TO STUDY SICK EUTHYROIDSTATE AS A PROGNOSTIC INDICATOR IN ACUTE ORGANOPHOSPHORUS POISONING

by

Dr Poongulali M D S

Dissertation submitted to

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

KOLAR, KARNATAKA

In partial fulfilment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

Under the Guidance of

DR Raveesha A

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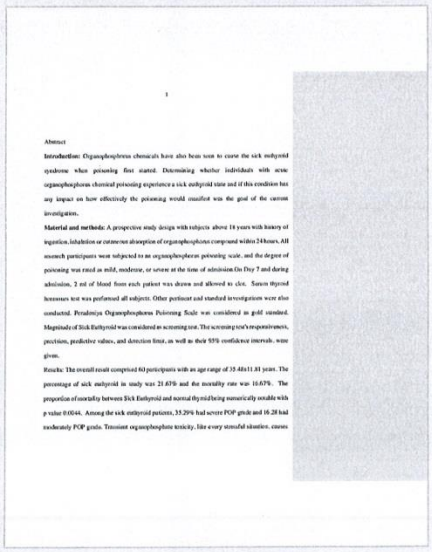

Abstract

Introduction: Organophosphorus chemicals have also been seen to cause the sick euthyroid syndrome when poisoning first started. Determining whether individuals with acute organophosphorus chemical poisoning experience a sick euthyroid state and if this condition has any impact on how effectively the poisoning would manifest was the goal of the current investigation.

Material and methods: A prospective study design with subjects above 18 years with history of ingestion, inhalation or cutaneous absorption of organophosphorus compound within 24hours. All research participants were subjected to an organophosphorus poisoning scale, and the degree of poisoning was rated as mild, moderate, or severe at the time of admission.On Day 7 and during admission, 2 ml of blood from each patient was drawn and allowed to clot. Serum thyroid hormones test was performed all subjects. Other pertinent and standard investigations were also conducted. Peradeniya Organophosphorus Poisoning Scale was considered as gold standard. Magnitude of Sick Euthyroid was considered as screening test. The screening test's responsiveness, precision, predictive values, and detection limit, as well as their 95% confidence intervals, were given.

Results: The overall result comprised 60 participants with an age range of 35.48±11.81 years. The percentage of sick euthyroid in study was 21.67% and the mortality rate was 10.67%. The proportion of mortality between Sick Euthyroid and normal thyroid being numerically notable with p-value 0.0344. Among the sick euthyroid patients, 35.29% had severe POP grade and 16.28 had moderately POP grade. Transient organophosphate toxicity, like every stressful situation, causes

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1 Abstract Introduction: Organophosphorus chemicals have also been seen to cause the sick euthyroid syndrome when poisoning first started. Determining whether individuals with acute organophosphorus chemical poisoning experience a sick euthyroid state and if this condition has any impact on how effectively the poisoning would manifest was the goal of the current investigation. Material and methods: A prospective study design with subjects above 18 years with history of ingestion, inhalation or cutaneous absorption of organophosphorus compound within 24 hours. All research participants were subjected to an organophosphorus poisoning scale, and the degree of poisoning was rated as mild, moderate, or severe at the time of admission.On Day 7 and during admission, 2 ml of blood from each patient was drawn and allowed to clot. Serum thyroid hormones test was performed all subjects. Other pertinent and standard investigations were also conducted. Peradeniya Organophosphorus Poisoning Scale was considered as gold standard. Magnitude of Sick Euthyroid was considered as screening test. The screening test's responsiveness, precision, predictive values, and detection limit, as well as their 95% confidence intervals, were given. Results: The overall result comprised 60 participants with an age range of 35.48±11.81 years. The percentage of sick euthyroid in study was 21.67% and the mortality rate was 16.67%. The proportion of mortality between Sick Euthyroid and normal thyroid being numerically notable with p value 0.0044. Among the sick euthyroid patients, 35.29% had severe POP grade and 16.28 had moderately POP grade. Transient organophosphate toxicity, like every stressful situation, causes 2 ill euthyroid syndrome in certain subjects. Sick euthyroid status gave a predictive validity in predicting the POP grade of poisoning a responsiveness of 35.29%, precision of 83.72%, positive predictive value 46.15%, negative predictive value 76.60% with a total diagnostic accuracy of 70.00%. Mortality rate is high among the sick euthyroid. Conclusions: Sick euthyroid state among OP poisoning subjects has found to be effective in predicting the mortality and grading of OP poisoning in these subjects. Key words: Organophosphorus Compound, euthyroid state, positioning, sick euthyriod Introduction Exposure to toxicity is among the most serious illnesses confronting lesser developed nations.1 Poisoning is a leading root of unexpected fatality in rural Asia. Insecticides, pesticides, herbicides, and chemical warfare agents all use organophosphorous (OP) compounds, also known as anticholinesterases.2 Because OP compounds are so readily available, there are more cases of pesticide poisoning and more deaths as a result, which is a problem for public health in developing nations like India3,4 According to estimates from the WHO and other studies, a significant portion of self-attempted deaths in the developing world were caused by OP pesticides. 5,6 Organophosphate compounds (OPC) seem to be a primary cause of poisoning-related deaths and rate of illness in hospitals, according to

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Dr. Poongulali M D S

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ABBREVIATIONS

Glossary	Abbreviations
Ach	Acetylcholine
AChE	Acetylcholine esterase
ACTH	adrenocorticotropic hormone
AMI	anti-microsomal antibodies
APACHE	Acute Physiology and Chronic Health Evaluation
ATA	Antithyroglobulin antibodies
ATDs	Antithyroid medications
CAT	choline acetyltransferase
CBC	Completed blood cell count
CNS	central nervous system
CPK	creatine phosphokinase
ESS	euthyroid sick syndrome
FNAC	Fine needle aspiration cytology
GO	grave's orbitopathy
hCG	human chorionic gonadotropin
HTN	Hypertension
IGF-1	insulin-like growth factor 1
IQR	interquartile ranges
OP	Organophosphorous
OP	Organophosphate
OPC	Organophosphate compounds
OPP	organophosphate compound poisoning
PET	positron emission tomography
POP	The Peradeniya Organophosphorus Poisoning
rT3	Triiodothyronine
SAPS	Simplified Acute Physiology Score
SOFA	Sequential Organ Failure Assessment

T3	triiodothyronine
T4	free thyroxine
TBG	thyroid-binding globulin
Tg	Thyroglobulin
TSH	thyroid-stimulating hormone
WHO	world health organization

ABSTRACT

Introduction: Organophosphorus chemicals have also been seen to cause the sick euthyroid syndrome when poisoning first started. Determining whether individuals with acute organophosphorus chemical poisoning experience a sick euthyroid state and if this condition has any impact on how effectively the poisoning would manifest was the goal of the current investigation.

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Key words: **Organophosphorus Compound, euthyroid state, positioning, sick euthyriod**

INTRODUCTION

INTRODUCTION

Exposure to toxicity is among the most serious illnesses confronting lesser developed nations.[1]Poisoning is a leading root of unexpected fatality in rural Asia. Insecticides, pesticides, herbicides, and chemical warfare agents all use organophosphorous (OP) compounds, also known as anticholinesterases.[2] Because OP compounds are so readily available, there are more cases of pesticide poisoning and more deaths as a result, which is a problem for public health in developing nations like India.[3,4] According to estimates from the WHO and other studies, a significant portion of self-attempted deaths in the developing world were caused by OP pesticides.[5] Organophosphate compounds (OPC) seem to be a primary cause of poisoning-related deaths and rate of illness in hospitals, according to studies.[6] According to one of the review studies, self-poisoning mortality rates generally exceed 20%, while they hover around 46% for OPC [3] Following cutaneous, inhalation, or oral administration to lesser evaporative pesticides (e.g., chlorpyrifos, dimethoate) or highly evaporative chemical agents, acute organophosphorus poisoning develops (e.g., sarin, tabun). Acetylcholine is accumulated and acetylcholine receptors are overexcited at the nerve-muscle terminals, in both the division of peripheral nervous system, and at synapses where acetylcholinesterase is inhibited. Early clinical signs (overstimulation at nerve muscle terminals) comprise watery sputum, sudden contraction of the bronchial muscles, excessive constriction of the pupil, the extracellular fluid secreted by the salivary gland, feces, excretion of urine, and low blood pressure and show parasympathetic system involvement. At this stage, paresis and muscle contractions, in addition to signs of central nervous system involvement like seizures, coma, and respiratory failure, are frequent..[7]Early deaths are caused by respiratory failure, which includes cardiovascular collapse, central hypoventilation, myasthenia gravis, watery sputum, and sudden contraction of the bronchial muscles. Treatment calls for an urgent admission, the administration of enough atropine to achieve

"atropinisation" (the counteraction of overt cholinergic effects), as well as airway and ventilator support[8]The degree of poisoning correlates with the decline in pseudocholine esterase activity in severe instances of OP toxicity. Despite the availability of numerous assessment methods like Acute Physiology and Chronic Health Evaluation[.9] (APACHE) and Simplified Acute Physiology Score (SAPS), laboratory analysis is still essential for confirming poisoning, identifying the onset of system failure, and determining a severity of toxicity. The most accurate test for OP poisoning in a laboratory setting is the estimation of plasma cholinesterase.[9]

One of the many biochemical abnormalities linked to OP compound poisoning is hyperamylasemia, which is well known and may result from too much cholinergic stimulation of the pancreas. Studies by Matsumiya N et al. and Lee HC evaluated the serum amylase's prognostic value in OP poisoning.[10] Increased serum amylase is less sensitive and specific, and acute pancreatitis occurs frequently in OP poisoning. In order to make an early diagnosis of pancreatitis in subjects with increased amylase concentration, serum lipase measurement can be of assistance. As a result, numerous studies were conducted to assess a promising biochemical marker in OP poisoning. Measurement of creatine phosphokinase (CPK) in acute OP poisoning, according to a recent study, it may be used to anticipate along with the outcome evaluation by Bhattacharya K et al.[11]

Types of hormones such as thyroid-stimulating hormone (TSH), adrenocorticotropic hormone (ACTH), and insulin-like growth factor 1 (IGF-1) insufficiency, all of whom are linked to enzyme cholinesterase concentrations, are the acute hormonal effects of OP. At 3 months of monitoring, many subjects with OP-related hormone deficiencies recover. The prolonged impact of these chemicals are less understood, though. Problems of nervous

system, certain malignancies, adverse reproductive consequences, and hormonal issues are examples of known chronic effects.[6]

Need for the study

An NTI (non thyroid illness) without preexisting thyroid gland or hypothalamic-pituitary dysfunction can present with nonthyroidal illness syndrome, which is characterized by abnormal thyroid function test results. A reduced concentration of total triiodothyronine (T3) in blood, which may be seen within 2 hours after the commencement of significant physical strain, is the most common thyroid function deficiency in subjects with the onset of the condition. The onset of a further complicated condition linked to reduced concentrations of triiodothyronine and thyroxine (main hormone) occurs gradually as the illness worsens (T4). Thyrotropin (TSH) levels stay constant or marginally decrease. Thyroid hormone levels have been found to be modified in cases of hunger, of onset and persistent medical disorders, bone marrow transplantation, operation, trauma, heart attack, and, in reality, any potentially serious sickness. The more severe the hormonal pattern alterations, the less favourable the outcome. In acute organophosphorus chemical exposure, a nonthyroidal illness syndrome has also been shown. To investigate the incidence of nonthyroidal illness syndrome condition in patients suffering from acute organophosphorus chemical toxicity and to determine if certain nonthyroidal illness syndrome has a predictive value in the prognosis of acute organophosphorus chemical toxicity.

Thyrotropin (TSH) levels are either unchanged or slightly decreased. Onset and persistent conditions, bone marrow replacement, surgical intervention, injury, heart attack, and, in general, extremely serious conditions, have all been linked to altered thyroid hormone levels. The poorer the outcome, the more severe the alterations in hormonal patterns. In situations of onset of toxicity, the ill euthyroid condition has also been observed with organophosphorus

compounds. Hence the present study aimed to investigate whether patients with acute organophosphorus compound poisoning experience sick euthyroid state and to determine whether this sick euthyroid state has any bearing on how well the poisoning will turn out.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Acute organophosphorus poisoning:

Organophosphorus Compound:

Organophosphorus compounds are organic compounds containing phosphorus. They are the derivatives of phosphoric, phosphonic, or phosphinic acid in which the oxygen atoms directly bound to the phosphorus atom can be replaced by sulphur or nitrogen atoms. The sulphur atom forms a coordinative covalent link with the phosphor atom making organophosphate substance inhibit acetylcholinesterase. To become physiologically active, these chemicals must undergo spontaneously or biotransformation processes to become a functional group that contains a single oxygen atom. Organophosphorus compound biotransformation processes involve a broad range of enzymatic reactions that can render them poisonous, or non-poisonous for acetylcholinesterase. [12] Therefore, organophosphates attach to cholinesterase enzymes and inhibit them, acute poisoning causes a cholinergic crisis. [13]

"In 1921, Loewi established that acetylcholine (ACh) is a substance capable of transmitting neural signals from one neuron to another through synapses. Acetylcholine is a neurotransmitter made from acetyl coenzyme A. Acetyl COA is synthesised from glucose and choline via choline acetyltransferase (CAT)". Acetylcholine is housed in presynaptic membrane packets known as vesicles. When stimulated, each packet is discharged. Acetylcholine esterase (AChE) breaks down the neurotransmitter acetylcholine into acetate and choline via a hydrolytic mechanism, ultimately halting its impact on muscarinic and nicotinic receptors.[14] Organophosphates can bind to acetylcholine esterase irreversibly and restrict acetylcholine breakdown. The release of acetylcholine causes overstimulation of nicotinic and muscarinic receptors.

Muscarinic and Nicotinic receptors are found in abundance throughout the body.

Nicotine Receptors- are classified into 2 types, the peripheral and central where both the Nn/N1 and Nm/N2 (nicotinic receptors)are present. Nm or NI peripheral nicotinic receptors are found at neuromuscular junctions.

- Fasciculation and muscle weakening at the N1 neuromuscular junction
- Hypertension and tachycardia are symptoms of the N2 ANS.[15]

Muscarinic Receptors- are well-known for mediating a variety of processes throughout the central and peripheral neural systems. The exocrine glands, heart, and smooth muscles of the internal organs are all innervated by postganglionic peripheral muscarinic receptors. The sympathetic postganglionic fibres innervate the sweat gland.[16]It has long been recognised that the parasympathetic nervous system, which is regulated by acetylcholine, is critical in controlling cardiovascular function. [16]Clinical manifestations of organophosphates mostly occur in the digestive, circulatory, respiratory, and central neurological systems.Chemical agents called organophosphorus compounds are widely used across the globe, mostly in farming for pesticides, pesticides, and herbicides. Additionally, they are employed as medicinal agents like ecothiopate for the glaucoma treatment and as nerve gas in chemical warfare, such as Sarin gas.[17]These substances exhibit ecological persistence and accumulation, are extremely hazardous, and annually cause a large number of poisoning cases and fatalities.[17] Organophosphates are used as a generic name to include all the organic compounds containing phosphorus.[18] There are 13 different forms of organophosphates, and these substances are what most pesticides are made of. The most used pesticides are:[15]

1. Parathion
2. Chlorpyrifos
3. Diazinon
4. Dichlorvos

5. Phosmet
6. Fenitrothion
7. Tetrachlorvinphos
8. Azamethiphos
9. Azinphos-Methyl
10. Malathion
11. Methyl Parathion

Organophosphates are also commonly used as a nerve agent and are classified into 3 groups

G series (developed by the Germans during WWII)

- Sarin
- Soman
- Tabun

V series (developed by the British)

- VE
- VG
- VM
- VR
- VM
- VX

Novichok

“Newcomer" (developed in the former Soviet Union in the late 70s and early 80s)

There is a scarcity of data on nerve gas and organophosphate pesticide exposure. Most of the organophosphate pesticide exposure occurs in rural regions where insecticides pesticides, and herbicides, are widely used. Exposure may occur accidentally or on purpose. Consuming foods like rye, flour, and cooking oil might expose one to certain chemicals. Spray for ants and cockroaches might possibly expose people.[15] The following are examples of exposure routes:

- Inhalation
- Direct contact
- Ingestion

OPC poisoning – define/describe,

Organophosphate (OP) insecticides are the most important pesticides used worldwide. Poisoning of organophosphate compounds is caused due to exposure to organophosphate pesticides and causes a significant number of poisonings which may occur through inhalation, unintentional or purposeful consumption, or contact to, agrochemicals, or dermal contact.[17] The major characteristic of organophosphorus chemical toxicity is the restraint of carboxyl ester hydrolases, specifically "acetylcholinesterase" (AChE), which results in the excessive buildup of acetylcholine and hinders the two main types of cholinergic receptors at a neural connections in both part of nervous systems.[17,19]Acetylcholinesterase inhibits the release of neurotransmitters in brain synapses and neuromuscular junctions by hydrolysing the neurotransmitter acetylcholine. The cholinesterase inhibitors are classified into three types: reversible, irreversible, and pseudo-reversible. Organophosphate compounds block the important enzyme acetylcholinesterase in an irreversible manner (AChE). This causes a build-up of acetylcholine (ACh), which can lead to pulmonary breakdown or even mortality is possible. Restoration of Acetylcholinesterase blocking activity, on the other hand, is useful

in the therapeutic intervention of neurodegenerative disease, skeletal muscle weakness, and bladder malfunction, they can still produce severe cholinergic responses.[20]

Acetylcholinesterase inhibition by organophosphorus pesticides can cause acute parasympathetic system dysfunction. Thus, symptoms such as increased movement, excessive production of a bodily secretion, slow heart rate, excessive constriction of the pupil, loose/water bowel movement, and low blood pressure may be present.[21]

Epidemiology of poisoning– global, India, study area:

The initial toxicity is a serious health of population concern in several nations worldwide.[22]The WHO has found that organophosphate compound pesticides which are potentially lethal pesticides pose a specific danger to young people and are recognized as of worldwide importance. Their extensive usage has resulted in health issues and casualties in several places around the world, typically because of workers contact and unintentional or purposeful consumption of toxic substances.[23] According to the World Health Organisation annually, 200,000 individuals strive self-poisoning, while 100,000 individuals accidentally poison. Yet, many fatalities are caused by intentional self-poisoning, because of the enormous demand that pesticides place on healthcare facilities, mainly in Asia.[24]

An estimated 350,000 people every year are self-poisoned by organophosphate poisoning pesticides.Self-poisoning with organophosphorus chemicals ranged from 10.3 to 43.8% in India.[24]In Bangladesh, organophosphorus toxicity is the most prevalent aetiology (27.64%) and has the greatest fatality percentage (13.88%).[25]The commonness of hospitalizations in Sri Lanka on a basis of yearly range between 10,000 and 20,000.[26]Although the actual frequency of organophosphate toxicity is unclear, it is the most prevalent suicide trigger targeting women in Pakistan.[24] However, this percentage is not equivalent to the quantity of chemicals distributed in every region; rather, it is a function of chemical consumption patterns and

tolerability. The proportion of all herbicide suicides ranges from 4% mostly in European Region to > 50percent of the overall in the Pacific Northwest.[27]

Classification and Guidelines:

For the evaluation of intoxication, there are prognostic scoring techniques in the literature, such as the "Acute Physiology and Chronic Health Evaluation and Simplified Acute Physiology Score", as well as the grading of organophosphate poisoning.[28]The APACHE-II grading system was proven to be beneficial for categorising patients based on illness intensity. There existed reverse relationship among a top category and duration of service and an increased mortality rate.[29]Humans who attempted suicide are often known to take a strive of self-harming substances and/or inoculated throughout their veins. In suicidal patients, organophosphate compound poisoning (OPP) may cause immediate organ failure owing to toxicity. Furthermore, the toxicity of organophosphates (OPs) hurts tissue and system activity. The signs and symptoms and investigations are reliable for poisoning detection.[30]

Table 1. The degree of organophosphate intoxication [28]

Grade 0: No clinical manifestations
Grade 1: Hypersecretion, fasciculations, consciousness
Grade 2: Grade 1 + hypotension, unconsciousness
Grade 3: Grade 2 + stupor, abnormal chest x-ray, pO2< 10 mmHg

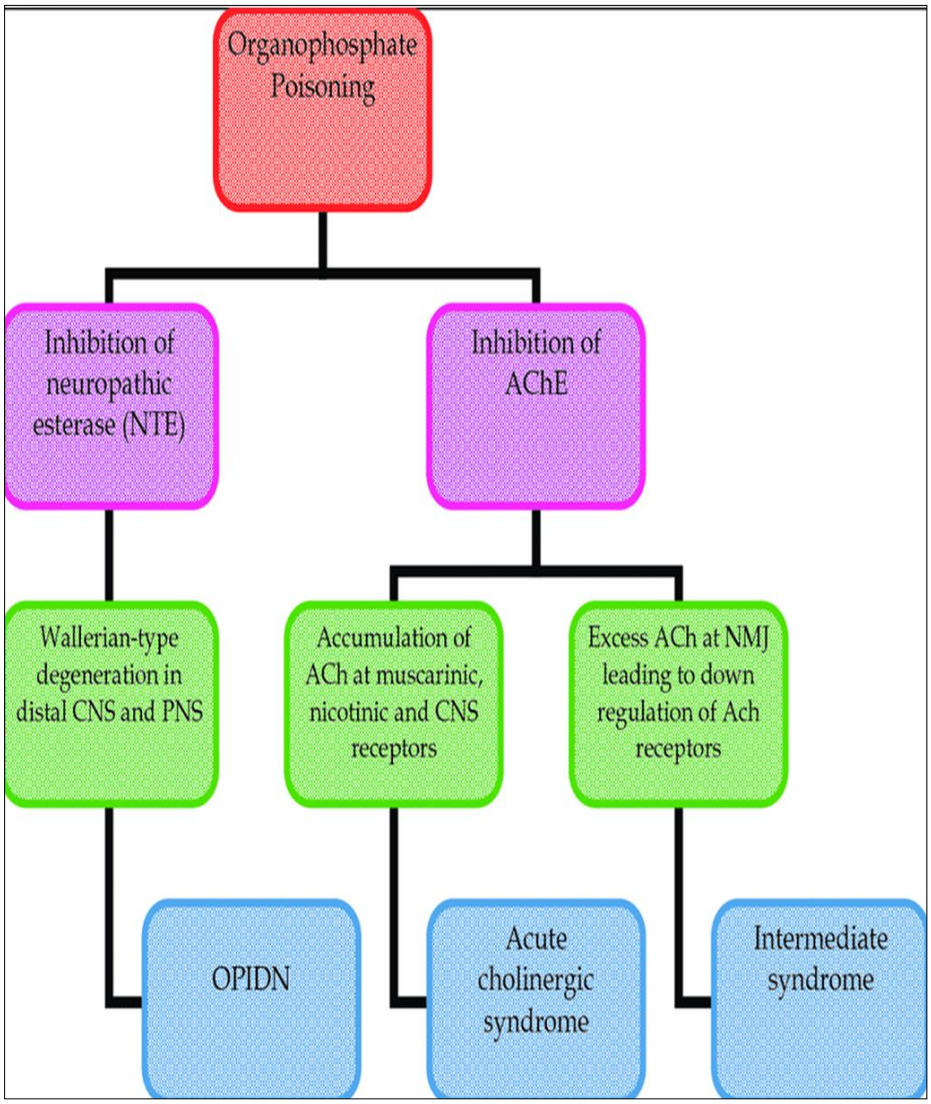

Figure 1: "Organophosphate toxicity is classified.

