"A HOSPITAL BASED LONGITUDINAL STUDY ON THE ELECTROCARDIOGRAPHIC FINDINGS IN ACUTE ORGANOPHOSPHORUS POISONING (OPP) WITH SPECIAL REFERENCE TO CORRECTED QT INTERVAL (QTC)."

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ENDORSEMENT

This is to certify that the dissertation entitled **"A HOSPITAL BASED LONGITUDINAL STUDY ON THE ELECTROCARDIOGRAPHIC FINDINGS IN ACUTE ORGANOPHOSPHORUS POISONING (OPP) WITH SPECIAL REFERENCE TO CORRECTED QT INTERVAL (QTC)."** is a bonafide research work done by **R EG NO. BG0 1 1 5 0 0 7**

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LIST OF ABBREVIATIONS USED

Ach	–	Acetylcholine
AChE	-	Acetyl Cholinesterase
APACHE II	-	Acute Physiology and Chronic Health Evaluation II
CNS	-	Central Nervous System
CPK	-	Creatine Phosphokinase
DEF	-	Tribufos
DNA	-	Deoxyribonucleic Acid
e.g.	-	For Example
ECG	-	Electrocardiogram
GCS	-	Glasgow Coma Scale
GI	-	Gastrointestinal
hrs	-	Hours
i.e.	-	That is
IPCSPSS	-	International Program on Chemical Safety Poison Severity Score
ICU	-	Intensive Care Unit
IM	-	Intramuscular
IMS	-	Intermediate Syndrome
LD	-	Lethal Dose
LDH	-	Lactate Dehydrogenase
MPM II	-	Mortality Prediction Model II
mEq/L	-	Milli Equivalents Per Liter
mg/dL	-	Milligrams Per Deciliter

mg/kg	-	Milligrams Per Kilogram
ml	-	Milliliter
mm	-	Millimeter
mRNA	-	Messenger Ribonucleic Acid
n	-	Total Number
NPIC	-	National Poison Information Center
NTE	-	Neuropathy Target Esterase
OP	-	Organophosphorus
p	-	Probability
P=O	-	Phosphorous Oxygen Bond
P2AM	-	Pralidoxime
PAM	-	Pralidoxime
POP	-	Peradenya Organophosphorus Poisoning
Qtc	-	Corrected Qt interval
RBCs	-	Red Blood Cells
SAPS II	-	Simplified Acute Physiology Score II
ChE	-	Serum Cholinesterase
SGOT	-	Serum Glutamic Oxaloacetic Transaminase
SGPT	-	Serum Glutamic Pyruvic Transaminase
U/L	-	Units per Liter
WHO	-	World Health Organization
VPC	-	Ventricular Premature Complies

ABSTRACT

Aims and Objectives

To study the Electrocardiographic findings in Acute OPP and to correlate the changes in corrected QT(QTc) interval with the severity of organophosphorous poisoning and to evaluate this relationship as a prognostic utility in OPP.

Methodology

The present one year hospital based longitudinal study was done on a total of 102 patients admitted with Acute organophosphorus compound poisoning in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2016 to December 2016. Standard 12 lead ECG done at admission in Casualty or ICU and at the time of discharge was studied and QTc interval was calculated using Bazett's formula. The **severity of OP poisoning** was assessed based on the Peradeniya Organophosphorous Poisoning (POP) Scale, The number of days of hospitalization, requirement of Mechanical ventilation and Glasgow Comma Scale scoring at admission.

Results

Maximum number of cases had an age of 21-30 years (47.06%) and the mean age was 31.50980392 ± 3.57years. There was male preponderance with a male to female ratio of 1.4:1. The most commonly consumed compound was chlorpyrifos (46.08%). Majority of the patients (75.49%) presented after 3 to 6 hours of consumption. The most common symptom was nausea and vomiting (98.04%) and POP score revealed mild intoxication in 53.892% of the patients. ECG Tracing revealed 61 patients (59.80%) had tachycardia while 3 patients (2.94%) had

bradycardia and in remaining 38 patients (37.25%) normal heart rate was noted. Prolonged Qtc was noted in 90 patients (88.23%) while a prolonged PR interval was noted in 14 patients (13.72%). We observed that the Mean QTc interval at Admission was higher (489.14ms) that than the QTc interval at Discharge (452.22ms). T inversion in 7 patients (6.86%), ST elevations in 6 patients (5.88%), VPCs in 4 patients (3.92%) and atrial Fibrillation was seen in 2 patients (1.96%). Sepsis was noted to be the commonest complication in the study i.e 21 patients (52.50%), Mortality was noted in 12.75% of the patients and ventricular tachycardia was the commonest cause (69.23%).

Conclusion

The commonest ECG abnormality noted was prolonged QTc followed by tachycardia and prolonged PR interval. We found that patients with a higher severity of poisoning had a longer corrected Qtc interval.

Patients with longer corrected QT intervals had lower GCS scores on admission and were more likely to require Mechanical ventialation. They also had a longer duration of hospital stay, a longer duration of atropine use, with an increase in complications and a poorer outcome . This observation suggests that corrected Qtc interval is a useful prognostic utility in Organophosphorus poisoning.

