 months period, spanning from May 2023 to October 2024, in the Department of Emergency Medicine at Sri Devaraj Urs Medical College, Tamaka, Kolar. The research focused on patients presenting with organophosphate (OP) poisoning, either through direct admission to the emergency department or subsequent transfer to the ICU during the study duration.

A total of 10p patients aged over 18 years were enrolled, all of whom had a clinical diagnosis of OP poisoning. Inclusion criteria required a documented history of exposure to an OP compound, corroborated by typical clinical signs and symptoms, along with serum pseudocholinesterase levels obtained within 24 hours of hospital admission. Prior to participation, informed consent was obtained from either the patient or a legally authorized representative.

Objectives:

- To investigate the relationship between the Peradeniya Organophosphorus Poisoning (POP) scale and the clinical severity of organophosphate poisoning in affected patients.
- To evaluate the prognostic utility of the Poisoning Mortality Score (PMS) in predicting key clinical outcomes, including morbidity and mortality.
- To analyze the correlation between serum pseudocholinesterase (PChE) levels and the severity of organophosphate poisoning, and to determine their potential role in forecasting clinical progression and outcome.

Demographics: Majority of subjects were aged 21–40 years (58%) with a mean age of 35.99 ± 13.687 years; 75% were male.

Type of Poison: Chlorpyrifos was the most common compound (62%), followed by Propofenos (20%).

POP Severity: Moderate severity: 41%, Mild: 39%, Severe: 20%

AVPU Scale: Alert: 68%, Verbal response: 15%, Pain response: 10%, Unresponsive: 7%

PMS Risk Level: Low risk (31–50): 55%, Intermediate (51–65): 32%, High risk (>65): 13%, No subjects in very low-risk category.

Proud Foot Classification: Severe: 47%, Moderate: 28%, Mild: 25%.

Correlation with Outcome Parameters:

- **POP Score**: Positively correlated with ventilator stay (r=0.615), ICU stay (r=0.472), hospital stay (r=0.294)
- **PMS Score**: Positively correlated with ventilator stay (r=0.678), ICU stay (r=0.319); no correlation with hospital stay
- **Pseudocholinesterase**: Negatively correlated with ventilator (r=-0.566), ICU (r=-0.717), and hospital stay (r=-0.485)

Outcome: 88% of patients were discharged; 12% mortality rate

Treatment & Hospital Stay by POP Severity:

- Severe POP cases required significantly more atropine (84.45 mg) and PAM (17.00 g)
- Longer ventilator (4.35 days), ICU (6.40 days), and hospital stays (10.85 days)

By PMS Severity:

- Higher PMS linked to more atropine ($p<0.001$) and PAM ($p=0.027$)

Longer ventilator (5.46 days) and ICU stay (6.69 days); no difference in hospital stay

By Pseudocholinesterase Level:

- Severe cases required more atropine (87.18 mg), PAM (20.45 g), and had prolonged ventilator (4.23 days), ICU (7.95 days), and hospital stay (12.82 days)

By Proud Foot Classification:

- Severe classification linked with more atropine (69.26 mg), PAM (17.62 g), and extended ventilator (2.40 days), ICU (7.23 days), and hospital stay (14.32 days)

Mortality Prediction Accuracy:

- **PMS Score**: AUC 0.987, best predictor with 100% sensitivity, 89.77% specificity (cut-off >57)
- **POP Score**: AUC 0.968, 100% sensitivity, 90.91% specificity (cut-off >7)

Pseudocholinesterase Level: AUC 0.965, 100% sensitivity, 89.77% specificity (cut-off ≤900)

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ANNEXURES

ANNEXURE – I: PROFORMA

POP SCORE

PARAMETERS			**SCORE**
Heart rate	>60 41 - 60 <60	0 1 2	
Respiratory rate	>20 <20 <20 with central cyanosis	0 1 2	
Pupil size	>2mm <2mm pinpoint	0 1 2	
Fasciculations	Absent Present Generalised/Continuous Generalised And Continuous	0 1 2	
Level of consciousness	Conscious and Coherent Impaired No Response to Verbal Commands	0 1 2	
Seizures	Absent Present	0 1	

Total Score: the total points are graded as

- Mild (Score 0-3)
- Moderate (Score 4-7)
- Severe (Score 8-11).