ACh is for acetylcholine; CNS stands for the central nervous system; NMJ stands for neuromuscular junction; OPIDN stands for organophosphate-induced delayed neuropathy; and PNS stands for the peripheral nervous system. Davidson's Principles and Practice of Medicine, Jones and Karalliedde 2006, 20th edition".[31]

Diagnosis:

The following tests should be run: full blood count (CBC), blood glucose, troponin, hepatic and renal function, and arterial oxygen gas. The ECG will show sinus bradycardia as a result of parasympathetic activity.[17] With a growing incidence of both intentional and accidental

poisonings, particularly in the developing world, there is a need for a reliable and low-cost way to help emergency workers diagnose patients and forecast outcomes. At admission, the Peradeniya scoring system was used, and the patients were intensively monitored to see how they fared in terms of morbidity and death. The severity of poison was calculated by Peradeniya scoring grade and was found to have a high connection with the outcome and may be used as a predictor of respiratory failure, the length of intensive care unit stays, and fatality. [32]

Table 2:The Peradeniya Organophosphorus Poisoning (POP) scale

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

Six criteria are assessed in the Peradeniya organophosphorus poisoning scale and assigned a score of 0, 1, or 2 based on the outcomes. The severity is determined by adding together the scores. A score of 0-3 indicates mild poisoning, 4-7 suggests moderate poisoning and 8-11 signifies severe poisoning[33]

Pathogenesis

The anticholinesterase OP are tetracoordinate pentavalent compounds having three singly linked substituents (often but not always organic), and in the case of nerve agents, an oxygen atom synchronously connected to the central phosphate group. These organophosphate substances have a strong affinity for acetylcholinesterase and are serine esterase and serine enzyme inhibitors. During the phase when the organophosphate makes a covalent connection with the activated serine site of the enzyme system or protease, the singly bound substitutions separate from phosphorus. Thus, a stoichiometric contact between the organophosphate molecule and the enzyme causes it to be removed during this phosphorylation process. Organophosphate suppression is long-lasting, lasting from hours to several days, and could not be recoverable if a dealkylation caused by "ageing," a nonenzymatic dealkylation, takes place. Reactivation does not work on the phosphorylated anticholinesterase that has aged.

Though these later compounds are often either weaker anticholinesterases or need bioactivation (typically "mediated by cytochromes P450") to the functional anticholinergic metabolites, insecticidal organophosphorus compounds were also produced thanks to Second World War chemical science. Since they are milder and/or take much longer to bioactivate than nerve agents, insecticidal OP are less dangerous than biological agents because they allow the body to employ its defences more effectively. Because nerve agents act immediately and do not need bioactivation, internal defence mechanisms can be effectively defended against them.[34]

Pathophysiology:

The suppression of carboxyl ester hydrolases, particularly acetylcholinesterase, is a major characteristic of organophosphate pesticides. This enzyme is essential for the breakdown of acetylcholine, a neurotransmitter present in both nervous systems.

By phosphorylating the enzyme's serine hydroxyl group, the organophosphate pesticide inactivates acetylcholinesterase (AChE). This is accompanied by acetylcholine build-up, which agitates the two main types of cholinergic receptors.[17]

Clinical presentation:

The standard method of clinical characteristics of initial toxicity has focused on receptor-specific impacts on cholinergic and central nervous system (CNS) receptors, which lead to a variety of symptoms and indications.[27] For unexpected cases or critical management subjects, organophosphorus poisoning is usually a dangerous situation. It is well recognized that doctors ought to be on the lookout for organophosphorus chemical toxicity and how to manage the condition.

Poisoning with organophosphorus results in widely diverse images of cholinergic crises often referred to as SLUDGE syndrome. [35]

The abbreviation of SLUDGE relates to the aforementioned:

- **S:** Salivation
- **L:** Lacrimation
- **U:** Urination
- **D:** Diaphoresis
- **G:** Gastrointestinal upset
- **E:** Emesis

Which are typically linked with clinical consequences. The intensity of symptoms in acute poisoning corresponds to the level of acetylcholinesterase (AChE) activity. Therefore,

Clinical characteristics were categorized as (a) receptor-specific symptoms, (b) incidence period, and (c) organ system inclusion.

Figure 2: (a) Organophosphorus toxicity features and indicators depending on receptor-specific: [35]

Type of receptor	Receptor sub-type	Action on	Manifestation
Nicotinic receptor stimulation	N1 (Nm) receptors	Neuromuscular junction	Weakness, fasciculations, cramps, paralysis
	N2 (Nn) receptors	Autonomic ganglia Adrenal medulla	Tachycardia, hypertension
Muscarinic receptor stimulation	M1-M5*	Central nervous system	Anxiety, restlessness, ataxia, convulsions, insomnia Dysarthria, tremors, coma, respiratory depression Circulatory collapse
	M2 receptor	Heart	Bradycardia, hypotension
	M3, M2 receptor*	Pupils	Blurred vision, miosis
	M3, M2 receptors*	Exocrine glands	Respiratory-rhinorrhea, bronchorrhea Gastrointestinal-increased salivation, diarrhea Ocular-increased lacrimation Others-excessive sweating
	M3, M2 receptors*	Smooth muscles	Bronchospasm, abdominal pain, urinary incontinence

*M1 receptors play a critical role in cognitive function; M3 receptor effect predominates in the pupils, airway smooth muscles and mucus glands. Nicotinic receptors are sub-typed as N1 or Nm receptors and N2 or Nn receptors. Muscarinic receptors are sub-typed from M1 to M5

Figure 3: (b)Organophosphorus toxicity features and indicators depending on the incidence period:[35]

Time of manifestation	Mechanism	Manifestation
Acute (minutes to 24-h)	Nicotinic receptor action	Weakness, fasciculations, cramps, paralysis
	Muscarinic receptor action	Salivation, lacrimation, urination, defecation, gastric cramps, emesis, bradycardia, hypotension, miosis, bronchospasm
	Central receptors	Anxiety, restlessness, convulsions, respiratory depression
Delayed (24-h to 2-week)	Nicotinic receptor action	Intermediate syndrome
	Muscarinic receptor action	Cholinergic symptoms-bradycardia, miosis, salivation
	Central receptors	Coma, extra-pyramidal manifestations
Late (beyond 2-week)	Peripheral-neuropathy target esterase	Peripheral neuropathic process

Organophosphorus toxicity features and indicators depending on the organ system inclusion such as brain, the spinal cord and the peripheral nerves, heart, and pulmonary manifestations and other systems:[35]

Figure 4: Brain, the spinal cord and the peripheral nerves features of Organophosphorus toxicity.[36]

Weakness or paralysis
Type I paralysis-acute paralysis
Type II paralysis-intermediate syndrome
Type III paralysis-delayed paralysis or OPIDP
Localized permanent paralysis at sites of dermal exposure
Cranial nerve palsies
Diaphragmatic paralysis
Isolated laryngeal paralysis
Supranuclear gaze palsy
Unconsciousness or impaired consciousness
Unconsciousness or coma at admission
Delayed onset organophosphate induced encephalopathy or coma
Cerebellar
Self-limiting ataxia-early (8-day) onset
Ataxia as a delayed neurotoxic manifestation
Neuropsychiatric symptoms and signs
Chronic organophosphate induced delayed neuropsychiatric disorder
Impaired memory
Confusion
Irritability
Lethargy
Psychoses
Extra-pyramidal findings
Dystonia
Resting tremor
Cog-wheel rigidity
Chorea, choreo-athetosis
Mask like facies
Bradykinesia
Ocular
Ophthalmoplegia
Supranuclear gaze palsy
Opsoclonus
Optic neuropathy
Degeneration of retina
Defective vertical smooth pursuit
Myopia
Cortical visual loss
Other features
Fasciculations
Convulsions
Delirium
Guillain-Barre syndrome
Sphincter involvement
Ototoxicity

OPIDP: Organophosphate induced delayed polyneuropathy; DOPE: Delayed organophosphate encephalopathy; COPIND: Chronic organophosphate induced neuropsychiatric disorder

Figure 5: Cardiac effect of organophosphate poisoning.[36]

Finding	Karki et al.[13] (n=23)	Saadeh et al.[14] (n=46)	Vijayakumar et al.[88] (n=20)	Taira et al.[89] (n=39)	Yurumez et al.[90] (n=85)
Electrocardiographic					
Prolonged QT interval	37.8	67	60	56.4	55.5
ST-T changes	29.7	41	40	89.7	17.6
Conduction defects	5.4	9	-	-	-
T-wave inversion	-	17	40	-	-
Prolonged PR interval	-	9	0	10.2	-
Rhythm abnormalities					
Sinus tachycardia	40.5	35	60	-	31.8
Sinus bradycardia	18.9	28	10	5.1	-
Ventricular tachycardia including polymorphic	2.7	9	-	-	-
Ventricular fibrillation	2.7	4.4	-	-	-
Supraventricular arrhythmia	-	9*	-	33.3	-
Other features					
Hypertension	13.5	22	35	-	-
Hypotension	10.8	17	10	-	-
Non-cardiogenic pulmonary edema	21.6	43	-	-	-

Values in parentheses indicate references. All values are expressed as percentages. *n*: Number of patients evaluated in the individual studies. *Patients who developed atrial fibrillation

Morbidity and Mortality:

Organophosphate poisoning (OPs) can lose life within minutes after contact. The kind of substance consumed, the quantity consumed returns.[33,37], the mode of consumption, the subject's overall wellness, and quick detection all contribute to fatality. The clinical severity, levels of cholinesterase activity, and organ dysfunction are also directly proportional. Before time detection, prescription of atropine combined with fluid administration through the vein, and effective mechanical ventilation, involving oxygenation treatment, resulted in positive Organophosphorus (OP) toxicity is a severe medical issue in emerging economies. Annually, there are many incidents of serious toxicity and around 220,000 fatalities. 45 In subjects with organophosphorus toxicity, the independent factors of death were slow heart rate, age, high blood sugar, enzyme lactic acid dehydrogenase level, and excess of acids in the body fluid.[39] There are few studies found to predict mortality and morbidity – the grade of the severity, the clinical manifestations assessments, and the requirement of ventilation and hospital stay to

observe the morbidity and the rate of death of organophosphate poisoning are considered. Therefore, it helps the clinician to identify to provide the appropriate management whether the need for a critical management setting or following the subjects or need transfer to a tertiary centre. [19]

Complications:

The initial organophosphate (OP) toxicity consequences are far more common and are associated with a substantial rate of occurrence and death, while delayed sequelae are lightly common and slightly potentially fatal.[26] Cardiovascular consequences are more serious than initial kidney damage. Self-harming ideation and direct consumption are strongly linked to heart issues. In comparison to OP toxicity, atropine therapy may result in an even more irregular heart rate. Kidney damage can occur because of direct toxicity or because of various indications of pre-renal failure such as parasympathetic activation or heart illness. Heart complaints might appear in three stages. First, sympathetic activity has risen. Two, there is extended parasympathetic activation, and three, repolarisation irregularities such as ST-segment elevation and T wave inversion, as well as QT prolongation, which can cause deadly ventricular arrhythmias, are the most common cardiac symptoms of organophosphorus toxicity. [40]

The increased levels of serum amylase are frequently seen with organophosphate poisoning and the predictive diagnosis of acute pancreatitis is a potentially fatal complication. 49 Following, the seizure was a complication presented with miosis and the need for ventilation and its duration was more among the seizure complication caused by organophosphate poisoning.

Management:

The initial procedure for treating organophosphorus toxicity subjects is to take on personal safety gear to cleanse the subject's clothing and drain the water to remove the harmful agents from the skin. To disinfect the skin, dry agents such as flour, bentonite and sand, or can be applied. In the event of consumption, emesis and loose or watery bowel movements may reduce the quantity of material swallowed but should never be provoked.

If the subject arrives around 60 minutes of intake, processed carbon can be administered, however, research has still not shown an advantage. Because respiratory management is important, an artificial ventilation tube may be necessary due to seizures, bronchospasm,or bronchorrhea, and supportive treatment should be preserved by supporting excellent intravenous access, and monitoring of vital signs, oxygen saturation and pulse rate. [17]

Atropine remains the basis of therapy, but other intriguing medicines have lately emerged. Although oximes are commonly used in the treatment of organophosphorus poisoning, clinical effectiveness has yet to be shown. "Treatment options include magnesium sulphate, calcium channel blockers (nimodipine), plasma alkalinizing drugs, -2 agonists, nicotinic receptor antagonists, clonidine, and lipid emulsions". Large phase III studies, however, are necessary to prove their effectiveness.[42] Next to the use of atropine, pralidoxime is suggested by the world health organization (WHO) as an antidote, where it regenerates the acetylcholinesterase after it has been inactivated by organophosphate poisoning (OP) and patients with seizures may benefit from benzodiazepines.[43]

Thyroid dysfunction:

In vertebrates, the thyroid gland is an endocrine gland. It is the human body's biggest organ specialised for endocrine activity. In infancy and youth, the gland is critical to optimal body growth. It absorbs iodine from the diet and produces thyroid hormones, which are iodine-

containing substances that assist regulate the body's metabolic rate. Its overall activities regulate protein, fat, and carbohydrate catabolism in all cells and manage body temperature. The metabolic symptoms of thyroid dysfunction are caused by either excessive or insufficient thyroid hormone production (hyperthyroidism and hypothyroidism, respectively).[43]

Thyroid dysfunction and pregnancy:

Hypothyroidism:

Thyroid-stimulating hormone (TSH) values above the demographic and trimester-specific reference ranges indicate hypothyroidism during pregnancy. When it is not accessible, a reference range with a higher upper limit than 4.0 mU/L must be used. Elevated trimester-specific TSH and relatively low T4 levels are two signs of overt hypothyroidism in pregnancy. Subclinical hypothyroidism, which has elevated trimester-specific TSH and regular free T4 levels, can also occur.

Hyperthyroidism:

Thyroid-stimulating hormone thyroid-stimulating hormone concentrations are lower and free thyroxine concentrations are higher in overt hyperthyroidism during pregnancy. Subclinical hyperthyroidism is distinguished by low thyroid-stimulating hormone (TSH) and normal free thyroxine (T4) levels. Due to thyroid physiological adaptation, transient subclinical hyperthyroidism can happen in the first trimester of pregnancy. TSH levels in gestational thyrotoxicosis typically decrease over the first trimester as a result of the tsh hormone (TSH) receptor being activated by human chorionic gonadotropin (hCG). At Seven and Eleven weeks of gestation, it hits its peak. Graves' disease and gestational thyrotoxicosis can be distinguished with a thorough history and physical. Blood testing for Graves' illness reveal elevated levels of TSH receptor antibodies.[44]

Clinical presentation:

Due to the fact that thyroid hormones may affect a wide range of systemic symptoms, hyperthyroidism can have a number of clinical presentations. Palpitations, exhaustion, trembling, anxiety, restless sleep, losing weight, heat intolerance, perspiration, and fluid retention are symptoms that are frequently described. Tactile tremors in the limbs, tachycardia, and weight loss are common physical symptoms. Weight loss, weariness, and heat intolerance are caused by the biological effects of T3 coupling to alpha and beta receptors, which raise core body temperature and basal metabolic rates. Weakness, accelerated bone resorption, weakened bone, and a higher chance of fracture are among musculoskeletal symptoms. Patients may experience swollen lymph nodes, abnormal breast enlargement, or infrequent menstruation. Swallowing disorders, pseudo diarrhea, and appetite are examples of gastrointestinal symptoms. Significant cardiovascular symptoms are frequent in hyperthyroidism, thus it's crucial to identify them and provide them the right care.[45] Although adrenergic receptors may be low or average in hyperthyroidism, the most frequent cardiovascular signs of thyroid disease are hypertension (HTN), tachycardia, and increased cardiac output, which are comparable in presentation to enhanced adrenergic activity.[46]Other possibilities include CHF and atrial fibrillation. Aging, greater T4 levels, male sex, toxic nodules, and atrial flutter are associated with an increased risk of developing heart problems on their own. According to preliminary study, heart failure occurs in 6–16% of hyperthyroid people, but far higher rates are anticipated if a cardiovascular condition is present as well.[47]Patients with graves' disease may exhibit signs and symptoms such as lid abduction and infiltrative grave's orbitopathy.[48] Age, smoking, greater symptom duration, and gender of the patient are risk factors for grave's orbitopathy (GO).102 A rare extrathyroidal sign of Graves' disease called thyroid dermopathy affects 1-4% of those with thyroid ophthalmopathy. Ophthalmopathy is present in almost all patients concurrently.[50] Skin that is

somewhat more pigmented and thicker and primarily affects the pretibial area distinguishes the lesions. [51]

Table 3: Clinical manifestation of thyrotoxicosis:[52]

System	Symptoms	Signs
Constitutional	Weight loss despite increased appetite; heat-related symptoms (heat intolerance, sweating, and polydipsia)	Weight loss
Neuromuscular	Tremor; nervousness; anxiety; fatigue; weakness; disturbed sleep; poor concentration	Tremor of the extremities; hyperactivity; hyper-reflexia; pelvic and girdle muscle weakness
Cardiovascular	Palpitations	Tachycardia; systolic hypertension; irregular heartbeat (atrial fibrillation)
Pulmonary	Dyspnoea, shortness of breath	Tachypnoea
Gastrointestinal	Hyper-defecation; nausea, vomiting	Abdominal tenderness
Skin	Increased perspiration	Warm and moist skin
Reproductive		Menstrual disturbances
Ocular (Graves' disease)	Diplopia; a sense of irritation in the eyes; eyelid swelling; retro-orbital pain or discomfort	Proptosis; eyelid retraction and lag; periorbital oedema; conjunctival injection and chemosis; ophthalmoplegia

Hypothyroidism:

Because the clinical features of an underactive thyroid can be moderate and ambiguous, and various symptoms may be found in different people, it is essential to keep an elevated threshold of caution. Typical symptoms such as cold sensitivity, puffiness, reduced sweating, and skin abnormalities are often not apparent. Therefore, a thorough medical, surgical,

medication, and family history should be obtained to diagnose hypothyroidism.[53]A history of unfavourable gestation and birth complications should be explored as well.[54]

Depression, anxiety, and psychosis are psychiatric and cognitive symptoms, and there may also be cognitive deficits including memory loss.[55] Occasionally, edema, rhabdomyolysis, and pericarditis may be present. [56]

Additionally, patients may exhibit sleep apnea, CHF a prolonged QT interval, low sodium levels, elevated cholesterol, and compression of the median nerve.[57]

Autoimmune disorders affecting the thyroid gland s difficult to distinguish; nonetheless, several characteristics are unique to this disorder, such as:

- Acid reflux
- Malaise
- Silent lymphocytic thyroiditis
- Periodic neck aches and/or pharyngitis

Because the signs and symptoms of an underactive thyroid are vague, a diligent physical examination may offer some hints. Physical examination may be necessary for the following reasons:

- Goitre
- Increase in body weight
- Ataxia
- Xeroderma
- Coarse and brittle hair
- Yellowing of the skin and eyes, pale
- Dull facial expressions
- Abnormal enlargementt of tongue
- Slow heart rate

- Pericardial effusion
- Protracted ankle reflex relaxation time.[53,57]

Complication:

The patient's age, gender, comorbidities, length of the disease, and the cause all have an impact on the clinical presentation. Despite having fewer and milder symptoms than younger people, elderly patients are more prone to have cardiovascular issues.[58]Adults with hyperthyroidism are three times more likely to get atrial fibrillation than individuals over 60 with such a healthy thyroid. A hyperthyroid-induced atrial dysrhythmias embolic stroke is significantly more frequent than one brought on by non-thyroidal causes.[59] However, the use of anticoagulants in individuals with atrial fibrillation caused by hyperthyroidism is still debatable. Atrial fibrillation is also thought to be a reliable indicator of the onset of CHF in people with hyperthyroidism.[60]

Management:

Antithyroid medications (ATDs), radioactive iodine ablation, and surgery are the three effective treatments for hyperthyroidism patients. Individuals with Graves' disease would benefit from all three therapeutic choices, however, subjects with benign tumours of the thyroid gland or multinodular toxic goitre should get either radioactive iodine treatment or surgical intervention, as reduction is rare in these subjects. Anti-thyroid drugs are mainly used to reinstate euthyroidism in subjects with multinodular toxic goitre prior to therapeutic interventions with surgical procedure or radioactive iodine and are rarely used as persistent treatment when the alternative two procedures are possibly unsafe or the patient has a short lifespan. [45]

Levothyroxine single-drug medication is the most often used treatment for underactive thyroid. Levothyroxine alternative therapy, starting at 1.6 mcg/kg per day, relieves symptoms and normalises thyroid-stimulating hormone concentrations. Even in individuals with prolonged symptoms and appropriate thyroid-stimulating hormone levels, adding triiodothyronine is not advised. Elder patients, as well as those with established or suspected ischemic heart disease, should start with a smaller dose of levothyroxine. Women with an underactive thyroid who get conceived should increase their weekly intake by 30%, up to nine dosages a week (take one added boost twice a week), with monthly adjustments review and treatment. Subjects who continue to experience symptoms despite the appropriate levothyroxine dose should be evaluated for additional reasons or the need for referral. [53]

Sick euthyroid syndrome:

Definition:

Non-thyroidal sickness syndrome, mentions alterations in thyroid activity panels performed on patients in critical management settings during the occasion of critical illness. Though it does not represent a genuine state, and around 75% of hospitalised patients have substantial changes in the connection between the hypothalamus, pituitary gland, and adrenal cortex. This syndrome is frequently encountered in people suffering from acute critical illness, calorie shortage, or following major surgery. The most common hormone rhythm in "sick euthyroid syndrome" is minimal triiodothyronine and free triiodothyronine concentrations with normal thyroxine and TSH concentrations.[61,62]

Epidemiology – global, India, study area:

Triiodothyronine (T3) decrease is the most prevalent anomaly, occurring in around 40% to 100% of patients, with approximately 10% having minimal thyroid stimulating hormone. The

most chronic patients have the highest incidence. The concentrations of total thyroxine (T4) in blood correspond with the chance of mortality. When thyroxine concentration in blood fall beneath two micro g/dL, the likelihood of mortality rises to 80%.[61]Non-thyroidal illness syndrome is quite frequent in persons suffering from cardiovascular disease. In cardiovascular patients, Euthyroid sick syndrome is an independent prognostic factor that is related to higher chances of death from all factors, cardiovascular fatality, and severe coronary impact.[63]

Criteria for diagnosis:

Two broad criteria should be followed while examining a critically sick patient.