Keywords

Organophosphorus compound poisoning; Peradeniya Organophosphorus Poisoning (POP) Scale; Prolonged Corrected QT interval

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

INTRODUCTION

Organophosphorus compounds constitute a varied array of chemicals formulated for pest control, weeds in the agriculture industry. Their use has contributed to substantial increase in agricultural productivity and crop yields and is still the most effective and efficient means for plant protection from pests [1]. In the field of medicine, Some organophosphorus compounds have also been used in the medical treatment of myasthenia gravis, glaucoma and Alziehmer's disease and Parkinson's disease. [2,3].

Common OP compounds used in agriculture are parathion, malathion, chlorpyrifos, and dichlorvos and the population of India being largely dependant on the agriculture industry, coupled with easy availability of organophosphorus (OP) compounds, makes it a most common modality of poisoning. [4]

Organophosphorus poisoning is a world wide public health issue. According to World Health Organization (WHO), the number of pesticide poisoning cases mounts to 3 million every year, resulting in more than 250,000 deaths. [5]

The National Crime Bureau of India estimates that 19.4 and 19.7% of suicides due to all causes of poisoning in the year 2006 and 2007 occurred due to consumption of pesticides alone.[6]

Organophosphorus agents or their metabolites cause poisoning by halting the enzyme acetylcholinesterase [7] which is responsible for breaking down and inactivating the neurotransmitter acetylcholine in the synaptic junction. This causes an increase in the accumulation of acetylcholine at the synapses and increased binding to the muscarinic receptors and the nicotinic receptors and in the central and peripheral nervous system. As a result, Cardiovascular manifestations are commonly observed

after exposure to organophosphorus compounds, but their exact nature is not fully explained. [8].

On studying the electrocardiogram (ECG) in patients with OP poisoning, a variety of abnormalities may be revealed, such as ST segment and T-wave abnormalities, sinus tachycardia or bradycardia, atrioventricular block and more uncommonly extreme QT interval prolongation and ventricular tachycardia following torsades de pointes. [9]

Karki P et al. in their study reported that ECG changes can be seen as early as the first hour after exposure to organophosphate compounds, Sinus tachycardia and prolonged QTc interval being the most common abnormalities in their study. [10]

The QT interval represents the total duration of ventricular depolarization and ensuing repolarization (11). But this value cannot be used as a comparative variable as it varies with the heart rate. The QT interval is inversely proportional to the heart rate and thus is longer at slower heart rates and shorter at faster heart rates. Many formulae have been discovered to adapt for this variation. The corrected QT interval (QTc) as formulated by Bazett is most frequently used. Studies have been carried out which show that the QTc interval has prognostic implications in several conditions, such as stroke and migraine, but its role in organophosphate poisoning is ambiguous (12,13). However several studies have emerged in the recent past to explore this previously ambiguous association.

Grmec et al. reported that patients with QTc-interval prolongation had respiratory failure, indicating a severe poisoning, with increased in hospital morbidity and mortality compared with those with normal QTc interval in OPP. (14)

Chuang et al. reported that patients with QTcinterval prolongation had a higher mortality ratio, compared with those without QTc-interval prolongation, and that increased QTc interval had a prognostic value in OPP. (15)

Archana Deshpande et. Al reported that QTc intervals have shown to be good in predicting respiratory failure and hospital mortality in patients with organophosphorous poisoning. [16]

Therefore, this prospective study was carried out to study the Electrocardiographic findings in Acute OPP and to correlate the changes in corrected QT(QTc) interval with the severity of organophosphorous poisoning and to evaluate this relationship as a prognostic utility in OPP in a tertiary care hospital.

AIMS AND OBJECTIVES

To study the Electrocardiographic findings in Acute OPP and to correlate the changes in corrected QT (QTc) interval with the severity of organophosphorous poisoning and to evaluate this relationship as a prognostic utility in OPP.

REVIEW OF LITERATURE

ORGANOPHOSPHATE COMPOUNDS

Organophosphorus compounds (OPs) are the chemical compounds containing carbon-phosphorus bond. Organophosphate (OP) is a term that can be utilized to depict every single substance compound in which a phosphate croup, or phosphate subsidiary, is in some portion of a natural (i.e. carbon-containing) particle. Practically speaking, it is used to refer to those organophosphorus mixes which restrain the compound cholinesterases namely Red blood cell acetylcholinesterase (RBC AChE), plasma or pseudocholinesterase and neuropathy target esterase (NTE) in humans and animals. Human exposure leads to both muscarinic and nicotinic receptor stimulation leading to various manifestations.[17] They have been utilized as a part of agrarian and agricultural pesticides, a few veterinary meds (especially in sheep to avoid and treat sheep scab and other parasitic infestations), in human pharmacy malathion was used as a treatment for head lice, and in different open cleanliness items, both for use by pest control e.g. for the control of cockroaches and other bugs in housing areas and for use in populated buildings like schools and hospitals.

The OP compounds are not only used as insecticides and pesticides but also in petroleum industry and more infamously in warfare.

Epidemiology

Poisoning with OP compounds is a worldwide phenomenon. The global burden of fatalities has been estimated by the World Health Organization (WHO) to about 300,000 out of the 3,000,000 exposed to OP compounds annually and in 2008 about 8000 reported cases were from the United states alone of which there were less than 15 deaths. [18,19,20]. Out of the 3 million annual Op poisonoing cases , one

million serious unintentional poisonings occur every year and an additional two million people are hospitalized for suicide attempts with pesticides.[21,17] Chlorpyrifos, the organophosphate agent used widely in farming and found in many popular household cockroach and ant sprays, including the Raid brand is a common cause of OP compound poisoning in India. The compound was outlawed in 2001 by the United States Environmental Protection Agency (EPA) in household use and imposed strict limits on its use in the agriculture industry, especially in growing common fruits like tomatoes, apples, and grapes [22]. However With the invention of carbamate compounds, the use of OP compounds has significantly declined in the last 2 decades. [23].

