SERUM CREATINE PHOSPHOKINASE LEVEL AS A SEVERITY MARKER IN ACUTE ORGANOPHOSPHATE POISONING- A CROSS SECTIONAL STUDY

By:

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In partial fulfillment of the requirement for the degree of

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IN

GENERAL MEDICINE

Under The Guidance Of

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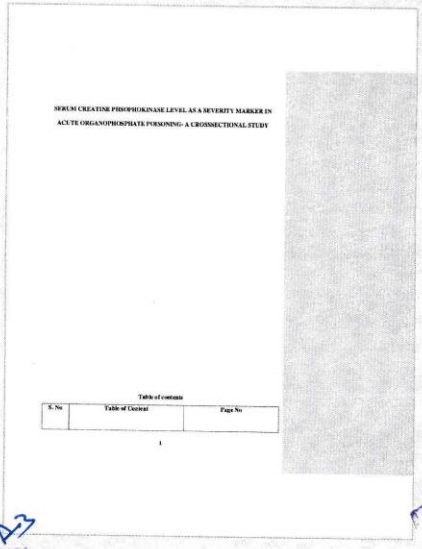
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Glossary	Abbreviations
ACh	Acetylcholine
AChE	Acetylcholinesterase
AMI	
CPK	Creatine Kinase Phosphokinase
EchE	Erythrocyte Cholinesterase
IMS	Intermediary Syndrome
IMS	Intermediary Syndrome
LDH	Lactate Dehydrogenase
NTE	Neuropathic Target Esterase
O	Oxygen
OP	Organophosphorus
OPIDN	OP-Induced Delay Neuropathy
OPIDN	Organophosphate Pesticides
POP	Peradeniya Organophosphorous
POPS	Peradeniya Organophosphorus Poisoning Scale
REM	Rapid Eye Movements
SAPS	Simplified Acute Physiology Score
SPECT	Solitary Emission Computed Tomography
UAPP	Unintentional, Acute Pesticide Poisoning

ABSTRACT

Background: When a patient has acute organophosphorus (OP) poisoning, the pseudo cholinesterase levels are often checked since they are anticipated to decline. The creatine kinase phosphokinase (CPK) levels are raised in acute poisoning, and new, less expensive biochemical indicators for OP poisoning are required. The current study aims to evaluate and connect the blood CPK level with the Peradeniya Organophosphorous Scale-determined severity of OP poisoning (POP).

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Conclusions: The difference amongst serum CPK at admittance (mcg/l) and pseudo cholinergic (U/L) is scientifically substantial with a p value of 0.001, and there was a low negative correlation amongst serum CPK at admittance (mcg/l) and false cholinesterase

(U/L), with a r value of -0.040. The risk of death was increased by a greater POP score, a significant decrease in serum cholinergic levels, and an increase in serum CPK levels.

Keywords: acute organophosphorus poisoning, acetylcholine, serum creatinine phosphokinase, CPK, mortality, Peradeniya Organophosphorous Scale (POP).

INTRODUCTION

INTRODUCTION:

A wide range of chemicals made from phosphoric, phosphonic, and phosphinic acids are referred to as "organophosphorus" or "OP."[1] OP amalgams have been used as insecticides and are being studied for use as nerve agents in chemical warfare. Through skin contact, ingestion, and inhalation, they are easily absorbed.[2] Worldwide, OP chemicals are used extensively in domestic gardens and agriculture. There has been a marked rise in poisoning with these chemicals as a result of their ease of access and over-the-counter sales.[3] A serious global health concern, OP chemical poisoning is perhaps the most common kind of acute in developing countries. There have been generated more than 100 different OP compounds. The trio malathion, fenthion, dimethoate, chlorpyrifos, diazinon, and paraoxon and soman are the most well-known.[4]

Organophosphate exposure is expected to affect 3 million or more individuals annually, killing about 300,000. In the US, there are about 8000 exposures each year with extremely few fatalities. The majority of interaction is with agricultural chemicals, however certain household products, such ant and roach spray, contain organophosphate compounds.[5] Agrochemical poisoning (94.5%) prevailed in India when compared to several other categories of toxins, according to a comprehensive evaluation of the literature. Aluminum phosphide remained the most prominent fatal toxin from 2001, to 2010, before actually falling dramatically. Throughout the historical period, OP insecticides were crucial, but over the last ten years, as the prevalence of aluminium phosphide has diminished, they have become prevalent.[6]

The three principal symptoms resulting from OP poisoning are "acute cholinergic syndrome", intermediary syndrome (IMS), then OP-induced delay neuropathy (OPIDN).[7,8] IMS begins

48-96 hours after ingesting the OP chemical and recovering from the acute cholinergic crisis. It is distinguished by skeletal muscular weakness.[9] Respiratory paralysis in IMS, if detected in time, can minimize the requirement for ventilator assistance and allow proper therapy to begin as soon as possible.[10] Monitoring those who are at a significant risk for IMS may thus result in a decrease in morbidity and mortality. Acetylcholinesterase (AChE), an enzyme involved in the breakdown of acetylcholine (ACh) at synapses, is permanently inhibited by OPs, which reduces the function of the cholinergic nervous system. ACh builds up at synapses in the peripheral and central and neural systems as a result of OPs' inhibition of AChE, and cholinergic receptors are activated more than necessary to maintain homeostatic tolerances.[11] Inducing cholinergic neuronal excitability and instability, which are significant factors to the cholinergic urgent situation in the acute stage of OP pressures (in min), acute, raised activation of neurotransmitter acetylcholine (chiefly the muscarinic binding site, mAChR, in the brain), may result in minor neuronal injury and long-term neurobehavioral effects.[12,13]

The reductions in cholinesterase activity are frequently used to corroborate laboratory evidence of OP poisoning. However, a substantial cholinesterase activity drop might emerge due to large inter-individual variability while remaining within the "normal" range.[14] This then defies specificity and contains no link to the severity of the poisoning, therefore it is not suitable for expecting the prognosis. In the case of OP poisoning, new options for affordable and/or more easily measurable biochemical indicators are emerging. Creatine phosphokinase (CPK), lactate dehydrogenase (LDH), amylase, and lipase are examples.[14] A rise in total serum CPK levels can be used instead of cholinesterase levels to determine the severity of OP poisoning. Presences of muscle fiber necrosis in OP poison leads to increase in CPK level. In addition to this there is Rhabdomyolysis in intermediate syndrome leads to increase

in CPK level.[15] Estimating CPK is simple, because levels rise throughout both the acute phase and the intermediate condition as an outcome of the premature death of muscle fibres. According to several reports, the greatest and most accurate sign of muscle injury is high blood CPK levels, which show the severity of acute muscular necrosis.[16,17]

There are many severity rating methods for acute organophosphorus poisoning. Senanayake N put forward the "Peradeniya Organophosphorus Poisoning" (POP) scale to grade the severity of organophosphorus poisoning, which is based on 5 basic manifestations of OP: pupillary contraction, heart rate, fasciculations, respirational rate, and level of awareness. Each indication is assigned a severity score, which is then total up to determine the severity on a scale of 1 to 11. A score of 0-3 indicates mild poisoning, 4-7 indicates moderate poisoning, and 8-11 indicates severe poisoning.[18] A single-center observational investigation on OP poisoned subjects discovered that POP score, SChE, and blood CPK levels on admission were valid indicators of prognosis and survival, with serum CPK levels being a cheaper and most effective indiactors.[19] Another study discovered that high blood CPK levels were connected with a heightened incidence of intermediate syndrome and fatality, and so may be used to determine the severity of poisoning and early detection, such as setting on mechanical breathing, and minimizing death.[20] A prospective observational research conducted at a hospital revealed a numerically consequential definite connection between early CPK levels and the sternness of OP exterminating (as measured by the POP scale) on the day of admission.[21]

Need for the study

Poisoning with organophosphorus (OP) may result in life-threatening circumstances that lead to respiratory failure. Because it is so readily available, in India, OP chemicals are routinely

used as insecticides and, more frequently, for suicide intentions. To patients and critical care providers, early detection and effective response to toxic effect from these drugs is vital. OP substance functions by blocking AchE at muscarinic and nicotinic receptors. The levels of EchE and PchE drop in OP poison. However, their estimate is expensive. Increase the total S. Cholinesterase levels can be substituted with CPK levels to determine the severity of OP toxicity. The presence of muscle fibre necrosis in OP toxin causes an increase in CPK levels. Because of its low cost and ease of use, serum CPK levels can be a viable biomarker in instances of acute OP poisoning, as well as the fact that its value may be predicted by serial monitoring during the therapy process. The current study aims to assess blood plasma CPK levels in acute severe organophosphorus poisoning and associated with the severity of OP poisoning as evaluated by the POP scale.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES:

Objectives of the study:

- Determine the Blood Plasma Creatinine Phosphokinase [CPK] levels in the acute severe organophosphate intoxication.
- To correlate the serum creatinine phosphokinase [CPK] level with severity of organophosphate determined by the Peradeniya Organophosphorous Scale [POP].

REVIEW OF

LITERATURE

REVIEW OF LITERATURE

Organophosphorus poisoning

Organophosphates (OP) are chemical compounds formed by the esterification of phosphoric acid and alcohol. Ethanol from the ester group can be released when organophosphates are degraded. The main constituents in fertilizers, insecticides, and herbicides are those. Additionally, the primary components of poison gas are OPs. Organophosphate exposure, whether acute or chronic, can have varied levels of harmful effects on people, animals, plants, and insects. Numerous solvents and polymers are produced using organophosphates.[22]

Organophosphorus pesticides are probably among the most frequent causes of illness and death from poisoning globally, especially in emerging countries like India, due to their accessibility of availability. The amount of time between exposed and the start of care affects morbidity and mortality. As a result, it is critical to detect the full range of symptoms. Organophosphorus (OP) chemicals block the enzymes acetyl cholinesterase and butyryl cholinesterase, causing overactivity of cholinergic synapses.[23] Over 100 distinct OP chemicals have been produced. Malathion, fenthion, parathion, dimethoate, diazinon, chlorpyrifos, soman, and paraoxon, are the most prominent.[4]

Annually, many subjects die because of OP chemical poisoning. More than 90% of fatal poisoning cases, according to WHO estimates, occur in, developing nations in over-all and agricultural nations in particular. In India, the estimated death rate from OP poisoning is 7-12%. [24,25]

The frequency of unintentional, acute pesticide poisoning (UAPP), which was thoroughly studied, was anticipated to occur 740,000 times annually, with 7446 fatalities and 733,921

quasi cases. UAPP is thought to cause about 385 million cases and over 11,000 fatalities annually throughout the world. Based on an estimated 860 million farmers worldwide, this translates to almost 44% of agriculture being killed by pesticides every year. South-eastern Asia, east Africa, and southern Asia had the largest expected numbers of non-fatal UAPP cases, respectively.[26] A Indian government record[27] indicated around 6500 deaths, many of which were most likely caused by workplace exposure, but India did not upload this data to the WHO Mortality Database, nor did the government mention the number of occupational poisonings in their report.

Chemical structure

Organophosphorus insecticides are a chemical family that is similar but varies in structure. "They are often ester, amide, or thiol derivatives of phosphoric or phosphonic acids".

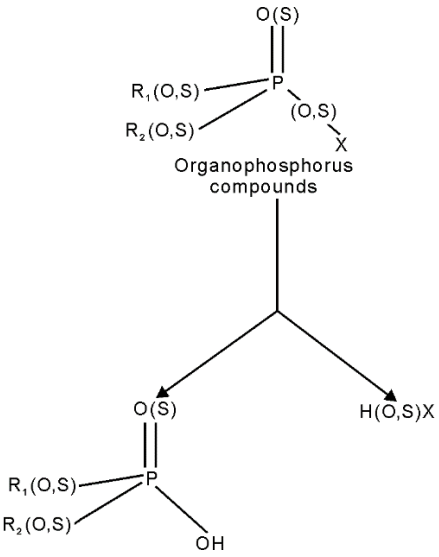


Figure 1: General formula of organophosphate pesticides and their biotransformation[28]

Phosphorus is coupled to oxygen (O) via a double bond in the basic structure, and R1 and R2 can be alkyl, alkoxy, aryloxy, amido, mercaptan, or other groups. The leaving group is represented by X, and a conjugate base of weak acid is identified as a halide, cyanide, thiocyanate, phenoxy, thiocholine, or carboxylate group. Because the P=S form is

fundamentally very firm, many insecticides are synthesised in that form, which may then be changed in vivo to the biologically active form oxon. [29,30]

Chemical structures and toxicities of organophosphorus insecticides differ. Phosphorate, phosphorothioate, O-alkyl phospho-rothioate, and phosphorodithioate are the major groups. Compared to phosphorodithioate compounds like Malathion, phosphor-thioate compounds like parathion are far more hazardous. Aside from OP insecticides and chemical warfare nerve agents, very few OP chemicals, notably glyphosate and merphos, were utilised as herbicides. Organophosphorus herbicides are structurally distinct from OP insecticides, and they have far less ability to block AChE than other OPs. [31]

Pharmacokinetics/dynamics

OP molecules can be absorbed by the skin, breath, or the digestive tract. The chemical binds to a cholinesterases substance in RBCs after ingestion, inactivating the enzyme. As a result, synapses and nerve terminals have an overabundance of acetylcholine. Neural stimulation of nicotinic receptor sites at neurons and muscle connections can result in fasciculations and myoclonic jerks. Depolarizing block eventually results in flaccid paralysis because of this. Nicotinic receptors can cause hypertension, perspiring, tachycardia, and left shifting leukocytosis since they are also found in the adrenal glands.[32, 33, 34, 35]

Because of their lipophilic nature, organophosphorus chemicals may easily pass respiratory epithelium and skin membranes and so form mostly as aerosol.[36, 37] Additionally, the stomach mucosa is relatively porous to Ops therefore is as a typical route for absorption in people who are suicidal.[38] Organophosphorus chemicals are found throughout the body, particularly in fatty tissues, and their rapid breakdown prevents their buildup. Certain OPs are removed without much metabolism. They are normally destroyed and removed in urine, faeces, and

breathed air. The majority of OP pesticides are triggered in the liver by enzymes of the cytochrome P450 family and monooxygenases that include flavin. Nerve poisons like soman, sarin, and others are active by nature. The main enzymatic systems involved in OP detoxing are antioxidant enzyme, carboxylesterases, and phosphotriesterases. Hydrolysis by esterases known as 'phosphotriesterases' is a major detoxifying mechanism for OPs (PTEs). Because the reaction's products have no phosphorylating capacity, the hydrolysis of OPs by PTEs is regarded a detoxification. Human serum paraoxonases are the most well-known PTEs.[39]

Phases, pathophysiology

It is common practice to utilise OP pesticides, which are irretrievable blockers of COO- ester hydrolases such AChE, plasma EChE, or BChE, and added indiscriminate proteases. The chief source of these chemicals' toxicity is the buildup of acetylcholine, which causes over-activation of nicotinic and muscarinic cholinergic receptors inside the central and parasympathetic nervous systems as well as in skeletal neuromuscular junctions.[40] Acetyl cholinesterase is hindered by organophosphorus chemicals. By engaging with the esteratic region on the esterase molecule, the enzyme is hampered. The connection that forms between the phosphate group and the creating appropriate site of the protein is sturdy and takes a while to weeks to break, based on the type of organophosphates molecule. The activity of phosphorylated enzyme is inhibited due to the presence of its active site. It is unable to carry out its usual function of hydrolytic acetyl choline. This phosphorylated enzyme can spontaneously hydrolyze or dealkylate. Reactivation occurs when active enzyme cholinesterase is released as a result of spontaneous hydrolysis. Phosphorylated enzymes can also be dealkylated. When this happens, recovery will be difficult. This is referred to as ageing.[41] Recovery of cholinesterase function after ageing is dependent on the production of a new enzyme by the liver, which might take days or weeks. These three distinct

mechanisms, namely phosphorylation of cholinesterase by organophosphorus chemicals, reactivation, and ageing, control the rate of occurrence and severity of poisoning.