- To begin, only thyroid stimulating hormone should be monitored in the event of a significant clinical indication of thyroid illness. Further testing is carried out if the thyroid-stimulating hormone concentration is unexpected. If the thyroid stimulating hormone concentration is more than 20 micro-Units/mL or not able to be detect, the euthyroid unwell syndrome is minimal to be the root cause, and overt thyroid illness should be checked more thoroughly.
- In individuals with established thyroid illness and low serum-free T4, the euthyroid sick syndrome should be investigated when serum TSH is not raised.

There are four types of the euthyroid ill syndrome:

(1) Minimal thyroxine abnormality,

(2) Minimal triiodothyronine and thyroxine abnormality

(3) Elevated thyroxine abnormality

(4) various abnormalities.

The most prevalent anomaly in non-thyroidal illness syndrome is minimal serum total triiodothyronine, which is detected in around 70% of hospitalised patients. Except in renal

failure, the blood contraction of contrary triiodothyronine (rT3) is elevated in euthyroid sick syndrome. Elevated reverse triiodothyronine (rT3) is primarily caused by a reduction in the iodothyronine 5'-monodeiodinase function (deiodination of thyroxine to triiodothyronine and reverse triiodothyronine to 3,3'-diiodothyronine). In severely sick patients hospitalised in intensive care units, both minimal triiodothyronine (T3) and thyroxine (T4) syndromes are noted.[64]

TSH levels are lowered in non-thyroidal illness syndrome subjects treated with neuromodulator molecules and cortisone, lowering free thyroxine (T4) levels. Blood thyroid binding globulin levels rise in individuals with a severe for of initial porphyria (vampire disease) and persistent inflammation of the liver, resulting in normal free thyroxine levels but elevated serum total thyroxine. Human immunodeficiency virus subjects show atypical alterations in thyroid function, resulting in an elevation in thyroxine (T4) and thyroid-binding globulin (TBG), a decrease in reverse triiodothyronine and the reverse triiodothyronine/thyroxine (rT3/T4) ratio, and normal thyroid function.

Chronic lymphocytic thyroiditis, overt thyroid, thyrotoxicosis, decreased secretion of hormones from the pituitary gland, and thyroid dysfunction caused by amiodarone medication are all alternative explanations for the non-thyroidal illness syndrome.[61]

Thyroid function tests:

Thyroid activity panels used (TFTs) to help in the diagnosis of thyroid dysfunction, give information at three levels: physiological, pathological, and anatomical. They owe to several findings linked with thyroid functioning, in addition to history and physical examination. "As a result, an attempt has been made to present a summary of thyroid function testing. Thyroid function tests in serum include total thyroxine (T4), total triiodothyronine (T3), free thyroxine (FT4), free triiodothyronine (FT3), reverse triiodothyronine (rT3), thyroid

stimulating hormone (TSH), serum calcitonin, and protein thyroglobulin (Tg)". Antithyroglobulin antibodies (ATA) and anti-microsomal antibodies (AMI) are the serological assays (AMA). Fine needle aspiration cytology (FNAC) is an invasive test for histologic evaluation, whereas non-invasive tests include ultrasonography, magnetic resonance imaging, and positron emission tomography (PET). [65]

Aetiology:

"Critical illness, inflammatory condition of the lung, the extreme form of malnutrition, eating disorder type-Anorexia Nervosa, septicemia, stress, history of trauma, cardiopulmonary bypass, heart attack, malignancies, congestive cardiac failure, hypothermia, inflammatory bowel disease, cirrhosis, major surgery, kidney damage, and diabetic ketoacidosis are some of the causative factors of non-thyroidal illness syndrome". [61]

Clinical presentation:

The subject's record and medical examination findings are distinctive for the causative factors, without obvious indications of the non-thyroidal illness syndrome. Subjects who have previously had thyroid diseases may be impacted, and concomitant non-thyroidal illness syndrome may mask the typical medical examination indications of underactive and overt thyroid.[61,66]

Mortality and morbidity:

The "euthyroid sick syndrome" is quite common in severely unwell subjects and reduced triiodothyronine (T3) or thyroxine (T4) levels are linked to death in elderly subjects hospitalized in the critical management setting. There are now various well-established prognostic indicators in the critical management setting to evaluate mortality and illness

severity, such as “Acute Physiology and Chronic Health Evaluation IV, Simplified Acute Physiology Score, and Sequential Organ Failure Assessment”. The measurement of organs which does not perform an unexpected function sequentially over the first days after critical management setting admission is a strong predictor of the outcome. The mean and highest Sequential Organ Failure Assessment (SOFA) values are both excellent indicators of outcome. A rise in Sequential Organ Failure Assessment scores within the initial 2 days in critical management settings is associated with a 50% death rate regardless of the baseline score.[67]

Complications:

The euthyroid sick syndrome often gets misdiagnosed as hypothyroidism due to low concentration of triiodothyronine and high concentration of the thyroid-stimulating hormone. There are no noted complications for the euthyroid sick syndrome (ESS); however, the underlying causative factors for illness must be properly diagnosed and treated to prevent secondary complications related to the disorder.[68]

Sick Euthyroid state in subjects of initial organophosphorus compound toxicity Blood thyroid hormone concentration in organophosphorus compound poisoning

There are two types of cholinesterase: acetylcholinesterase and cholinesterase, often known as pseudocholinesterase. Acetylcholinesterase is mostly present in red blood cells. Cholinesterase is produced in the liver and detected in the bloodstream; this is the variant of the protein that is commonly investigated. Cholinesterase is routinely tested as a biomarker of anticholinesterase exposure (organophosphates, including many insecticides). cholinesterase Herbicides and pesticides are made from organophosphorus (OP) compounds. Most organophosphate poisoning is caused by self-ingestion, particularly in the adult population,

because very poisonous agrochemicals are widely accessible during periods of crisis, such as family troubles, marital breakdown, and test fear, among other things. Adults are also exposed to occupational hazards because of unprotected usage. Children are prone to accidental poisoning owing to their inventive and curious nature. Exposure to these toxic substances produces impacts on thyrotrophic feedback control, changing thyroid hormone concentrations in the short term.

The impacts of organophosphates on blood thyroid hormone concentrations are dependent on agrochemical dosage and vulnerability. The overall triiodothyronine (T3) concentrations were determined to fall within the proper biological limits among organophosphorus poison, hyper- and hypo-thyroid patients, but the results from the healing process were superior to those from the exposure time, and this variation was numerically relevant. Total thyroxine levels were greater than the biological baseline interval during the acute exposure, stimulating hyperthyroidism, and then returned to biological reference levels following recovery. Elevated Tetra-iodothyronine (TT4) levels were statistically significant during the acute phase of intoxication. Thyroid-stimulating hormone (TSH) levels were lower during the exposure period than during the recovery phase, although a statistically significant increase was detected during the recovery phase. Thus, thyrotoxicosis was found during the subjects recovered from initial organophosphorus exposure and returned to euthyroid condition.[69]

Prevalence/Incidence/Frequency of non-thyroidal illness syndrome in subjects with initial organophosphorus compound toxicity

Thyroid hormones are vital in various physiological processes such as right growth, development, and metabolism, and such differences in their concentrations can result in a variety of clinical illnesses. The sick euthyroid syndrome is an aberrant result of thyroid activity panels in individuals with non-thyroid sickness with low levels of hormones such as

T3 (Triiodothyronine) during the acute illness of poisoning and can be found in blood within 2 hours of acute illness. As the condition progresses, there is a significant presentation of the symptoms associated with hypothyroidism, particularly with T3 and T4, but TSH levels are modestly increased or unaffected.[70,71]

Sick euthyroid hormone syndrome is highly common in males and females in their forties. The research found that ill euthyroid syndrome had a higher frequency in the age bracket of 20-30 years, with men being more prevalent. Because the mechanism of the non-thyroidal illness syndrome is identical to that of the non-thyroidal illness syndrome in other intensive management disorders. As a result, further research should be conducted to improve our understanding of the prognostic usefulness of sick euthyroid syndrome in critically sick patients. Thyroid function should be often checked in patients to prevent the sick euthyroid syndrome.[72]

Pathophysiology

The action of type III deiodinase, which catabolizes thyroxine or triiodothyronine instead of triiodothyronine, is involved in the pathophysiology of NTIS. Type I deiodinase, which typically converts thyroxine or triiodothyronine, is also expressed at a lower level. 1 "Additionally, thyrotrophic feedback control is blocked by a hormone inhibits hunger-leptin production decreases throughout starving times or by an increase in type II deiodinase in the third ventricle, which promotes the conversion of thyroxine or triiodothyronine in the hypothalamus and hence results in a decrease in thyrotropin-releasing hormone production. Furthermore, blood concentrations of thyroid hormone transport proteins fall, inhibiting thyroxine transport in triiodothyronine-producing organs". Euthyroid sickness syndrome may show the body's attempt to reduce basal and resting metabolic rates, as a result, it may be an appropriate reaction to an environmental stimulus that needs no treatment.[66]

Sick euthyroid state as a prognostic indicator/significance in the outcome of acute organophosphorus compound poisoning:

The sick euthyroid syndrome is most common in patients hospitalised in critical management settings and is intimately linked to the condition status and likely course of the condition.[73]Reduced free triiodothyronine concentrations in NTI subjects are usually associated with a poor prognosis. Low triiodothyronine syndrome, for example, has been proven to be a powerful predictive predictor of mortality in subjects with cardiovascular problems. In addition to free triiodothyronine concentrations, free thyroxine or thyroid-stimulating hormone concentrations may influence sick euthyroid syndrome mortality.[74] The research found that individuals with septicemia and non-thyroidal illness who had minimal thyroxine and triiodothyronine concentrations had a poorer prognosis than those who just had low T3 levels. Another Turkish study found that reduced free triiodothyronine and elevated free thyroxine concentrations are not dependent on indicators of long-standing death hazards in persistent subjects sick euthyroid syndrome.[75,68]

Recent studies

Yadav I et al 2022, aimed to assess the frequency of nonthyroidal illness syndrome with acute organophosphorus compound toxicity in addition to analyses of biochemical markers among the population. The study considered 74 subjects who have detected OPC toxicity and their biochemical markers (Thyroid hormone, hepatic and kidney panel blood tests, and medical laboratory full blood tests and gastric lavage) were evaluated. The study population found that the frequency of nonthyroidal illness syndrome with acute organophosphorus compound toxicity was 53% in the study population, significantly higher among 62% of males aged 20 to 40 years. The study results found that the prevalence of sick euthyroid is

frequently emerging in onset of organophosphorus compound toxicity and in addition to prevalence among men aged 20 to 40 years, who have a suicidal drive. To determine the fatality and rapid therapy of OPC toxicity it is recommended to use the biochemical markers thyroid hormonal level and blood cholinesterase as the biochemical findings suggested an increase in OPC toxicity.[71]

A cross-sectional, analytical observational Aditya Sukma Pawitra et al 2022 intended to determine the relationship among the thyroid profile, organophosphate compound poisoning and unaffected. The study considered 150 subjects out of all only 50 subjects were affected and the other 100 were unaffected. The study population noticed the concentration of thyroid hormone profile comprising thyroid-stimulating hormone was elevated whereas thyroxine and triiodothyronine were lowered among the affected group. The study results found that the thyroid stimulating hormone value increases in the organophosphorus compound poisoning and the value of thyroxine and triiodothyronine lowers.[76]

Pornpimol Kongtip et al 2021, aimed to assess the link between OPC toxicity and transient difference in concentrations of thyroid hormones in Thailand farmer subjects. The study considered 78 subjects on two occasions, one before using the organophosphate compounds and the second after the use. Their hormone levels and urine results were evaluated on both occasions. The study population found that thyroid hormone levels comprising Total triiodothyronine (TT3), and free triiodothyronine (FT3) were considerably lowered. The day following the use of organophosphate compounds, total thyroxine (T4) elevated dramatically. While the levels of metabolites in urine were elevated the day following the use of organophosphate compounds. The study results found that the use of organophosphate compounds leading to OPC toxicity fluctuates the levels of thyroid hormone. [77]

A retrospective study by Jing Gong et al 2021 aimed to assess variations in thyroid profile values that affect the death rate in non-thyroidal illness with covid-19. The study considered 150 subjects where blood thyroid profile values were evaluated and based on were divided into 4 categories. The data were analysed and assessed among the categories with appropriate indicators. The study population found that the reduced thyroid-stimulating hormone has increased risks of death and progression of the illness, whereas in a reduced thyroxine category only the risk of death was substantially elevated. The study results found that risks of death are in connection to decreased levels of thyroid-stimulating hormone and thyroxine. However, the reduced levels of the thyroid-stimulating hormone are the predictive indicators to assess the incidence of death in non-thyroidal illness with covid-19.[78]

A prospective observational study by Jianying Guo et al 2021, aimed to assess the low levels of thyroid profile comprising triiodothyronine and clinical biomarkers and its association with the sick euthyroid syndrome. The study considered 305 subjects placed in the critical care unit. The serum thyroid profile, specific triiodothyronine were measured, and clinical biomarkers in association with the sick euthyroid syndrome were observed. The study population found that the triiodothyronine was reduced among the sick euthyroid syndrome along with the variation results of the clinical biomarker. The study results found that the occurrence of a sick euthyroid syndrome is 38.7% among the subjects placed in the critical care unit with reduced values of triiodothyronine. The variation of clinical biomarkers is also linked whereas brain natriuretic peptide, platelet and albumin are the predictive indicators.[68]

Ranjith Kumar C et al 2020, aimed to assess the levels of the thyroid hormones of acute OPC (organophosphate compound) toxicity. The study considered subjects who ingested organophosphate compounds consciously and unknowingly and were admitted to ER at SVS Medical College, Mahabubnagar. Ingestion of those same chemicals was validated by evaluating the blood cholinesterase levels and subjects with blood cholinesterase levels

<4000 U/L respectively. After admission and 30 days after rehabilitation, subjects' thyroid hormonal levels comprising Total triiodothyronine (TT3), Total tetraiodothyronine (TT4), and Thyroid stimulating hormone (TSH) were evaluated. The study population found that the concentrations of thyroid hormones varied substantially during the OPC toxic stage and 30 days post-recovery period. The study results found that in the acute OPC toxic stage the level of Total tetraiodothyronine (TT4) was elevated, resembling hyperthyroidism, and reverted to the normal level during the rehabilitation period. But Thyroid stimulating hormone (TSH) concentrations dramatically lowered in the OPC toxic stage compared to the rehabilitation period. [69]

Dattatray Hambhire et al 2019, aimed to assess the prevalence of decreased levels of thyroid hormone without thyroid gland dysfunction in non-thyroidal illness. The study considered 60 subjects. The study considered subjects diagnosed with low thyroid hormone without thyroid gland dysfunction. Relevant biochemical indicators were evaluated and recorded. The study population found that the vicenarian categories are 30% and 33.3% of the tricenarian categories. Only 15 of the 60 subjects were found to have low values of thyroid hormone without thyroid gland dysfunction where 60% of the subjects had reduced T3 values solely, whereas 40% had abnormalities in triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH) values. The study results found that the prevalence of decreased levels of thyroid hormone without thyroid gland dysfunction in non-thyroidal illness is prevalent in males in contrast with females. It is recommended to frequently monitor the thyroid hormonal values to evaluate non-thyroidal illness syndrome. As a result, additional research should be conducted to improve the predictive indicators of sick euthyroid in severely unwell subjects.[72]

A prospective observational study by Sohair Sayed Abu El-Ella et al 2018, aimed to assess the occurrence and the genetic factors' ability to project the natural history of sick euthyroid state in severely unwell children in relation to another factor. The study considered 70 subjects admitted under the paediatric critical care unit. Following admittance, the blood thyroid profile was assessed within 1 day. The study population found that reduced free triiodothyronine was the most prevalent occurrence with the stable value of thyroxine and thyroid-stimulating hormone. As the TSH was elevated among 3 subjects it was validated to measure the degree of illness. Reduced thyroxine was substantially higher in deaths than in live subjects. Seven per cent of subjects' complete thyroid profile values were lowered which was validated as a predictive marker of death. Hence, the study results found that the occurrence of a sick euthyroid state in severely unwell children is linked to death and degree of illness.[66]

Srishti Shrestha et al 2018, aimed to evaluate the occurrence of underactive thyroid conditions among organophosphate compound users. The study considered 829 volunteer subjects with an occurred underactive thyroid condition. The study population found that the frequency of underactive thyroid conditions was substantially higher among various organophosphate compound users. Hence, the study results found that an underactive thyroid condition is an adverse disorder that occurs among organophosphate compound users.[79]

Wang et al 2018 aimed to assess the association between the role of thyroid profile in sick euthyroid syndrome and the degree of illness among the Chinese population. The study considered 51 subjects according to a severity-of-disease classification system degree 2 of the ICU scoring system. The subjects were divided into 3 categories depending on their blood thyroid profiles value and analysed with the severity-of-disease classification system. Additionally, the sick euthyroid syndrome and degree of illness were evaluated. The study

population found that the values of triiodothyronine and second-degree illness were significantly higher among males than females. The study results found that the elevated values of triiodothyronine are a greater prevalence in sick euthyroid syndrome and the progression of the illness. [80]

A retrospective global population-based cohort study Hung-Sheng Huang et al 2017, aimed to assess the correlation between an underactive thyroid condition and organophosphate compound poisoning-inducing cholinesterase inhibitors The study considered 2 categories in the ratio of 1:3, which included OPC poisoning-inducing cholinesterase inhibitors and non-inducing cholinesterase inhibitors. These were contrasted with the occurrence of underactive thyroid conditions and thyroid profiles were evaluated among the 2 categories. The study population found that organophosphate compound poisoning-inducing cholinesterase inhibitors were detected with underactive thyroid conditions compared to non-induced ones in relation to various appropriate investigations. It is also found that women's gender, carcinoma, kidney conditions, inflammation of the thyroid gland, swollen thyroid, psychiatric condition, and organophosphate compound poisoning-inducing cholinesterase inhibitors without atropine medication were the major indicators for the underactive thyroid condition. The study results found that organophosphate compound poisoning-inducing cholinesterase inhibitors are linked to a higher incidence of an underactive thyroid condition. Therefore, it is recommended to assess the thyroid profile in OPC poisoning. [81]

Whitney S. Goldner et al 2013 aimed to assess the correlation between the overactive and underactive thyroid condition with organophosphate compounds and their fifty subtypes within the users. The study considered 22,246 men subjects. The study population found that the impact of various use of chemical agents was an elevated incidence of an underactive thyroid condition. By using dose-response relation data analysis found that the alachlor-herbicide type and 2,4-Dichlorophenoxyacetic acid, as well as aldrin, organochlorine,

Dichlorodiphenyltrichloroethane, gamma-hexachlorocyclohexane, and parathion-ethyl insecticides were greatly responsible for this condition. The study results found that exposure to certain organophosphate compound poisoning increases the incidence of an underactive thyroid condition.[82]

Lacunae in literature:

Many nonthyroidal illness conditions, both onset and persistent condition can have an impact on thyroid function measurement. This phenomenon, commonly called "euthyroid sick syndrome" or "nonthyroidal illness syndrome," leads to the detecting negative thyroid function tests in a patient with nonthyroidal illness who does not have pre-existing thyroid problems. As documented in the situation of critical care unit admissions for infection, injury, burns, heart attack, and cancer. It also occurs in normally healthy subjects who are starving. The pathophysiology of euthyroid syndrome is not clearly understood, therefore more and more studies should be done for a better evaluation of the non-thyroidal illness syndrome prognostic value in severely sick subjects.

AIMS AND OBJECTIVES

OBJECTIVES OF STUDY

- To estimate Serum thyroid hormone levels in organophosphorus compound poisoning
- To study the occurence of sick Euthyroid state in patients with acute organophosphorus compound poisoning
- To study sick euthyroid state as a prognostic indicator in acute organophosphorus compound poisoning

MATERIALS AND METHODS

MATERIALS AND METHODS

SOURCE OF DATA:

Individuals coming to R.L.Jalappa Hospital and Research Centre attached to Sri DevarajUrs Medical College affiliated to Sri DevarajUrs Academy of Higher Education and Research Centre, Tamaka, Kolar who fulfil the inclusion and exclusion criteria.

STUDYDESIGN:

- Prospective study
- Cohort study of the patients recruited according to the inclusion and exclusion criteria

STUDY PERIOD

- 1 year from january 2021 to december 2021 or till the sample size is met.

INCLUSION CRITERIA:

- Patients aged above 18 years of age
- Patients with history of ingestion,inhalation or cutaneous absorption of organophosphorus compound within 24 hours

EXCLUSION CRITERIA:

- Patient with past or present H/o. thyroid Dysfuntion
- Patients taking drugs that will affect thyroid function
- Patients with liver disease
- Patients with Renal Dysfuntion.
- Patients with mild grade of poisoning according to organophosphorus poisoning scale
- Patients with organophosphorus poisoning mixed with any other poison/alcohol

METHOD OF COLLECTION OF DATA

The whole operation was described to the patient or patient attendees, and informed permission was obtained in their own easily understood language. Each patient's information

was gathered using a pre-tested proforma. The patient, a relative, or a close bystander who was with the patient were asked for a thorough history. Patients who met the criteria had a clinical examination and the necessary biochemical testing.

A thorough clinical examination was performed, paying close attention to vital signs, pupil size, a central nervous system evaluation, and the cardiovascular system in accordance with the recommended proforma.

Paradeniya

All research participants were subjected to an organophosphorus poisoning scale, and the degree of poisoning was rated as mild , moderate, or severe at the time of admission.

On Day 7 and during admission, 2 ml of blood from each patient was drawn and allowed to clot. Centrifugation was performed to separate the serum before it was used to measure the amounts of thyroid hormones in the serum. Other pertinent and standard investigations were also conducted.