Data from the National Poison Information Center (NPIC) in All India Institute of Medical Sciences, New Delhi suggests that suicidal poisoning with household agents such as OP compounds pyerthrinoids, rodenticides and corrosives was the commonest modality of poisoning. Pesticides used in agricutre accounted for 12.8% of all cases of poisoning. [24,25]

Hospital based data suggests that Op compounds have become the most commonly used agents in central and south India for suicidal poisoning, replacing the earlier agents like copper sulfate and barbiturates in the 1970s. [17, 26]

In 2012 a cross sectional study of 1 year duration to study the Clinico-Epidemiological Characteristics of Patients Presenting with Organophosphorus Poisoning, conducted in a tertiary medical college in Kolkata consisting of 968 patients revealed that the average age of presentation was 34.47 years and there was a female preponderance in the Male to female ratio of 1:1.38.About 82 % with poisoning had a suicidal intent versus a 18% to those who presented with accidental exposure. The average age of presentation was 34.47 years and there was a female

preponderance in the Male to female ratio of 1:1.38. Majority of the patients were housewives (42%) followed by farmers, shopkeepers, laborers, students respectively and the commonest poison consumed was methyl parathion (35.74%) followed by diazinon, chlorpyriphos, dimicron respectively. A total of 56 patients (5.78%) died in the study with the most common cause being respsiratory failure.[27]

In contrast to this, Another study from Andhra Pradesh in which more than half the study group were males with more than two-thirds of patients less than 30 with suicidal intent being the most common form of exposure. Majority of the deaths were due to monocrotophos. [25]

More common that accidental exposure to OP compounds is their intentional use for self harm. These are the most commonly used agents for suicidal poisoning in India [30] being an agrarian country where such compounds are available over the counter. Oral ingestion is usually the most common route of exposure [31] and a study from Sri Lanka showed that young age, poverty ,unemployment, marital disharmony, alcohol abuse and psychiatric disorders were the main problems associated with suicidal poisoning which consisted of 85% of the study group.[32]

A Brief History

Lassaigne combined OP mixes around the mid 1800s. In any case, the earliest recorded portrayal of their sysnthesis was by De Clermont in 1854. In this manner, Michaelis in Germany and Arbusov in Russia depicted combination of countless op mixes by the mid 1900s. Be that as it may, the harmful impacts of OP mixes were not perceived until 1932, when Lange and Von Kruger depicted the lethal impacts of OP vapors.[33]

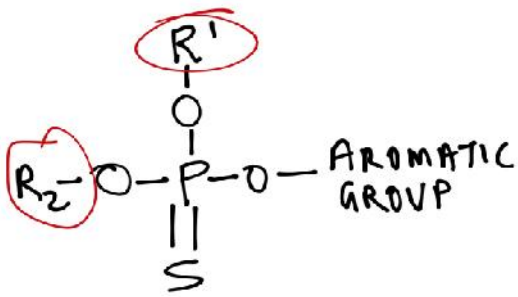
Triorthocresyl phosphate (TOCP) was isolated in the gigantic epedemic of Ginger-Jake plaralysis in individuals who devoured polluted alcoholic concentrate of Jamaican ginger in the United States in 1930s. The toxicity of OPs used in agriculture was first noted in the UK among sheep workers was recognized as a cause for concern only in the early 1950s.

The infamous OP nerve agents (eg, tabun , sarin , soman) developed in Germany during world war 2 as a result of Schrader's research, were not actually used for widescale military purposes [34]. It was not a public concern until The 1995 Tokoyo Subway Attacks using Sarin nerve gas which brought about the recognition of such agents being a tool for bioterrorism and resulted in the consequent research on prevention and treatment of exposures to such agents. [35-37]

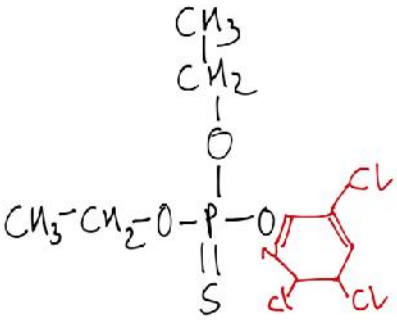
Classification

Organophosphorus compounds were initially classified by the WHO in 1973 and this has been periodically updated to include a distinction between the toxicity of these compounds . [38].The below classification of organophosphorus compounds has been adapted to classify commonly encountered compounds based on their toxicity, active or inactive form and structure i.e based on Diethyl (CH3CH2),Dimethyl (CH3) groups and S-Alkyl groups: [38,39]

WHO CLASSIFICATION	DIETHYL	OXON/ THION	DIMETHYL	OXON/ THION	S-ALKYL	OXON/ THION
Ia Extremely hazardous (LD50: 1 to 50 mg/kg) Ib Highly hazardous (LD50: 51 to 500 mg/kg)	Phorate Triazophos Parathion	Thion Thion Thion	Monocrotophos Methyl parathion Dichlorvos	Oxon Thion Oxon		
II Moderately hazardous (LD50: 501 to 5000 mg/kg)	Quinalphos Chlorpyrifos Ethion	Thion Thion Thion	Dimethoate Fenthion Phenthoate	Thion Thion Thion	Profenfos	Oxon
III Slightly hazardous (LD50: more than 5000 mg/kg)			Malathion Acephate	Thion Oxon		



GENERAL ORGANOPHOSPHORUS STRUCTURE



CHLORPYRIFOS

Figure showing the General structure of an Organophosphorus compound and that of the Diethyl compound - chlorpyrifos.