Diagnosis

Organophosphorus compound toxicity can indeed be recognized by a history of by occupational or chemical warfare attack, purposeful or unintentional oral OP pesticide consumption, and clinical signs. Specific biomarkers of exposure are provided by the enzymes that are inhibited by OPs up until the enzyme turnover under advantageous circumstances. Red blood cells and plasma both contain accessible AChE and BChE.[42] Butyrylcholinesterase, while being less specific, is commonly used as an early indicator due to its higher prevalence and sensitivity than AChE. It is crucial to check the AChE levels in red blood cells in people exposed to these drugs.[43]

Some OS have a unique garlic or fuel odour, which may aid in identification. If OP poisoning is suspected but not established, an atropine trial may be used. If symptoms improve with atropine, acetylcholinesterase blocker toxicity is more likely.[5]

Progression, Complications, prognosis

There are two categories of clinical outcomes following OP exposure: acute and chronic symptoms. OPs can cause acute effects through ingestion, skin or eye contact, inhalation, or other routes, depending on the site of exposure. However, high exposure levels across all pathways have similar consequences**.**[44] More rapid development of symptoms are seen through ingestion and inhalation route than by skin exposure. After consumption, symptoms will develop within 30-90 minutes, with a maximum of 24 hours in the incidence of extremely lipophilic substances that necessitate metabolic biotransformation.[45]

Before the onset of systemic symptoms the GI symptoms appear first by ingestion route whereas inhalation typically exhibits respiratory effects. Three clinical phases are seen in systemic effects:

1) The first cholinergic phase
2) Intermediary syndrome (IMS)
3) Late polyneuropathy

In moderate to severe oral OP poisoning, vomiting, nausea, stomach discomfort, and diarrhoea, as well as cholinergic condition, CNS, and cardiovascular issues, might result.[46] Cholinergic common signs of OPs, which appear within the first few hours of exposure, are brought on by overactivity of Receptor sites (nicotinic and muscarinic).[43] Dizziness, nausea, vomiting, stomach discomfort, diarrhoea, miosis, impaired vision, salivation, urine, lacrimation and respiratory distress are all symptoms of muscarinic receptors. Bronchoconstriction and increased bronchial secretion have huge impacts on the respiratory system, resulting to lung failure, which is the most frequent cause of death.

Irritability, anxiousness, giddiness, ataxia and widespread weakness are all symptoms of central nervous system depression, as lethargy, memory loss, confusion, convulsions, coma, respiratory depression, metabolic alkalosis, hypoventilation, and hypotension.[47,48,49,50] Suppression of the brain's pulmonary and autonomic centers may occur as a result, escalating the clinical symptoms.[51] Nicotinic and main stimulation overshadow the majority of the neuromuscular junction effects at low to high OP doses. The most common causes of mortality are respiratory and cardiovascular failure.[52]

Following acute poisoning, there is an intermediate phase that lasts 1-4 days and is distinguished by distal muscle atrophy, pulmonary function limitation, and nerve palsy.[53,10] It

has been established that OP-induced myopathy and intermediate syndrome are related. Intermediate syndrome is caused by overextension of the cholinergic system at the neuromuscular junction. Studies from the 1990s show that intermediate syndrome is associated with both a significant fall in cholinesterase levels and the outflow of the metabolite of the cholinesterase inhibitor in the urine. It has been proven to occur with exposure to parathion as well as a variety of other pesticides containing dimethyl phosphate, including dimethoate, dichlorvos, fenthion and methylparathion.[10,54]

Laborers, mostly agricultural workers, who are exposed to OP chemicals on a daily basis run the risk of developing chronic poisoning. Organophosphate-induced delayed neuropathy can be brought on by several organophosphate pesticides (OPIDN). It is an identical sensory axonopathy that appears 7 to 14 days following revelation and is especially bad in long axons. Peripheral nerves contain more than 70% of the functional neuropathic target esterase (NTE), which is phosphorylated and gradually matures the nerves, causing organophosphate-induced delayed neuropathy to start. It is hypothesized that the process involves spinal cord ornithine decarboxylase insufficiency or NTE suppression.[55]

Repetitive revelation to OP and at least 4 of the preceding are screening test for chronic OPIND.; 1. (a) Mood swings and personality changes, (b) a lifelong decline in concentration, (c) a reduction in exercise tolerance, (d) a reduction in alcohol tolerance, and (e) an increase in vulnerability to organophosphate exposure; 3-at least 3 of the ones that follow: "Dipper's flu" getting worse, "spur of the moment" suicidal thoughts, "linguistic issue," "heightened sense of smell," and "degenerative handwriting" are some examples.[54]

Table 1: The assessment of clinical grading of organophosphate poisoning[56]

Grade	Symptoms	Signs
Mild	Dizziness, headache, anxiousness, and breathlessness	Rhinorrhea, perspiration, salivation, nausea, asthenia, coughing, tears, a moderate type of sluggish heartbeat, low blood pressure, and urges to vomit.
Moderate	Inability to fall asleep, disorientation, shortness of breath, agitation, nausea, vomiting, and sleepiness	respiratory stress, bronchorrhea, bronchoconstriction, muscular twitching, fasciculation, meiosis/ mydriasis*, bradycardia/ tachycardia, hypertension*, and loss of consciousness
severe		Involuntary urination/defecation, flaccid paralysis, convulsions, respiratory distress, pulmonary edema, and convulsions Cyanosis and a profound coma
Fatal		Within minutes of exposure, coma, convulsions, excessive secretions, and apnoea might occur.

* Miosis or mydriasis, tachycardia or bradycardia, hypertension, or hypertension may ensue depending on whether the cholinergic or nicotinic syndrome predominates. deterioration of the aforementioned traits plus the following; Severe: The aforementioned traits getting worse plus the following Neuropsychological consequences of poisoning include impaired memory and attentiveness, poor synthesis of information and psychomotor efficiency, memory deficit, linguistic problems, sorrow, anxiety, and a propensity to become enthusiastic, angry, or irritated easily. Those who had not been revealed to poison for a year had their brain electrical activity studied by Duffy et al. Significant deviations from the placebo group were seen, including elevated activity, increasing delta and slowing, decreased activity, and an increase in rapid eye movements (REM) sleep. After OP poisoning, solitary emission computed tomography of the brain (SPECT) revealed perfusion abnormalities, particularly in the parietal lobe.[57,58]

Extra pyramidal manifestations might occur after 4 – 40 days of OP poisoning. In those who survive the symptoms might disappear within 1-4 weeks. The features include dystonia, resting Unintentional shaking, cog-wheel stiffness, and the incidence of involuntary movement are frequently bilateral, however they can also be unilateral or asymmetrical in

some circumstances. The human extrapyramidal system, which is populated with cholinergic neurons and anticholinesterase, is known to have acetyl cholinesterase inhibitory activity, which has been connected to this phenomena. After exposure to pesticides, Glutathione Enzyme Polymorphisms may reduce a patient's body's capacity to detoxify insecticides and raise their risk of developing Parkinson's disease.[18,59]

Following exposure to a nerve agent and serious OP pesticide poisoning, breathing collapse brought on by a depressed respiratory center , paralyzed respirational muscles, and obstruction induced by bronchoconstriction and respiratory secretions is the main cause of death.[60] The global death rates from organophosphate insecticides range from 2 to 25%. The most common pesticides linked to mortality are dichlorvos, fenitrothion, trichlorfon and malathion, The major reason of mortality is breathing collapse.[5]

Markers for prediction of severity of acute OPC poisoning

Predicting prognostic factors would be helpful for doctors to standardize individuals based on their chance of progression because timely screening and therapy are frequently lifesaving measures. To determine the seriousness of OPC exterminating and forecast the prognosis of patients hospitalized with OPC poisoning, a variety of diagnostic and biochemical signs were used.

The Peradeniya Organophosphorus Poisoning Scale was one of several programs (POPS).[61] This is a single indicator that incorporates five indicators of OP poisoning (respiratory rate , pulse rate, fasciculations, level of awareness, pupil size, and confiscation activity). Each variable is given a rating between 0 and 2 during the initial presentation.[18]

The decrease in pseudo choline esterase is correlated with the level of poisoning in acute OP poisoning. Despite the availability of various assessment techniques like the “Simplified Acute Physiology Score” (SAPS) and the “Acute Physiology and Chronic Health Evaluation” (APACHE), biochemical analysis is crucial for establishing intoxication, locating the first signs of acute organ damage, and determining the level of intoxication. Plasma cholinesterase estimation is the method that works well for overdose exposure to OP intoxication in a lab setting.[62]

The most common of these biochemical changes, higher blood amylase levels, are linked to OP compound poisoning and may be brought on by the pancreas' improper cholinergic activity. In their study, Matsumiya et al. evaluated the predictive value of serum amylase in cases of OP poisoning and discovered that the beginning of respiratory failure was correlated with an increase in plasma amylase levels above the range of normal on the day of admission. They reached the conclusion that a rise in amylase levels signaled impending respiratory failure.[63] Increased serum amylase is much less specific and sensitive, and acute pancreatitis is typically associated with OP poisoning. In those with rising amounts of amylase, serum lipase tests may be useful for the early detection of pancreatitis.[64]

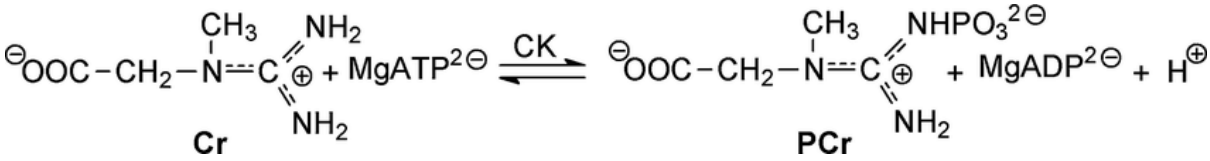
Bhattacharya et al study’s found that monitoring the patient prognosis in acute OP poisoning may be accomplished by detecting the creatine phosphokinase (CPK) level. Additionally, it has been shown that the "intermediate syndrome" results in the disintegration of skeletal muscle, which is trailed by a proportional rise in amylase levels.[65] Because it is readily available and affordable, serum CPK level might be a helpful diagnostic in the case of severe OP intoxication. Additionally, serial surveillance of its levels during the course of therapy may be capable of predicting the result. However, this marker's quasi is its principal

drawback.[66] In individuals with intermediate syndrome, serum CPK levels are still increased, which is indicative of premature muscle fiber loss, as shown by muscle biopsy. The CPK level remains increased if there is enduring muscle injury as a result of problems that first appear. CPK has a ½ of around 1.5 days, but after a single muscle damage, it recovers to normal in 5–6 days.[67]

The quantity of creatine phosphokinase in the blood as a measure of severity in acute organophosphate poisoning

"creatine phosphokinase, creatine phosphotransferase, Adenosine-5-triphosphate, phosphocreatine phosphotransferase, and creatine N-phosphotransferase" are all names for the enzyme creatine kinase (EC 2.7.3.2), which catalyzes the consequences that effect of a phosphoryl group from magnesium ATP to creatine (Cr), resulting in phosphocreatine.[68] It belongs to the guanidino kinase phospahgen kinase family (ATP-guanidino-phosphotransferases).[69]

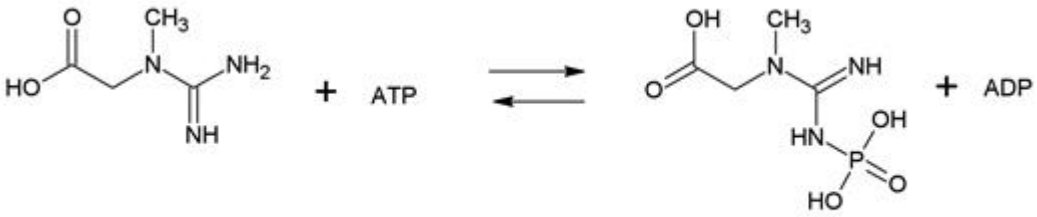
Figure 2: Reaction catalyzed by creatine kinase[68]



The four major CK isozymes were originally discovered for the tissue from which they were collected and isolated on an agar plate by centrifugation in 196371. Other two are mitochondrial, while two are cytoplasmic matrix. under physiological conditions.[70,71] The tertiary structure and highly conserved sequence of the cytoplasmic CKs (MMCK and BBCK) are substantially similar.[72] In solution, they unite to form a dimer with two domains: a larger C-terminal region with both b-sheets and a-helix structural properties, and a shorter N-terminal domain with just a-helices.

All CK isoenzymes catalyze the phosphorylated of creatine to produce phosphocreatine. Extremely high levels of CK are seen in skeletal muscle, especially the MM type. In muscle tissue, phosphocreatine acts as an energy storage device, whereas CK catalyzes the opposite process—the rapid synthesis of ATP from creatine phosphate and ADP—when the tissue needs ATP.[73]

Figure 3: Conversion of creatine to phosphocreatine



Creatine Phosphocreatine

The enzyme active site is thought to facilitate substrate and inhibitor entry since it is located in the gap between the two zones 69. Heart muscle frequently contains CK-MB, skeletal muscle frequently contains CK-MM, and most quasi tissues, such as the brain, typically have CK-BB. An octamer of Mi-CK and a heterodimer of Mi-CK are the mitochondrial variants in the brain and muscle, respectively. CK-MB divides into two varieties when it enters the bloodstream: CK-MB1 and CK-MB2.[68,74,75] The brain, skeletal muscles, heart tissue, and other tissues all contain creatine phosphokinase. However, CPK escapes into the bloodstream when muscles are injured. CPK thus denotes a muscular damage. Compared to CK-MM, which is a marker of musculoskeletal muscle injury, CK-MB is a more accurate indicator of cardiac muscle damage.[76]

The phosphocreatine created by this process is used to transport ATP to cells and tissues that need a lot of it, such the heart, skeletal muscles, and the brain. The normal range of CPK concentrations is 20 to 200 IU/L. A number of diseases, such as the breakdown of skeletal

muscle, heart disease, kidney disease, as well as several medications, can have an impact on CPK levels.[77] The half-life of CK enzyme is 1.5 days and it will be reduced by 40-50% compared to the first value.