SAMPLE SIZE:

- Sample size was estimated by using the proportion of sick euthyroid in acute pesticide poisoning was 16.66% from the study by Thirumal Valavan K et al.[83] using the formula

$$\text{Sample size} = \frac{Z_{1-\alpha/2}{}^2 p(1-p)}{d^2}$$

Here

$Z_{1-\alpha/2}$ = Is standard normal variate (at 5% type 1 error ($P<0.05$) it is 1.96 and at 1% type 1 error ($P<0.01$) it is 2.58). As in majority of studies P values are considered significant below 0.05 hence 1.96 is used in formula.
p = Expected proportion in population based on previous studies or pilot studies.
d = Absolute error or precision – Has to be decided by researcher.

- P = 16.66% or 0.166
- q = 83.34 or 0.833
- d = 10% or 0.10
- Using the above values at 95% Confidence level a sample size of 54 subjects will be included in the study.
- Considering 10% Nonresponse a sample size of 54+ 5.4 ≈ 60 subjects will be included in the study.

STATISTICAL METHODS:

Mechanical ventilation, ICU stay, No. of days in hospital and Recovered were considered as outcome variable. Magnitude of Sick Euthyroid was regarded as a type of independent variable (explanatory variable). Age, sexual preference, T3 (ng/mL), T4 (g/dL), TSH (uI/mL), and other factors were deemed research essential. Descriptive analysis was performed on quantitative data using mean and standard deviation, and categorical variables using frequency and percentage. The median and interquartile range were used to describe non-normally distributed quantitative values (IQR). Data was also depicted through relevant

layouts such as a circle chart, bar graphs, Cluster bar chart, and boxplot. The Mann Whitney u test was used to evaluate medians and interquartile ranges (IQR) between research categories for non-normally distributed quantitative data (2 groups). The Chi square test was employed to assess categorical results between research categories. Peradeniya Organophosphorus Poisoning Scale was considered as gold standard. Magnitude of Sick Euthyroid was considered as screening test. The screening test's responsiveness, precision, predictive values, and detection limit, as well as their 95% confidence intervals, were given. Numerical significance was defined as a P value of 0.05. Data was analysed by using coGuide software:[84]

RESULT

Result:

A total of 60 subjects were included in the final analysis.

Table 4: Descriptive analysis of Age in the study population (N=60)

Name	Mean ± S. D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Age	35.48±11.81	35.00	18.00	65.00	32.50	38.47

The study population's mean age was 35.48 11.81; the lowest level was 18 and the highest level was 65 (95% CI 32.50 to 38.47). (Table 4)

Table 5: Descriptive analysis of Gender in the study population (N=60)

Gender	Frequency	Percentage
Male	43	71.67%
Female	17	28.33%

Among the study population, 43 (71.67%) participants were male and remaining 17 (28.33%) were female. (Table 5 & Figure 6)

Figure 6: Pie Chart of Gender in the study population (N=60)

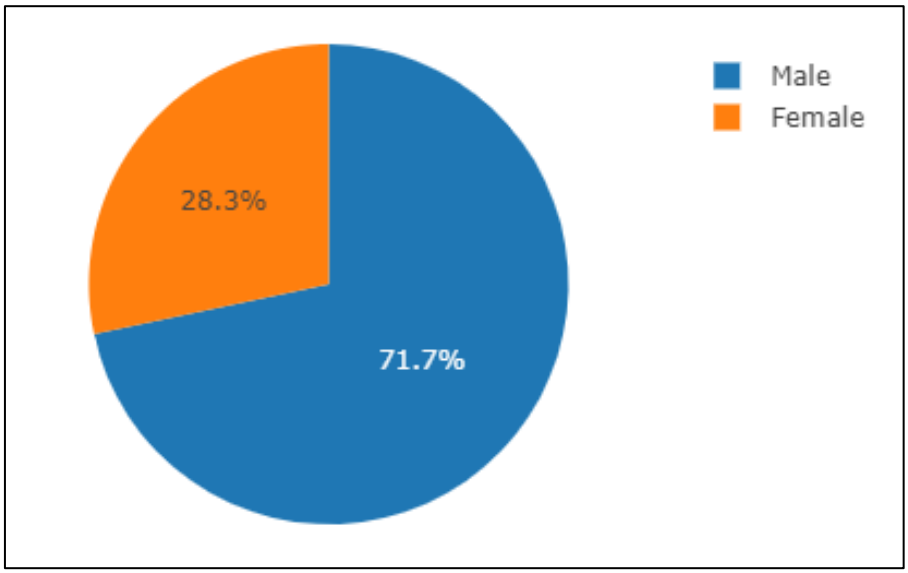

Table 6: Descriptive analysis of Magnitude of Sick Euthyroid in the study population (N=60)

Magnitude of Sick Euthyroid	Frequency	Percentages
Sick Euthyroid	13	21.67%
Normal Thyroid	47	78.33%

Among the study population, 13 (21.67%) participants had Sick Euthyroid and 47 (78.33%) had Normal Thyroid (Table 6 & Figure 7)

Figure 7: Bar Chart of Magnitude of Sick Euthyroid in the study population (N=60)

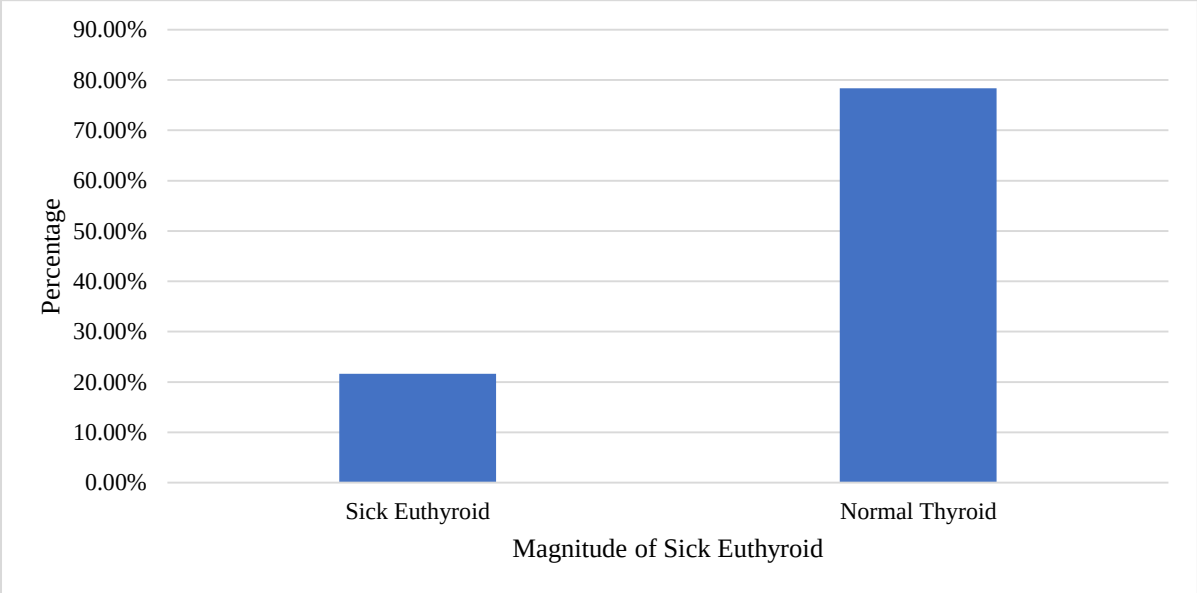

Table 7: Descriptive analysis of Day 1 and Day 7 T3, T4 and TSH in the study population (N=60)

Parameters	Frequency	Percentage
Day 1 T3 (ng/mL)		
Normal	48	80.00%
Abnormal	12	20.00%
Day 1 T4 (µg/dL)		
Normal	57	95.00%
Abnormal	3	5.00%
Day 1 TSH (uI/mL)		
Normal	57	95.00%
Abnormal	3	5.00%
Day 7 T3 (ng/mL)		
Normal	48	80.00%
Abnormal	12	20.00%
Day 7 T4 (µg/dL)		
Normal	59	98.33%
Abnormal	1	1.67%
Day 7 TSH (uI/mL)		
Normal	58	96.67%
Abnormal	2	3.33%

Among the study population, 12(20.00%) participants had abnormal T3 (ng/mL), 3 (5.00%) had abnormal T4 (µg/dL) and 3 (5.00%) had abnormal TSH (uI/mL) on day 1. On Day 7, 12 (20.00%) participants had abnormal T3 (ng/mL), 1 (1.67%) had abnormal T4 (µg/dL) and 2 (3.33 %) had abnormal TSH (uI/mL). (Table 7)

Table 8: Descriptive analysis of Serum magnesium (mg/dL) in the study population (N=60)

Serum Magnesium (mg/dL)	Frequency	Percentage
Normal	45	75.00%
Abnormal	15	25.00%

Among the study population, 45 **(**75.00%) participants were normal Serum Magnesium and 15 (25.00%) were abnormal Serum Magnesium. (Table 8)

Table 9: Descriptive analysis of Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

Peradeniya Organophosphorus Poisoning Scale	Frequency	Percentage
Moderate	43	71.67%
Severe	17	28.33%

Among the study population, 43 **(**71.67%) participants had moderate Peradeniya Organophosphorus Poisoning Scale and 17 (28.33%) had severe Peradeniya Organophosphorus Poisoning Scale. (Table 9 & Figure 8)

Figure 8: Bar Chart of Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

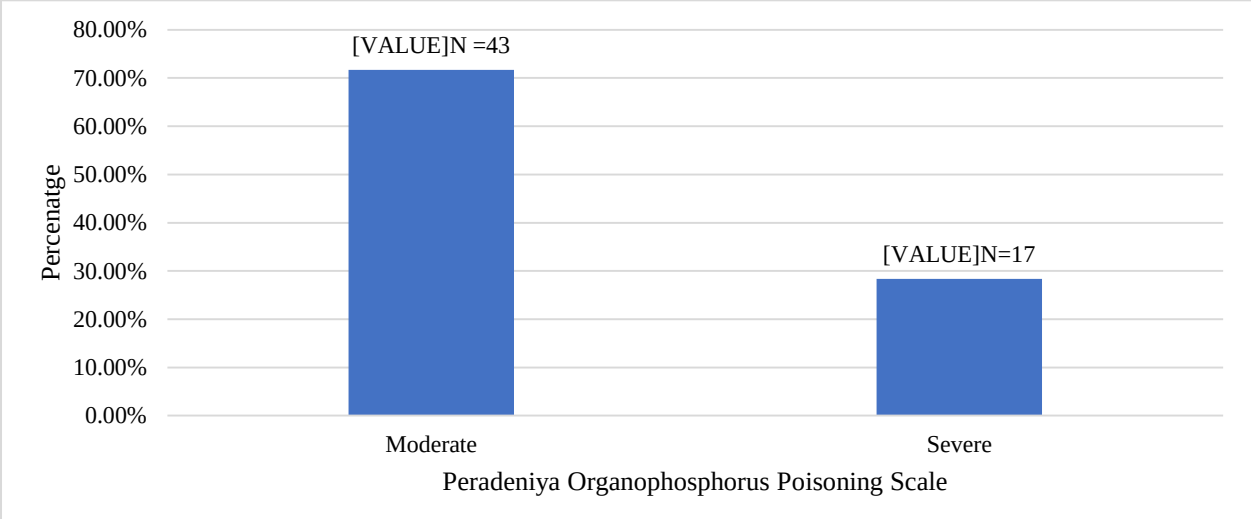

Table 10: Descriptive analysis of Mechanical ventilation in the study population (N=60)

Mechanical ventilation	Frequency	Percentage
Yes	22	36.67%
No	38	63.33%

Among the study population, 22 (36.67%) participants had mechanical ventilation. (Table 10 & Figure 9)

Figure 9: Bar Chart of Mechanical ventilation in the study population (N=60)

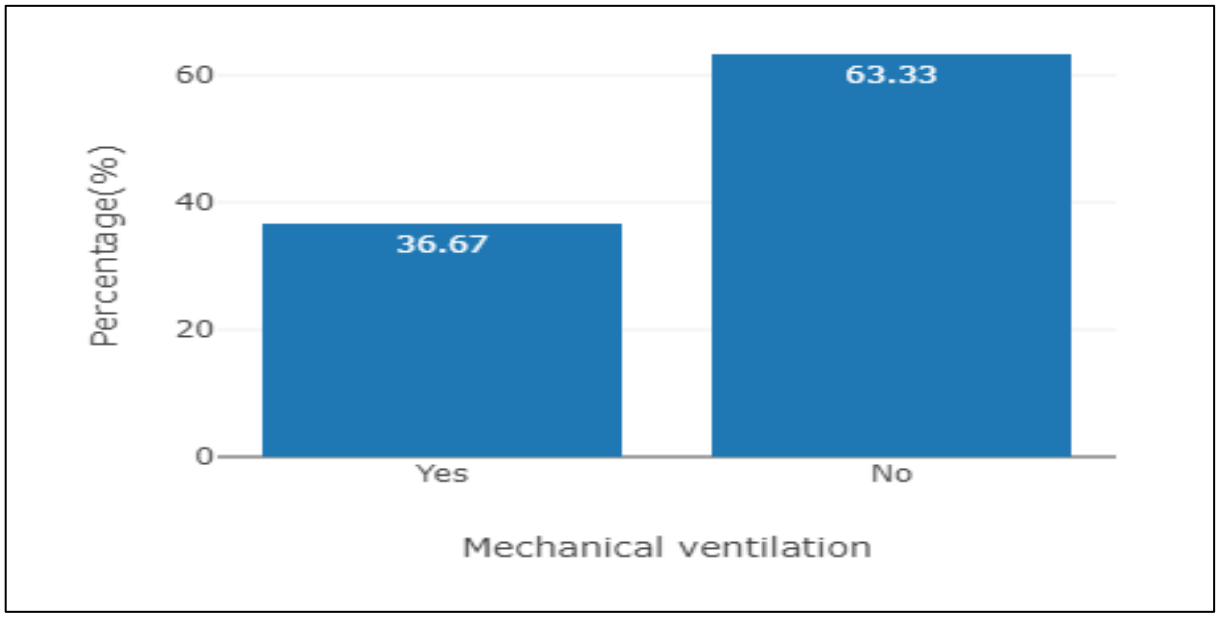

Table 11: Descriptive analysis of High dependency unit stay in the study population (N=60)

High dependency unit stay	Frequency	Percentage
Yes	57	95.00%
No	3	5.00%

Among the study population, 57 (95.00%) participants were high dependency unit stay. (Table 11 & Figure 10)

Figure 10: Pie Chart of High dependency unit stay in the study population (N=60)

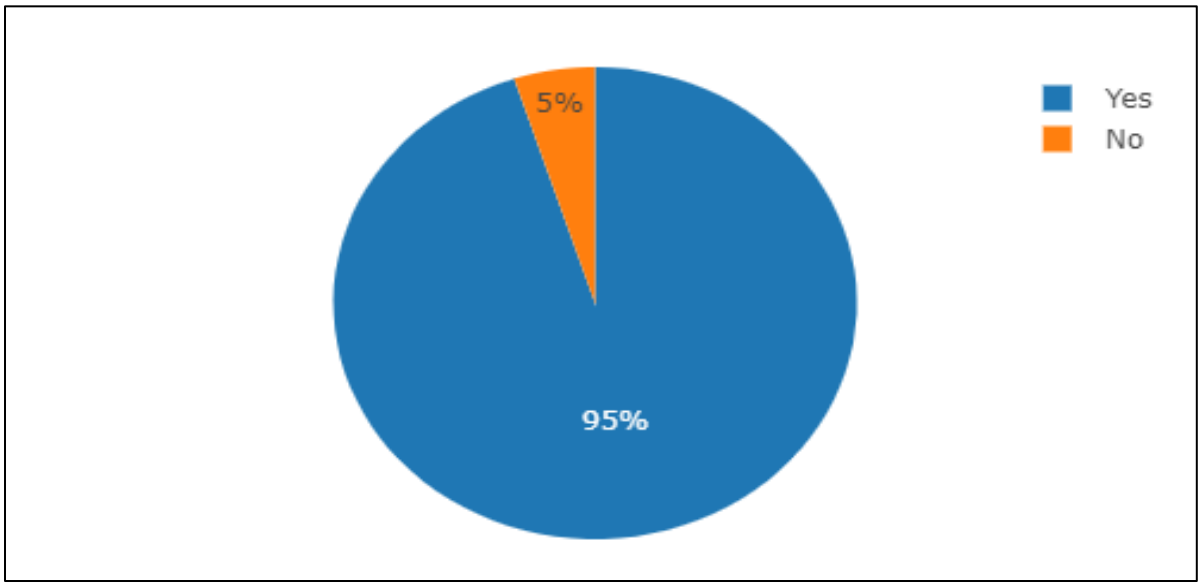

Table 12: Descriptive analysis of ICU stay in the study population (N=60)

ICU stay	Frequency	Percentage
Yes	35	58.33%
No	25	41.67%

Among the study population, 35 (58.33%) participants were Stay in ICU. (Table 12 & Figure 11)

Figure 11: Pie Chart of ICU stay in the study population (N=60)

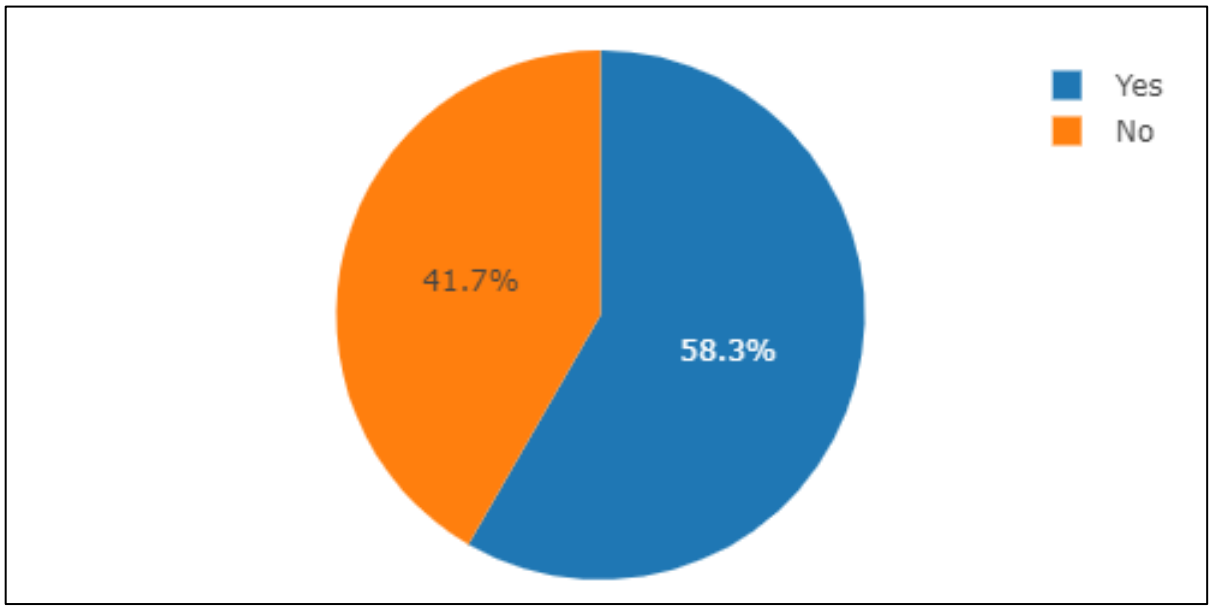

Table 13: Descriptive analysis of No. of days in hospital in the study population (N=60)

Name	Mean ± S. D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
No. of days in hospital	8.85±1.57	9.00	7.00	15.00	8.45	9.25

The mean Number of days in hospital was 8.85±1.57 in the study population, minimum level was 7 and maximum level was 15 in the study population (95% CI 8.45 to 9.25). (Table 13)

Table 14: Descriptive analysis of outcome Parameters in the study population (N=60)

Parameters	Frequency	Percentage
Recovered		
Recovered	50	83.33%
Death	10	16.67%
Outside treatment		
Stomach wash	10	16.67%
Atropine	7	11.67%
Stomach wash + Atropine	6	10.00%
Stomach wash + Atropine + Pralidoxime	5	8.33%
1 Day treatment with Atropine	1	1.67%
Nil	31	51.67%
Compound		
Chlorpyrifos + Cypermethrin	13	21.67%
Phorate	10	16.67%
Chlorpyriphos	8	13.34%
Dichlorvas	8	13.33%
Malathion	6	10.00%
Fenthion	4	6.67%
Profenos + Cypermethrin	2	3.33%
Parathion	3	5.00%
Quinolphos	qu	1.67%
Triazophos	1	1.67%
Diazion	1	1.67%
Profenofos	1	1.67%
Monochlorophos	1	1.67%
Unknown	1	1.67%

Among the study population, 50 (83.33%) participants were Recovered and 10 (16.67%) reported death. In Outside treatment, 10 (16.67%) participants were given Stomach wash, followed by Atropine in 7 (11.67%), both stomach wash & Atropine given were 6 (10.00%), stomach wash, atropine and pralidoxime in 5 (8.33%). In compound, 13(21.67%) participants had Chlorpyrifos + Cypermethrin, 10 (16.67%) had Phorate and 8 (13.34%) had Chlorpyriphos, 8 (13.34%) had Dichlorvas. (Table 14)

Table 15: Descriptive analysis of Baseline Parameters in the study population (N=60)

Baseline Parameters	Mean ± S. D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Pulse (Bpm)	96.07±23.23	88.00	60.00	170.00	90.19	101.94
SBP (mmHg)	115.93±14.88	111.00	90.00	160.00	112.17	119.70
DBP (mmHg)	70.83±10.30	70.00	60.00	100.00	68.23	73.44

The mean Pulse was 96.07±23.23 Bpm in the study population, Ranged between was 60 Bpm to 170 Bpm (95% CI 90.19 to 101.94). The mean SBP was 115.93±14.88 mmHg in the study population. Ranged between 90 to 160 mmHg (95% CI 67.43 to 70.13). The mean DBP was 70.83±10.30 mmHg in the study population. Range between 60 mmHg to 100 mmHg (95% CI 68.23 to 73.44). (Table 15)

Table 16: Descriptive analysis of Consciousness based on Glascow Coma Scale score out of 15 in the study population (N=60)

GCS out of 15	Frequency	Percentage
Severe (3-8)	6	10%
Moderate (9-12)	1	1.67%
Mild (13-15)	53	88.33%

Among the study population, 6 (10%) had severe, 1 (1.67%) had moderate and mild in 53 (88.33%)

Table 17: Descriptive analysis of Pupils in the study population (N=60)

Pupils	Frequency	Percentage
<= 3mm	48	80.00%
> 3mm	12	20.00%

Among the study population, 48 (80.00%) participants were <=3mm pupils, 12 (20%) were >3 mm, (Table 17)