Mechanism of action

The amount of of OP compound that can be consumed with a certainty of zero harmful effects is as low as 0.02 mg/kg for malathion, and 0.004 mg/kg for parathion [40-42]

After exposure to an OP compound the duration of onset of signs and symptoms depends on the route of exposure and the type of compound involved. Due to the lipophilic nature of majority of the OP compounds they are easily absorbed across the Gastric and intestinal mucosa, the respiratory epithelia and skin. Oral ingestion, which is the most common route and inhalational exposure take less than 3 hours for onset of signs and symptoms while in skin exposure the onset may be delayed for up to 12 hours.

After absorption into the system the compound itself is broken down by the cytochrome p- 450 sysetm in the liver and various ezymes that hydrolise its ester bond. Organophosphorus pro-pesticides have a sulphur atom attached to the phosphorus atom (P=S) and are called phosphorothioates or thions. This sulphur must be replaced with an oxygen (P=O) to make the active metabolite called oxon or phosphate. Some compunds like monocrotophos already exist in the oxon for and are active the moment they are absorbed systemically. Pro-pesticides like chlorpyrifos (thions) are converted to its active metabolites which are distributed throughout the body and build up quickly in the adipose tissue and organs of the salivary glands, liver and kidneys and is able to cross the blood brain barrier. This property is owed to the general lipophilic nature of OP compunds however some are more lipophilic than others, examples like fenthion and parathion which are a phosphorothioates (thions) and are more lipophilic than dichlorvos which is phosphate and so due to excessive fat

deposition accounts for the sustained intoxication and clinical relapse after some initial improvement seen with Parathion.[43]

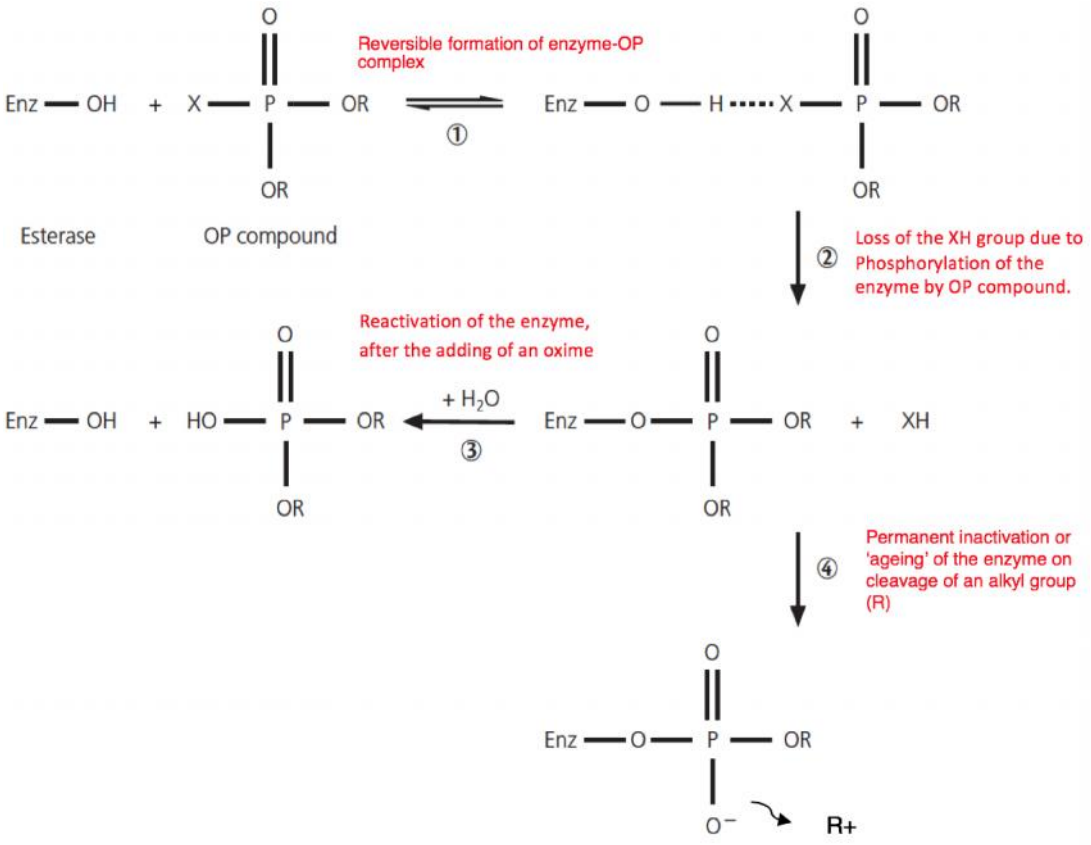


Figure showing the various stages of interaction between AChE enzyme and OP compound.

Hence the plasma half life in OP compounds also varies which is dependent on the type of compound and also on quantity of administration but most are eliminated within 48 hours via rapid clearance through urine and faeces.