Estimation

Due to the number of distinct test methods available, there is no reference standards value for serum CK. Normal values are set locally since that is the most practical choice given the approach and the permissible ranges for healthy controls. The values are provided in liters and international units. A common method of determining the rate of the previous reaction is by using spectrophotometry, which also determines the total CK content. Due to changes in analysis techniques, aging, gender, race, and level of physical activity, the results are very variable. To separate CK into its isoenzymes, one can utilize, radioimmunoassay, column chromatography, or electrophoresis. The majority of tests use cellulose acetate or agarose gel electrophoresis, with band quantification done using spectrophotometric or fluorometric techniques. The capillary electrophoresis bands can also be quantified using extraction. The most mobile channel is CK-BB, followed by CK-MB and CK-MM.[78]

Significant events have demonstrated that electrophoresis is appropriate for routine medical examination, despite the fact that it may not be as exact as radioimmunoassay or column chromatography. The most used method in investigations is sensitive column chromatography. As technology develops, radioimmunoassay techniques for isoenzymes could be speedy and the way of the future.[78]

Causes for altered levels of CPK

Burns, chronic muscle diseases, skeletal muscle breakdown, and even intense exercise can increase CK activity. As a result, the CK-MB isoenzyme was employed to assist in the AMI diagnosis. The CK-MB score can still rise in circumstances including acute muscular damage, CCF, and arrhythmias while being superior than the CK test alone. One of the early symptoms of an acute heart attack is the presence of creatine kinase (AMI).After 24 h of AMI symptoms, creatine kinase production increases, increases at between 24 and 36 hours, and returns to baseline at 48 to 72 hrs.[79]

Individuals with Alzheimer's and Pick illness may have less CPK activity in their brains. These people had mostly decreased BB-CK activity, which decreased net CPK activation.[75] Patients with rhabdomyolysis also have elevated CPK levels. Rhabdomyolysis can be brought on by a crush injury, medication use, viral infections, and strenuous activities. Weakness and pain in the muscles are frequent signs, as is urine that has a dark tint. CPK, as well as ALT, AST, and electrolytes, are produced as a result of the breakdown of skeletal muscle. The dark urine is brought on by myoglobinuria. Rhabdomyolysis is indicated by a CPK level more than 1000 IU/L; values higher than 5000 IU/L indicate severe rhabdomyolysis.[80] Simvastatin, a drug used to decrease cholesterol, can cause extremely high CPK levels in patients, which can lead to rhabdomyolysis.[81]

Low CPK levels can occur in people with connective tissue disorder such SLE or arthritis. Low levels can also be detected in those who don't do much physical exercise, including elderly individuals who are bedridden.[82]

Role of CPK in OPC poisoning

Depending on which receptor is affected, a variety of symptoms of organophosphorus intoxication can be categorized as being either muscarinic or nicotinic. Excessive salivation, lacrimation, incontinence, diarrhoea, vomiting, stomach pains, distention, narrow pupils, bradycardia, and wheezing are examples of muscarinic symptoms. Fasciculation, paralysis, elevated blood pressure, and palpitations are examples of nicotinic symptoms. One of the signs of central receptor malfunction is ataxia, which can also cause anxiety, confusion, convulsions, and psychosis. Plegia can have one of three different forms.[83,84] Continuous stimulation at the neuromuscular junction causes type I, intermediate syndrome causes type II, and late polyneuropathy causes type III. In mild to severe OP toxicity, the muscle fibers will recover to normal after a single shock in 5–6 days. Testing for CPK is affordable and easy to measure. Serum CPK can be used to forecast and evaluate OP poisoning patients' prognoses. In animal tests, Calore et al. found that OP poisoning causes muscle fiber necrosis.[85] In a subgroup of their severe poisoning cases, serum creatine phosphokinase was shown to be high, and rhabdomyolysis was discovered in the "intermediate syndrome," which led to an elevated level of CPK.[86] In OP poisoning, there are three different types of muscle injuries that can occur: type I, which is brought on by continual depolarization at the NM junction and likely results in muscle fiber injury (going to occur during the initial cholinergic crisis); type II, which is brought on by intermediate syndrome; and type III, which is brought on by late polyneuropathy.[87] Individuals within 24 and 96 hours after an acute OP exposure develop transitional syndrome.[10] CPK is the appropriate biomarker for diagnosing and monitoring skeletal muscle damage, since Schneider et al. found that muscle tissue injuries caused CPK to infiltrate into the urine and blood very quickly.[88]

The severity and outcome of those with sever OP toxicity are significantly influenced by acidosis.[89] The fact that acidosis may cause small increases in CPK concentrations in the blood suggests that CPK levels in acidosis could actually be higher.[90] Additionally, because acidosis is a common side effect of acute OP poisoning and can be brought on by respiratory acidity or metabolic acidosis), CPK levels are linked to both the severity of acidosis and the amount of acute OP intoxication.[91] In severe OP poisoning, particularly in severely intoxicated patients, Bhattacharyya et al. and Calore et al. found a rise in blood CPK levels that was likely related to the premature death of muscle fibres.[65,85]

After a solitary muscle injury, CPK recovers to baseline in 5–6 days. Following a day to 2days following the start of tissue injury or rhabdomyolysis, CPK levels peak and then steadily decline to 39% of the levels from the day before.[88] When compared to instances with lower serum enzyme levels, patients with higher creatine kinase values had a higher mortality rate. In contrast, only 12% of patients improved inside the group with greater CPK levels, or levels > 180 IU/L, whereas 80% of cases recovered inside the group with reduced CPK levels, or 180 IU/L upon admission. It was notable that the correlation between plasma cholinesterase and serum CPK upon admission was -0.522. After one week, survivors had a median serum CPK level of 201.0IU/L compared to 2498.0IU/L for quasi (p0.014).[92]

The majority of individuals with lower tiers of creatine kinase experienced complete recovery with no issues. The mean marker enzymes levels in various phases of intoxication showed higher values on future days in individuals who had respiratory failure and required tube insertion for breathing. Patients died more frequently when their creatine kinase levels were higher. Early plasma creatine kinase levels and levels of intoxication were correlated significantly, and those with initial high CPK concentrations died. The individuals who

survived after intubation had the overall average CPK levels throughout all three days, followed by the mortality group. This was proven to be statistically significant.[93]

Peradeniya Organophosphorus Poisoning [POP] scale evaluates CPK association with poisoning severity

Bhattacharyya et al. discovered a significant correlation between the initial CPK value as well as the Op score, serum EChE concentrations, and total atropine dosage in cases of acute OP poisoning. Muscle biopsy evidence showed that CPK levels were high even in the absenteeism of intermediate condition, which is likely related to the early mortality of muscle tissue.[65] An observational study found that the plasma CPK concentrations and multiple doses of atropine needed for treatment increased as well as the degree of OP intoxication as determined by the POP score. BChE concentrations dropped ,POP found that mild and moderate OP-impaired individuals had elevated blood Creatine kinase levels in at least one of three serial tests.[94] According to the positive connection of first serum CPK with POP score and the negative link of early serum CPK with pseudo cholinesterase, a care facility prospective investigation indicated a significant correlation between early blood CPK levels as well as the severity of OP poisoning. According to the POP scoring system, the average creatine kinase readings at different phases of poisoning on subsequent days revealed greater values in patients who required intubation because of respiratory distress. Patients died more frequently when their creatine kinase levels were higher. The patients who lived after the insertion of a tube for ventilation had the overall average CPK levels on each of the three days, followed by the mortality group.[93]

Lokesh and others In a prospective observational research study of OP poisoning, the diagnostic severity was evaluated and categorized according to the POP scale. There was a

substantial association amongst the early plasma CK and the degree of inebriation, and those with high initial Creatine kinase levels died. According to the considerable link between CPK and POP score, the study discovered a robust correlation among blood Creatine kinase levels (3rd day levels) and the amount of OP intoxication (p0.001).[20]

CPK actions was markedly altered in cases of poisoning and even more so in subjects who passed away as a consequence of poisoning. Patients with light poisoning had average blood plasma Creatine kinase levels of 153.41 units/liter, moderate poisoning had mean levels of 344.94 units/liter, and severe poisoning had mean levels that were raised to 280.53 units/liter. It was determined to be numerically important (p=0.00) when the relationships of serum CPK among the groups defined by the POP scale were subjected to statistical analysis.[95]

Association of clinical severity (peradeniya score) of patients with raised total serum CPK level at end day 1 shows that CPK levels positivity was seen in 16 patients where as in 184 patients there was CPK negative levels. Maximum patients had mild symptoms and very few patients had severe symptoms. Statistical analysis shows that the difference between the groups categorized according to POP score it was confirmed to be numerically relevant with p value 0.01. Serum CPK shows significant difference between mild, reasonable and severe poison groups.[96] Upon correlation, POP scale and serum CPK values exhibited strong positive correlation between CPK and the degree of the intoxication. A substantial difference between the lesser form of poisoning and the severe and moderate forms, when CPK concentrations are raised, was seen in the research patients' CPK levels.[97]

According to Chellappan et al., serum creatinine phosphokinase concentrations and the requirement for atropine increased as the Op score increased. The initial CPK level was 240.5

±102.61 in cases of light poisoning (POP SCORE 0 - 3), compared to 448.05 ±72.40 in cases of medium poisoning and 1123.65 corresponding slot IU/L in cases of severe toxicity (POP 8 – 11). This demonstrated that there was a significant association amongst the early CPK value as well as the POP grade.[98] Using POP Scale, individuals with minor, medium, and acute cases were categorized according to the level of toxicity in their bodies. It was found that as the level of toxicity grew, so did the average value of S. CPK. If a patient is critically poisoned, blood CPK levels rise even in the absence of intermediate syndrome, which is probably related to the premature death of muscle. Early blood CPK levels and the disease severity of intoxication are significantly linked. The CPK level stays elevated if difficulties have caused permanent muscle injury. To put it another way, increased and persistent blood Creatine kinase levels can be utilized to anticipate future issues.[66]

Table 2: 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
	≥20/min with central cvanosis	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		

According to a prospective clinical investigation, there is a significant association between the initial blood CPK level and the seriousness of acute OP poisoning. According to the study, the initial serum CPK level may be used to rule out any other illnesses or disorders that can cause a rise in CPK levels since it is similar to the Butyrylcholinesterase level and can be used as a replacement screening method in the assessment of acute OP poisoning.[14]

Levels in subjects with moderate POP ranged between 128 IU/L to 344 IU/L. POP levels varied from 372 IU/L through 994 IU/L in individuals with moderate POP scores. The range of the severe grade POP score, from 978 IU/L to 1057 IU/L, reveals a significant association between both the POP score and the blood's initial CPK. Even without IMS, there is a rise in serum CPK levels.[99] A case-control study found that there was a significant difference in CPK across groups with varying POP severity at the time of hospitalization , at 6, and at 24 hours. Cholinesterase, CPK, and lactate were the three variables, and CPK exhibited the best association coefficient (0.69), versus the other two, with the severity of POP.[100]

MOST RELEVANT STUDIES:

Rayannavar et al. (2022)[19] POP scoring system, plasma cholinesterase, and plasma CPK levels on admittance were observed to be make it fairly of outcome in patients to OP poisoning from a this investigation, with serum Creatine kinase levels being a more practical and affordable interval estimate.

Chellappan et al. (2022)[98] According to a descriptive investigation, blood CPK, serum pseudocholinesterase levels, and total atropine dosage were all significantly correlated with the clinical level of OP toxicity. As a backup indication, serum CPK is strongly advised.

Yaduraj et al. (2022)[95] concluded from a cross-sectional study that POP scale applied at admission along with serum pseudocholinesterase and CPK levels serve as an simple and effective method for shaping early demand of ventilation and mortality in rural, peripheral centres in developing nations.

Chauhan et al. (2021)[96] Eight out of every 100 individuals with a history of OP compound use exhibited higher full blood plasma CPK levels. Seven of them experienced respiratory failure, died, and their levels of total blood plasma CPK significantly increased. According to the study, the initial rise in the total serum Creatine kinase level was strongly correlated with the prognosis and severity of OP chemical poison, suggesting that it could be employed as a diagnostic indicator for OP compound poison.

Wali et al. (2020)[97] correlated POP scale and serum CPK values and has a considerable positive suggestion with the grade of the intoxication.

Das et al. (2018)[21] To ascertain the correlation amongst plasma CPK concentration and the amount of organophosphorus intoxication, a prospective observational research including 100 patients with acute OP poisoning was done. This study implies that because blood CPK level is accessible and affordable, it may be used as a substitute biomarker in stratifying the degree of acute OP poisoning.

Balasubramaniyan et al. (2018)[101] On admission, patients with OP poisoning had their estimated blood CPK levels assessed clinically using Peradeniya OPC Poisoning methods, and the severity of the cases was categorised. At both periods of assessment, the correlations between serum CPK and outcomes like ventilatory functioning and survival of patients was statistically significant and favourable.

N.K. Lokesh et al. (2018)[20] found that a greater blood CPK level was related with a higher frequency of intermediate syndrome and death in persons with acute organophosphorus poisoning. According to the study, serum CPK can be used to determine the level of poisoning and to guide early treatment options including mechanical breathing and life-saving measures.

Mural et al. (2017)[93] discovered that individuals with acute organophosphorus poisoning have difficulty and death, and that increased blood CPK levels are related with a serious stage of poisoning.

Manar et al. (2017)[100] Clinical severity was graded using the POP scale in a research to look at the link between blood lactate, CPK concentration, and the seriousness of OP poisoning. AChE and the severity of the poisoning were significantly inversely correlated, while CPK and lactate were significantly positively correlated. They discovered that blood CPK and serum lactate can be utilised to predict the outcomes of OP poisoning and help identify situations that need for more research.

Kumar, Amith et al. (2017)[92] Repeated examinations of blood CPK levels discovered a substantial correlation with severity of individuals with acute OP poisoning. The CPK levels showed a positive predictive ability of 92%, a specificity was 81%, and a sensitivity of 74%. According to the study, high CPK levels are associated with severe organophosphorus poisoning. This study recommends evaluating CPK levels as a substitute to choline esterase as a prognostic indicator for patients with organophosphorus substances.

Nagarajan et al. (2016)[102] Serum CPK level served as added biomarker to analyse or stratify the severity of severe OP poisoning in a prospective longitudinal study of individuals with the condition since it is affordable and readily accessible, especially in poor countries.

Khan et al. (2016)[84] conducted a clinical study on 80 patients with OP poisoning and discovered no significant association between creatine regulator and patient prognosis (p = 0.15), but a robust relationship among serum cholinesterase and treatment outcomes (p = 0.005). Their research revealed a clear correlation between blood cholinesterase levels and the severity and outcomes of OP poisoning, but not with serum phosphokinase levels.

Dubey et al. (2016)[61] serum amylase and serum CPK levels correlate favourably with the degree of poisoning, according to observational study, and serum amylase is a key predictor of the requirement for respiratory assistance and demise.

Kumar et al. (2015)[84] Except for three patients who had blood CPK levels greater than 1500 IU/L and reported with IMS, all patients with OP poisoning had high levels 48 hrs after poisoning, according to a one-year prospective observational study on these patients. They proposed that fast care and minimising extra potential life implications may be aided by early diagnosis of IMS to use a serial estimation of CPK.

Sen et al. (2014)[66] Serum Cholinesterase, Creatine kinase Phosphokinase, and Ldh levels were evaluated for connexion with the degree of organophosphate poisoning at the time of admission. Even though serum CPK shows a substantial +ve correlation with poisoning magnitude and can serve as a predictor of outcomes in OP poisoning, they found that while serum cholinesterase continues to serve as a diagnosing variable for organophosphates intoxication and associates with intensity, it cannot be utilized as a outcome indicator.

Sumathi et al. (2014)[103] conducted a hospital-based observational research to connect plasma cholinesterase levels with amylase, lipase, and CPK levels in acute OP poisoning. They found that Serum amylase may be a better indicator of severity than CPK or lipase, in that order.

Madboly et al. (2013)[14] Patients who develop illness within 6hrs of acquaintance to OP but without prior treatment had their serum CPK evaluated. The POP scale was used to categorise the disease symptoms of the patients. The severity of acute OP exterminating was shown to be substantially correlated with early blood CPK levels, according to the study's findings. In this study, CPK is suggested as a substitute marker for severe OP poisoning.

Jayalaxmi (2013)[99] In the study, it was discovered that mild to severe POP grades had considerably higher serum CPK levels. Future respiratory failure has a significant risk when serum CPK levels are higher than 500 IU/L. Higher Peradeniya scores indicate a significant risk of future respiratory failure.

Bhattacharyya et al. (2011)[65] conducted an observational study to see whether blood CPK levels might be used to gauge the severity of OP poisoning instead of cholinesterase levels. The researchers found that clinical severity was significantly correlated with blood CPK, EchE level, levels of blood, and overall atropine dosage, and they proposed blood CPK as an alternative diagnosis.