Table 18: Descriptive analysis of Neck lift at admission in the study population (N=60)

Neck lift	Frequency	Percentage
Good	34	56.67%
Poor	26	43.33%

Among the study population, 34 (56.67%) participants had good neck lift and 26 (43.33%) were with poor neck lift. (Table 18 & Figure 12)

Figure 12: Pie Chart of Neck lift at admission in the study population (N=60)

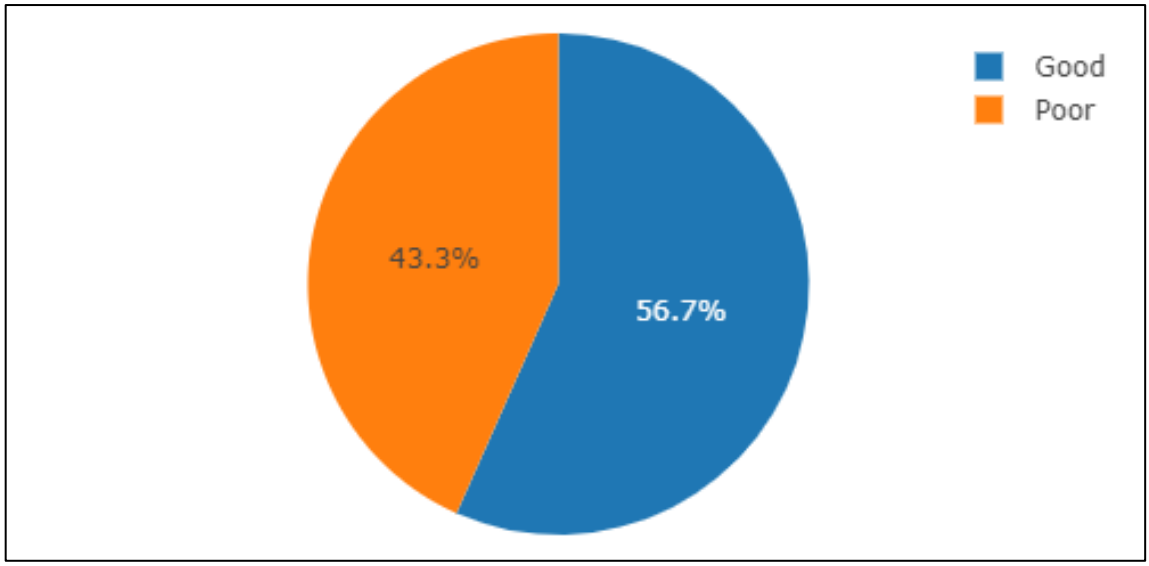

Table 19: Descriptive analysis of Seizures in the study population (N=60)

Seizures	Frequency	Percentage
Yes	8	13.33%
No	52	86.67%

Among the study population, 8 (13.33%) participants had seizures. (Table 19)

Table 20: Descriptive analysis of Fasciculations at admission in the study population (N=60)

Fasciculations	Frequency	Percentage
Yes	30	50.00%
No	30	50.00%

Among the study population, 30 (50.00%) participants had Fasciculations. (Table 20)

Table 21: Descriptive analysis of dry axilla at admission in the study population (N=60)

Dry axilla	Frequency	Percentage
Absent	26	43.33%
Present	34	56.67%

Among the study population, 34 (56.67%) participants had dry axilla at admission. (Table 21)

Table 22: Descriptive analysis of Systemic examination findings in the study population (N=60)

Systemic Examination	Frequency	Percentage
Cardiovascular system		
s1 s2 normal	60	100.00%
Respiratory system		
Bilateral normal vesicular breath sounds	46	76.67%
Crackles	14	23.33%
Central nervous system		
No focal neurological deficits	35	58.33%
Drowsy	16	26.67%
Irritable	6	10 %
Unresponsive	3	5 %
Per abdomen – GI symptoms (vomiting and diarrhea)		
Absent	60	100.00%
Present	0	0

Among the study population, all of them, 60 (100.00%) participants had s1 s2 normal Cardiovascular system. 46 (76.67%) had normal vesicular breath sounds in respiratory

system examination and 14 (23.33%) had crackles in Respiratory system examination. In Central nervous system, 35 (58.33%) participants had no focal neurological deficits, 16 (26.67 %) participants were drowsy, 6 (10%) were irritable and 3 (5%) were unresponsible. all of them, 60 (100.00 %) had no gastrointestinal symptoms like vomiting or diarrhea.

Table 23: Descriptive analysis of Parameters in the study population (N=60)

Parameters	Mean ± S. D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Pseudo cholinesterase (U/L)	340.70±221.88	232.00	200.00	960.00	284.56	396.84
Total bilirubin (mg/dL)	0.87±0.18	0.90	0.30	1.20	0.82	0.91
Aspartate Aminotransferase (U/L)	35.03±15.45	30.00	22.00	106.00	31.12	38.94
Alaline transaminase (U/L)	24.40±9.59	24.00	13.00	68.00	21.97	26.83

The mean Pseudo cholinesterase was 340.70±221.88 U/L in the study population. Ranged between was 200 cm to 960 U/L (95% CI 284.56 to 396.84). The mean Total bilirubin was 0.87±0.18 mg/dL in the study population. Ranged between 0.30 mg/dL to 1.20 mg/dL (95% CI 0.82 to 0.91). The mean Aspartate Aminotransferase was 35.03±15.45 in the study population. Range between 22 U/L to 106 U/L (95% CI 31.12 to 38.94). The mean Alaline transaminase was 24.40±9.59 in the study population. Range between 13 U/L to 68 U/L (95% CI 21.97 to 26.83). (Table 23)

Table 24: Descriptive analysis of Renal function tests, electrolytes and pseudo cholinesterase in the study population (N=60)

Parameters	Frequency	Percentage
Urea (mg/dL)		
Normal	60	100.00%
Creatinine (mg/dL)		
Normal	58	96.67%
Abnormal (reduced)	2	3.33%
Sodium mEq/L		
Normal	23	38.33%
Abnormal(reduced)	37	61.67%
Potassium mEq/L		
Normal	21	35.00%
Abnormal (reduced)	39	65.00%
Pseudocholinesterase (U/L)		
Reduced	60	100 %
Normal	0	0

Among the study population, 2 (3.33%) had reduced creatinine, 37 (61.67%) had hyponatremia, 39 (65%) had hypokalemia and all of them 60 (100 %0 had reduced pseudo cholinesterase levels. All of them 60 (100.00%) participants had normal Urea, 58 (96.67%) had normal creatinine, 23 (38.33%) participants had normal sodium and 21 (35.00%) had normal potassium. (Table 24)

Table 25: Comparison of Day 1 T3, T4 & TSH with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

Parameter	Peradeniya Organophosphorus Poisoning Scale		Chi square value	P value
	Moderate (N=43)	Severe (N=17)		
Day 1 T3 (ng/mL)				
Normal	39 (90.70%)	9 (52.94%)	10.85	0.0024
Abnormal (reduced)	4 (9.30%)	8 (47.06%)		
Day 1 T4 (µg/dL)				
Normal	41 (95.35%)	16 (94.12%)	0.04	1.0000
Abnormal	2 (4.65%)	1 (5.88%)		
Day 1 TSH (uI/mL)				
Normal	43 (100.00%)	14 (82.35%)	-	*
Abnormal	0 (0.00%)	3 (17.65%)		

=No statistical test was applied- due to 0 subjects in the cells

In moderate Peradeniya Organophosphorus Poisoning Scale, 39 (90.70%) participants had normal T3 and 4 (9.30%) had abnormal T3. In Severe Peradeniya Organophosphorus Poisoning Scale, 9 (52.94%) had normal T3 and 8 (47.06%) had abnormal T3, the difference in the proportion of Day 1 T3 (ng/mL) levels between Peradeniya Organophosphorus Poisoning Scale was statistically significant with P value 0.0024. In Moderate Peradeniya Organophosphorus Poisoning Scale, 41 (95.35%) participants had normal T4 and in severe Peradeniya Organophosphorus Poisoning Scale 16 (94.12%) participants had normal T4, the difference in the proportion of Day 1 T4 (µg/dL) between Peradeniya Organophosphorus Poisoning Scale was statistically not significant with P value 1.00. In Moderate Peradeniya Organophosphorus Poisoning Scale, 43 (100.00%) participants had normal TSH and in severe Peradeniya Organophosphorus Poisoning Scale 14 (82.35%) participants had normal TSH. (Table 25)

Figure 13: Cluster bar chart of Day 1 T3 (ng/mL) with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

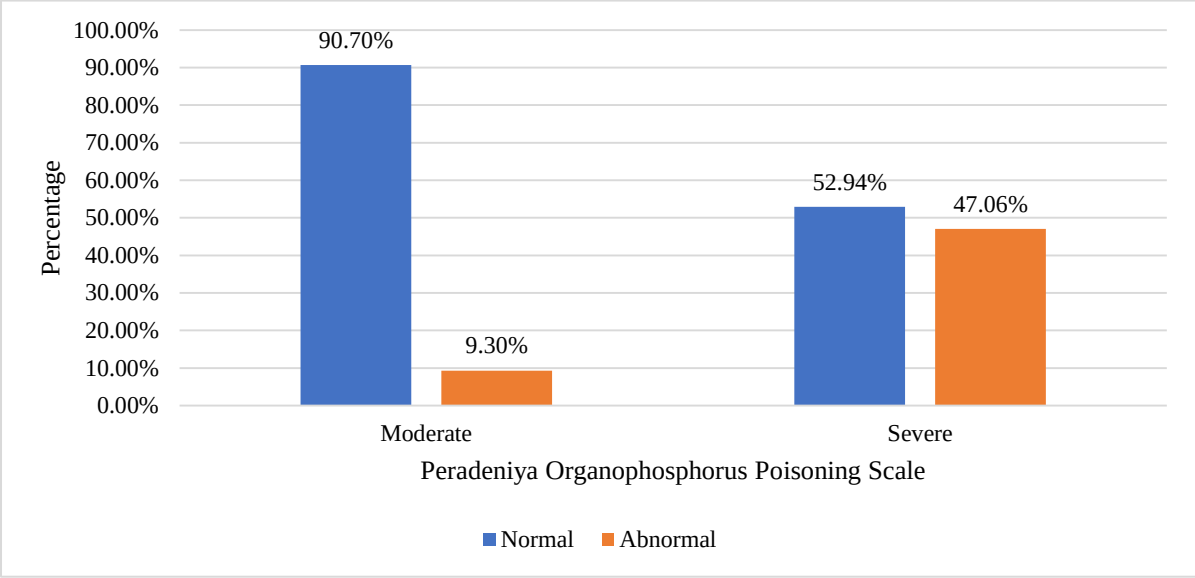

Figure 14: Cluster bar chart of Day 1 T4 (µg/dL) with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

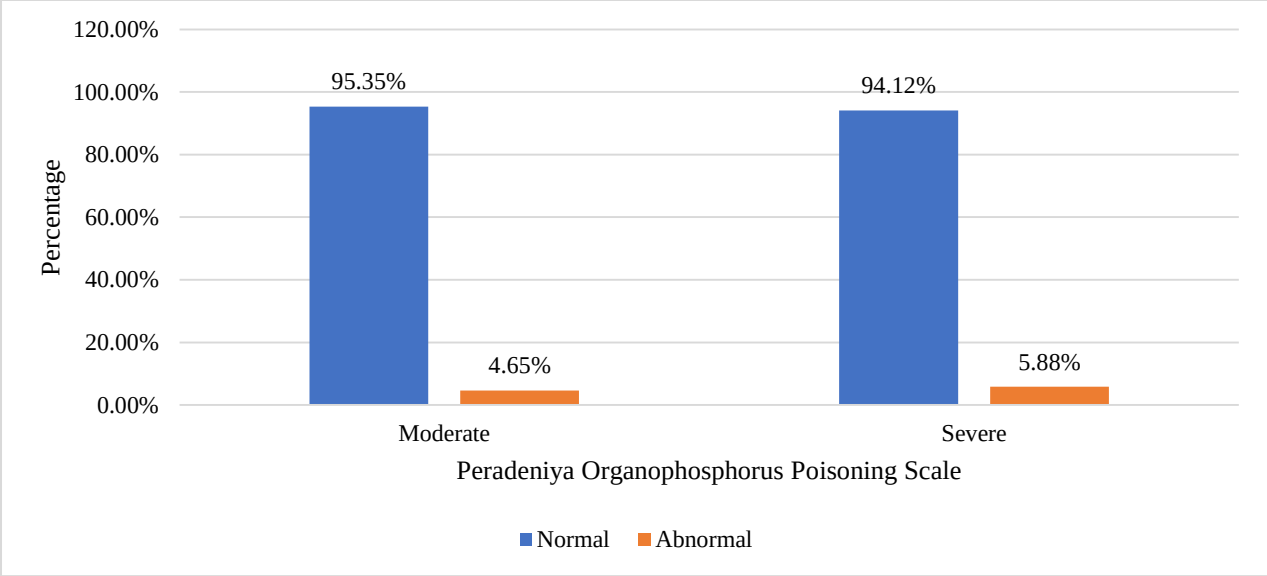

Figure 15: Cluster bar chart of Day 1 TSH (uI/mL) with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

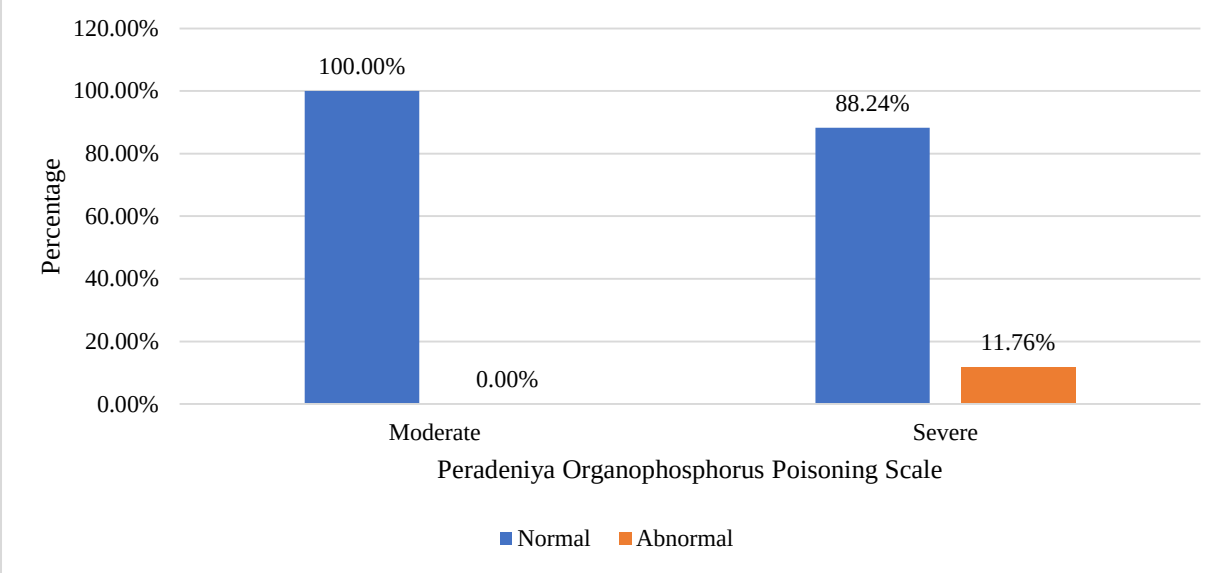

Table 26: Comparison of Day 7 T3, T4 & TSH with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

Parameter	Peradeniya Organophosphorus Poisoning Scale		Chi square value	P value
	Moderate (N=43)	Severe (N=17)		
Day 7 T3 (ng/mL)				
Normal	37 (86.05%)	11 (64.71%)	3.47	0.0806
Abnormal	6 (13.95%)	6 (35.29%)		
Day 7 T4 (µg/dL)				
Normal	42 (97.67%)	17 (100.00%)	-	*
Abnormal	1 (2.33%)	0 (0.00%)		
Day 7 TSH (uI/mL)				
Normal	43 (100.00%)	15 (88.24%)	-	*
Abnormal	0 (0.00%)	2 (11.76%)		

=No statistical test was applied- due to 0 subjects in the cells

In moderate Peradeniya Organophosphorus Poisoning Scale, 37 (86.05%) participants had normal T3 and 6 (13.95%) had abnormal T3. In Severe Peradeniya Organophosphorus Poisoning Scale, 11 (64.71%) had normal T3 and 6 (35.29%) had abnormal T3, the difference in the proportion of Day 7 T3 (ng/mL) between Peradeniya Organophosphorus Poisoning Scale was statistically not significant with P value 0.0806. In Moderate Peradeniya Organophosphorus Poisoning Scale, 42 (97.67%) participants had normal T4 and in severe Peradeniya Organophosphorus Poisoning Scale 17 (100.00%) participants had normal T4. In Moderate Peradeniya Organophosphorus Poisoning Scale, 43 (100.00%) participants had normal TSH and in severe Peradeniya Organophosphorus Poisoning Scale 15 (88.24%) participants had normal TSH in Day 7. (Table 26)

Figure 16: Cluster bar chart of Day 7 T3 (ng/mL) with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

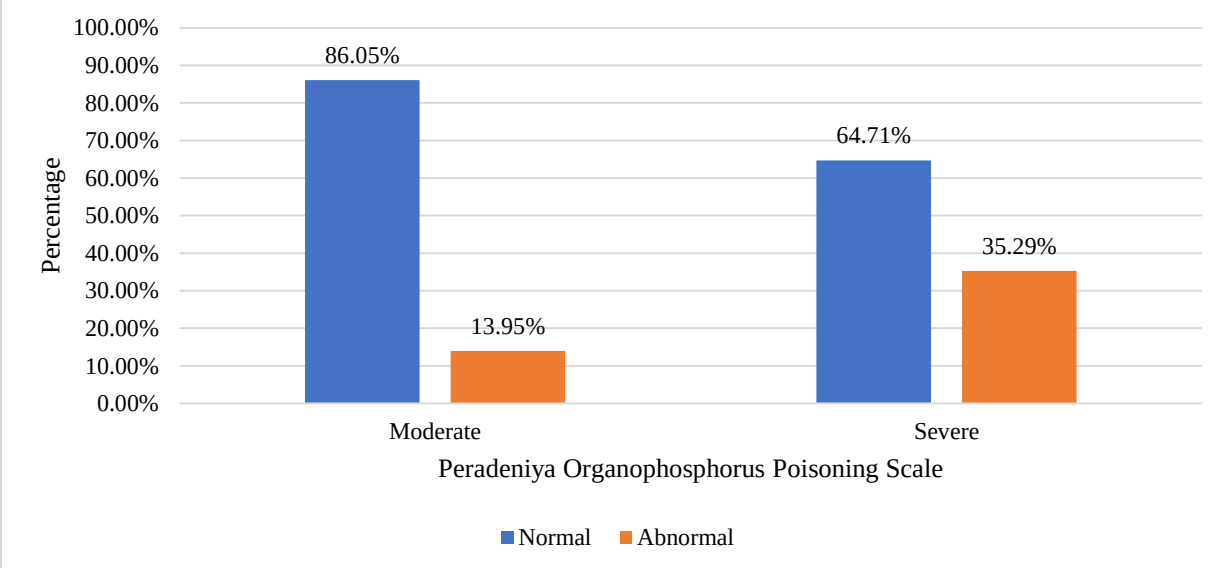

Figure 17: Cluster bar chart of Day 7 T4 (µg/dL) with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

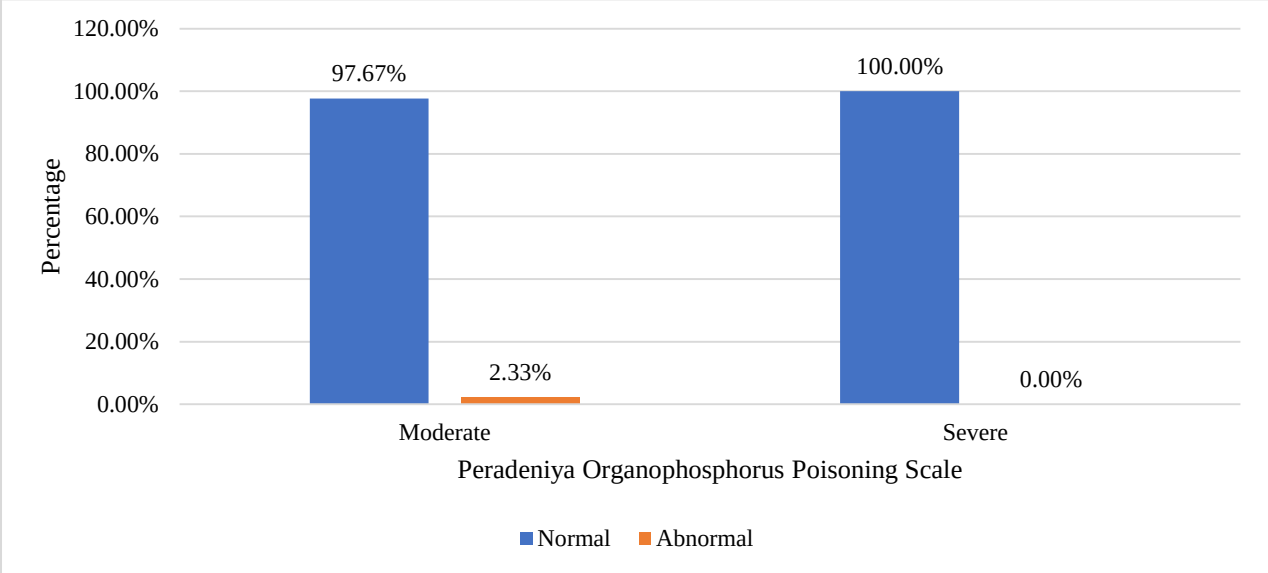

Figure 18: Cluster bar chart of 7 TSH (uI/mL) with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