Studies have shown a substantial difference in toxicity of OP compounds owing to its variable groups (R1 and R2) (see figure) which are composed of either methyl (CH3) or ethyl (CH3CH2) moieties. The mechanism of action of OP compounds renders acetylcholinesterase (AChE), more precisely RBC acetylcholinesterase, redundant. It does this by covalent binding of phosphate

radicals to enzymatic active site bringing about a structural transformational change thus forming a protein that is enzymatically ineffective.[1].

As a result there is a build-up of acetylcholine in the neuronal synapses and there is failure of its hydrolysis to choline and acetic acid which is brought about by the enzyme AChE. Depending on the chemical structure of OP compound this initial inhibition is reversible and the enzyme AChE can be reactivated (by the administration of oximes) for a certain time period after which it undergoes an irreversible structural change which is resistant to reactivation , known as "aging". [44]. This structural change is due to the loss of an alkyl group from the OP moiety and normal takes 48 to 72 two hours from the time of ingestion and so this period becomes the critical interval during which the enzyme can be reversed . [40-42]

For this reason, it is of vital importance to recognize the type of op compound involved as dimethyl (malathion, dimethoate) compounds undergo rapid aging, making the early administration of oxime therapy imperative and diethyl (parathion, chlorpyrifos) compounds delayed aging and toxicity which required prolonged oxime therapy. [19]

Spontaneous Cholinesterase recovery occurs very slowly via denovo synthesis of enzyme and spontaneous dephosphorylation at the rate of approximately 1 % a day. This varies between plasma AChE and RBC AChE, taking upto 4 weeks for plasma levels and longer for the RBC AchE levels to normalize. [40-42]

Clinical manifestations

As mentioned earlier, the clinical presentation of OP is dependent on the time since exposure, quantity of compound exposed, type of compound and the route of exposure. After ingestion symptoms appear within 30 mins to as late as 24 hours in some pro-pesticides compounds that require metabolic biotransformation. Another factor that affects the toxicity of compounds is the presence of other solvents in the solution such as petroleum fractions, xylene and cyclohexane with can cause emesis and CNS depression. [40, 41, 45, 46]

There are three distinctive clinical phases observed in Oraganophosphorus poisoning.[41]

• The Initial cholinergic phase.

• The intermediate syndrome (IMS)

• The Delayed polyneuropathy

The cholinergic phase [41,47,48]

Acetylcholine is responsible for neurotransmission at both the parasympathetic and sympathetic ganglia, in the terminal endings of all postganglionic parasympathetic nerves and at all the neuromuscular junctions.

The anatomical network of the parasympathetic nervous system is particularly dependent on acetylcholine regulation, since it is the chief neurotransmitter in the synapses of the autonomic ganglia (nicotinic) and end organs (muscarinic) it supplies[49,50]

The Chloinergic crises is mainly due to accumulation of Ach at the Cholinergic synapses and may be classified into

1) Muscarinic (all postganglionic nerve endings)

2) Nicotinic (Autonomic ganglia and skeletal muscle end plates).

3) CNS manifestations (synapses in CNS)

Overstimulation of the muscarinic receptors at the end organs results in Muscarinic Features first described as Wadia Type 1Syndrome [51] and consists of features such as miosis, salivation, bronchorrhea, bronchospasm , lacrimation, urination vomiting ,diarrhea and bradycardia.

Excessive stimulation of the nicotinic receptors present in the post synaptic neurons of the neuromuscular junction (NMJ) results in uncontrolled neuronal depolarization and ensuing neuromuscular blockade, similar to that caused by succinylcholine. These nicotinic Features first described as the Wadia type 2 syndrome consists of features such as Hypertension, Tachycardia, fasciculations, progressive muscle weakness, Respiratory depression and paralysis. Both muscarinic and nicotinic receptors are also present in the brain which explains clinical features of central respiratory depression, lethargy, headache, nightmares, insomnia, involuntary movements, seizures and coma in OP poisoning. [52]

The intermediate syndrome (IMS)

Intermediate (neurologic) syndrome

The initial cholinergic crises is due to the early and rapid inhibition of AChE enzyme at the muscarinic synapses while the nicotininc syndromic features in Op poisoning occur later after atleast 80% of the synaptic AChE is rendered ineffective. This is the basis for the delayed effects described in the “intermediate syndrome” of

OP poisoning after the initial cholinergic crises seen in severe OP poisonoing.. [17,53]

The term Intermediate refers to the timing of a syndrome that occurs after the initial cholinergic crises and before the delayed neurological effects of OP poisoning. There may be an apparent recovery from the inital cholinergic crisis but then muscle paralysis occurs early before the expected onset of delayed polyneuropathy.[54] First described as the Wadia type 2 syndrome , it was later recognized as a seperate entity in 1987 by Karalliedde and Senanayake .It was seen to occurs in about 10 to 40 percent of patients within as early as 24 hours to about 96 hours post exposure and is characterized by persistent muscle weakness which is proximal more than distal, including neck muscle weakness, sluggish deep tendon reflexes and cranial nerve palsies and respiratory distress due to diaphragmatic paralysis.[43,55,56]. It can be differentiated from delayed neurotoxicity through nerve conduction studies which reveal distinctive postsynaptic abnormalities while the later would reveal decreased firing of motor conduction units [57].