PMS SCORE

Demographics	Age	<40	0
		40–59	7
		60–69	12
		70–74	16
		75–79	16
		≥80	19
	Sex	Male	0
		Female	4
Poisoning related factors	Intent of poisoning	Unintentional	0
		Intentional	8
		Unknown	9
	Route of poisoning	Contact	0
		Ingestion	8
		Inhalation	5
	Category of substances	OP	27
Initial vital signs in ED	SBP	>100	0
		70-99	6
		<69	15
	HR	70-119	0
		30-69	1
		120-159	4
		>160	8
	RR	12-24	0
		<11 / >25	5
	Temp	<39	0
		>39	6
	Mental status	Alert	0
		Verbal response	5
		Pain response	8
		Unresponsive	16

Total score:

- Very Low 0–27
- Low 28–40
- Intermediate 41–55
- High ≥56

ANNEXURE – I: PROFORMA

A STUDY OF CLINICAL SEVERITY AND OUTCOME OF ORGANOPHOSPHATE POISONING PATIENTS USING PERADENIYA ORGANOPHOSPHOROUS POISONING(POP) SCALE WITH POISONING MORTALITY SCORE(PMS) AND PSEUDOCHOLINESTERASE LEVELS.

Serial No: **Date:**

OP No: **IP No:**

1. **Name**
2. **Age:**
3. **Gender:**
4. **OP compound content:**
5. **Amount of ingestion:**
6. **Time of consumption:**
7. **Time of presentation:**
8. **Time interval between consumption and presentation:**
9. **Vitals - RR -**
    - HR -
    - Pupil size -
    - Fasciculation -
    - Seizures -
    - Level of consciousness-
    - SBP -
    - Temp -
    - AVPU-
10. **Pseudocholinesterase levels at the time of admission:**
11. **Total ATROPINE dose:**
12. **Total PAM dose:**
    - loading dose-
    - Infusion-

13. **Ventilator days:**

14. **ICU stay days:**

15. **Hospital stay days:**

16. **Discharge or mortality:**

ANNEXURE – II: PATIENT INFORMATION SHEET

Study Title: A STUDY OF CLINICAL SEVERITY AND OUTCOME OF ORGANOPHOSPHATE POISONING PATIENTS USING PERADENIYA ORGANOPHOSPHOROUS POISONING(POP) SCALE WITH POISONING MORTALITY SCORE(PMS) AND PSEUDOCHOLINESTERASE LEVELS.

Study Site: R. L. Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, affiliated to Sri Devaraj Urs Academy Higher Education & Research,

Aim: To correlate Peradeniya Organophosphorous Poisoning (POP) scale with pseudocholinesterase levels in predicting the severity and outcome of patients with a history of Organophosphorous compound poisoning.

This information is intended to give you the general background of the study. Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study, we will collect information (as per proforma) from you or a person responsible for you or both. Relevant history will be taken. General physical examination, systemic and local examination will be done. We will collect the treatment and relevant details from your hospital record. This information collected will be used for only dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide a thumb impression only if you voluntarily agree to participate in this study. The investigations needed are part of routine investigations.

For any further clarification you can contact the study investigator

Dr. Y Nikhil Reddy

Mobile No: 9515137238

E-mail id: josephnikhilreddy@gmail.com.

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಪೆರಡೆನಿಯಾ ಆರ್ಗನೊಫಾಸ್ಫರಸ್ ವಿಷಕಾರಿ (ಪಾಪ್) ಸ್ಕೇಲ್ ಅನ್ನು ಬಳಸುವ ಆರ್ಗನೊಫಾಸ್ಫೇಟ್ ವಿಷಕಾರಿ ರೋಗಿಗಳ ಕ್ಲಿನಿಕಲ್ ತೀವ್ರತೆ ಮತ್ತು ಫಲಿತಾಂಶದ ಅಧ್ಯಯನ.

ಅಧ್ಯಯನ ತಾಣ: ಆರ್.ಎಲ್.ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರವು ಶ್ರೀ ದೇವರಾಜ್ ಅರ್ಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿಗೆ ಲಗತ್ತಿಸಲಾಗಿದೆ, ಇದು ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಅಕಾಡೆಮಿ ಉನ್ನತ ಶಿಕ್ಷಣಕ್ಕೆ ಸಂಯೋಜಿತವಾಗಿದೆ

ಗುರಿ: ಆರ್ಗಾನೊಫಾಸ್ಫರಸ್ ಸಂಯುಕ್ತ ವಿಷದ ಇತಿಹಾಸ ಹೊಂದಿರುವ ರೋಗಿಗಳ ತೀವ್ರತೆ ಮತ್ತು ಫಲಿತಾಂಶವನ್ನು ಊಹಿಸಲು ಪೆರಾಡೆನಿಯಾ ಆರ್ಗಾನೊಫಾಸ್ಫರಸ್ ವಿಷ (ಪಿಒಪಿ) ಪ್ರಮಾಣವನ್ನು ಸೂಡೊಕೊಲಿನೆಸ್ಟರೇಸ್ ಮಟ್ಟಗಳೊಂದಿಗೆ ಪರಸ್ಪರ ಸಂಬಂಧಿಸುವುದು.