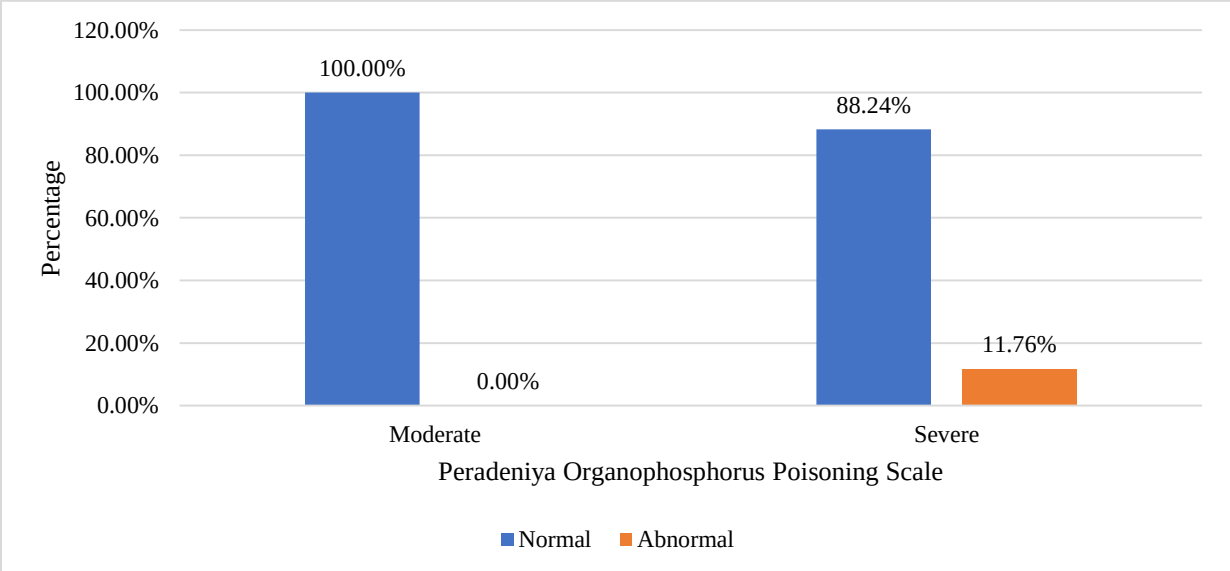

Table 27: Comparison of Serum Magnesium (mg/dL) with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

Serum Magnesium (mg/dL)	Peradeniya Organophosphorus Poisoning Scale (POP)		Chi square value	P value
	Moderate (N=43)	Severe (N=17)		
Normal	36 (83.72%)	9 (52.94%)	6.16	0.0207
Abnormal	7 (16.28%)	8 (47.06%)		

In Moderate POP scale, 36 (83.72%) participants had normal serum magnesium and 7 (16.28%) had abnormal serum magnesium. In Severe POP scale, 9 (52.94%) participants had normal serum magnesium and 8 (47.06%) had abnormal serum magnesium. The difference in Serum Magnesium (mg/dL) between Peradeniya Organophosphorus Poisoning Scale was statistically significant with P Value 0.0207. (Table 27 & Figure 18)

Figure 19: Cluster bar chart of Comparison of Serum magnesium (mg/dL) with Peradeniya Score in the study population (N=60)

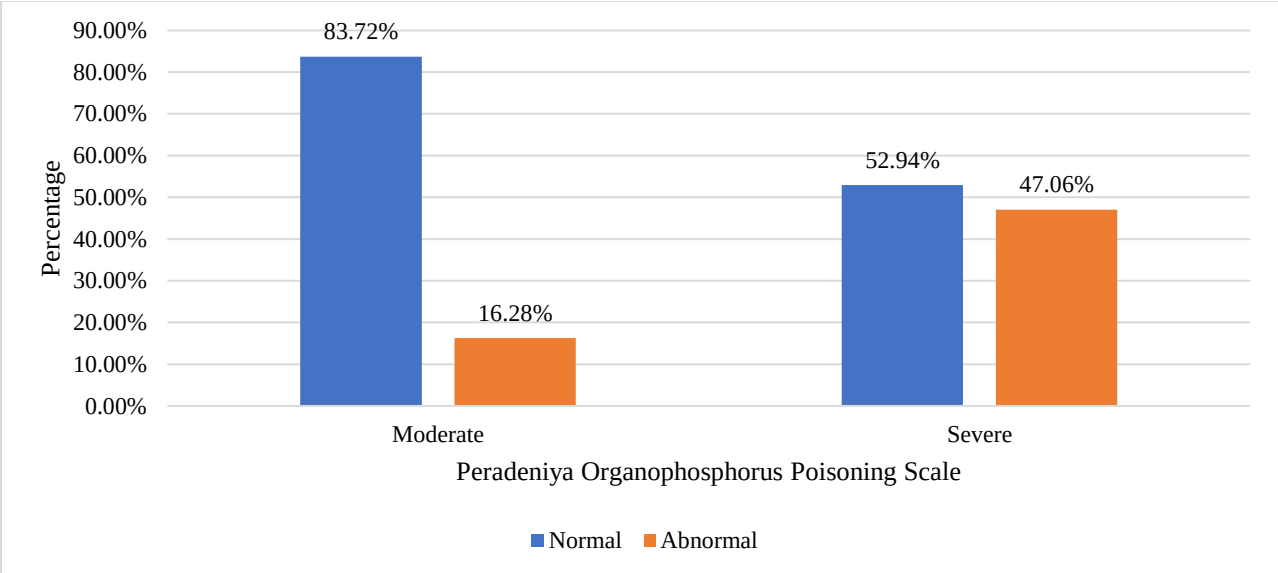

Table 28: Comparison of Mechanical ventilation, ICU stay, No. of days in hospital & Recovered with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

Parameter	Magnitude of Sick Euthyroid		Chi square value	P value
	Sick Euthyroid	Normal Thyroid		
Mechanical ventilation				
Yes (N = 22)	6 (27.27%)	16 (72.73%)	0.64	0.5200*
No (N = 38)	7 (18.42%)	31 (81.58%)		
ICU stay				
Yes (N = 35)	7 (20.00%)	28 (80.00%)	0.14	0.7108*
No (N = 25)	6 (24.00%)	19 (76.00%)		
No. of days in hospital	9.00(8.0 to 10.0)	9.00(8.0 to 9.0)		0.2207†
Recovered				
Recovered (N = 50)	7 (14.00%)	43 (86.00%)	10.39	0.0044*
Death (N = 10)	6 (60.00%)	4 (40.00%)		

***=Chi Square test P value; †= Mann Whitney U test P value**

Out of 22 participants with Mechanical ventilation, 6 (27.27%) had Sick Euthyroid and 16 (72.73%) had Normal Thyroid. The difference in Mechanical ventilation between Magnitude of Sick Euthyroid was statistically not significant with P Value 0.5200. Out of 35 participants with stay in ICU, 7 (20.00%) had Sick Euthyroid and 28 (80.00%) had Normal Thyroid. The difference in ICU stay between Magnitude of Sick Euthyroid was statistically not significant with P Value 0.7108. The median No. of days in hospital of Sick Euthyroid was 9.00(8.0 to 10.0) and it was 9.00(8.0 to 9.0) in Normal Thyroid, the median difference of No. of days in hospital between Magnitude of Sick Euthyroid was statistically not significant with P value 0.2207. Out of 50 participants with Recovered, 7 (14.00%) had Sick Euthyroid and 43 (86.00%) had Normal Thyroid. Out of 10 participants with death, 6 (60.00%) had Sick Euthyroid and 4 (40.00%) had Normal Thyroid. The difference in Recovered and death cases

between Magnitude of Sick Euthyroid was statistically significant with P Value 0.0044. (Table 28)

Figure 20: Cluster bar chart of Comparison of Mechanical ventilation with Magnitude of Sick Euthyroid in the study population (N=60)

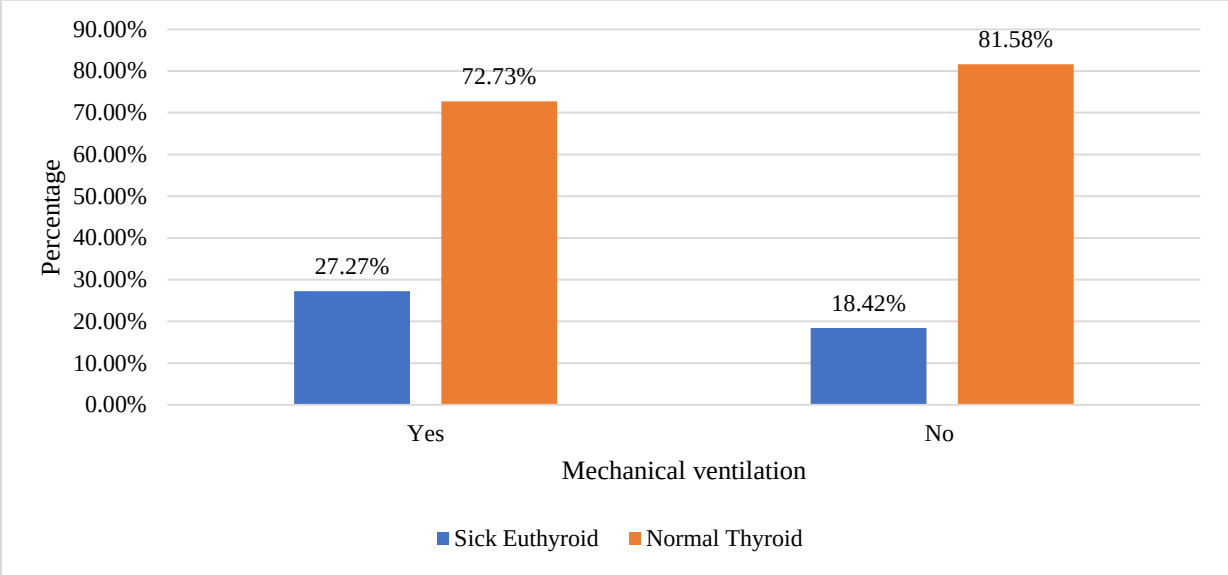

Figure 21: Cluster bar chart of Comparison of ICU Stay with Magnitude of Sick Euthyroid in the study population (N=60)

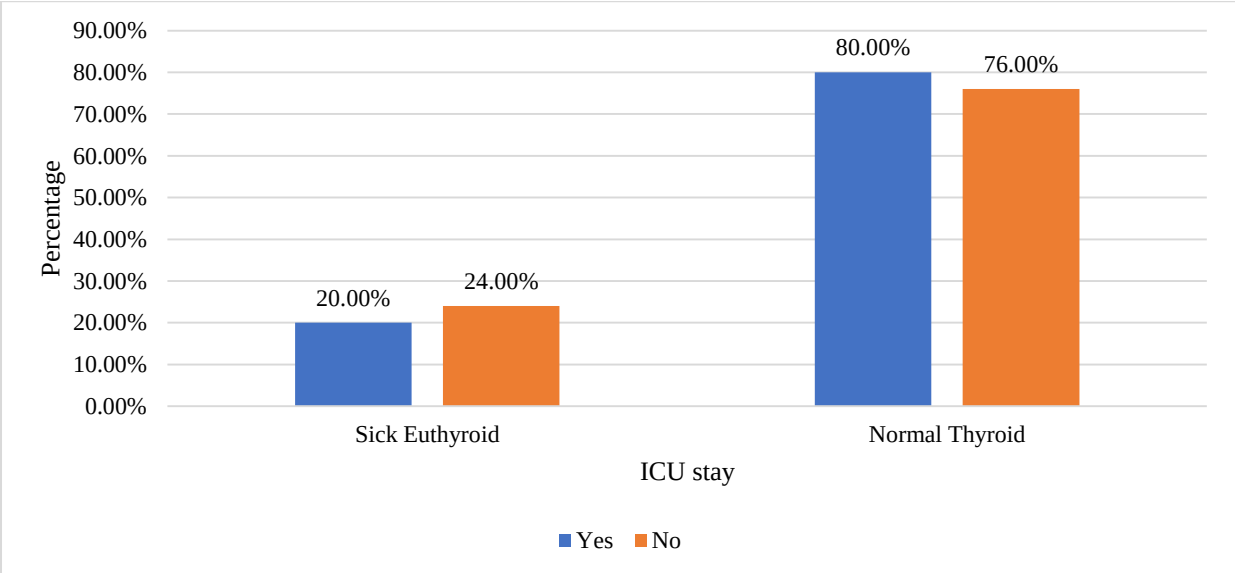

Figure 22: Boxplot of Comparison of No. of days in hospitals with Magnitude of Sick Euthyroid in the study population (N=60)

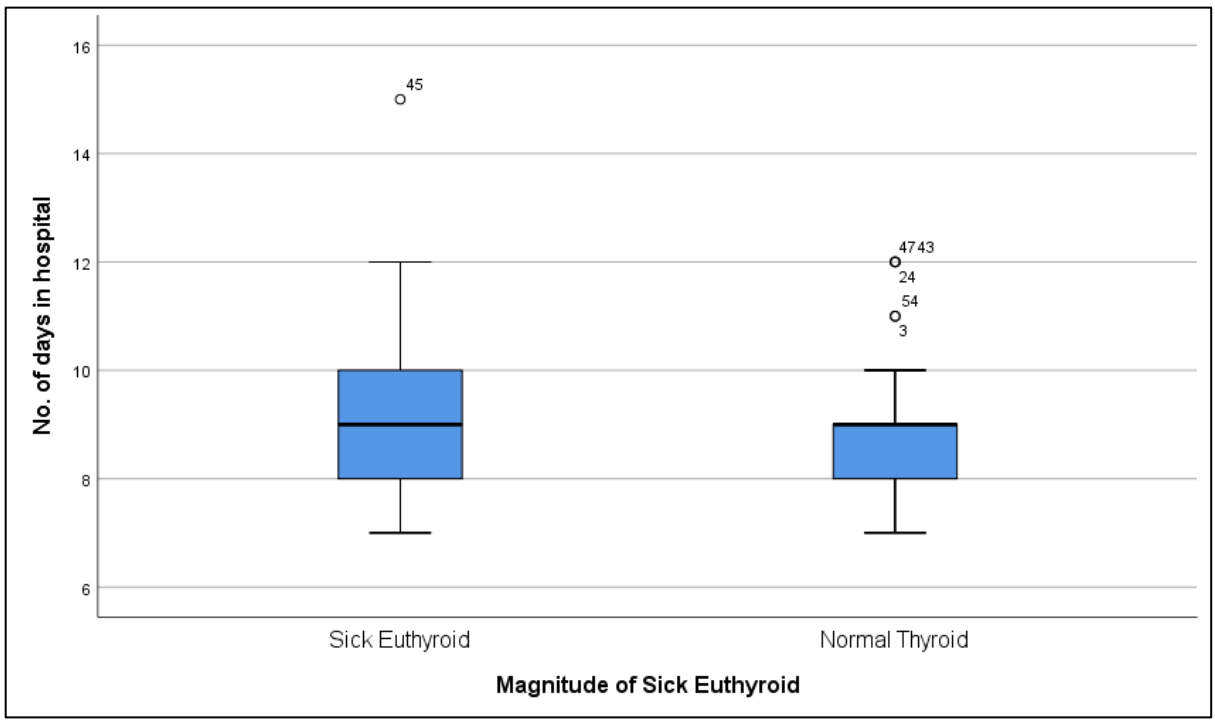

Figure 23: Cluster bar chart of Comparison of Recovered with Magnitude of Sick Euthyroid in the study population (N=60)

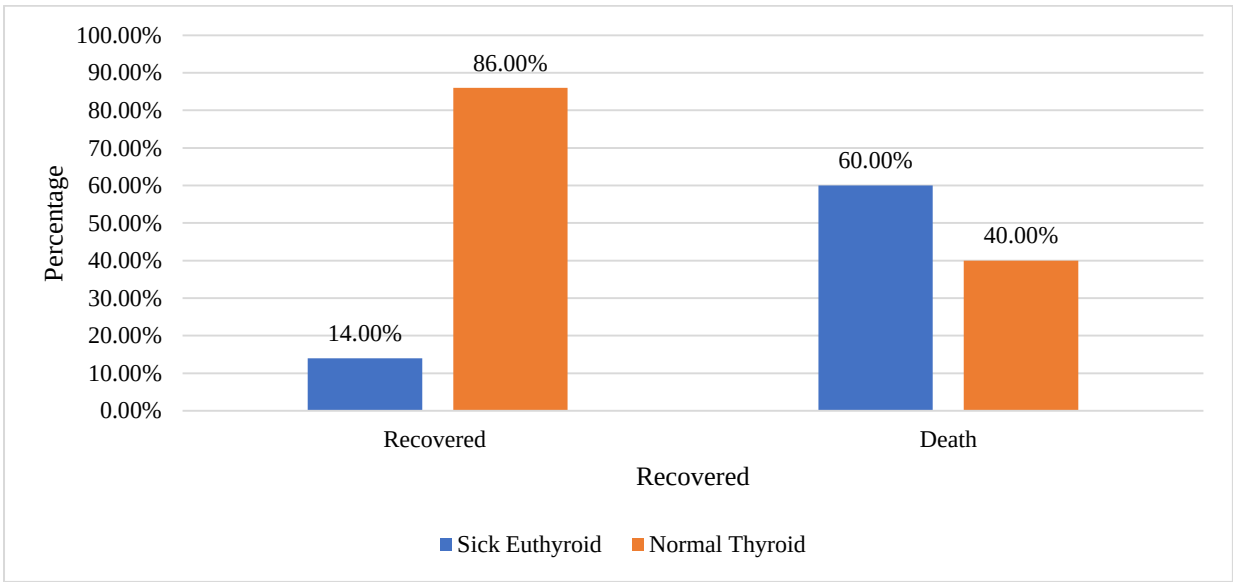

Table 29: Comparison of Magnitude of Sick Euthyroid with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

Magnitude of Sick Euthyroid	Peradeniya Organophosphorus Poisoning Scale		Chi square	P value
	Severe (N=17)	Moderate (N=43)		
Sick Euthyroid	6 (35.29%)	7 (16.28%)	2.596	0.163
Normal Thyroid	11 (64.71%)	36 (83.72%)		

In Severe POP scale, 6 (35.29%) participants had Sick Euthyroid and 11 (64.71%) had Normal Thyroid. In Moderate POP scale, 7 (16.28%) participants had Sick Euthyroid and 36 (83.72%) had Normal Thyroid. The difference in Magnitude of Sick Euthyroid between Peradeniya Organophosphorus Poisoning Scale was statistically not significant with P Value 0.163. (Table 29 & Figure 24)

Figure 24: Custer bar chart of Comparison of Magnitude of Sick Euthyroid with Peradeniya Score in the study population (N=60)

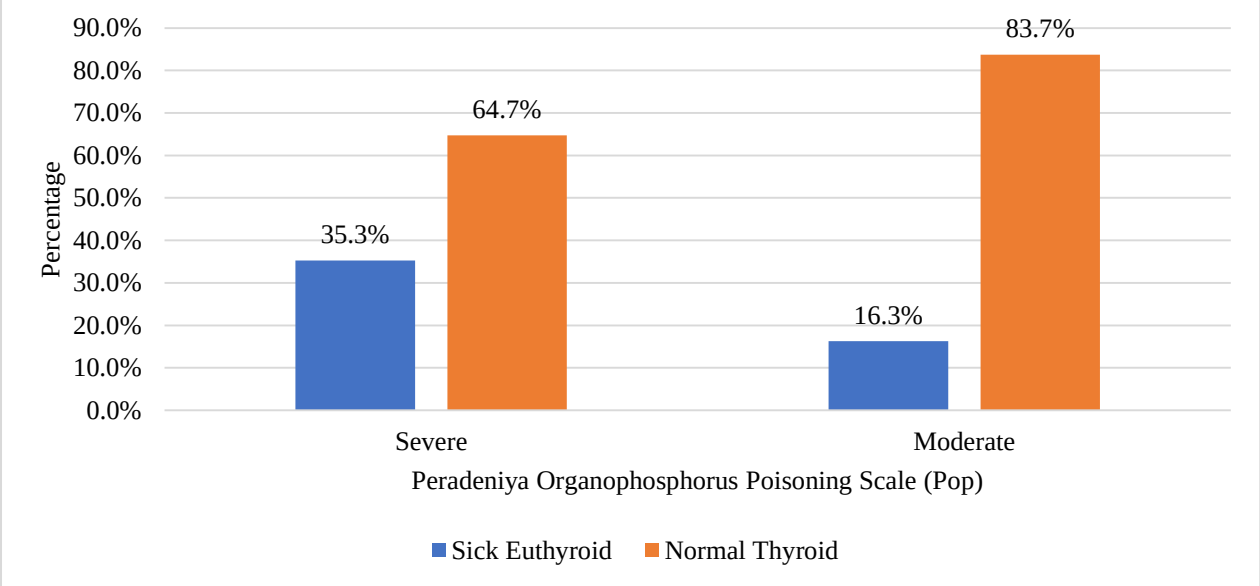

Table 30: Predictive validity of Magnitude of Sick Euthyroid in predicting Severe Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

Parameter	Value	95% CI	
		Lower	Upper
Sensitivity	35.29%	14.21%	61.67%
Specificity	83.72%	69.30%	93.19%
False positive rate	16.28%	6.81%	30.70%
False negative rate	64.71%	38.33%	85.79%
Positive predictive value	46.15%	19.22%	74.87%
Negative predictive value	76.60%	61.97%	87.70%
Diagnostic accuracy	70.00%	56.79%	81.15%

The Magnitude of Sick Euthyroid had sensitivity of 35.29% (95% CI 14.21% to 61.67%) in predicting Severe Peradeniya Organophosphorus Poisoning Scale. Specificity was 83.72% (95% CI 69.30% to 93.19%), false positive rate was 16.28% (95% CI 6.81% to 30.70%), false negative rate was 64.71% (95% CI 38.33% to 85.79%), positive predictive value was 46.15% (95% CI 19.22% to 74.87%), negative predictive value was 76.60% (95% CI 61.97% to 87.70%), and the total diagnostic accuracy was 70.00% (95% CI 56.79% to 81.15%). (Table 30)

Table 31: Comparison of Parameters with Peradeniya Organophosphorus Poisoning Scale in the study population (N=60)

Parameter	Peradeniya Organophosphorus Poisoning Scale		Chi square value	P value
	Moderate (N=43)	Severe (N=17)		
Urea (mg/dL)				
Normal	43 (100.00%)	17 (100.00%)	-	*
Creatinine (mg/dL)				
Normal	42 (97.67%)	16 (94.12%)	0.48	0.4898
Abnormal	1 (2.33%)	1 (5.88%)		
Sodium (mEq/L)				
Normal	17 (39.53%)	6 (35.29%)	0.09	0.7608
Abnormal	26 (60.47%)	11 (64.71%)		
Potassium (mEq/L)				
Normal	15 (34.88%)	6 (35.29%)	0.00	0.9760
Abnormal	28 (65.12%)	11 (64.71%)		

Test is not applicable due to nature of the data

The difference in Creatinine (mg/dL) between Peradeniya Organophosphorus Poisoning Scale was statistically not significant with P Value 0.4898. The difference in Sodium (mEq/L) between Peradeniya Organophosphorus Poisoning Scale was statistically not significant with P Value 0.7608. The difference in Potassium (mEq/L) between Peradeniya Organophosphorus Poisoning Scale was statistically not significant with P Value 0.9760. (Table 31)

DISCUSSION

DISCUSSION

Organophosphorus toxicity is a major public health issue in developing nations with a significant morbidity and death rate. The majority of OP poisonings and subsequent deaths take place as a result of intentional self-ingestion, especially in the adult population because highly toxic pesticides are easily accessible at times of stress brought on by issues with family, failed relationships, exam phobia, etc. Due to unprotected use, occupational exposure is also seen in adults. Children are more susceptible to accidental poisoning due to their exploratory nature. Acetylcholine accumulation is linked to initial organophosphorus toxicity and its adverse impacts on the central nervous system. Prior to mortality, this causes symptoms such as seizures, pulmonary arrest, anxiety, headaches, impaired coordination, unintentional trembling, and asthenia. OP-based insecticide toxicity is a dangerous medical illness which requires prompt detection and management. Organophosphate impacts the hypothalamus gland, pituitary gland, and thyroid glands also known as the suprarenal gland, causing biochemical alterations that are particular to a condition found in post-critical condition subjects known as "sick euthyroid syndrome" or "non-thyroidal impairment syndrome."[85] The current study is being carried out to investigate the happening of non-thyroidal illness syndrome in subjects suffering from onset organophosphorus chemical toxicity and to determine if certain non-thyroidal illness syndrome has a predictive value in the eventual result of onset organophosphorus chemical toxicity.