LACUNAE IN LITERATURE:

Common tests for OP poisoning include estimates of plasma and serum cholinesterase (EchE) and erythrocyte cholinesterase (EchE). Additionally, OP poisoning causes abnormalities in other blood markers that can be used to gauge the severity and outcome of the condition. One of these indicators, serum CPK, which measures muscle damage, is now being studied. According to certain research, serum CPK can be used to assess the severity and prognosis of diseases in people. The current work thus compares blood CPK values to the POP grade of the level of OP toxicity.

MATERIALS & METHODS

MATERIALS AND METHODS-

Source of data: All the patients who admitted in R L Jalappa hospital with acute Organophosphorus toxicity was used as the study population.

Study design: The current study was a cross sectional study

Study period: Jan 2021 to Dec 2022

Method of collection of data:

Inclusion Criteria:

- Patient having a history of organophosphorus chemical exposure during the previous 24 hours.
- Patient without receiving any prior treatment outside the hospital which alter POP score.

Exclusion Criteria:

- Other pesticide poisoning, mixing with poisons and alcohol.
- Prior IM injection before reporting.
- Chronic liver disease, myopathy, trauma, renal failure, and autoimmune disease are all medical conditions.
- Patient is on drugs like -statin, fibrates, steroids.

7.5 Sample size:

Sample size was estimated by using correlation coefficient (r) the relationship between early CPK levels and the intensity of OP poisoning as 0.847 (i.e. r = 0.847) from the study by Das P et al. Using these values at 90% power and 95% confidence level and substituting in the below formula, sample size of 9 was obtained.

Complete sample size = N = [(Z? +Z?)/C] 2 + 3

The typical normal deviation for? = Z? = 1.960

The typical normal deviation for? = Z? = 1.28

r = Correlation coefficient = 0.847

C = 0.5 * In [(1+r)/(1-r)]

N = 9

Considering 10% Non-response rate a sample size of 9 + 0.9 = 10 subjects minimum to be included in the study. We will include 100 subjects in our study.[7]

7.6 Methodology:

- There were 100 subjects who met inclusion and exclusion requirements
- A complete history was taken and detailed examination was performed
- All subjects have undergone investigations including 2cc of blood drawn into a simple vacutainer to gather serum creatinine phosphokinase using automated machine
- After acquiring the Sr CPK values it was used to correlate the POP scale with Sr. CPK to assess the severity and prognosis in acute organophosphorous poisoning patients

STATISTICAL METHODS

The primary outcome variable was decided upon as serum creatinine phosphokinase. The primary explanatory variable was the Peradeniya Organophosphorus Poisoning Scale.

On quantitative data, using mean and standard deviation, as well as on categorical variables, using frequency and percentage, descriptive analysis was carried out. Additionally, information was presented using appropriate formats, such pie charts, bar graphs, and so on. Using independent-samples T tests, the mean and standard deviation of continuous data were computed. The link between the quantitative explanatory and outcome variables was analyzed using the Pearson correlation coefficient, and the data was shown in a scatter plot. P value less than 0.05 was deemed statistically significant.

RESULTS AND OBSERVATIONS

RESULTS:

A total of 100 subjects were included

Table 3: Age distribution in the research subjects (N=100).

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Age	46.33±13.47	46.50	19.00	78.00	43.69	48.97

The study's participants had an average age of 46.3313.47. With a 95% C, the minimum and maximum values are 19 and 78, respectively (43.69 to 48.97). (Table 3)

Table 4: Gender review illustration in the research subjects (N=100)

Gender	Frequency	Percentage
Male	72	72.00%
Female	28	28.00%

The research subjects consisted of 72 (72%) males and 28 (28%) females. (Figure 4 and Table 4)

Figure 4: Gender distribution in the research subjects (N=100) as a pie chart

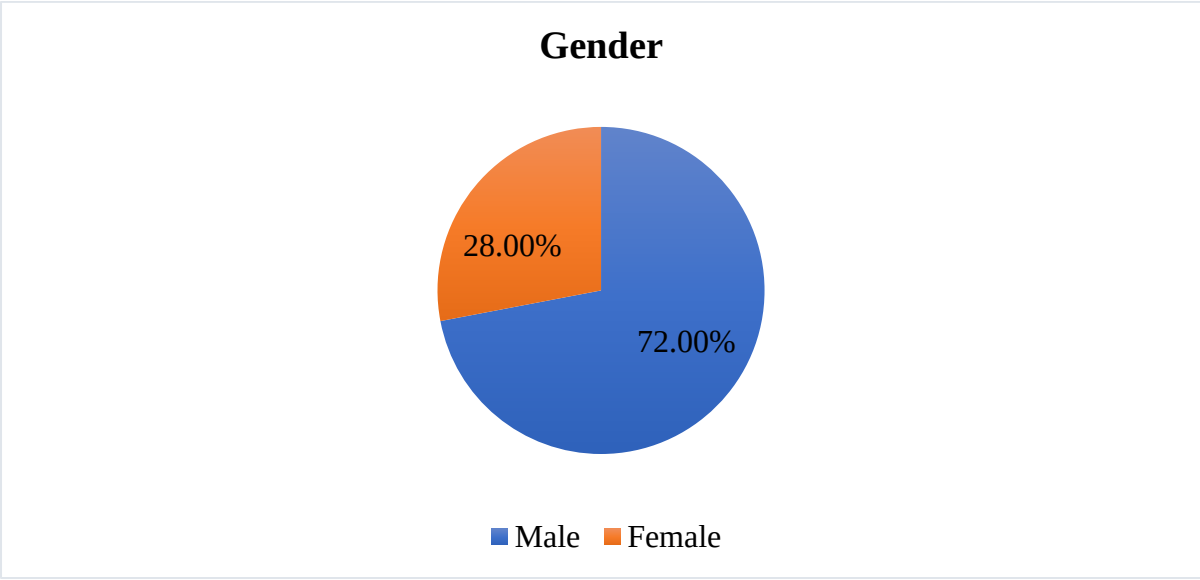


Table 5: Occupation review illustration in the research subjects (N=100)

Occupation	Frequency	Percentage
Farmer	21	21.00%
House wife	19	19.00%
Shop keeper	15	15.00%
Student	13	13.00%
Driver	10	10.00%
Daily labourer	9	9.00%
Employee	6	6.00%
Teacher	3	3.00%
Engineer	2	2.00%
Watchmen	1	1.00%
Business man	1	1.00%

Among the study population, 21 (21%) were farmers, 15 (15%) were shop keepers, 13 (13%) were students, 10 (10%) were drivers. (Table 5)

Table 6: Miosis review illustration in the research subjects (N=100)

Miosis	Frequency	Percentage
< 2mm	45	45.00%
2 mm	1	1.00%
>2mm	20	20.00%
pin point	34	34.00%

Among the study population, 45 (45%) had miosis <2mm, 1 (1%) had 2mm miosis, 20 (20%) had >2mm miosis and 34 (34%) had pin point. (Table 6 & Figure 5)

Figure 5: Miosis in the study population (N=100) is depicted as a bar diagram.

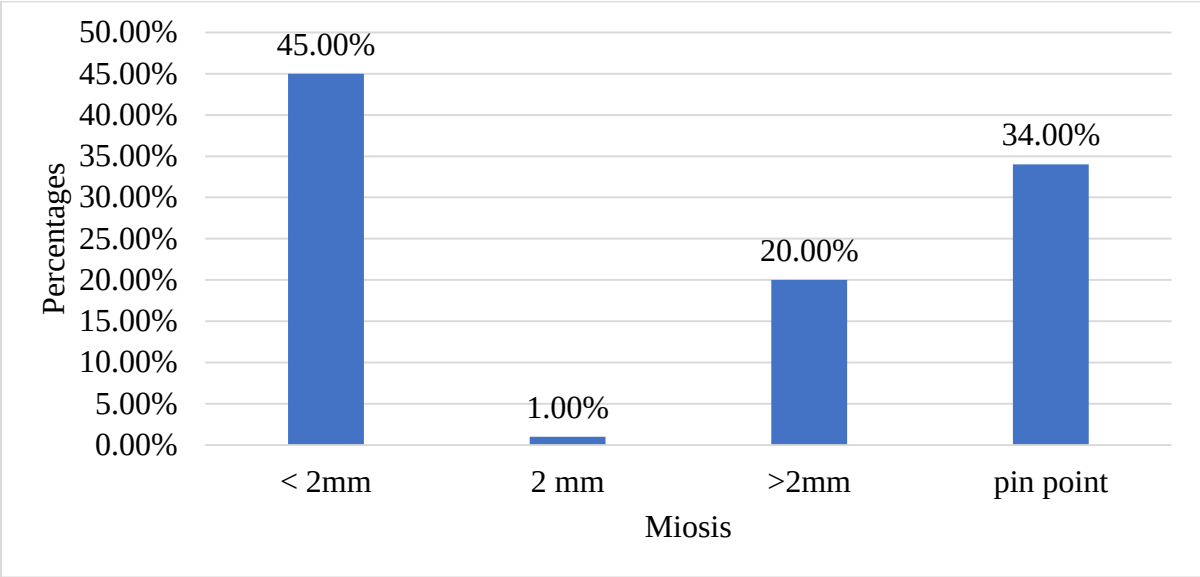


Table 7: PCO2 (partial carbon dioxide) Review illustration in the research subjects (N=100)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
PCO2	38.82±8.88	37.00	20.00	75.00	37.08	40.56

The mean PCO2 was 38.82±8.88 in the study population. The minimum and maximum was 20 and 78 respectively with 95% C. I (37.08, 40.56) (Table 7)

Table 8: hco3 (bicarbonate) review illustration in the research subjects (N=100)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
hco3	22.92±4.25	24.00	10.00	30.00	22.09	23.75

The mean hco3 was 22.92±4.25 in the study population. The minimum and maximum was 10 and 30 respectively with 95% C. I (22.09, 23.75) (Table 8)

Table 9: Perspiration in the research subjects (N=100): Review illustration

Sweating	Frequency	Percentage
Present	92	92.00%
Absent	8	8.00%

Perspiration was reported by 92 (92%) of the research subjects. (Table 9)

Table 10: Salivation Review illustration in the research subjects (N=100)

Salivation	Frequency	Percentage
Present	54	54.00%
Absent	46	46.00%

54 (54%) of the research subjects salivated. (Table 10)

Table 11: Review illustration of Fasciculations in the research subjects (N=100)

Fasciculations	Frequency	Percentage
Present	59	59.00%
Absent	39	39.00%
None	2	2.00%

Among the study population, 59 (59%) had fasciculations. (Table 11)

Table 12: Review illustration of Neck lift in the research subjects (N=100)

Neck lift	Frequency	Percentage
Present	14	14.00%
Absent	86	86.00%

Among the study population, 14 (14%) had neck lift. (Table 12)

Table 13: Review illustration of Vomiting in the research subjects (N=100)

Vomiting	Frequency	Percentage
Present	89	89.00%
Absent	11	11.00%

With the subjects in the research, 89 (89%) had vomiting's. (Table 13)

Table 14: Review illustration of Blood PH in the research subjects (N=100)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Blood PH	7.32±0.06	7.32	7.10	7.46	7.31	7.34

The average Blood PH in the research subjects was 7.320.06. With 95% C, the lowest and highest were 7.10 and 7.46, respectively (7.31, 7.34) (Table 14)

Table 15: Review illustration of Respiratory rate in the research subjects (N=100)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Respiratory rate (breath per minute)	31.98±4.39	32.00	22.00	43.00	31.12	32.84

The mean respiratory rate was 31.98±4.39 in the study population. The minimum and maximum was 22 and 43 respectively with 95% C. I (31.12, 32.84) (Table 15)

Table 16: Heart rate review illustrations in the research subjects (N=100)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Heart rate (bpm)	53.66±11.36	56.00	22.00	72.00	51.43	55.89

The mean heart rate (bpm) was 53.66±11.36 in the study population. The minimum and maximum was 22 and 72 respectively with 95% C. I (51.43, 55.89) (Table 16)

Table 17: Descriptive analysis of serum creatinine phosphokinase at admission (mcg/l) in the research subjects (N=100)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Serum creatinine phosphokinase at admission (mcg/l)	1,034.38±1,169.13	659.00	89.00	6,789.00	805.23	1,263.53

The mean Serum creatinine phosphokinase at admission (mcg/l) was 1,034.38±1,169.13 in the study population. The minimum and maximum was 89 and 6,789 respectively with 95% C. I (805.23, 1263.53) (Table 17)

Table 18: Pseudocholinesterase (U/L) review illustrates in the research subjects (N=100).

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Pseudocholinesterase (U/L)	2,188.68±2,567.03	1,144.50	200.00	7,890.00	1,685.54	2,691.82

The mean Pseudocholinesterase (U/L) was 2,188.68±2,567.03 in the study population. The minimum and maximum was 200 and 7,890 respectively with 95% C. I (1685.54, 2691.82) (Table 18)

Table 19: Descriptive analysis of Peradeniya organophosphorus poisoning Scale in the study population (N=100)

Peradeniya organophosphorus poisoning Scale	Frequency	Percentage
Mild	36	36.00%
Moderate	51	51.00%
Severe	13	13.00%

Among the study population of Peradeniya organophosphorus poisoning Scale, 36 (36%) were mild, 51 (51%) were moderate and 13 (13%) were severe. (Table 19 & Figure 6)

Figure 6: Pie chart of Peradeniya organophosphorus poisoning Scale in the study population (N=100)

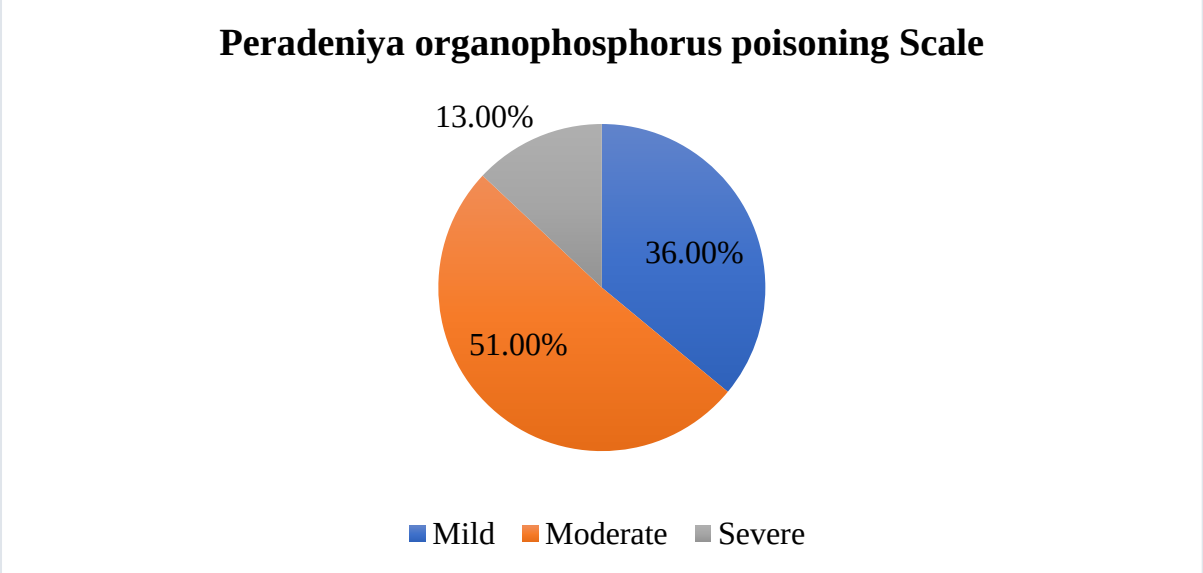


Table 20: Seizures in the research subjects (N=100): a review illustartion

Seizures	Frequency	Percentage
Present	20	20.00%
Absent	80	80.00%

With the subjects in the research, 30 (30%) had seizures. (Table 20)

Table 21: Review illustration of the research subjects state of consciousness (N=100).