Among the proposed mechanisms for the development of this syndromes include the varying susceptibilities of various cholinergic receptors as mentioned earlier, prolonged AChE inhibition, failure to administer adequate doses of oximes before enzyme “aging” occurs, down regulation or desensitization of postsynaptic acetylcholine receptors, failure of postsynaptic acetylcholine release, and oxidative stress-related myopathy and exposure to a highly fat soluble (lipophilic) organophosphorus agent causing its deposition in fat stores and subsequent delayed release. [58,59]

Delayed polyneuropathy

This is a neuropathy that typically occurs 2 to 4 weeks after the initial cholinergic crises in certain specific organophosphorus agents, commonly chlorpyrifos but also described in malathion, trichlorfon and triorthocresylphosphate (TOCP) poisoning. [60,61]. This complication is unrelated to the initial severity of cholinergic crises as observed in parathion poisoning which often causes a severe cholinergic crises but seldom results in Organophosphorus agent induced delayed neuropathy (OPIDN). However, the opposite is seen in TOCP poisoning which causes only mild features of cholinergic crises but is often associated with OPIDN.[62]

The predominant enzyme affected resulting in this syndrome is neuropathy target esterase (NTE) which is found in the Central nervous tissue and Peripheral nervous tissue and lymphocytes and is responsible for cell homeostasis [63]. The enzyme dysfunction results in cell dysfunction and death which is seen in as Wallerian degeneration of large distal axons on histopathology sections.[64]

The clinical features results due to involvement of the distal muscle and nerve groups but can progress to involve the proximal muscle and nerve groups if the poisoning is severe. Initially there is often a temporary glove and stocking paresthesia associated with pain which is ensued by a bilateral weakness of the lower extremities associated with flaccid deep tendon reflexes which progresses in an ascending fashion to involve the upper limbs. This symmetrical polyneuropathy is mainly motor and sensory involvement is less .[65].

The cause of long term sequelae in severe OP poisoning is controversial and is difficult to ascertain if it is due to the neurotoxicity of the agent itself or due to hypoxic effects. Patients often end up with features of an upper motor neuron syndrome with spastic paralysis, wasting of distal muscles like the small muscles of

the hands and feet and permanent disability. However, In majority of the cases with mild neurodeficits, show improvement of motor and sensory functions with time. Other Long term sequelae described include cognitive deficits such as reduced memory, calculation and development of parkinsonism.[66]

Other effects of OP poisoning

Cardiovascular system

Bradycardia and low blood pressure occur as a result of muscarinic effect. Patients may also present with nicotinic features such as tachycardia and hypertension.[41]

Respiratory system

Respiratory — OP poisoning affects the respiratory system in an intrinsic manner by depression of the central respiratory center and in a extrinsic manner by causing neuromuscular paralysis of the diaphragm and other respiratory muscles, bronchospasm and excessive respiratory secretions. As mentioned earlier, bronchorrea and bronchospasm are the muscarininc effects ,while intercoastal and diapgagmatic muscle weaknes leading to respiratory paralysis is due to the nicotinic action.[41] Fatalities in acute organophosphorus agent poisoning generally result from its actions on the respiratory system leading to acute respiratory failure. Uncontrolled vasodilation leading to vasodilatory shock may also play a role in fatalities [67].

Other affected systems

Altered immunity to infection

Alteration in immunity in the form of immunosuppression is believed to be to due alterations in the neuropathy target esterase (NTE) present in lymphocytes , due to severe cholinergic stimulation and toxic chemical stresses on the bonemarrow.[41]

Effects on temperature regulation

Temperature regulation abnormalities in the form of hypothermia (incidence 7%) has been noted in several studies at the intial cholinergic phase of OP poisoning. Later this is reversed due to the anticholinergic effects of atropine. However a biphasic response has also been noted after the initial hypothermia when some patients may experience fever lasting for many days. [41,68]

Changes in glucose metabolism.

Patients with acute OP poisonoing are noted to have a transient hyperglycaemia and glycosuria which may be in part due to the increased sympathetic stimulation and due to the release of stress hormones during the acute stressful state. In comatose patients with a high random blood sugar level it is differentiated by Diabetic ketoacidosis by the absence of acetone bodies but may be difficult to differentiate from the hyperglycemic hyperosmolar non-ketotic coma state in diabetic patients.[41]

Management- Diagnosis and Treatment

Any patient who presents to the emergency with a history of poison consumption should be received and a quick evaluation should be carried out. The priority should be the immediate emergency and focus should be ABC of Cardiopulmonary resuscitation. Two IV lines should be secured and blood samples taken for haematological and biochemical analysis .ECG should be recorded. Profuse oral secretions, sweating, Laboured breathing, pin point pupil would suggest OP/carbamate poisoning.

Diagnosis of Organophosphorus poisoning

This is based on the history of ingestion of the OP compound or by identification of the OP compound based on the container label brought by the Patients' attender at hospital arrival, in the setting of the typical signs and symptoms of cholinergic crises. The quantification of acetylcholinesterase levels is routinely done in patients with OP poisoning. There are two types of acetylcholinesterase enzymes that can be quantified. On is the Direct measurement of the acetylcholinesterase activity in the RBC (RBC AChE) which provides a measure of the degree of toxicity. Serial quantification of RBC AChE can also be used to assess the success of oxime therapy in reactivation of the enzyme and help ascertain the need for prolonged therapy .However, the problem lies in that most hospital laboratories are not equipped to perform this test. Another more easily done and more regularly performed test is the assay of plasma cholinesterase also called butylcholinesterase [BuChE] or pseudocholinesterase activity reflects indirectly the RBC Ache, but is not associated with the severity of poisoning and should not be used to monitor oxime therapy [69]. Bio chemical evidence of depressed RBC cholinesterase or butyrylcholinesterase may only be present initially in less than 50% of patients.