ಈ ಮಾಹಿತಿಯು ಅಧ್ಯಯನದ ಸಾಮಾನ್ಯ ಹಿನ್ನೆಲೆಯನ್ನು ನಿಮಗೆ ನೀಡಲು ಉದ್ದೇಶಿಸಲಾಗಿದೆ. ದಯವಿಟ್ಟು ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬದ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದರೆ, ನಾವು ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆಪ್ರೊಫಾರ್ಮಾ ಪ್ರಕಾರ) ನಿಮ್ಮಿಂದ ಅಥವಾ ನಿಮ್ಮಿಂದ ಅಥವಾ ಇಬ್ಬರಿಗೂ ಜವಾಬ್ದಾರರಾಗಿರುವ ವ್ಯಕ್ತಿಯಿಂದ. ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. ಸಾಮಾನ್ಯ ದೈಹಿಕ ಪರೀಕ್ಷೆ, ವ್ಯವಸ್ಥಿತ ಮತ್ತು ಸ್ಥಳೀಯ ಪರೀಕ್ಷೆಯನ್ನು ಮಾಡಲಾಗುತ್ತದೆ. ನಿಮ್ಮ ಆಸ್ಪತ್ರೆಯ ದಾಖಲೆಯಿಂದ ನಾವು ಚಿಕಿತ್ಸೆ ಮತ್ತು ಸಂಬಂಧಿತ ವಿವರಗಳನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ.

ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿಯು ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಸಹಿ ಮಾಡುವ ಅಗತ್ಯವಿದೆ. ಅಗತ್ಯವಿರುವ ತನಿಖೆಗಳು ವಾಡಿಕೆಯ ತನಿಖೆಗಳ ಭಾಗವಾಗಿದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು

ಡಾ. ವೈ ನಿಖಿಲ್ ರೆಡ್ಡಿ
ಮೊಬೈಲ್ ನಂ: **9515137238**
ಇ-ಮೇಲ್ ಐಡಿ: josephnikhilreddy@gmail.com

ANNEXURE – III: WRITTEN INFORMED CONSENT

Study Title: A STUDY OF CLINICAL SEVERITY AND OUTCOME OF ORGANOPHOSPHATE POISONING PATIENTS USING PERADENIYA ORGANOPHOSPHOROUS POISONING(POP) SCALE WITH POISONING MORTALITY SCORE(PMS) AND PSEUDOCHOLINESTERASE LEVELS.

If you agree to participate in the study, we will collect information (as per proforma) from you or a person responsible for you or both. We will collect the treatment and relevant details from your hospital record. This information collected will be used for only dissertation and publication. This study has been reviewed by the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

I understand that I remain free to withdraw from the study at any time and this will not change my future care. I have read or have been read to me and understand the purpose of the study, the drug that will be used, the risk and benefits associated with my involvement in the study and the nature of information that will be collected and disclosed during the study. I have had the opportunity to ask my questions regarding various aspects of the study and my questions were answered to my satisfaction. I, the undersigned agree to participate in this study and authorize the collection and disclosure of my personal information for dissertation.

Subject Name: **DATE:**

Signature / Thumb impression:

Parents / Guardians name: **DATE:**

Signature / Thumb impression:

Name and Signature of the person taking consent:

Slno	UHID no	Age(yrs)	Gender	Compound	Time interval	HR	RR	PUPILS (mm)	SEIZURES	Fasciculations	LEVEL OF CONSCIOUSNES	Total POP	SBP
1	270637	70	M	C	4	54	16	1	N	Y	Y	6	110
2	268499	23	M	Ph	4	56	18	1	N	Y	N	7	105
3	265975	35	F	M	2	80	18	3	N	N	Y	4	140
4	265201	18	F	C	3	120	18	3	N	N	Y	3	130
5	253263	28	M	C	1	38	15	3	N	Y	N	8	120
6	279102	23	M	Dz	2	56	18	1	N	N	Y	4	120
7	259608	28	M	D	3	58	20	2	N	N	Y	3	140
8	254658	20	M	C	1	89	18	1	N	Y	Y	5	140
9	250786	20	M	D	3	110	21	3	N	N	Y	1	135
10	257781	35	M	C	2	93	24	1	N	N	Y	2	155
11	252724	35	F	C	2	40	20	1	Y	Y	Y	6	90
12	228070	40	M	C	2	110	24	1	N	N	Y	4	100
13	23026	28	F	C	1	40	22	3	N	N	Y	2	150
14	236792	24	M	C	2	80	20	2	N	N	Y	4	140
15	202336	38	M	P	1	40	17	3	N	N	Y	4	130
16	222383	30	M	C	1	80	22	1	N	Y	N	7	80
17	236118	22	M	C	3	86	22	3	Y	Y	Y	6	90
18	225169	18	F	E	4	34	14	1	Y	Y	N	10	70
19	207372	39	M	C	1	78	20	3	N	N	Y	1	130
20	277140	18	F	C	3	44	14	1	Y	Y	Y	6	70
21	281001	56	M	D	9	38	18	1	Y	Y	N	10	60
22	281792	38	M	C	3	44	18	1	N	N	Y	4	110
23	291142	32	M	C	2	65	20	2	N	N	Y	3	120
24	287452	38	F	P	4	39	16	1	Y	Y	N	9	90
25	299258	32	F	P	1	42	18	1	Y	Y	N	10	80
26	299965	42	M	P	1	74	18	2	N	Y	Y	5	120
27	304044	21	F	C	7	84	21	2	N	N	Y	3	140
28	312520	38	F	C	1	100	20	2	N	N	Y	3	130
29	311220	52	M	C	2	58	18	1	N	Y	Y	7	130
30	310605	30	F	E	3	49	17	1	N	Y	Y	5	120
31	314502	24	M	C	2	52	16	2	Y	Y	Y	8	130
32	312102	29	M	C	4	40	17	1	Y	Y	N	9	140
33	315977	48	M	P	3	58	18	2	N	N	Y	4	130
34	314104	45	M	C	1	42	18	1	N	Y	N	7	100
35	334324	28	M	C	4	56	15	1	Y	Y	N	9	100
36	349731	25	F	C	3	56	16	1	N	Y	Y	6	90
37	356764	40	F	D	5	70	20	2	N	N	Y	3	110
38	346442	23	F	C	3	55	16	1	N	Y	Y	6	100
39	363168	42	F	C	5	60	18	2	N	N	Y	3	90
40	363233	35	M	C	4	54	16	1	Y	Y	N	9	90
41	366205	35	M	C	3	55	18	2	N	Y	Y	6	120
42	368137	24	M	C	1	48	16	1	Y	Y	N	8	80
43	367962	30	M	P	1	58	18	2	N	Y	N	7	140
44	376954	40	M	D	3	44	16	1	N	Y	N	8	130
45	374522	58	M	C	5	30	14	1	Y	Y	N	10	60
46	385459	30	F	C	3	75	18	2	N	N	Y	4	110
47	385462	45	M	C	4	52	15	1	N	Y	Y	7	100
48	385502	32	M	P	4	45	16	1	Y	Y	N	9	90
49	386693	19	F	C	2	70	18	2	N	N	Y	4	130