Level of consciousness	Frequency	Percentage
State of awareness	39	39.00%
Impaired response to commands	36	36.00%
There is no reaction to vocal requests.	25	25.00%
Defective response to vocal requests	6	6.00%
Responsive to oral commands	2	2.00%

Among the study population, 39 (39%) were conscious, 36 (36%) had impaired response to commands, 6 (6%) had impaired response to verbal commands. (Table 21)

Table 22: Fatality in the study population (N=100): a review illustrated

Mortality	Frequency	Percentage
Death	9	9.00%
Survived	91	91.00%

Nine (9%) of the research subjects were deceased and 91 (91%) survived. (Table 22 & figure 7)

Figure 7: Fatality in the research subjects (N=100) illustrated in a pie chart

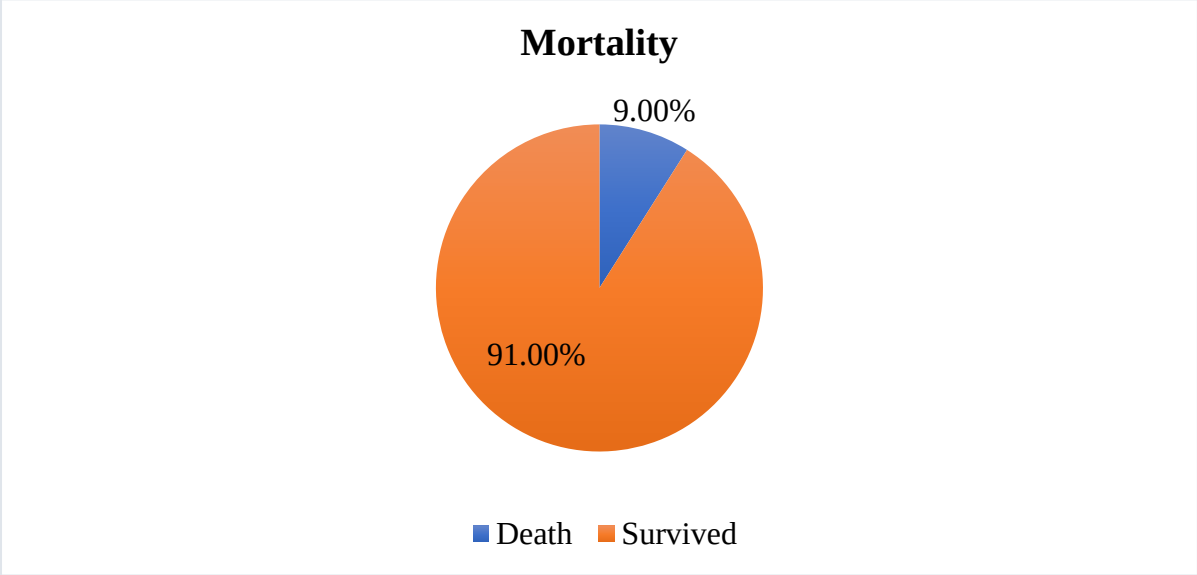


Table 23: A review illustration of the research population's (N=100) duration of stay (in days).

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Duration of stay (in days)	11.78±6.21	10.00	5.00	32.00	10.56	13.00

The mean Duration of stay (in days) was 11.78±6.21 in the study population. The minimum and maximum was 5 and 32 respectively with 95% C. I (10.56, 13) (Table 23)

Table 24: Descriptive analysis of op smell in the study population (N=100)

OP smell	Frequency	Percentage
Present	100	100.00%

Table 25: In the research subjects (N=100), review illustration of serum creatinine phosphokinase after one week or upon discharge was performed.

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Serum creatinine phosphokinase after one week or at discharge	1,110.54± 1,811.23	403.00	70.00	8,789.00	755.54	1,465.54

The mean Serum creatinine phosphokinase after one week or at discharge was 1,110.54±1,811.23 in the study population. The minimum and maximum was 70 and 8789 respectively with 95% C. I (755.54, 1465.54) (Table 25)

Table 26: Intermediate syndrome review illustrated in the research subjects (N=100)

Intermediate syndrome	Frequency	Percentage
Present	14	14.00%
Absent	86	86.00%

With the subjects in the research,14 (14%) had intermediate syndrome. (Table 26)

Table 27: Descriptive analysis of Intubation in the study population (N=100)

Intubation	Frequency	Percentage
Yes	17	17.00%
No	83	83.00%

Among the study population, 17 (17%) had intubation. (Table 27)

Table 28: Hemoglobin review illustrated in the research subjects (N=100)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Hemoglobin (g/dl)	11.86±1.19	11.60	8.90	14.70	11.63	12.10

The mean Hemoglobin (g/dl) was 11.86±1.19 in the study population. The minimum and maximum was 8.9 and 14.70 respectively with 95% C. I (11.63, 12.10) (Table 28)

Table 29: Descriptive analysis of Total leukocyte count in the research subject (N=100)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Total leukocyte count (cells/cmm)	12.21±3.00	11.70	5.80	18.90	11.62	12.79

The mean Total leukocyte count (cells/cmm) was 12.21±3.00 in the study population. The minimum and maximum was 5.8 and 18.90 respectively with 95% C. I (11.62, 12.79) (Table 29)

Table 30: Urea review illustrated in the study population (N=100)

Name	Mean ± S.D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
Urea (mg/dL)	46.57±15.35	43.00	22.00	128.00	43.56	49.58

The mean Urea (mg/dL) was 46.57±15.35 in the study population. The minimum and maximum was 22 and 128 respectively with 95% C. I (43.56, 49.58) (Table 30)

Table 31: Creatinine review illustrated in the study population (N=100)

Name	Mean ± S.D	Median	Lowest	Highest	95% CI	
					Lower CI	Upper CI
Creatinine (mg/dL)	0.92±0.24	0.90	0.50	2.10	0.87	0.96

In the study population, the mean Creatinine (mg/dL) level was 0.920.24. With 95% C, the lowest and maximum were 0.50 and 2.10, respectively (0.87, 0.96) (Table 31)

Table 32: comparison of serum creatinine phosphokinase at admission (mcg/l) with Peradeniya organophosphorus poisoning Scale (N=100)

Parameter	Peradeniya organophosphorus poisoning Scale (Mean ± SD)			P Value
	Mild (N=36)	Moderate (N=51)	Severe (N=13)	
Serum creatinine phosphokinase at admission (mcg/l)	387.53 ± 410.34	1059.18 ± 864.52	2728.38 ± 1817.88	<0.001

The mean Serum creatinine phosphokinase at admission (mcg/l) was 387.53 ± 410.34 in mild POP scale, it was 864.52 in moderate POP scale and it was 2728.38 ± 1817.88 in severe POP scale. The difference in Serum creatinine phosphokinase at admission (mcg/l) among Peradeniya organophosphorus poisoning Scale was numerically relevant (P value: <0.001) (Table 32)

Table 33: The blood creatinine phosphokinase level at admittance (mcg/l) was evaluated to mortality (N=100).

Parameter	Mortality (Mean ± SD)		P Value
	Death (N=9)	Survived (N=91)	
Serum creatinine phosphokinase at admission (mcg/l)	3132.11 ± 2039.02	826.91 ± 806.48	<0.001

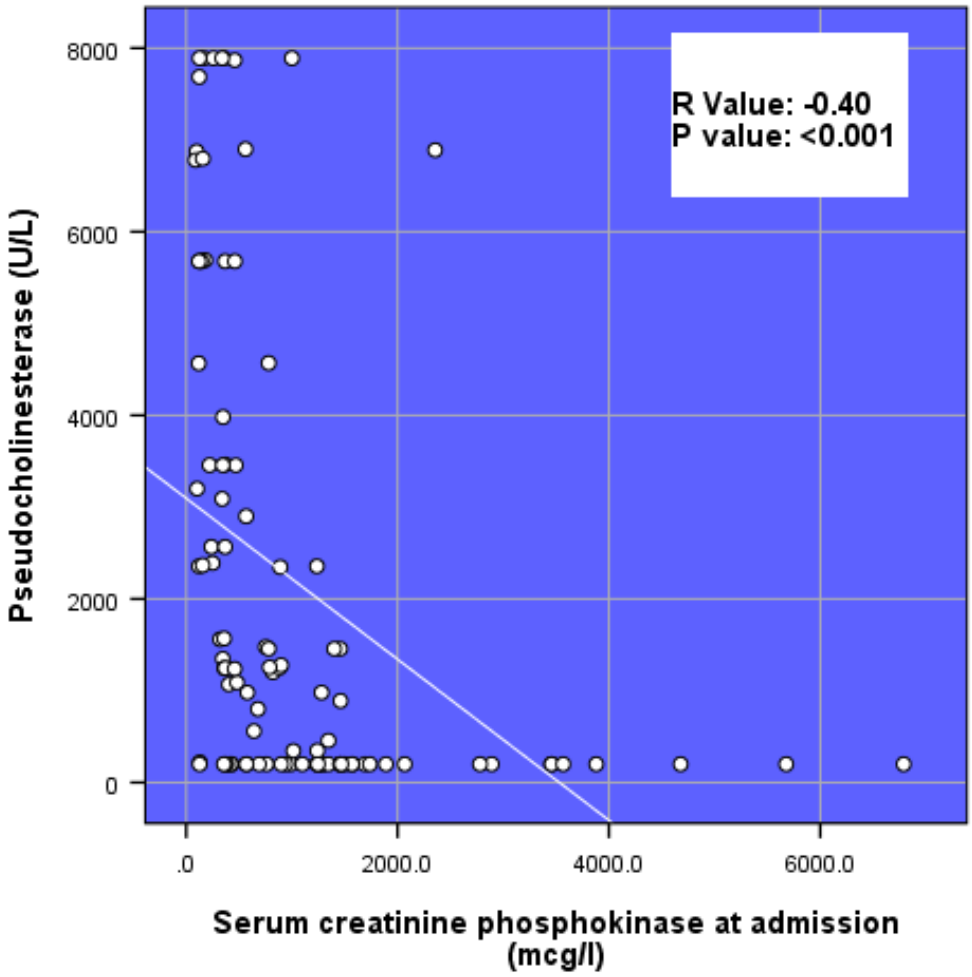
The mean Serum creatinine phosphokinase at admission (mcg/l) was 3132.11 ± 2039.02 in dead people and it was 826.91 ± 806.48 in survived people. The difference in Serum creatinine phosphokinase at admission (mcg/l) between mortality was numerically relevant (P value: <0.001) (Table 33)

Table 34: Pseudocholinesterase (U/L) vs serum creatinine phosphokinase (mcg/l) upon hospitalization (N=100)

Parameter	Pseudocholinesterase (U/L)	P Value
	r Value	
Serum creatinine phosphokinase at admission (mcg/l)	-0.40	<0.001

Serum creatinine phosphokinase (mcg/l) and Pseudocholinesterase (U/L) upon admission had a slight negative relationship. The difference between plasma creatinine phosphokinase at admission (mcg/l) and Pseudocholinesterase (U/L) was statistically significant. (Table 34 & Figure 8)

Figure 8: Scatter plot between Comparison of pseudocholinesterase (U/L) with serum creatinine phosphokinase at admission (mcg/l) (N=100)



DISCUSSION

DISCUSSION:

In especially in poor nations like India, OP insecticides are frequently cited as one of the major causes of disease and fatalities from poisoning globally. The length of time after exposure determines the likelihood of morbidity and mortality as well as the start of care. Due to their widespread usage in industry and agriculture, as well as their accessibility and reduced cost, OP compounds are becoming a significant source of health risk. It is crucial to understand the whole range of symptoms as a result. Patient screening, vulnerability analysis, early identification, and prompt treatment are all crucial.[65] The dominant and autonomic neural systems, as well as the skeletal neuromuscular link, are all affected by excessive acetylcholine accumulation, which is the main cause of these medications' toxic effects. It's common to observe pseudo cholinesterase levels, which should decline in people with severe organophosphorus poisoning. It cannot be used as a marker to forecast the level of toxicity because it is generic and has no link.[104] Alternatives for novel economically viable and quantifiable laboratory indications of OP poisonings are being developed. These indicators include serum immunoglobulins, lactate dehydrogenase, and creatine phosphokinase (CPK) (IgG, IgA).[65] The objective of the current cross-sectional investigation is to assess blood plasma CPK levels in acute severe organophosphorus poisoning and correlate them with the degree of poisoning as assessed by the POP scale. The main outcome variable is serum CPK, and the main explanatory factor is POP scale.

A total of 100 OP poisoned subjects meeting the inclusive criteria are incorporated into the final analysis. The average age was 46.33±13.47 years in our study population with 72% males and 28% females. Rayannavar et al. had a younger age group in their study with a mean age of 33.86 ± 14.46 years with the gender distribution being 67% males and 33% females. Suicidal poisoning accounted for 100% of the poisoning approaches in their study,

which explains why young adults were so prevalent and reflects the ubiquitous psychological disorders in this age group.[19] Similar gender distribution was observed in Chellappan et al.'s study with 67 % males and 32.6 % females and an average age was 35.5 years[98] and Mural et al.'s[93] study among those under the age of 20, women outnumbered men. The majority of patients in Chauhan et al study .'s were under 30 years old, with an average age of 33.23 10.12 and 62% males and 38% females.[96]

Table 35: Mean age across studies:

Study	Mean age (years)
Current study	46.33±13.47
Rayannavar et al.[19]	33.86 ± 14.46
Chellappan et al.[98]	35.5
Chauhan et al.[96]	33.23 ± 10.12
Balasubramaniyan et al.[101]	36.66± 14.15

Occupation wise, 21% are farmers, 19% housewives, 15% are shop keepers, 13% students, 10% drivers, 9% daily laborers, 6% employed in organizations, 3% teachers and 2% engineers in our study. In the study by Chellappan et al, majority of the subjects were farmers, but there were also some university students, businesspeople, experts, and housewives.[98]

The SLUDGE symptoms, which stand for Saliva, Rhinorrhea, Urinary incontinence, Defecation, Gastrointestinal tract Cramps, and Emesis, are indicative of OP poisoning. In our study, 45% had miosis with < 2mm pupils, 1% had 2 mm miosis, 20% had > 2 mm miosis and 34% had pin point pupils. Among the OP toxidrome symptoms, sweating is manifested in 92%, 54% had excessive salivation, 59% had fasciculations, 14% had neck lift, 89% had emesis, 30% had seizures. According to Rayannavar et alstudy, .'s emesis was the most prevalent symptom in 84% of cases, followed by miosis in 78.4%, bradycardia in 60.2%,

quick breathing in 77.3%, muscular spasms in 43.2%, altered perception in 42%, with convulsions in 5.7%.[19] In our study, 39% are conscious, 36% had defective instruction responding, whereas 6% had limited verbal instruction reactions. Similar observation was made by Chellappan et al. where the most common presentation was emesis in 88.3 %, followed by miosis in 81.95 %, bradycardia in 70.84 %, increased salivation in 60.84 %, sweating in 49.97 %, altered sensorium in 49.08 %, tachypnea in 28.5 % and muscle fasciculations in 10.03 %.[98] Slow heart rate (33%) is the most commonly diagnosed symptom seen in the Chauhan et al research, which may be related to increased parasympathetic stress.[96]

The mean Serum CPK at admission (mcg/l) is 1,034.38±1,169.13 ranging from 89 to 6,789 with 95% C. I (805.23, 1263.53). The average blood plasma CPK level was 401.27 ± 298.28 IU/L in Rayannavar et al.'s study.[19]

The mean Pseudo cholinesterase (U/L) is 2,188.68±2,567.03 ranging from 200 to 7,890 respectively with 95% C. I (1685.54, 2691.82). The mean serum pseudo cholinesterase was much higher in Rayannavar's et al.'s study at 4239.53 ± 2706.21. [19]

The POP Scale shows that 36% of people have mild poisoning, 51% have moderate poisoning, and 13% have severe poisoning. In the research by Rayannavar et al., the POP score identified 45.5% of patients as having light poisoning, 39.8% as having similar inebriation, and 14.8% as experiencing severe inebriation.[19] According to the POP score, 27 %, 50.8 % and intoxication was classified as mild, moderate, or severe in 18.2% of all cases, respectively in Chellappan et al.'s study. [98] Per POP scale, 61% experienced mild poisoning, 30% had serious poisoning, and 9% had severe intoxication in Chauhan et al.'s study.[96]

Mural et al. reported 83% were mild, 11% moderate and 6% severe cases of poisoning per POP in their study.[93] Wali et al. also noted that the mild form (51%) of OP poisoning was most common followed by moderate (29%) and severe form (18%).[97] according to Lokesh et al.' POP's study, intoxication resulted in 67.3% of cases as mild, 16.4% as severe, and 16.4% as severe.[20] On applying POP score, Balasubramaniyan et al.[101] discovered that 42% of cases had low severity, 16% had severe POP scores, and 42% had moderate POP scores.