However, treatment should not be delay in view of pending lab values when OP poisoning is suspected.

Patients who present to the hospital with alleged history of an unknown poison ingestion often have a garlic like/ petroleum odor about them ,especially on their stained clothes or in their gastric contents and are high suspects for OP compound poisoning. If doubt exists as to whether an organophosphate has been ingested, a trial of 1 mg atropine in adults (or 0.01 to 0.02 mg/kg in children) may be administered and absence of anticholinergic signs such as an increase in heart rate by more than 20-25 beats/min ,flushing, pupillary dilation strongly suggests a state of cholinergic excess.

Antidote

Atropine causes inhibition of the muscarinic receptors at the post synaptic memberane and is direct competition with the levels of acetylcholine in the synapse. It is the only life saving antidote in OP poisoning. A total of 38 regimes for atropinisation has been found in literature such as give a bolus loading dose followed by boluses after a fixed time interval varying from 5-15-30 minutes till atropinisation; Bolus loading dose followed by infusion. [70,71] The current recommended dosing for OP poisoning is to be individualized with an initial bolus dose of 2 to 5 mg IV for adults after which the dose is doubled every three to five minutes until the criteria for atropinisation is met.

The criteria for atropinisation is said to be met when there is clearance of respiratory secretions, bronchospasm and the Pupils no longer pin point and dry axilla is noted. [70,72].Tachycardia and Mydriasis are NOT useful therapeutic end points for atropinisation as they may persist due to hypoxia, shock or excessive sympathetic activity.[50]

There was a controversy that oxygen must be given before atropine bolus due to the risk of inducing ventricular arrhythmias in hypoxic patients however this was disproved in a cohort study showing it was not necessary to provide oxygen prior to initiating treatment with atropine [73].

A personalized atropine regimen based on individual therapeutic end points (atropinisation) was supported by a randomized trial in which a 156 patients who were treated with increasing doses of atropine followed by infusion, experienced lower mortality (6 deaths) and lower incidence of atropine toxicity than patients given a standard bolus dose followed by infusion (18 deaths) [74]. The duration of atropine therapy depends on the severity of poisoning and clinical response to treatment. Frequent assessment is required to detect early signs on Intermediate syndrome in which case it has to be continued [50]. It is important to remember that the requirement of atropine may be very high in patients with severe poisoning, amounting to near or more than hundred milligrams per day for over several days.

Once atropinisation is achieved its blood levels are to be maintained by atropine infusion as this reduces the fluctuation in atropine concentration associated with repeated bolus doses. The rate to be infused per hour is 10-20% of the total atropine required for atropinisation.[70] Adequate fluid replacement should also be done at about 500- 1000 ml of normal saline (10-20 ml/kg) should be given over 10-20 minutes to compensate fluid loss due tosweating, diarrhoea and increased secretions. This stays true even if the patients has features of bronchorrea as there is no evidence of harm as long as atropine is being given.[70]

Atropine administration by nebulisation also improves respiratory distress and oxygenation. If the patient is hypotensive and an IV access is difficult, atropine can be

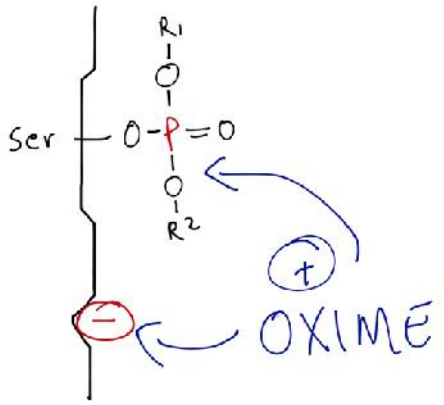
given through an endotracheal tube, intratracheally which in addition to local effects is absorbed systemically through the peribronchial vessels.[29,30]

Atropine toxicity

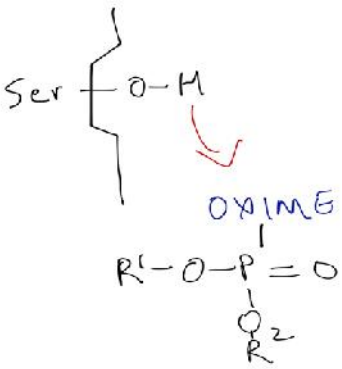
Patients on atropine infusion may develop signs of ileus, delirium , and tachycardia, which would suggest levels beyond the therapeutic end point of atropinisation, and would warrant a reduction in dose to 60%-70% of the previous infusion rate or temporary discontinuation. Hyperthermia is a serious complication in patients undergoing atropine therapy in hot wards which needs active cooling and prevention.[70]

Cholinesterase Reactivators

The role of Oximes as an antidote in OP poisoning is significant due to the inability of atropine to blockade the nicotinic Ach receptors, rendering it ineffective in treating neuromuscular dysfunction. However it does not cross the blood brain barrier and so is not useful in treating the CNS toxicity of OP compounds and has its highest activety in the skeletal muscles than the autonomic ganglia. Therefore its is clear that their use in OP poisoning is only an adjunct to that of atropine. Oximes act as cholinesterase reactivators and hence are effective in treating both the muscarinic and nicotinic adversities of OP poisoning [19,75,76]. Examples are Pralidoxime (P2AM), Asoxime Chloride (HI6) and obidoxime. These agents bind to the Cholinesterae enzyme by attaching to the free positively charged site on them and then react with phosphorus atom of the OP compound which is itself attached to the active site of the enzyme. This oxime phosphate complex diffuses away leaving the enzyme intact and hence reactivated for hydrolysis on acetylcholine.[77]



GRAPHICAL REPRESENTATION OF A POSITIVELY CHARGED OXIME ATTACK ON THE PHOSPHORYLATED SERINE RESIDUE OF AN INACTIVATED ACHE ENZYME.