Individuals with history of ingestion, inhalation or cutaneous absorption of organophosphorus compound within 24 hours coming to R. L. Jalappa Hospital and Research Centre above 18 years of age are considered in the research. To assess the intensity of organophosphorus toxicity, the clinical scale is used to measure the selected clinical manifestation parameters.

The overall result comprised 60 participants considering an age category of 35.48±11.81 years. Ours is a male predominant population with 71.67% males and 28.33% females. Kumar et al.'s study included 30 patients with organophosphate poisoning with a much younger age group with a mean age of 25.9 ± 10.99 yrs. Their study also had a male predominant population with 60% males and 40% females.[69]In the occupational field are majoring filled by men in India, thus they are more susceptible to excessive stress and harmful routines, which may explain why there are more incidences among men. Şimşek's study group included 29 OP poisoned patients with a slightly older age group than our study with a mean age of 41.9±16.7 (range: 18–69) years. Males were 55.17% with 44.82% females.[86]All included patients in Masoud et al.'s study were at middle age ranged between 16 and 37 years old with a female predominance at 62% females.[87] This may be due to sociocultural factors as lower education and lower economic income than males in Egypt. In Dutta et al study,.'s the average age was 30.1 10.3 years, with an age rank of 18 to 49 years old.[88] Hundekari et al.'s study, 57.33% of men were shown to be more inclined to self-harm than women. Exposure to poison was much more likely in individuals aged 15 to 35 years old.[89]The mean age in Huang et al.'s study among the anticholinesterase pesticide poisoning (ACCP) group was much older compared to our study at 54.27 ± 16.20 years. Men outnumbered females in the ACPP cohort at 72.13%.[81] The mean age of Cobilinschi et al.'s research subject was 49 years old, showing that the subject was geriatric to our study with the number of males higher than female patients.[90]

At admission on day one and day 7 majority had normal thyriod profile whereas, the percentage of sick euthyroid in study is 21.67%. The major compounds in our study are 21.67% had Chlorpyrifos + Cypermethrin, 16.67% had Phorate and 13.34% had Chlorpyriphos. The compounds Hundekari et al.'s study were dimetoate, monocrotophos, chlorpyriphos, paraoxan, mevinphos, triazophos.[89]

The mean Pulse was 96.07±23.23 Bpm, mean SBP was 115.93±14.88 mmHg, mean DBP was 70.83±10.30 mmHg in the study population in our study. All patients had normal blood pressure, 34.5% had tachycardia in Masoud et al.'s study [87]Among the study population, 36.67% required mechanical ventilation, 95.00% needed a high dependency unit stay and 58.33% required ICU admission. The mean number of days in hospital is 8.85±1.57 in the study population. Duration of hospitalization in medical intensive care unit (day) 7.0±5.0 in Şimşek's study.[86] Dutta et al. reported a mean of 9.5 ± 7.6 days duration of hospital stay in their study.[88]

During the analysis, majority recovered (83.33%) and mortality was 16.67%. Mortality rate was only 6.9% in Masoud et al.'s study, the low mortality rate may be explained by early administration to hospital with early intervention with antidotes. Also, 93.1% were moderate cases as regards poison severity score in their study.[87]Mortality rate was 20.67% in Hundekari et a.'s study.[89] Among our study population, 6 (10%) had severe, 1 (1.67%) had moderate and mild in 53 (88.33%). The GCS was 13.5 ± 2.7 (range; 6 ‑ 15) in Dutta et al.'s study.[88]

The mean Pseudo cholinesterase was 340.70±221.88 U/L in the current study. The minimum value of pseudocholinesterase was 1509.2±2544.4 (range: 2550–6800) U/mL in Şimşek's study.[86]The mean serum cholinesterase in poisoning was 913±15.3 in Yadav's study.[71]

Hypokalemia is noted among 65.00% and hyponatremia in 61.67% of our study group. Masoud et al. reported hypokalemia was observed in 57.6% studied patients.[87] The degree of organophosphorus exposure was determined by the Peradeniya organophosphorus poisoning measure revealed 71.67% had moderate poisoning and 28.33% had severe poisoning. On day 1, among the moderately poisoned patients as per POP, 90.70% had normal T3 and 9.30%

had irregular T3. Among the severely poisoned patients, 52.94% had normal T3 on day and 47.06% had abnormal T3. With a p-value of 0.0024, the variation in the part of day 1 triiodothyronine between Peradeniya organophosphorus poisoning grades was significant statistically. The overall T3 concentrations were determined to fall within the acceptable biological limit in both instances, however, the recuperation cycle readings were greater than the response stage readings, and this was numerically relevant in Kumar et al.'s study [69]Triiodothyronine and thyroxine levels were not different substantially between the base point and at 90 days as reported by Dutta et al. in their study.[88] According to Hundekari et al.'s study, when organophosphate toxicity subjects were contrasted with controls, there was a minor and insignificant drop in blood triiodothyronine and thyroxine levels but no big difference in serum thyroid stimulating hormone concentrations.[89] With a p-value of 1.00, the variation in the part of day 1 triiodothyronine between Peradeniya organophosphorus poisoning grades was numerically insignificant. On day 1, among the moderately poisoned patients, all had normal TSH and in severely poisoned patients, 82.35% of subjects with standard thyroid-stimulating hormone. With a p-value of 0.0806, the variation in the part of day 7 triiodothyronine between Peradeniya organophosphorus poisoning classes is statistically insignificant. T4 on day 7 was normal in all of the severely poisoned patients and 97.67% of the moderately poisoned patients. TSH was normal on day 7 in all of the moderately poisoned patients and among the severely poisoned, 88.24% had normal TSH on day 7.

Majority of patients had normal TSH levels, which is in line with Dutta et al[88], Masoud et al.[87] and Gundogan et al.[91]s studies, this may be explained by non-thyroidal illness syndrome which is distinguished by acceptable thyroid-stimulating hormone and thyroxine values and decreased triiodothyronine level which normalize after treatment.[92] Dutta et al. reported that

the blood thyroid-stimulating hormone at starting (0.7 0.5) was reduced at the point of admission compared to the TSH after 3 months of follow-up (2.9 ± 2.1) (P = 0.02).[88] Total triiodothyronine levels were greater than the acceptable biological limit during the initial exposure, imitating hyperthyroidism, and then returned to normal following recuperation. Total triiodothyronine levels were numerically notable during the initial stage of intoxication in Kumar et al.'s study[69] Thyroid stimulating hormone levels were less during the exposure period than during the healing process, although a statistically significant increase was detected during the recovery phase in Kumar et al investigation. They determined that hazardous dosages of an organophosphorus chemical such as chlorpyrifos, phosate 10% granules, and monocrotophos may act on thyroid follicles and release triiodothyronine from storage. Another idea is that the opiate substance derived from opium limit peripheral conversion of triiodothyronine to thyroxine by hindering deiodinase enzymes in the liver and renal, resulting in hyperthyroidism after acute OP poisoning. This discovery is supported by a substantial variation in TT3 readings between the two periods of their investigation.[69]

Dutta et al. revealed that alteration in thyroid stimulating hormone levels in major conditions, the intensity and length of the sickness, the patient's age, and the number of cytokines all play a role.[88] This was not observed in our study which may be due to the fact that majority of the cases were moderate per POP grade of poisoning. Contrary to the above, Hundekari et al.'s study did not indicate any disruption in thyroid hormone metabolism during acute OP poisoning.[89] During the prolonged era, no subjects reported adverse thyroid function findings in Şimşek et al.'s study.[86]Huang et al.'s study ACPP was linked to a higher likelihood of underactive thyroid, particularly in the age subgroup of 40-64 years, female sex, and history of goitre. During the follow-up intervals, the elevated risk for underactive thyroid was greatest in the first month following poisoning and continued to exhibit a pattern after 30

days.[81] Anticholinesterase pesticides disrupt the process by which thyroid hormones are produced, which may be how ACPP raises the risk of hypothyroidism. The elevated risk for hypothyroidism was greatest in the first month following poisoning compared to subsequent follow-up periods, indicating that the short-term rather than long-term effects of ACPP on hypothyroidism are more noticeable.In Cobilinschi et al.'s the initial assessment on admittance demonstrated a rise over the maximum bound of standard limit, accompanied by a considerable drop after a day in the study group. Although the initial levels of free thyroxine and thyroid stimulating hormone did not surpass the maximum bound of normal, there was a considerable drop after a day in the both assays[90].

Among those who have recovered, 14.00% had Sick Euthyroid and 60.00% of the patients who died were Sick Euthyroid with the proportion of mortality between Sick Euthyroid and normal thyroid being statistically significant with p value 0.0044. Thyroid function may vary throughout major illness based on the extent of the disease, the course of the ailment, cytokine levels, and the age of the subject. In contrast to the cytokine-mediated reduction of thyroid-stimulating hormone in major condition, elevated induction of liberated acetylcholine in somatostatin tone may also promote thyroid stimulating hormone suppression in patients with organophosphate chemical toxicity. Furthermore, the type of OPC may influence thyroid hormone levels in the blood.[88]Among the sick euthyroid patients, 35.29% had severe POP grade and 16.28 had moderate POP grade. The predictive validity of Sick Euthyroid in predicting the POP grade of poisoning gave a responsiveness of 35.29%, precision of 83.72%, probability of true disease 46.15%, negative predictive value 76.60% with a total diagnostic accuracy of 70.00%.

Hundekari et al. found a small and clinically insignificant drop in triiodothyronine and thyroxine concentrations in organophosphate-poisoned subjects, but no major shift in serum thyroid stimulating hormone concentrations in their research of 150 organophosphate-poisoned subjects.[89]Rao and Bhavana discussed two case reports in which there was decreased TSH measurement and hyperthyroidism at stage of acute poisoning and normalized at time of discharge.[93]Kumar et al.'s study reported that subjects suffering from initial organophosphorus chemical toxicity have hyperthyroidism because opiates affect the endocrine system, storing and releasing total thyroxine, as well as preventing the peripheral conversion of total thyroxine to total triiodothyronine by blocking peroxidase enzyme-deiodinases. These subjects sustained euthyroid after therapy for the initial period. The consequences of organophosphate on thyroid hormone concentrations in blood are dependent on pesticide dosage and length of vulnerability.[69] Yadav et al. concurred with this stating that thyroid investigation and serum cholinesterase can both be utilised as predicting indicators in determining the intensity of organophosphate toxicity.[71]Şimşek et al.'s study[86] showed that organophosphate toxicity can impair endocrine performance both acutely and chronically. Most hormones improve following the initial stage of recuperation. Our study is in agreement with the above that thyroid tests and euthyroid status is useful in determining OP poisoning mortality and providing prompt treatment. Mortality rate is high in those with sick euthyroid status. With a specificity of 83.72%, sick euthyroid can be depended upon to not give any false positive results, hence can be used as a biomarker in predicting mortality in OP poisoning cases.

CONCLUSION

CONCLUSION

- The overall result comprised 60 subjects with an average age of 35.48±11.81 years. Ours is a male predominant population with 71.67% males and 28.33% females.
- At admission on day 1, 12(20.00%) participants had abnormal T3 (ng/mL), 3 (5.00%) had abnormal T4 (µg/dL) and 3 (5.00%) had abnormal TSH (uI/mL).
- The percentage of sick euthyroid in study is 21.67%.
- When the degree of organophosphate toxicity was rated using the Peradeniya organophosphorus poisoning scale, 71.67% had moderate toxicity and 28.33% had severe toxicity.
- In our study, 83.33% recovered and the mortality rate was 16.67%.
- The mean Pseudo cholinesterase was 340.70±221.88 U/L in the study population.
- With a p-value of 0.0024, the variation in the part of day 1 triiodothyronine between Peradeniya organophosphorus poisoning grades was numerically relevant. However, with a p-value of 1.00, the variation in the part of day 1 thyroxine between Peradeniya organophosphorus poisoning grades was not numerically relevant. On day 1, among the moderately poisoned patients, all had normal TSH and in severely poisoned patients, 82.35% subjects had an average level of thyroid stimulation hormone.
- With a p-value of 0.0806, the variation in the part of day 7 triiodothyronine between Peradeniya organophosphorus poisoning classes is numerically insignificant. T4 on day 7 was normal all of the severely poisoned patients and 97.67% of the moderately poisoned patients. TSH was normal on day 7 in all of the moderately poisoned patients and among the severely poisoned, 11.76 % had abnormal TSH on day 7.
- The difference in Serum Magnesium between POP grades of poisoning was statistically significant with p value 0.0207.

- Among those who have recovered, 14.00% had Sick Euthyroid and 60.00% of the patients who had mortality were Sick Euthyroid with the proportion of mortality between Sick Euthyroid and normal thyroid being numerically notable with p-value 0.0044.
- The predictive validity of Sick Euthyroid in predicting the POP grade of poisoning gave a responsiveness of 35.29%, precision of 83.72%, probability of true disease 46.15%, negative predictive value 76.60% with a total diagnostic accuracy of 70.00%.

Limitations and Recommendations:

Because the number of patients in this study is minimal, application of study outcomes should be cautiously applied.

SUMMARY

SUMMARY:

During critical illness, the hypothalamic-pituitary-thyroid (HPT) axis may undergo dramatic alterations. The most frequent alteration is a reduction in serum triiodothyronine (T3) levels, although in severe disease, serum thyroxine (T4) and thyroid stimulating hormone (TSH) levels may also decline.[94] Organophosphate toxicity is characterised by "sick euthyroid syndrome," a thyroid dysfunction peculiar to severe diseases such as multiple traumatic injuries or blood poisoning."[95] Physiologically, this 'non-thyroid' condition is distinguished by low free triiodothyronine, normal or low free thyroxine 4, and low thyroid-stimulating hormone.[94] However, the initial few hours of stressful situations are marked by a rise in thyroid hormone release, accompanied by a gradual decline [95] This is a prospective study done in rural india to find the incidence of non-thyroidal illness syndrome in patients with acute organophosphorus compound toxicity on individuals coming to R. L. Jalappa Hospital and Research Centre with history of ingestion, inhalation or cutaneous absorption of organophosphorus compound within 24 hours. The overall result comprised 60 participants with an age range of 35.48±11.81 years. The percentage of sick euthyroid in study was 21.67% and the mortality rate was 16.67%. The proportion of mortality between Sick Euthyroid and normal thyroid being numerically notable with p value 0.0044. Transient organophosphate toxicity, like every stressful situation, causes ill euthyroid syndrome in certain subjects. Sick euthyroid status gave a predictive validity in predicting the POP grade of poisoning a responsiveness of 35.29%, precision of 83.72%, positive predictive value 46.15%, negative predictive value 76.60% with a total diagnostic accuracy of 70.00%. Mortality rate is high among the sick euthyroid.

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ANNEXURES

PROFORMA

Particulars of the patients

NAME

AGE

SEX

OCCUPATION

LOCATION

HOSPITAL NUMBER

DATE AND TIME OF ADMISSION : __/__/20__ AT __:__ AM/PM

DATE OF DISCHARGE :__/__/20__

ADMISSION DIAGNOSIS

BRIEF HISTORY

AMOUNT INGESTED

DATE OF INGESTION

TIME OF INGESTION

TIME SINCE CONSUMPTION

SYMPTOMS ON PRESENTATION :

SYMPTOMATOLOGY

- □ Nausea,
- □ Vomiting
- □ Cough
- □ Burning sensation in the chest
- □ Headache
- □ Dizziness
- □ Weakness

- □ Abdominal Pain
- □ Diarrhea
- □ Tightness in chest
- □ Difficulty in breathing
- □ Salivation,
- □ Lacrimation
- □ Sweating
- □ Pupillary Changes
- □ Bradycardia
- □ Confusion
- □ Tremor
- □ Restlessness
- □ Respiratory depression
- □ Generalized weakness
- □ Cyanosis
- □ Peripheral circulatory failure
- □ Convulsions
- □ Coma

PRIOR TREATMENT :

- □ YES
- □ NO

PROVIDER:

SUPPORTIVE:

TREATMENT : PAM : _____ML/HR

ATROPINE : _____ ML/HR

TIME TAKEN TO REACH FIRST TREATMENT CENTRE:

PAST HISTORY :

- □ DIABETES MELLITUS
- □ HYPERTENSION
- □ CARDIOVASCULAR DISEASE
- □ RENAL DISORDER
- □ TUBERCULOSIS
- □ BRONCHIAL ASTHMA
- □ PSYCHIATRIC DISORDER
- □ THYROID DISORDER
- □ EPILEPSY
- □ LIVER DISORDER

PERSONAL HISTORY :

- □ DIET :
- □ APPETITE :
- □ SLEEP :
- □ HIGH RISK BEHAVIOUR :
- □ BOWEL AND BLADDER :
- □ HABITS :

PREVIOUS HISTORY OF POISONING [YES / NO] IF YES _DETAILS:

FAMILY HISTORY OF POISONING [YES/NO]

IF YES DETAILS:

INTENTION OF THE POISON :

□ SUICIDAL

□ ACCIDENTAL

□ HOMICIDAL

MODE OF EXPOSUR E :

□ INGESTION

□ INHALATION

□ CUTANEOUS ABSORPTION

GENERAL PHYSICAL EXAMINATION:

- Height:

_____cms

- Weight : ____ kgs
- BMI : _____kg / m2
- Pallor/Cyanosis/Icterus/Clubbing/edema/Generalized lymphadenopathy

- Thyroid examination :

VITAL DATA

- Pulse:
- Temperature:
- BP:
- Respiration rate:
- SpO2 : ____ @ RA _____

Systemic examination

- Per abdomen:
- Respiratory system:

VENTILATORY SUPPORT

- Cardio vascular system:
- Central nervous system

GCS

INVESTIGATIONS:

Routine:

CBC

- HB
- TC
- DC
- PCV
- PLATELETS

ESR

RFT

- UREA
- SERUM CREATININE
- NA+
- K+
- MG2+

RBS

LFT

- TOTAL BILIRUBIN

- DIRECT BILIRUBIN
- AST
- ALT
- ALP
- TOTAL PROTEIN
- GGT

SERUM PSEUDOCHOLINESTERASE LEVEL

THYROID PROFILE

- TOTAL T3
- TOTAL T4
- TSH

ECG

CHEST X-RAY

TREATMENT:

FINAL OUTCOME:

PATIENT INFORMATION SHEET

STUDY TITLE:"To study sick euthyroid state as a prognostic indicator in acute organophosphorus compound poisoning **"**

GUIDE:DR.RAVEESHA.A

STUDY CONDUCTED BY: DR.POONGULALI MDS

STUDY LOCATION: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

Organophosphorus compound poisoning is the most commmon poisoning in India because of its easy availability.There are approximately about 20-25 cases of Organophosphorus compound every month in our institute.
There are many than 3,00,000 deaths each year in developing countries .

All Patients diagnosed with Acute organophosphorus poisoning will be included in this study. Patients in this study will undergo routine investigations,cbc,rft,lft,serum magnesium and thyroid profile

Your participation is the study will help us to use the outcomes of this study for future subjects and will bring to limelight the importance and potentiate the clinical application od sick euthyroid state as a prognostic indicator in Acute organophosphorus compound poisoning .

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. This information collected will be used only for 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 thumb impression only if you voluntarily agree to participate in this study.

The purpose of the study is explained in detail to us and all information collected is for study purpose only. The data collected is submitted to the department of General Medicine, SDUMC, Kolar and confidentiality ensured .The merits and demerits explained briefly to us

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: TyG ಇಂಡೆಕ್ಸ್ ಒಂದು ಪ್ರೊಗ್ನೋಸ್ಟಿಕ್ ಆಗಿ ಕೋವಿಡ್ - 19 ಸೋಂಕು ಹೊಂದಿರುವ ರೋಗಿಗಳಲ್ಲಿ ಸೂಚಕ

ಅಧ್ಯಯನ ನಡೆಸಿದವರು: ಡಾ.ಪೂಂಗುಲಾಲಿ ಎಂಡಿಎಸ್
ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್ ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರವು ಶ್ರೀ ದೇವರಾಜರ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು, ತಮಕ, ಕೋಲಾರ.

ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯು ಭವಿಷ್ಯದ ವಿಷಯಗಳಿಗೆ ಈ ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಬಳಸಲು ನಮಗೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ ಮತ್ತು ಪ್ರಾಮುಖ್ಯತೆಯನ್ನು ಬೆಳಕಿಗೆ ತರುತ್ತದೆ ಮತ್ತು ಕೋವಿಡ್ 19 ಸೋಂಕಿನಲ್ಲಿ ಪೂರ್ವಭಾವಿ ಮಾರ್ಕರ್ ಆಗಿ TyG ಇಂಡೆಕ್ಸ್‌ನ ಕ್ಲಿನಿಕಲ್ ಅಪ್ಲಿಕೇಶನ್ ಅನ್ನು ಪ್ರಬಲಗೊಳಿಸುತ್ತದೆ.