Table 36: POP severity across studies

Study	Mild	Moderate	Severe
Current study	36%	51%	13%
Wali et al. [97]	51%	29%	18%
Lokesh et al. [20]	67.3%	16.4%	16.4%
Balasubramaniyan et al.[101]	42%	42%	16%
Rayannavar et al.[19]	45.5%	39.8%	14.8%

In our study, mortality rate is 9% which is similar to that in Chellappan et al.'s study at 10.86%.[98] In Rayannavar et al.'s study 86.36% survived and 13.6% patients succumbed.[19] Mortality rate was 14.2% in Nagarajan et al.'s study.[102]

The mean Serum CPK after one week or at discharge is 1,110.54±1,811.23 ranging from 70 to 8789 with 95% C. I (755.54, 1465.54). Intermediate syndrome is observed in 14% of the study group and 17% required intubation which is much higher than that noted in Chellappan et al.'s[98] study which was 7.94 %. In Wali et al.'s [97] study 18% developed the intermediate syndrome which was similar to that in Lokesh et al.'s[20] study, which was 18.1%.

According to the inquiry, admission blood plasma CPK levels are greater in cases of severe organophosphorus poisoning. This conclusion is in line with past findings from studies by

Rayavannar et al.Chellappan et al. The mean Serum CPK at admission (mcg/l) is 387.53 ± 410.34 in those with mild poisoning according to the POP scale, 864.52 in the moderately poisoned group and 2728.38 ± 1817.88 in the severely poisoned patients. The difference in Serum CPK at admission (mcg/l) among different grades of poisoning on POP scale is numerically relevant with a p value < 0.001. Higher the serum CPK, higher is the POP score severity. The mean serum CPK levels in the mild, moderate, and severe groups were 210.35 ±146.63 IU/L, 460.17± 245.59 IU/L, and 830.15± 270.89 IU/L, respectively, in Rayannavar et al study's As the poisoning became more severe, the serum CPK levels increased ('p' value 0.001). For mild, moderate, and severe poisoning, the rate of death of cases was 0%, 11.4%, and 61.5%, respectively. The variance in case fatality rate is numerically important with a p value of 0.001.[19] Additionally, Chellappan et al. found that the serum CPK levels increased along with the POP score. For mild cases (POP SCORE 0 - 3), initial CPK levels was 240.5 102.61; for moderate cases (448.05 72.40); and for severe cases (1123.65 210.08 IU/L) (POP 8 - 11).[98] Serum CPK showed significant difference between mild, moderate and severe poison cases in Chauhan et al.'s study.[96] Supporting the observations made in our study, Mural et al investigation .'s discovered a significant link among earlier and earlier serum CPK levels and the severity of OP poisoning, which is demonstrated by the initial CPK's positive correlation with POP score and its negative correlation with pseudo cholinesterase in the preliminary blood plasma. These connections have numerical significance.[93] POP scale and blood CPK levels were connected by Wali et al. They also discovered a sturdy optimistic correlation between CPK and the acuteness of the poisoning.[97] According to the research by Das et al., there is a statistically significant link between the baseline CPK level and the degree of OP poisoning (as determined by the POP scale) on the day of hospitalization.[21]

The mean Serum CPK at admission (mcg/l) is 3132.11 ± 2039.02 in those who are dead and it was 826.91 ± 806.48 in the survivors. The link between mortality and the increase in Serum CPK upon admittance (mcg/l) with a p - values of 0.001 is quantitatively significant. A greater blood CPK level upon admission indicates a higher chance of death. This discovery is comparable to that made in Rayannavar et al study's which found that patients with case fatalities were 0% for blood CPK levels under 100 IU/L and 63.60% for levels over 800 IU/L. Case fatalities increased as serum CPK concentrations climbed. Number-wise, the variation in rates of case fatalities is significant ('p' value 0.001).[19] In concordance with the above studies, Chauhan et al. also reported the difference in CPK level to be numerically relevant (p=0.002) among people who survived and deceased. In non-survivors CPK level greatly elevated throughout the course of disease in their study.[96] Mural et al. also found that the death group had the highest average CPK levels over the course of the three days, followed by those who survived with a breathing tube in place.[93] The difference in CPK levels was found to be noteworthy (p.001) in the Wali et al study, highlighting the fact that CPK levels could be used as a predictive biomarker in subjects who might have consumed OPC poison. CPK levels were lower in individuals who began to recover purely than in those who frozen to death.[97] Incidences with greater starting CPK levels fared poorly in Das et al.'s study.[21] Higher death rate was detected in subjects with higher CPK levels ($p<0.01$) in Lokesh et al.'s study.[20]

In our study, serum CPK upon admission had a slight negative connection (mcg/l) and pseudo cholinesterase (U/L) with r value -0.040 and the difference between Serum CPK at admission (mcg/l) and pseudo cholinesterase (U/L) is substantial with a p value < 0.001. Similar results were obtained by Rayavannar et al., who discovered a moderately negative correlation (r = -0.398; p value 0.001) between plasma CPK and blood cholinesterase

levels.[19]Severe organophosphorus poisoning caused pseudo cholinesterase levels to decrease, showing a strong correlation between initial CPK values and plasma pseudo cholinergic levels. This finding was statistically significant (P 0.001) in the study by Chellappan et al. Plasma volume CPK and serum cholinesterase showed a very substantial inverse connection, according to Wali et al.[97] Lokesh et al. also noted that when the severity of OP poisoning increases, less pseudocholinesterase is produced.

Our research's findings were in agreement with those of Rayavannar et alstudy .'s in that a high blood plasma CPK level, a large drop in serum ChE levels, and a rise in POP score were all determined to be risk factors for mortality. In the study by Chellappan et al., it was shown that people with high starting CPK levels had a considerable fatality rate.[98] Mural et al. found that severe degrees of poisoning, difficulties, and mortality are all associated with high initial serum CPK levels.[93] Serum CPK levels must be evaluated at the time of initial presentation, and they must be monitored throughout time since they have a significant role in how patients' conditions develop, according to Balasubramaniyan et al.[101]

CONCLUSIONS

CONCLUSIONS

- A total of 100 OP poisoned subjects meeting the inclusive criteria are incorporated into the final analysis. The mean age was 46.33±13.47 years in our study population with 72% males and 28% females.
- Occupation wise, 21% are farmers, 19% housewives, 15% are shop keepers, 13% students, 10% drivers, 9% daily laborers, 6% employed in organizations, 3% teachers and 2% engineers.
- In our study, 45% had miosis with < 2mm pupils, 1% had 2 mm miosis, 20% had > 2 mm miosis and 34% had pin point pupils. Among the OP toxidrome symptoms, sweating is manifested in 92%, 54% had excessive salivation, 59% had fasciculations, 14% had neck lift, 89% had emesis, 30% had seizures.
- With regard to the blood parameters, the mean PCO2 is 38.82±8.88, mean HCO3 is 22.92±4.25, mean hemoglobin (g/dl) is 11.86±1.19, mean total leukocyte count (cells/cmm) 12.21±3.00, mean Urea (mg/dL) 46.57±15.35, mean Creatinine (mg/dL) 0.92±0.24.
- Among the study population, 39% were conscious, 36% had a poor reaction to instructions, and 6% had a poor response to vocal instructions.
- The mean Blood PH is 7.32±0.06, mean respiratory rate 31.98±4.39, mean heart rate (bpm) 53.66±11.36 in the study population.
- The mean Serum creatinine phosphokinase at admission (mcg/l) is 1,034.38±1,169.13 ranging from 89 to 6,789 with 95% C. I (805.23, 1263.53).
- The mean Pseudocholinesterase (U/L) is 2,188.68±2,567.03 ranging from 200 to 7,890 respectively with 95% C. I (1685.54, 2691.82).
- According to the POP Scale, 36% are mildly poisoned, 51% moderately and 13% had severe poisoning.

- In our study, 9% are dead and the survival rate is 91%.
- The mean duration of stay (in days) is 11.78±6.21.
- OP smell is observed in all the patients.
- The mean Serum CPK after one week or at discharge is 1,110.54±1,811.23 ranging from 70 to 8789 with 95% C. I (755.54, 1465.54).
- Intermediate syndrome is observed in 14% of the study group and 17% required intubation.
- The mean Serum CPK at admission (mcg/l) is 387.53 ± 410.34 in those with mild poisoning according to the POP scale, 864.52 in the moderately poisoned group and 2728.38 ± 1817.88 in the severely poisoned patients. The difference in Serum CPK at admission (mcg/l) among different grades of poisoning on POP scale with a p value of <0.001, it is numerically relevant.
- The mean Serum CPK at admission (mcg/l) is 3132.11 ± 2039.02 in those who are dead and it was 826.91 ± 806.48 in the survivors. The difference in Serum CPK at admission (mcg/l) between mortality is statistically significant with p value < 0.001.
- There was a weak negative correlation between Serum CPK at admission (mcg/l) and pseudo cholinesterase (U/L) with r value -0.040 and the difference between Serum CPK at admission (mcg/l) and pseudo cholinesterase (U/L) with a p value of 0.001, it is numerically relevant.
- The likelihood of dying was raised by an elevated POP score, a marked decline in serum ChE levels, and an increase in CPK levels.

Limitations and Recommendations

Because our study had a limited number of participants and was directed in a single institution in South India, we may be biased in our assessment of the correlation, efficacy, and accuracy of CPK values on the degree of OP toxicity in other racial groups. The absence of serial CPK measurements is a flaw in this study. We need more multicentric research with larger sample sizes to confirm our findings.

SUMMARY

SUMMARY

Early evaluation and management of OP intoxicated incidences is of paramount importance in reducing mortality and morbidity. The mean Serum CPK at admission (mcg/l) is 387.53 ± 410.34 in those with mild poisoning according to the POP scale, 864.52 in the moderately poisoned group and 2728.38 ± 1817.88 in the severely poisoned patients showing a there is a substantial positive connection between admission CPK and poisoning severity (p value 0.001). The mean blood plasma CPK at admission (mcg/l) is 3132.11 ± 2039.02 in those who are dead and it was 826.91 ± 806.48 in the survivors, higher the initial serum CPK, higher is the risk for mortality. The difference in Serum CPK at admission (mcg/l) between mortality is statistically significant with p value < 0.001. There was a weak negative relationship between blood plasma CPK at admission (mcg/l) and pseudo cholinesterase (U/L) with r value -0.040 and the difference between Serum CPK at admission (mcg/l) and pseudo cholinesterase (U/L) is numerically relevant with a p value < 0.001. The risk of mortality was heightened by greater POP scores, a significant drop in serum ChE levels, and an increase in serum CPK levels. In conclusion, early serum CPK can be suggested as a developing indication for predicting the severity of OP poisoning and identifying patients who need intensive care first due to its affordability, ease of accessibility, and strong correlation with mortality. It is important to rule out possible origins of increased CPK before interpreting blood CPK in individuals with acute OP poisoning.

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ANNEXURES

PROFORMA FOR DATA COLLECTION

Name:

Age:

Sex:

Occupation:

UHID number:

Phone number:

Address:

DOA:

DOO:

DOD:

Complaints with duration:

Previous history:

Family history:

Past history:

General physical examination:

- Built and nourishment:
- Pallor/Cyanosis/Icterus/Clubbing/edema/Generalized lymphadenopathy
- OP smell

Vital data:

- Pulse:
- Temperature:
- BP:
- Respiration rate:

Systemic examination:

- Per abdomen:
- Respiratory system:
- Cardio vascular system:
- Central nervous system:

  Consciouness

Neck lift

Pupill

Fasiculations

INVESTIGATIONS

COMPLETE HEMOGRAM

PERIPHERAL SMEAR

RANDOM BLOOD SUGAR

BLOOD UREA

SERUM CREATININE

SERUM ELECTROLYTES

ECG

CHEST X RAY

SPECIFIC PARAMETER

CREATININE PHOSPHOKINASE (CPK)

CONSENT FORM

Title: -SERUM CREATININE PHOSPHOKINASE LEVEL AS A SEVERITY MARKER IN ORGANOPHOSPHOROUS POSIONING

Principal investigator: Dr.P.AMULYA

I, Mr/Ms/Mrs. …….. Have been explained in my own understandable language, that I will be included in a study which is SERUM CREATININE PHOSPHOKINASE LEVEL AS A SEVERITY

MARKER IN ORGANOPHOSPHOROUS POSIONING in RL Jalappa Hospital.

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

I have been explained my participation in this study is entirely voluntary and 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.P.AMULYA

Phone number- 9963083256

Participant’s signature/ thumb impression

Name:

Signature/thumb impression of the witness: Date:

Name:

Relation to patient

ಒಪ್ಪಿಗೆ ಪತ್ರ

ಶೀರ್ಷಿಕೆ: -ಸೆರಮ್ ಕ್ರಿಯೇಟಿನೈನ್ ಫಾಸ್ಫೋಕಿನೇಸ್ ಮಟ್ಟವನ್ನು ತೀವ್ರತೆಯಂತೆ

ಆರ್ಗನೊಫಾಸ್ಫೊರಸ್ ಸ್ಥಾನದಲ್ಲಿ ಮಾರ್ಕರ್

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ.ಪಿ.ಅಮುಲ್ಯ

ನಾನು, ಶ್ರೀ / ಕುಮಾರಿ / ಶ್ರೀಮತಿ. .... ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಇದನ್ನು ಸೀರಮ್ ಕ್ರಿಯೇಟಿನೈನ್ ಫಾಸ್ಫೊಕಿನೇಸ್ ಮಟ್ಟSವು ಒಂದು ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗುವುದು

ಆರ್.ಎಲ್. ಜಲಪ್ಪ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಆರ್ಗನೊಫಾಸ್ಫೊರಸ್ ಸ್ಥಾನದಲ್ಲಿ ಮಾರ್ಕರ್.

ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಆವಿಷ್ಕಾರಗಳು, ತನಿಖೆಗಳು, ಸಂಶೋಧನೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದು ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

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

ಅಧ್ಯಯನದ ಅಪಾಯ / ಲಾಭದ ಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

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

ಈ ಅಧ್ಯಯನವು ಉದ್ಭವಿಸುವ ಯಾವುದೇ ಡೇಟಾ ಅಥವಾ ಫಲಿತಾಂಶದ ಬಳಕೆಯನ್ನು ನಿರ್ಬಂಧಿಸದಿರಲು ನಾನು

ಒಪ್ಪುತ್ತೇನೆ, ಅಂತಹ ಬಳಕೆಯು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ.