TEMP(F)	AVPU	PMS SCORE	Pseudocholinesterase level	Proud foot classification	Total ATROPINE	Total PAM (g)	Ventilator stay days	ICU stay days	Hospital stay days	Discharge or mortality
37	V	65	560	S	60	12	0	12	21	D
36	A	44	750	S	52	10	0	11	24	D
36	A	47	2100	S	41	6	0	6	20	D
39	A	57	2600	Mo	40	12	0	6	18	D
38	A	44	470	S	111	16	6	9	24	D
38	A	44	750	S	50	18	0	11	24	D
38	A	44	2800	Mo	35	12	0	6	11	D
35	A	43	1942	S	35	14	0	10	18	D
37	A	43	5142	Mi	10	4	0	2	14	D
36	A	43	5041	Mi	10	4	0	1	14	D
36	P	62	1285	S	89	16	4	10	22	D
39	A	56	2633	Mo	26	22	3	5	18	D
39	A	54	4700	Mi	15	5	0	1	18	D
38	A	43	3890	Mo	20	5	0	2	14	D
37	V	43	1127	S	52	20	0	5	13	D
39	U	49	1229	S	70	25	0	4	23	D
39	U	49	1248	S	74	26	0	2	11	D
39	P	82	200	S	78	20	6	6	6	M
35	A	43	6055	Mi	26	8	0	4	9	D
39	P	50	200	S	67	18	2	10	17	D
38	U	82	245	S	40	4	1	1	1	M
38	A	44	1150	S	35	15	0	5	21	D
37	A	44	2475	S	26	12	0	2	18	D
39	V	60	226	S	75	10	4	4	4	M
37	V	50	310	S	92	26	5	11	27	D
38	A	50	1900	S	46	15	2	9	21	D
36	A	47	4100	Mo	32	10	0	4	16	D
36	A	47	3900	Mo	25	6	0	3	15	D
38	A	51	775	S	64	10	2	7	19	D
38	A	50	1450	S	48	16	2	9	21	D
39	A	50	2100	S	38	14	3	6	19	D
38	A	43	1850	S	44	16	4	9	24	D
37	A	43	2200	S	64	14	2	5	16	D
38	V	47	1950	S	58	12	0	7	14	D
38	A	50	2100	S	54	12	0	4	12	D
39	V	51	3000	Mo	76	14	0	3	14	D
37	A	47	5400	Mi	40	10	0	2	10	D
38	A	47	3500	Mo	74	18	2	5	20	D
38	A	50	4700	Mi	46	11	0	2	5	D
39	P	82	450	S	68	8	3	3	3	M
37	V	47	4100	Mo	66	12	0	1	4	D
36	V	50	4800	Mi	46	10	0	1	5	D
39	A	47	3900	Mo	57	12	0	2	3	D
39	V	51	2700	Mo	58	12	0	5	10	D
39	U	87	240	S	90	22	2	2	2	M
37	A	47	4100	Mi	56	14	0	3	6	D
38	V	56	1500	Mo	76	10	0	4	6	D
39	P	89	900	S	90	22	2	6	15	M
36	A	47	4700	Mi	30	12	0	2	6	D

50	397000	38	M	P	2	50	17	1	Y	Y	N	8	80
51	349731	25	F	P	2	65	16	2	N	N	Y	4	80
52	363168	42	M	C	3	66	16	2	N	N	Y	3	100
53	363221	35	M	C	5	70	18	3	N	N	Y	3	95
54	364091	63	M	C	4	72	20	3	N	N	Y	2	110
55	366205	35	M	C	5	58	19	2	N	N	Y	4	105
56	368137	24	M	C	2	56	18	1	N	Y	N	5	100
57	367962	30	M	Ph	2	50	16	1	Y	Y	N	6	140
58	376954	40	M	Di	1	52	16	2	Y	Y	N	6	130
59	374522	58	M	C	4	82	18	2	N	N	Y	2	110
60	381509	70	M	C	6	66	16	2	N	N	Y	1	115
61	381673	30	F	C	3	75	16	2	N	N	Y	2	120
62	385459	30	F	C	6	76	17	3	N	N	Y	1	130
63	385462	45	M	C	8	78	18	2	N	N	Y	2	110
64	385502	32	M	C	7	88	19	2	N	N	Y	1	120
65	386693	19	F	C	5	90	20	2	N	N	Y	1	115
66	391099	48	M	C	2	70	22	2	N	N	Y	0	125
67	393634	30	M	C	3	68	16	2	N	N	Y	2	120
68	460931	32	M	C	2	66	18	2	N	N	Y	2	100
69	478047	30	M	M	3	58	17	1	Y	Y	N	5	95
70	492715	32	M	C	2	60	18	2	N	N	Y	4	100
71	493182	51	M	C	5	96	16	2	N	N	N	2	110
72	495255	43	M	C	4	92	16	2	N	N	Y	2	110
73	498757	34	M	C	6	82	18	3	N	N	Y	1	120
74	499185	18	M	C	2	78	17	2	N	N	Y	1	130
75	499234	58	M	P	2	60	15	2	N	N	Y	4	120
76	499727	45	F	P	3	56	18	2	Y	Y	Y	6	100
77	503612	36	F	Di	2	60	16	2	N	N	N	4	110
78	504883	55	M	P	5	48	22	2	Y	Y	N	8	70
79	504911	18	M	P	4	80	16	2	N	N	Y	3	100
80	504919	39	M	C	4	70	18	2	N	N	Y	2	110
81	484497	43	M	C	5	75	16	2	N	N	Y	3	120
82	485814	40	M	P	2	64	17	2	N	N	Y	4	110
83	486315	27	M	C	1	62	18	2	N	N	Y	3	140
84	486391	18	F	C	2	90	16	2	N	N	Y	3	130
85	488742	49	M	C	3	80	16	2	N	N	Y	2	130
86	491861	30	M	P	2	84	18	2	N	N	Y	4	100
87	453218	18	M	C	3	70	16	2	N	N	Y	3	110
88	465187	28	M	P	4	48	22	1	Y	Y	N	9	70
89	466106	34	M	P	4	80	19	2	N	N	Y	4	120
90	467078	18	M	Ph	4	76	17	3	N	N	Y	3	130
91	437639	77	M	P	5	44	24	1	Y	Y	N	8	80
92	437705	83	M	C	3	94	16	2	N	N	Y	2	125
93	438731	50	M	C	3	52	20	1	Y	Y	N	8	90
94	443665	36	M	Ph	4	62	18	2	N	N	Y	3	135
95	528714	49	M	C	4	48	16	1	Y	Y	N	8	70
96	521113	58	M	P	2	76	18	2	N	N	Y	4	110
97	523229	20	M	Di	3	60	16	2	N	Y	Y	6	100
98	523705	41	F	C	6	80	18	3	N	N	Y	4	120
99	523783	49	M	Ph	5	44	16	1	Y	Y	N	9	80
100	524072	23	M	P	5	64	17	3	N	N	Y	3	130