ಕೋವಿಡ್ 19 ಸೋಂಕಿಗೆ ಒಳಗಾದ ಎಲ್ಲಾ ರೋಗಿಗಳನ್ನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ರೋಗಿಗಳು ವಾಡಿಕೆಯ ತನಿಖೆಗಳಿಗೆ ಒಳಗಾಗುತ್ತಾರೆ,cbc,rft,lft,fbs , ppbs , ಉಪವಾಸ ಟ್ರೈಗ್ಲಿಸರೈಡ್ ಮಟ್ಟಗಳು , ಮೂತ್ರ ದಿನಚರಿ

ದಯವಿಟ್ಟು ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದರೆ, ನಾವು ನಿಮ್ಮಿಂದ ಅಥವಾ ನಿಮ್ಮಿಂದ ಅಥವಾ ಇಬ್ಬರಿಗೂ ಜವಾಬ್ದಾರರಾಗಿರುವ ವ್ಯಕ್ತಿಯಿಂದ ಮಾಹಿತಿಯನ್ನು (ಪ್ರೊಫಾರ್ಮಾ ಪ್ರಕಾರ) ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. 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. ನಿಮಗೆ ಸಿಗುವ ಕಾಳಜಿ ಇರುತ್ತದೆ
ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸಿದರೆ ಮಾತ್ರ ನೀವು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಒದಗಿಸಬೇಕಾಗುತ್ತದೆ.
ಅಧ್ಯಯನದ ಉದ್ದೇಶವನ್ನು ನಮಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯು ಅಧ್ಯಯನ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ. ಸಂಗ್ರಹಿಸಿದ ಡೇಟಾವನ್ನು ಜನರಲ್ ಮೆಡಿಸಿನ್, ಎಸ್‌ಡಿಯುಎಂಸಿ, ಕೋಲಾರ ಇಲಾಖೆಗೆ ಸಲ್ಲಿಸಲಾಗಿದೆ ಮತ್ತು ಗೌಪ್ಯತೆಯನ್ನು ಖಾತ್ರಿಪಡಿಸಲಾಗಿದೆ. ಅರ್ಹತೆ ಮತ್ತು ದೋಷಗಳನ್ನು ನಮಗೆ ಸಂಕ್ಷಿಪ್ತವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ.

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

Title: **To study sick euthyroid state as a prognostic indicator in Acute Organophosphorus compound poisoning**

Principal investigator: Dr.Poongulali MDS

I, Mr./Mrs./Miss . …….. have been explained in my own understandable language,that I will be included in the above mentioned study ,being conducted in RL JALAPPA HOSPITAL.

I have been explained that my clinical findings,investigations,treatment and prognosis will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntaryand I can withdraw from the study any time and this will not affect my relation with my doctor or treatment for my ailment.

I have been explained about the risk/benefit of the study.

I understand that the medical information produced by this study will become part of institutional records and will be kept confidential by my said institute.

I agree not to restrict the use of any data or result that arise from this study provided such a use is only for scientific purpose(s).

I have principal investigator mobile number for enquiries.

I have been informed that standard of care will be maintained throughout the treatment period.

I in my sound mind give full consent to be added in the part of this study.

Investigator: Dr.Poongulali MDS

Phone number : 8095920050

Name	Signature	Date	Time
Patient:			
Witness:			
Primary Investigator/ Doctor:			

ತಿಳಿವಳಿಕೆ ಸಮ್ಮತಿ ನಮೂನೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: TyG ಇಂಡೆಕ್ಸ್ ಒಂದು ಪ್ರೊಗ್ನೋಸ್ಟಿಕ್ ಆಗಿ ಕೋವಿಡ್ - 19 ಸೋಂಕು ಹೊಂದಿರುವ ರೋಗಿಗಳಲ್ಲಿ ಸೂಚಕ

ಅಧ್ಯಯನ ನಡೆಸಿದವರು: ಡಾ.ಪೂಂಗುಲಾಲಿ ಎಂಡಿಎಸ್
ನಾನು, ಶ್ರೀ/ಶ್ರೀಮತಿ/ಮಿಸ್. …….. ನನ್ನದೇ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಅದು ಆರ್ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ನಡೆಸಲಾಗುತ್ತಿರುವ ಮೇಲೆ ತಿಳಿಸಿದ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಿಕೊಳ್ಳಲಾಗುವುದು.

ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಸಂಶೋಧನೆಗಳು, ತನಿಖೆಗಳು, ಚಿಕಿತ್ಸೆ ಮತ್ತು ಮುನ್ನರಿವುಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುತ್ತದೆ ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಮತ್ತು ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ನನ್ನ ಕಾಯಿಲೆಯ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದ ಅಪಾಯ/ಪ್ರಯೋಜನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಿಂದ ಉತ್ಪತ್ತಿಯಾಗುವ ವೈದ್ಯಕೀಯ ಮಾಹಿತಿಯು ಸಾಂಸ್ಥಿಕ ದಾಖಲೆಗಳ ಭಾಗವಾಗುತ್ತದೆ ಮತ್ತು ನಾನು ಹೇಳಿದ ಸಂಸ್ಥೆಯು ಗೌಪ್ಯವಾಗಿಡುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಈ ಅಧ್ಯಯನದಿಂದ ಉಂಟಾಗುವ ಯಾವುದೇ ಡೇಟಾ ಅಥವಾ ಫಲಿತಾಂಶದ ಬಳಕೆಯನ್ನು ನಿರ್ಬಂಧಿಸದಿರಲು ನಾನು ಸಮ್ಮತಿಸುತ್ತೇನೆ, ಅಂತಹ ಬಳಕೆಯನ್ನು ಕೇವಲ ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶ(ಗಳಿಗೆ)

ವಿಚಾರಣೆಗಾಗಿ ನಾನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಮೊಬೈಲ್ ಸಂಖ್ಯೆಯನ್ನು ಹೊಂದಿದ್ದೇನೆ.

ಚಿಕಿತ್ಸೆಯ ಅವಧಿಯುದ್ದಕ್ಕೂ ಆರೈಕೆಯ ಗುಣಮಟ್ಟವನ್ನು ನಿರ್ವಹಿಸಲಾಗುವುದು ಎಂದು ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ನನ್ನ ಉತ್ತಮ ಮನಸ್ಸಿನಲ್ಲಿ ನಾನು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ.

ತನಿಖಾಧಿಕಾರಿ: ಡಾ.ಪೂಂಗುಲಲಿ ಎಂಡಿಎಸ್
ದೂರವಾಣಿ ಸಂಖ್ಯೆ : 8095920050

Mastersheet:

Sr.No	Age	Gender	Day 1 T3 (ng/mL)	Day 1 T4 (µg/dL)	Day 1 TSH (ul/mL)	Day 7 T3 (ng/mL)	Day 7 T4 (ng/mL)	Day 7 TSH (ul/mL)	Magnitude of Sick Euthyroid	Serum Magnesium (mg/dL)	Peradeniya Organophosphorus Poisoning Scale (POP)	Mechanical ventilation	High dependency unit stay stay	ICU stay	Recovered	No. of days in hospital	Outside treatment	Compound	Pulse	SBP	DBP	GCS out of 15	Pupils	Neck lift
1	40	1	2	1	1	2	1	1	1	2	2	1	1	1	2	8	3	Chlorpyrifos + Cypermethrin	114	120	70	15	1	2
2	30	1	1	1	1	1	1	1	2	1	1	2	1	2	1	7	2	Fenthion	118	112	80	15	2	2
3	36	1	1	1	1	1	1	1	2	1	1	2	1	1	1	11	3	Phorate	75	90	60	3	2	2
4	43	1	1	1	1	1	1	1	2	1	1	2	1	2	1	8	4	Profenos + Cypermethrin	85	100	60	15	1	1
5	35	1	2	1	1	2	1	1	1	2	2	1	1	1	2	8	6	Chlorpyrifos + Cypermethrin	84	120	80	15	1	2
6	42	1	2	1	1	2	1	1	1	2	2	1	1	1	2	11	2	Dichlorvas	64	120	80	15	1	2
7	35	1	1	1	1	1	1	1	2	1	2	2	1	1	1	8	3	Malathion	110	110	60	15	2	2
8	50	1	1	1	1	1	1	1	2	1	1	2	1	2	1	8	6	Unknown	90	110	70	15	1	2
9	55	1	1	1	1	1	1	1	2	1	1	2	1	2	1	9	6	Chlorpyriphos	100	104	60	15	1	1
10	25	1	1	1	1	2	1	1	1	1	1	2	1	1	1	9	6	Triazophos	98	90	60	15	4	2
11	45	2	2	1	1	2	1	1	1	2	1	2	1	2	1	9	6	Dichlorvas	86	100	60	13	1	1
12	24	2	1	2	1	1	1	1	2	2	1	2	1	2	1	9	6	Malathion	87	110	60	15	1	1
13	41	1	1	1	1	1	1	1	2	1	1	2	1	1	1	7	6	Phorate	87	100	60	13	1	2
14	26	2	1	1	1	2	1	1	1	1	1	2	1	2	1	8	6	Dichlorvas	86	110	70	13	1	1
15	29	2	2	1	1	1	1	1	2	2	2	1	1	1	1	7	3	Parathion	88	148	90	15	1	1
16	21	2	1	1	1	1	1	1	2	1	2	1	2	1	2	9	6	Chlorpyrifos	123	110	70	12	1	2
17	35	1	1	1	1	1	1	1	2	2	1	2	1	2	1	8	6	Diazion	65	110	90	15	1	1
18	38	1	1	1	1	1	1	1	2	1	2	2	1	1	1	9	5	Phorate	114	100	60	15	1	1
19	20	1	2	2	2	2	1	2	1	2	2	1	2	1	2	7	1	Malathion	65	110	70	6	1	2
20	19	2	1	1	1	1	1	1	2	1	2	1	1	1	1	9	6	Chlorpyrifos + Cypermethrin	123	110	70	15	1	1

21	18	1	1	1	1	1	1	1	2	2	1	1	1	1	1	9	2	Dichlorvas	88	120	70	15	2	1
22	30	1	1	1	1	1	1	1	2	2	1	2	1	2	1	8	4	Phorate	88	150	100	3	5	2
23	50	2	1	1	1	1	1	1	2	1	2	1	1	1	1	8	5	Chlorpyrifos + Cypermethrin	78	120	70	15	1	1
24	20	1	1	1	1	1	1	1	2	1	1	2	1	1	1	12	6	Malathion	124	100	60	15	1	1
25	30	1	1	1	1	1	1	1	2	1	1	2	1	1	1	10	5	Parathion	113	130	80	15	1	1
26	25	1	1	1	1	1	1	1	2	1	1	2	1	2	1	8	3	Profenofos	67	100	60	15	1	1
27	25	1	1	1	1	1	1	1	2	1	1	1	1	1	1	8	6	Phorate	77	130	80	3	2	2
28	48	1	2	1	1	2	1	1	1	2	2	1	1	1	2	12	3	Fenthion	64	120	70	15	1	2
29	20	1	1	1	1	1	1	1	2	1	1	1	1	1	1	7	6	Phorate	124	130	70	15	1	1
30	25	1	1	1	1	1	1	1	2	1	1	2	1	2	1	7	6	Monochlorophos	88	130	80	13	2	1
31	45	2	1	1	1	1	1	1	2	1	1	2	1	1	1	9	2	Chlorpyriphos	78	100	60	15	1	1
32	28	2	2	1	2	1	1	1	2	2	2	1	2	1	2	9	2	Fenthion	78	110	70	15	1	2
33	48	1	1	1	1	1	1	1	2	2	1	2	1	1	1	9	3	Dichlorvas	65	130	80	15	1	1
34	40	1	1	1	1	1	1	1	2	1	1	1	1	1	1	10	6	Phorate	88	130	80	15	1	2
35	18	1	1	1	1	1	1	1	2	1	1	1	1	1	1	8	4	Chlorpyriphos	88	110	60	15	2	1
36	26	1	1	1	1	1	1	1	2	1	2	1	1	1	2	9	3	Chlorpyrifos + Cypermethrin	114	100	60	15	1	1
37	38	1	1	1	1	1	1	1	2	1	1	2	1	2	1	8	3	Quinolphos	88	120	70	15	2	1
38	50	1	1	1	1	1	1	1	2	1	1	2	1	2	1	9	3	Chlorpyrifos + Cypermethrin	126	130	80	15	1	1
39	22	2	2	1	2	2	1	2	1	1	2	1	1	1	2	8	5	Parathion	123	100	70	15	1	2
40	60	1	1	2	1	1	2	1	1	1	1	2	1	2	1	8	2	Chlorpyrifos + Cypermethrin	113	120	70	15	1	1
41	25	2	1	1	1	1	1	1	2	1	2	1	1	1	1	7	6	Phorate	118	108	60	6	1	2
42	30	1	1	1	1	1	1	1	2	1	2	1	1	1	2	10	4	Malathion	60	110	70	15	4	2
43	65	1	1	1	1	1	1	1	2	1	1	2	1	2	1	12	2	Chlorpyriphos	60	130	80	15	1	1
44	45	1	1	1	1	1	1	1	2	1	1	1	1	1	1	9	6	Dichlorvas	77	100	60	15	1	1
45	27	2	2	1	1	2	1	1	1	1	1	2	1	2	1	15	6	Chlorpyrifos + Cypermethrin	108	120	70	15	1	2
46	34	2	1	1	1	1	1	1	2	2	1	2	1	1	1	7	6	Chlorpyrifos + Cypermethrin	100	90	60	15	1	1
47	28	1	1	1	1	1	1	1	2	1	1	1	1	1	1	12	6	Chlorpyrifos + Cypermethrin	126	110	60	13	3	2
48	51	1	2	1	1	2	1	1	1	1	1	2	1	2	1	10	6	Profenos + Cypermethrin	170	150	100	13	1	2
49	26	1	1	1	1	1	1	1	2	2	2	1	1	1	1	9	6	Phorate	66	120	60	15	1	1
50	24	1	1	1	1	1	1	1	2	1	1	2	1	2	1	9	4	Dichlorvas	100	160	80	15	1	1

51	46	1	1	1	1	1	1	1	2	1	1	2	1	2	1	7	6	Phorate	132	120	70	14	4	1
52	38	1	1	1	1	1	1	1	2	1	1	2	1	2	1	8	6	Chlorpyriphos	76	110	70	15	1	1
53	37	1	1	1	1	1	1	1	2	1	1	2	1	2	1	9	6	Chlorpyrifos + Cypermethrin	76	120	70	15	1	1
54	52	1	1	1	1	1	1	1	2	1	1	2	1	1	1	11	6	Chlorpyrifos + Cypermethrin	76	120	80	15	1	2
55	45	2	1	1	1	1	1	1	2	1	1	2	1	1	1	9	6	Chlorpyiphos	114	120	70	15	4	1
56	28	2	1	1	1	1	1	1	2	1	1	2	1	2	1	8	6	Malathion	90	110	60	15	3	2
57	49	1	2	1	1	2	1	1	1	1	1	2	1	2	1	9	5	Dichlorvas	109	140	90	14	1	2
58	56	2	1	1	1	1	1	1	2	1	1	2	1	2	1	8	6	Chlorpyriphos	124	110	70	15	1	1
59	43	2	1	1	1	1	1	1	2	1	1	2	1	1	1	7	6	Fenthion	124	120	70	15	1	1
60	25	1	1	1	1	1	1	1	2	1	1	2	1	2	1	10	4	Chlorpyrifos + Cypermethrin	124	124	80	3	1	2

Sr.No	Seizures	Fasciculations	Secretions	Cardiovascular system	Respiratory system	Central nervous system	Per abdomen	Pseudo cholinesterase (U/L)	Urea (mg/dL)	Creatinine (mg/dL)	Sodium mEq/L	Potassium mEq/L	Tb (mg/dL)	AST (U/L)	ALT (U/L)
1	2	1	1	1	1	Drowsy	1	370	1	1	2	1	1.2	23	20
2	2	1	1	1	2	Drowsy	1	800	1	1	2	2	0.3	83	17
3	1	2	1	1	2	Drowsy	1	200	1	1	2	1	0.9	82	21
4	2	2	2	1	1	Nfnd	1	456	1	1	2	2	0.8	26	16
5	2	1	1	1	1	Drowsy	1	200	1	1	1	2	1.2	34	28
6	2	1	1	1	2	Drowsy	1	200	1	1	1	2	0.8	28	24
7	1	1	1	1	2	Fasciculations	1	480	1	1	1	2	0.7	26	15
8	2	2	1	1	1	Nfnd	1	690	1	1	2	2	0.9	23	13
9	2	2	2	1	1	Nfnd	1	200	1	1	1	1	1	24	14
10	2	1	1	1	1	Atropinized	1	240	1	1	2	2	0.9	22	17

11	2	2	2	1	1	Nfnd	1	890	1	1	2	1	0.8	60	40
12	1	1	2	1	2	Nfnd	1	650	1	1	2	1	1	34	14
13	2	2	2	1	1	Nfnd	1	200	1	1	1	1	0.9	67	47
14	2	1	2	1	1	Nfnd	1	205	1	1	1	1	0.8	29	15
15	1	1	2	1	1	Nfnd	1	200	1	1	2	2	0.8	35	21
16	1	1	1	1	2	Drowsy	1	406	1	1	2	2	0.7	34	32
17	2	2	2	1	1	Nfnd	1	250	1	1	2	2	1	106	68
18	2	2	2	1	1	Nfnd	1	259	1	1	2	2	0.9	48	40
19	2	2	1	1	2	Drowsy	1	200	1	1	2	1	0.8	26	18
20	2	2	2	1	1	Nfnd	1	210	1	1	2	1	0.8	30	26
21	2	1	2	1	1	Nfnd	1	200	1	1	2	2	0.7	24	15
22	2	2	1	1	2	Drowsy	1	200	1	1	1	2	0.9	32	17
23	2	2	2	1	1	Nfnd	1	234	1	1	2	2	0.7	32	24
24	2	2	2	1	1	Atropinized	1	200	1	1	2	1	0.8	30	26
25	2	1	2	1	1	Nfnd	1	240	1	1	1	2	0.9	28	24
26	2	2	2	1	1	Nfnd	1	340	1	2	1	2	1	32	30
27	2	2	1	1	2	Unresponsive	1	200	1	1	2	2	0.6	30	17
28	2	1	1	1	1	Drowsy	1	337	1	2	2	1	0.8	26	16
29	2	2	2	1	1	Nfnd	1	960	1	1	2	2	0.8	28	16
30	2	2	2	1	1	Nfnd	1	230	1	1	2	2	1.1	26	18
31	2	1	2	1	1	Nfnd	1	240	1	1	2	2	1.2	24	20
32	2	2	1	1	1	Drowsy	1	450	1	1	2	2	0.7	26	15
33	2	1	2	1	1	Nfnd	1	228	1	1	1	2	1.1	34	24
34	2	2	2	1	1	Nfnd	1	200	1	1	1	2	0.8	32	18
35	2	1	1	1	1	Nfnd	1	200	1	1	2	2	0.6	32	28
36	2	2	2	1	1	Nfnd	1	440	1	1	2	1	0.8	30	28
37	2	2	2	1	1	Nfnd	1	241	1	1	2	1	0.7	34	30
38	2	1	1	1	1	Nfnd	1	793	1	1	2	1	1	34	26
39	2	1	1	1	1	Nfnd	1	200	1	1	1	1	0.9	26	16
40	2	1	1	1	1	Nfnd	1	200	1	1	1	1	0.6	25	15

41	2	2	1	1	2	Unresponsive	1	200	1	1	1	2	1.1	43	36
42	1	1	1	1	2	Drowsy	1	240	1	1	1	2	0.9	40	38
43	2	2	2	1	1	Nfnd	1	200	1	1	2	2	0.8	28	18
44	2	1	2	1	1	Nfnd	1	200	1	1	1	2	0.9	32	28
45	2	2	1	1	1	Drowsy	1	515	1	1	1	2	1.1	34	28
46	2	1	2	1	1	Nfnd	1	200	1	1	1	2	0.9	36	24
47	1	1	2	1	1	Restless, Fasciculations	1	200	1	1	2	1	0.9	30	28
48	2	2	1	1	1	b/l Flaccid paralysis	1	240	1	1	2	2	0.6	30	28
49	2	1	2	1	1	Nfnd	1	200	1	1	2	2	1	26	25
50	2	1	2	1	1	Nfnd	1	240	1	1	1	1	0.9	24	20
51	2	1	2	1	1	Nfnd	1	200	1	1	2	2	0.7	40	36
52	2	2	2	1	1	Drowsy	1	200	1	1	2	2	0.8	34	30
53	2	1	2	1	1	Iritable	1	660	1	1	2	2	1.2	28	25
54	1	1	1	1	2	Delirious	1	200	1	1	1	1	0.6	42	17
55	2	2	2	1	1	Nfnd	1	260	1	1	2	1	1	46	24
56	2	2	1	1	2	Drowsy	1	200	1	1	1	2	0.8	28	18
57	2	1	1	1	1	Drowsy	1	918	1	1	2	2	0.9	26	20
58	2	1	2	1	1	Nfnd	1	870	1	1	2	2	0.9	42	36
59	2	2	1	1	1	Nfnd	1	660	1	1	1	1	1	28	26
60	2	2	2	1	2	Drowsy	1	200	1	1	1	2	1.1	40	30

KEY OF THE MASTER CHART:

Variable Name	
Gender	1=Male, 2=Female
Day 1 T3 (ng/mL)	1=Normal, 2=Abnormal
Day 1 T4 (µg/dL)	1=Normal, 2=Abnormal
Day 1 TSH (uI/mL)	1=Normal, 2=Abnormal
Day 7 T3 (ng/mL)	1=Normal, 2=Abnormal
Day 7 T4 (µg/dL)	1=Normal, 2=Abnormal
Day 7 TSH (uI/mL)	1=Normal, 2=Abnormal
Magnitude of Sick Euthyroid	1=Sick Euthyroid, 2=Normal Thyroid
Serum Magnesium (mg/dL)	1=Normal, 2=Abnormal
Peradeniya Organophosphorus Poisoning Scale (POP)	1=Moderate, 2=Severe
Mechanical ventilation	1=Yes, 2=No
High dependency unit stay stay	1=Yes, 2=No
ICU stay	1=Yes, 2=No
Recovered	1=Recovered, 2=Death
Outside treatment	1=1 Day treatment with Atropine, 2=Atropine, 3=Stomach wash, 4=Stomach wash + Atropine, 5=Stomach wash + Atropine + Pam, 6=Nil
Pupils	1=<= 3mm, 2=> 3mm, 3=Dilated, 4=Constricted, 5=Pinpoint
Neck lift	1=Good, 2=Poor
Seizures	1=Yes, 2=No
Fasciculations	1=Yes, 2=No
Secretions	1=Present, 2=Absent
Cardiovascular system	1=s1 s2 normal
Respiratory system	1=b/l nvbs,2=Crepts
Per abdomen	1=Soft
Urea (mg/dL)	1=Normal
Creatinine (mg/dL)	1=Normal, 2=Abnormal
Sodium mEq/L	1=Normal, 2=Abnormal
Potassium mEq/L	1=Normal, 2=Abnormal