ವಿಚಾರಣೆಗಾಗಿ ನನ್ನ ಬಳಿ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ ಇದೆ.

ಚಿಕಿತ್ಸೆಯ ಅವಧಿಯುದ್ದಕ್ಕೂ ಗುಣಮಟ್ಟದ ಆರೈಕೆಯನ್ನು ನಿರ್ವಹಿಸಲಾಗುವುದು ಎಂದು ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ.

ನಾನು, ನನ್ನ ಉತ್ತಮ ಮನಸ್ಸಿನಲ್ಲಿ, ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ತನಿಖಾಧಿಕಾರಿ: ಡಾ.ಪಿ.ಅಮುಲ್ಯ

ದೂರವಾಣಿ ಸಂಖ್ಯೆ- 9963083256

ಭಾಗವಹಿಸುವವರ ಸಹಿ / ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ

ಹೆಸರು:

ಸಾಕ್ಷಿಯ ಸಹಿ / ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ದಿನಾಂಕ:

ಹೆಸರು:

ರೋಗಿಗೆ ಸಂಬಂಧ

PATIENT INFORMATION SHEET

Study title : SERUM CREATINE PHOSPHOKINASE LEVEL AS A SEVERITY MARKER IN ACUTE ORGANOPHOSPHATE POISONING- A CROSS SECTIONAL STUDY

Principal investigator: Dr PENUMATSA AMULYA, Dr. B.N.RAGHAVENDRA PRASAD I Dr.PENUMATSA AMULYA, Post graduate student in Department of general medicine at Sri Devraj Urs Medical College, will be conducting a study titled"SERUM CREATINE PHOSPHOKINASE LEVEL AS A SEVERITY MARKER IN ACUTE ORGANOPHOSPHATE POISONING- A CROSS SECTIONAL STUDY" . This study will be useful for further management of Acute organophosphorous poisoning in the near future. The funds needed for the serum creatine phosphokinase levels will be done at my own expense .2 ml of blood will be drawn for estimation of serum creatine levels , from each of the participating patients in this study . This study will be done under the guidance of Dr.B.N.RAGHAVENDRA PRASAD,HOD & Professor of Department of GENERAL MEDICINE .

All the data will be kept confidential and will be used only for purpose specified by the institution. You are free to provide consent for the participation of yourself in this study. You can also withdraw yourself from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution.

In case of any clarifications are needed you are free to contact me on this mobile number - 9963083256

Name and Signature of the Principal Investigator
Date-

Patient or patient bystanders Signature

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನ ಶೀರ್ಷಿಕೆ: ಸೀರಮ್ ಕ್ರಿಯೇಟೈನ್ ಫಾಸ್ಫೋಕಿನೇಸ್ ಮಟ್ಟ ಒಂದು ತೀವ್ರತೆಯ ಮಾರ್ಕರ್ ಆಗಿ ತೀವ್ರವಾದ ಆರ್ಗನೊಫಾಸ್ಫೇಟ್ ವಿಷಕಾರಿ - ಒಂದು ಅಡ್ಡ ವಿಭಾಗೀಯ ಅಧ್ಯಯನ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ ಪೆನುಮತ್ಸ ಅಮೂಲ್ಯ, ಡಾ. ಬಿ.ಎನ್.ರಾಘವೇಂದ್ರ ಪ್ರಸಾದ್

ಶ್ರೀ ದೇವರಾಜ ಅರಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನ ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗದಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿಯಾದ ಡಾ ಪೆನುಮತ್ಸ ಅಮೂಲ್ಯ ಅವರು "ಸೆರುಮ್ ಕ್ರಿಯೇಟಿನೈನ್ ಫಾಸ್ಫೋಕಿನೇಸ್" ಎಂಬ ಅಧ್ಯಯನವನ್ನು ತೀವ್ರ ಅಂಗೊಫಾಸ್ಫೇಟ್ ವಿಷಪ್ರಾಶನ- ಅಡ್ಡ ಅಧ್ಯಯನದ ಭಾಗವಾಗಿ ನಡೆಸಲಿದ್ದಾರೆ. ಈ ಅಧ್ಯಯನವು ಮುಂದಿನ ದಿನಗಳಲ್ಲಿ ತೀವ್ರ ಅಂಗಜನ್ಯ ವಿಷತ್ವದ ಮತ್ತಷ್ಟು ನಿರ್ವಹಣೆಗೆ ನೆರವಾಗಲಿದೆ. ಸೀರಮ್ ಕ್ರಿಯೇಟಿನೈನ್ ಫಾಸ್ಫೋಕಿನೇಸ್ ಮಟ್ಟಗಳಿಗೆ ಅಗತ್ಯವಿರುವ ಹಣವನ್ನು ನನ್ನದೇ ಸ್ವಂತ ಖರ್ಚಿನಲ್ಲಿ ಮಾಡಲಾಗುತ್ತದೆ. ಸೆರಮ್ ಮಟ್ಟವನ್ನು ಅಂದಾಜು ಮಾಡಲು 2 ಮಿಲಿ ರಕ್ತವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವ ಪ್ರತಿ ರೋಗಿಗಳಿಂದ. ಡಾ. ಬಿ. ಎನ್. ರವೀಂದ್ರನಾಥ ಅವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಈ ಅಧ್ಯಯನ ನಡೆಯಲಿದೆ. ಮತ್ತು ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗದ ಪ್ರಾಧ್ಯಾಪಕರು.

ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿ ಇಡಲಾಗುತ್ತದೆ ಮತ್ತು ಸಂಸ್ಥೆಯು ನಿರ್ದಿಷ್ಟಪಡಿಸಿದ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಗೆ ಅನುಮತಿ ನೀಡಲು ನೀವು ಸ್ವತಂತ್ರರು. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ನಿಮ್ಮನ್ನು ಹಿಂದೆ ಸರಿಯಬಹುದು. ಇದರಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿಮ್ಮ ನಿರಾಕರಣೆಯು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿ ಯಾವುದೇ ವರ್ತಮಾನ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೈಕೆಯಲ್ಲಿ ನಿಮ್ಮನ್ನು ಪೂರ್ವಾಗ್ರಹಿಸುವುದಿಲ್ಲ.

ಯಾವುದೇ ಸ್ಪಷ್ಟೀಕರಣಗಳ ಅಗತ್ಯವಿದ್ದಲ್ಲಿ, ಈ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ - 9963083256 ರಲ್ಲಿ ನೀವು ನನ್ನನ್ನು ಸಂಪರ್ಕಿಸಲು ಉಚಿತ.

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಗಳ ಹೆಸರು ಮತ್ತು ಸಹಿ

ದಿನಾಂಕ-

ರೋಗಿ ಅಥವಾ ರೋಗಿಯ ಸಹಿ

MASTER CHART

S.No	Age	Gender	Occupation	Miosis	PCO2	hco3	Sweating	Salivation	Fasciculations	Neck lift	Vomiting	Blood ph	RR	Heart rate	CPK at admission (mcg/l)	PESUDOCHLINESTERASE (U/L)
1	34	Male	Farmer	pin point	35	18	Present	Present	None	Absent	Present	7.23	32	38	987	200
2	23	Male	Student	pin point	34	15	Present	Present	Present	Absent	Present	7.2	36	42	1013	345
3	56	Male	Farmer	pin point	20	15	Present	Present	Present	Absent	Present	7.3	38	30	1235	200
4	30	Female	House wife	2 mm	20	20	Present	Present	Present	Absent	Present	7.34	26	64	402	1068
5	24	Female	Student	< 2mm	25	25	Present	Present	Present	Absent	Present	7.35	30	54	820	1200
6	34	Male	Student	pin point	20	25	Present	Present	Present	Absent	Present	7.3	28	56	940	200
7	25	Male	Student	>2mm	35	30	Present	Absent	None	Present	Present	7.4	24	66	102	3200
8	43	Male	Daily labourer	< 2mm	30	28	Present	Present	Present	Absent	Present	7.3	30	60	640	560
9	56	Male	Farmer	< 2mm	34	24	Present	Present	Present	Absent	Present	7.4	33	56	430	200
10	38	Male	Shop keeper	pin point	60	12	Present	Present	Present	Absent	Present	7.26	40	35	2067	200
11	45	Male	Shop keeper	< 2mm	35	26	Present	Present	Absent	Absent	Present	7.35	34	67	569	200
12	57	Male	Farmer	pin point	34	25	Present	Present	Present	Absent	Absent	7.3	25	66	480	1089
13	35	Male	Daily labourer	< 2mm	38	28	Present	Absent	Absent	Absent	Absent	7.4	26	68	340	3090
14	22	Female	Student	>2mm	28	25	Present	Absent	Absent	Present	Absent	7.43	28	72	128	220
15	32	Female	House wife	pin point	48	18	Present	Present	Present	Absent	Present	7.2	38	32	1290	200
16	39	Female	House wife	< 2mm	26	22	Present	Absent	Absent	Absent	Absent	7.3	28	48	348	3980
17	27	Male	Student	< 2mm	28	26	Present	Absent	Present	Absent	Absent	7.32	32	52	249	2390
18	38	Male	bussiness man	pin point	40	20	Present	Present	Present	Absent	Present	7.12	39	38	3880	200

19	30	Male	Shop keeper	>2mm	26	28	Present	Absent	Absent	Present	Absent	7.34	28	72	179	5690
20	53	Male	Farmer	pin point	45	20	Present	Present	Present	Absent	Present	7.3	38	30	2890	200
21	46	Male	Daily labourer	pin point	25	20	Present	Absent	Absent	Absent	Absent	7.3	33	65	1280	980
22	34	Female	House wife	< 2mm	32	25	Present	Present	Present	Absent	Absent	7.34	28	68	880	1250
23	54	Male	Farmer	pin point	28	18	Present	Present	Present	Absent	Present	7.28	30	58	1890	200
24	56	Male	Shop keeper	>2mm	32	20	Present	Present	Present	Absent	Present	7.34	36	68	346	1348
25	67	Male	Farmer	pin point	52	14	Present	Present	Present	Absent	Absent	7.1	40	38	2780	200
26	56	Female	House wife	< 2mm	35	25	Absent	Absent	Absent	Present	Absent	7.32	26	68	368	3465
27	54	Male	Farmer	< 2mm	37	26	Present	Present	Present	Absent	Present	7.3	34	56	760	200
28	22	Male	Student	< 2mm	36	27	Present	Absent	Absent	Absent	Present	7.45	30	60	1456	1456
29	58	Female	House wife	pin point	40	20	Present	Present	Present	Absent	Present	7.26	36	58	420	200
30	19	Male	Student	>2mm	45	28	Present	Absent	Absent	Present	Present	7.46	28	72	367	5678
31	47	Female	Daily labourer	< 2mm	38	20	Absent	Absent	Absent	Absent	Present	7.34	30	66	578	980
32	49	Male	Daily labourer	pin point	46	19	Present	Present	Present	Absent	Present	7.27	34	56	1288	200
33	57	Male	Farmer	< 2mm	36	26	Present	Present	Absent	Absent	Present	7.35	30	62	890	2346
34	38	Female	House wife	< 2mm	38	20	Present	Absent	Absent	Absent	Present	7.36	28	68	237	2567
35	58	Male	Driver	pin point	48	18	Present	Present	Present	Absent	Present	7.28	38	58	3456	200
36	36	Male	Engineer	pin point	56	15	Present	Present	Present	Absent	Present	7.2	38	36	5678	200
37	47	Male	Shop keeper	< 2mm	36	26	Present	Absent	Present	Absent	Present	7.3	32	57	1246	200
38	45	Female	House wife	< 2mm	37	23	Present	Absent	Absent	Absent	Present	7.43	26	63	221	3457
39	67	Female	House wife	pin point	38	20	Present	Present	Present	Absent	Present	7.27	38	56	1345	200
40	24	Male	Student	< 2mm	35	26	Present	Absent	Present	Absent	Present	7.34	30	55	120	2356
41	69	Male	Shop keeper	< 2mm	39	20	Present	Absent	Present	Absent	Present	7.32	36	58	360	1245
42	56	Female	House wife	pin point	40	22	Present	Present	Absent	Absent	Present	7.29	32	40	689	200
43	47	Male	Farmer	< 2mm	42	26	Present	Absent	Absent	Absent	Present	7.32	30	47	369	1245
44	38	Male	Driver	< 2mm	35	22	Absent	Absent	Absent	Absent	Present	7.36	25	67	126	5678
45	26	Male	Student	pin point	56	14	Present	Present	Present	Absent	Present	7.28	35	42	4678	200
46	56	Male	Watchmen	>2mm	35	24	Present	Absent	Absent	Present	Present	7.4	32	50	98	6875
47	48	Male	Farmer	>2mm	35	27	Present	Absent	Absent	Present	Present	7.34	36	65	356	7890
48	39	Female	House wife	< 2mm	35	24	Absent	Absent	Absent	Absent	Present	7.34	32	56	468	3456

49	34	Male	Driver	>2mm	36	27	Absent	Absent	Absent	Present	Present	7.32	28	64	123	7686
50	28	Female	Student	< 2mm	38	20	Present	Present	Present	Absent	Present	7.3	36	54	1345	456
51	43	Male	Shop keeper	>2mm	37	24	Present	Absent	Present	Absent	Present	7.29	30	58	897	1278
52	44	Male	Driver	< 2mm	35	20	Present	Present	Present	Absent	Present	7.34	28	48	1456	200
53	38	Male	Engineer	pin point	40	18	Present	Present	Present	Absent	Present	7.27	32	52	752	1477
54	35	Female	House wife	< 2mm	36	20	Present	Absent	Absent	Absent	Present	7.3	29	67	560	6899
55	58	Male	Employee	>2mm	35	26	Present	Absent	Absent	Absent	Present	7.35	32	62	89	6780
56	46	Female	House wife	< 2mm	38	22	Present	Present	Present	Absent	Present	7.36	30	58	380	200
57	57	Male	Shop keeper	pin point	48	25	Present	Present	Present	Absent	Present	7.3	38	48	1460	889
58	48	Male	Farmer	>2mm	45	20	Present	Absent	Absent	Absent	Present	7.35	35	56	145	5678
59	32	Male	Employee	< 2mm	35	25	Present	Absent	Absent	Absent	Present	7.45	28	62	155	2366
60	39	Male	Shop keeper	pin point	48	15	Present	Present	Present	Absent	Present	7.28	38	42	1456	200
61	56	Male	Farmer	< 2mm	36	25	Present	Absent	Absent	Present	Present	7.34	28	58	320	1560
62	68	Male	Driver	pin point	68	15	Present	Present	Present	Absent	Present	7.22	42	22	6789	200
63	69	Male	Shop keeper	< 2mm	36	26	Absent	Absent	Absent	Present	Present	7.45	22	62	1000	7890
64	54	Male	Farmer	< 2mm	42	28	Present	Absent	Present	Absent	Present	7.38	30	50	458	1238
65	49	Male	Employee	pin point	48	30	Present	Present	Present	Absent	Present	7.35	33	43	1240	346
66	46	Male	Farmer	>2mm	35	25	Present	Absent	Absent	Absent	Present	7.34	26	63	458	7869
67	57	Female	House wife	< 2mm	38	22	Present	Present	Present	Absent	Present	7.4	36	45	1400	1456
68	45	Female	Teacher	< 2mm	35	26	Present	Present	Absent	Absent	Present	7.32	32	62	367	200
69	63	Male	Shop keeper	>2mm	35	26	Present	Absent	Absent	Present	Present	7.3	28	64	156	6796
70	46	Male	Driver	pin point	30	24	Present	Present	Present	Absent	Present	7.29	36	46	1690	200
71	56	Male	Farmer	< 2mm	40	20	Present	Present	Present	Absent	Present	7.3	32	44	1570	200
72	34	Female	Teacher	< 2mm	50	18	Present	Present	Present	Absent	Present	7.27	38	36	1236	2356
73	56	Female	House wife	pin point	75	10	Present	Present	Present	Absent	Present	7.2	43	32	3457	200
74	45	Female	Employee	>2mm	45	25	Present	Absent	Absent	Absent	Present	7.3	27	56	358	1568
75	67	Male	Shop keeper	< 2mm	34	26	Present	Present	Present	Absent	Present	7.45	34	47	678	800
76	57	Male	Farmer	pin point	47	22	Present	Present	Present	Absent	Present	7.34	36	50	1500	200
77	34	Male	Shop keeper	>2mm	36	26	Present	Absent	Absent	Present	Absent	7.35	26	64	167	7890
78	26	Female	Student	< 2mm	36	28	Present	Absent	Present	Absent	Present	7.34	36	58	789	1256