39	P	82	1400	S	88	18	3	7	11	D
38	A	56	4500	Mi	60	8	0	3	5	D
37	A	51	5200	Mi	8	6	0	1	3	D
38	A	47	4900	Mi	10	6	0	1	3	D
38	A	43	6200	Mi	10	6	0	1	2	D
38	A	47	4000	Mo	20	12	0	5	9	D
38	V	56	2400	S	38	14	0	6	10	D
39	P	60	650	S	88	18	4	8	14	D
39	U	82	720	S	92	22	6	8	11	D
38	A	47	4500	Mi	12	10	0	2	3	D
37	A	51	5900	Mi	10	8	0	1	2	D
38	A	43	5400	Mi	8	6	0	2	3	D
38	A	47	5920	Mi	6	6	0	2	3	D
38	A	47	4900	Mi	15	8	0	2	5	D
39	A	43	5100	Mi	10	6	0	1	3	D
38	A	43	4250	Mo	12	8	0	2	4	D
38	A	47	6100	Mi	6	6	0	1	3	D
38	A	51	5600	Mi	8	6	0	3	6	D
38	A	51	5450	Mi	11	6	0	1	3	D
39	V	62	1500	S	52	16	0	11	16	D
37	A	47	2420	S	42	12	0	6	11	D
38	A	47	4200	Mo	26	6	0	2	4	D
38	A	43	2650	Mo	58	12	0	3	6	D
38	A	43	4100	Mo	28	6	0	1	2	D
38	A	43	3900	Mo	18	6	0	2	3	D
39	A	51	1750	S	20	15	0	8	14	D
39	V	62	950	S	84	84	2	12	16	D
38	A	51	2150	S	44	12	0	6	12	D
39	P	82	360	S	105	22	7	11	11	M
37	A	51	3800	Mo	52	8	0	7	10	D
38	A	51	4500	Mi	38	6	0	4	7	D
38	A	47	2500	Mo	68	12	0	5	8	D
39	A	60	1500	S	76	16	0	9	12	D
38	A	47	3650	Mo	46	10	0	4	7	D
38	A	47	3200	Mo	60	8	0	5	8	D
39	A	43	3450	Mo	48	6	0	4	5	D
38	A	51	1500	S	66	16	0	8	10	D
37	A	51	2600	Mo	46	12	0	4	7	D
39	U	82	400	S	150	24	12	12	12	M
38	A	60	1900	S	65	12	0	6	12	D
38	A	56	2500	Mo	46	8	0	3	6	D
39	P	87	345	S	86	18	8	8	8	M
38	A	51	4250	Mo	22	10	0	2	4	D
39	V	82	680	S	146	20	10	12	12	M
38	A	56	4000	Mo	20	6	0	4	7	D
39	P	84	900	S	126	24	6	6	6	M
38	A	43	3800	Mo	56	12	0	4	8	D
39	V	47	1600	S	72	14	0	5	10	D
38	A	52	4600	Mi	36	8	0	3	8	D
40	U	87	380	S	104	22	5	5	5	M
38	A	51	4700	Mi	30	8	0	2	4	D