79	59	Male	Driver	< 2mm	36	26	Present	Absent	Present	Absent	Present	7.32	32	28	1738	200
80	47	Male	Daily labourer	< 2mm	38	24	Present	Present	Present	Absent	Present	7.3	37	48	367	2567
81	58	Male	Farmer	>2mm	34	26	Present	Absent	Absent	Present	Present	7.35	28	56	256	7890
82	48	Female	House wife	>2mm	37	27	Present	Absent	Present	Absent	Present	7.32	29	62	120	5678
83	67	Male	Daily labourer	pin point	43	29	Present	Absent	Absent	Absent	Present	7.29	30	62	356	200
84	35	Male	Driver	< 2mm	48	26	Absent	Absent	Absent	Absent	Present	7.35	26	46	2356	6889
85	29	Male	Shop keeper	< 2mm	34	25	Present	Absent	Absent	Present	Present	7.37	28	67	118	4566
86	36	Male	Employee	>2mm	36	28	Present	Present	Absent	Absent	Present	7.45	29	57	567	2900
87	48	Male	Driver	pin point	38	25	Present	Absent	Absent	Absent	Present	7.34	28	62	126	200
88	53	Male	Daily labourer	pin point	36	28	Present	Present	Present	Absent	Present	7.32	32	46	780	4569
89	57	Male	Shop keeper	< 2mm	38	20	Present	Present	Present	Absent	Present	7.28	36	35	1568	200
90	68	Male	Farmer	< 2mm	47	18	Present	Present	Present	Absent	Present	7.3	32	42	899	200
91	36	Female	House wife	< 2mm	39	20	Present	Absent	Present	Absent	Present	7.4	29	54	780	1457
92	45	Male	Daily labourer	pin point	42	24	Present	Present	Present	Absent	Present	7.35	32	48	569	200
93	56	Male	Driver	>2mm	43	26	Present	Present	Present	Absent	Present	7.32	36	62	125	7890
94	54	Male	Employee	< 2mm	36	27	Present	Present	Absent	Absent	Present	7.35	34	46	346	3456
95	67	Male	Farmer	pin point	48	22	Present	Present	Absent	Absent	Present	7.45	32	56	1245	200
96	68	Male	Teacher	< 2mm	48	26	Present	Present	Present	Absent	Present	7.35	28	36	3567	200
97	43	Male	Farmer	pin point	56	20	Present	Absent	Present	Absent	Present	7.32	32	46	460	5678
98	32	Female	Student	< 2mm	35	26	Present	Absent	Present	Absent	Present	7.28	30	57	1098	200
99	78	Female	House wife	>2mm	46	24	Absent	Absent	Present	Absent	Present	7.32	36	62	340	7890
100	65	Female	House wife	pin point	39	26	Present	Present	Present	Absent	Present	7.35	37	56	1467	200

S.No	POP SCALE	Seizures	LEVEL OF CONSCIOUNESS	Mortality	Duration of stay (in days)	op smell	cpk after one week or at discharge	Intermediate syndrome	Intubation	hb	tlc	urea	creatinine
1	Moderate	Absent	No response to verbal commands	Survived	15	Present	688	Absent	No	13.2	6.4	52	1
2	Severe	Present	No response to verbal commands	Death	20	Present	2457	Absent	Yes	12.4	7.8	70	1.2
3	Severe	Present	No response to verbal commands	Death	25	Present	5680	Present	Yes	14.5	11.8	62	0.9
4	Moderate	Absent	impaired response to verbal	Survived	7	Present	210	Absent	No	11.2	13.8	40	0.6
5	Moderate	Absent	impaired response to verbal	Survived	10	Present	524	Absent	No	12.2	8.8	28	0.6
6	Moderate	Absent	impaired response to verbal	Survived	15	Present	820	Absent	No	13.2	10.2	38	0.9
7	Mild	Absent	responsive to oral commands	Survived	6	Present	70	Absent	No	11	9.2	40	0.9
8	Moderate	Absent	No response to verbal commands	Survived	10	Present	450	Absent	No	11.4	10.2	23	0.5
9	Moderate	Absent	responsive to oral commands	Survived	10	Present	328	Absent	No	10.8	12.3	42	0.9
10	Severe	Present	No response to verbal commands	Death	28	Present	8789	Present	Yes	13.2	15.2	85	1.4
11	Mild	Absent	Conscious	Survived	6	Present	345	Absent	No	11.9	13.6	50	0.9
12	Moderate	Absent	impaired response to verbal	Survived	7	Present	670	Absent	No	10.9	11.2	40	0.8
13	Mild	Absent	conscious	Survived	7	Present	240	Absent	No	11.2	15.9	35	0.9
14	Mild	Absent	conscious	Survived	6	Present	80	Absent	No	10.2	11.2	45	0.8
15	Severe	Present	No response to verbal commands	Death	20	Present	3090	Present	Yes	12.3	15.2	50	1.4
16	Moderate	Absent	impaired response to commands	Survived	7	Present	156	Absent	No	11.9	10.2	40	0.7
17	Moderate	Absent	impaired response to commands	Survived	10	Present	110	Absent	No	12.7	11.9	35	0.6
18	Severe	Present	No response to verbal commands	Death	32	Present	4020	Present	Yes	8.9	18.9	70	1.2
19	Mild	Absent	conscious	Survived	6	Present	128	Absent	No	13.2	16.2	39	0.8

20	Severe	Present	No response to verbal commands	Survived	28	Present	2500	Present	Yes	11.8	16.7	70	1.2
21	Moderate	Absent	impaired response to commands	Survived	8	Present	890	Absent	No	12.5	11.8	42	0.9
22	Mild	Absent	conscious	Survived	10	Present	460	Absent	No	11.8	16.8	45	0.8
23	Moderate	Present	impaired response to commands	Survived	12	Present	878	Absent	No	11.6	14.7	38	0.7
24	Mild	Absent	impaired response to verbal	Survived	7	Present	130	Absent	No	12.8	11.7	40	0.6
25	Severe	Present	No response to verbal commands	Death	32	Present	4353	Present	No	12	14.7	128	2.1
26	Mild	Absent	Conscious	Survived	6	Present	135	Absent	No	9.2	11.3	43	0.7
27	Moderate	Absent	impaired response to commands	Survived	12	Present	589	Absent	No	11.2	8.7	38	0.6
28	Moderate	Absent	impaired response to commands	Survived	14	Present	659	Absent	No	13.5	6.8	25	0.8
29	Moderate	Absent	impaired response to commands	Survived	28	Present	3467	Present	Yes	11.2	10.3	48	0.7
30	Mild	Absent	conscious	Survived	8	Present	268	Absent	No	12.8	11.5	43	0.9
31	Mild	Absent	conscious	Survived	6	Present	189	Absent	No	13.6	13.6	36	0.8
32	Moderate	Absent	impaired response to commands	Survived	14	Present	870	Absent	No	10.9	11.5	45	1
33	Mild	Absent	conscious	Survived	10	Present	350	Absent	No	13.5	14.6	37	0.9
34	Mild	Absent	impaired response to commands	Survived	8	Present	267	Absent	No	11.3	11.6	40	0.9
35	Moderate	Present	No response to verbal commands	Survived	17	Present	4089	Present	Yes	11.4	16.7	56	1.2
36	Severe	Present	No response to verbal commands	Death	5	Present	6784	Absent	Yes	14.6	15.2	78	1.4
37	Moderate	Absent	impaired response to commands	Survived	15	Present	890	Absent	No	11.6	14.5	45	0.9
38	Mild	Absent	impaired response to commands	Survived	12	Present	127	Absent	No	11.8	13.5	43	0.6
39	Moderate	Absent	impaired response to commands	Survived	19	Present	2345	Present	No	11.9	11.8	38	0.7
40	Moderate	Absent	impaired response to commands	Survived	10	Present	110	Absent	No	10.7	14.6	39	0.8
41	Moderate	Absent	impaired response to commands	Survived	8	Present	258	Absent	No	14.6	5.8	46	0.7
42	Moderate	Absent	No response to verbal commands	Survived	16	Present	566	Absent	Yes	9.6	8.9	53	1.1
43	Mild	Absent	Conscious	Survived	10	Present	126	Absent	No	10.8	10.2	46	0.8
44	Mild	Absent	Conscious	Survived	7	Present	110	Absent	No	10.6	15.7	40	0.7
45	Moderate	Absent	No response to verbal commands	Survived	25	Present	5678	Present	Yes	11.5	11.6	89	1.7
46	Mild	Absent	Conscious	Survived	5	Present	100	Absent	No	11.7	17.2	46	1
47	Mild	Absent	Conscious	Survived	5	Present	124	Absent	No	12.5	11.5	43	0.9
48	Moderate	Absent	impaired response to commands	Survived	8	Present	123	Absent	No	11.6	12.6	38	0.8
49	Mild	Absent	Conscious	Survived	6	Present	97	Absent	No	11.6	13.7	39	1

50	Moderate	Present	impaired response to commands	Survived	13	Present	879	Absent	No	11.8	6.7	40	0.7
51	Moderate	Absent	impaired response to commands	Survived	12	Present	356	Absent	No	10.8	9.8	38	0.9
52	Moderate	Absent	impaired response to commands	Survived	10	Present	890	Absent	No	11.6	10.8	56	1
53	Moderate	Absent	No response to verbal commands	Survived	10	Present	540	Absent	Yes	10.6	16.7	46	0.9
54	Mild	Absent	Conscious	Survived	6	Present	126	Absent	No	11.8	12.6	43	1.1
55	Mild	Absent	Conscious	Survived	6	Present	92	Absent	No	11.3	14.6	26	0.6
56	Moderate	Absent	impaired response to commands	Survived	13	Present	240	Absent	No	11.5	11.6	39	0.9
57	Severe	Absent	No response to verbal commands	Survived	16	Present	1008	Absent	No	10.7	15.7	56	1
58	Mild	Absent	Conscious	Survived	7	Present	100	Absent	No	11.6	11.7	48	0.9
59	Mild	Absent	Conscious	Survived	6	Present	110	Absent	No	11.7	9.8	38	1
60	Moderate	Absent	No response to verbal commands	Survived	18	Present	1267	Absent	No	12.8	11.2	46	1.5
61	Mild	Absent	Conscious	Survived	10	Present	112	Absent	No	10.8	14.7	39	0.8
62	Severe	Present	No response to verbal commands	Death	7	Present	7898	Absent	No	12.7	17.8	80	0.9
63	Mild	Absent	conscious	Survived	8	Present	890	Absent	No	11.8	11.6	38	0.8
64	Moderate	Absent	impaired response to commands	Survived	16	Present	340	Absent	No	9.4	17.8	22	1
65	Severe	Present	No response to verbal commands	Survived	18	Present	1678	Absent	No	10.8	15.7	48	0.8
66	Mild	Absent	Conscious	Survived	5	Present	458	Absent	No	11.6	11.5	46	1
67	Moderate	Absent	impaired response to commands	Survived	10	Present	1256	Absent	No	14.6	7.6	50	0.9
68	Mild	Absent	Conscious	Survived	7	Present	125	Absent	No	11.8	12.7	89	0.9
69	Mild	Absent	Conscious	Survived	7	Present	126	Absent	No	14.7	11.6	37	0.8
70	Severe	Present	No response to verbal commands	Survived	17	Present	3456	Absent	No	12.5	15.7	42	0.9
71	Moderate	Absent	impaired response to commands	Survived	10	Present	1000	Absent	No	11.6	13.6	46	1
72	Moderate	Absent	impaired response to commands	Survived	12	Present	890	Absent	No	11.8	12.6	36	0.9
73	Severe	Present	No response to verbal commands	Death	5	Present	7890	Absent	Yes	9.8	16.8	58	1.3
74	Mild	Absent	Conscious	Survived	6	Present	146	Absent	No	11.9	10.8	47	0.9
75	Moderate	Absent	No response to verbal commands	Survived	13	Present	350	Absent	No	11.5	7.8	36	0.8
76	Moderate	Present	impaired response to commands	Survived	10	Present	982	Absent	No	10.8	10.8	49	1
77	Mild	Absent	Conscious	Survived	6	Present	89	Absent	No	14.7	16.8	26	0.9
78	Moderate	Absent	impaired response to commands	Survived	8	Present	567	Absent	No	11.6	15.7	46	1
79	Moderate	Absent	impaired response to commands	Survived	16	Present	578	Present	Yes	10.7	13.8	34	0.8

80	Moderate	Absent	impaired response to commands	Survived	18	Present	324	Absent	No	12.6	12.8	57	0.6
81	Mild	Absent	Conscious	Survived	7	Present	236	Absent	No	11.6	16.8	34	0.7
82	Mild	Absent	impaired response to commands	Survived	6	Present	145	Absent	No	12.4	11.7	45	0.8
83	Mild	Absent	Conscious	Survived	5	Present	125	Absent	No	11.6	8.9	42	0.9
84	Mild	Absent	Conscious	Survived	8	Present	2567	Absent	No	11.7	7.8	40	1
85	Mild	Absent	Conscious	Survived	7	Present	98	Absent	No	11.4	12.6	37	0.9
86	Mild	Absent	Conscious	Survived	10	Present	125	Absent	No	13.6	15.7	42	1
87	Mild	Absent	Conscious	Survived	9	Present	120	Absent	No	11.9	7.9	38	0.8
88	Moderate	Absent	impaired response to commands	Survived	14	Present	356	Absent	No	13	10.8	46	1
89	Moderate	Absent	impaired response to commands	Survived	20	Present	1250	Absent	No	11.2	9.8	49	0.9
90	Moderate	Present	No response to verbal commands	Survived	22	Present	540	Present	Yes	13.6	10.8	50	1.2
91	Moderate	Present	impaired response to commands	Survived	13	Present	460	Absent	No	11.5	9.8	47	0.9
92	Moderate	Absent	impaired response to verbal	Survived	16	Present	348	Present	Yes	10.8	10.7	43	0.5
93	Moderate	Absent	No response to verbal commands	Survived	12	Present	89	Absent	No	11.6	7.9	34	0.8
94	Moderate	Absent	No response to verbal commands	Survived	9	Present	256	Absent	No	12.5	8.7	45	0.9
95	Moderate	Absent	impaired response to commands	Survived	14	Present	678	Absent	No	10.8	13.6	56	1.2
96	Moderate	Absent	impaired response to commands	Survived	13	Present	689	Absent	No	13.6	7.8	46	0.8
97	Moderate	Absent	impaired response to commands	Survived	10	Present	213	Absent	No	11.5	8.9	57	1
98	Moderate	Present	impaired response to commands	Survived	16	Present	578	Present	Yes	10.8	10.2	48	1.1
99	Moderate	Present	No response to verbal commands	Survived	13	Present	134	Absent	No	13.4	11.3	37	0.8
100	Moderate	Absent	impaired response to commands	Survived	10	Present	897	Absent	No	11.4	9.7	78	0.9