FORMATION OF A PHOSPHORYLATED OXIME AND A REACTIVATED ACHE ENZYME.

However, it has been found that pralidoxime causes an initial, transient Ache inhibition and hence should not be administered alone, without simultaneous initiation of atropine therapy [78]. Also it should be noted that rapid IV boluses of Pralidoxime has been associated with sudden cardiac arrest and hence should be given slowly over half an hour which also reduces the initial , transient AChe inhibition and resulting muscle weakness [79].

The data on the usefulness of Pralidoxime in Acute OP poisoning is two faced and is not fully understood [44]. In a prospective study of 802 patients it was noted that those poisoned with diethyl compounds (eg, chlorpyrifos) had substantial lower death and intubation rates following treatment with pralidoxime than those poisoned with dimethyl agents (eg, dimethoate, fenthion) [46]. Conversely, in a smaller study of 235 patients, some were treated with pralidoxime and compared to other patients who were given placebo, irrespective of the type of op compound poisonoing. This was a double blinded placebo controlled randomized trial in which nil benefit with an additional detrimental tendency was observed in the patients treated with pralidoxime (in that mortality was nonsignificantly higher in the pralidoxime group) [80].

However, due to the lack of conclusive evidence of its possible hazards, its clear benefit in increasing levels of RBC Acetylcholinesterase and due to the lack of any better drugs, it is suggested that oxime therapy be given to all patients with evidence of cholinergic crises with neuromuscular dysfunction and those exposed to agents responsible for delayed neurotoxicity.

Oximes are to be administered as early as possible post ingestion to offer benefit but a patient arriving late is not a contraindication as the therapeutic window for Diethyl OP compound-AChe complex is 133 hours.[81,70]The intravenous bolus therapy as recommended by the WHO with pralidoxime is at least 30 mg/kg in adults which may be increased to 50 mg/kg based upon the severity of symptoms [44,82]. However it is most effective when this bolus dose is followed up with maintainence of a continuous infusion of at least 8 mg/kg per hour in adults.

The plasma oxime concentration that exerts a therapeutic effect is 4mg/litre but efficacy has also be been demonstrated in lower concentrations as well. In one study that Evaluated two treatment regimes of pralidoxime, the patients were randomized into two groups in which the first Group received a single bolus dose of 1gm P2AM at admission (Low dose group) followed by placebo infusion over the next four days and the second Group received placebo bolus at admission followed by P2AM 12gm as a continuous infusion over the next four days. The number of patients who developed Intermediate syndrome was significantly lesser in the low dose group suggesting that the time of administration of P2AM is a crucial factor which determines response to therapy.[83] The duration of therapy is recommended until clinical recovery i.e 12-24h after atropine is no longer needed or seven days have elapsed, whichever is later. Where highly lipophilic compounds such as dichlofenthion, fenthion, and malathion are involved (associated with delayed onset of symptoms up to 5 days and prolonged

illness of more than 30 days) and in severe poisoning the anti- AChE may be prolonged due to toxin redistribution to fat stores and maintenance infusion should be titred depending on the clinical response and amount of enzyme reactivation based on serial RBC acetylcholinesterase levels, if available [84]. Oximes have also been shown to have a beneficial effect in slowing progression to delayed complications of OP poisoning like the intermediate syndrome and delayed neuropathy (OIDN), but has not been effective in preventing it. [85].

Scoring systems in OP poisoning

There have been several studies carried out to asses the usefulness of various scoring systems to classify severity and predict mortality in Acute OP poisoning. A retrospective study involving 396 patients was done to asses the performance of various standard clinical scoring systems employed in the ICU setting. In this study the Mortality Prediction Model II (MPMII) scoring system, Simplified Acute Physiology Score II (SAPSII) and the Acute Physiology and Chronic Health Evaluation II (APACHEII) scoring systems did better than the International Program on Chemical Safety Poison Severity Score (IPCS PSS) in predicting mortality. [86] Also in another prospective study of 1365 patients to examine the prognostic factors in Acute op or carbamate poisoning showed that the Glasgow Coma Score (GCS) of less than 13 was as effective as the International Program on Chemical Safety Poison Severity Score (IPCS PSS) in indicating a poor prognosis.[87].

However, in all these comparative studies it is to be noted that the performace of these scoring systems were dependant of the OP agent involved. In the study mentioned the APACHEII scoring was most useful for the compounds quinalphos and chlorpyrifos least in in monocrotophos. On the other hand the non survivors poisoned with the compound fenthion had only mild symptoms at presentation.

Hence care must be taken when assessing severity in poisoning with lipophilic OPs such as fenthion, triorthocresylphosphate (TOCP) and parathion, as these patients may exhibit slow onset and drawn out poisoning symptoms.

Cardiotoxicity in OP poisoning

How do op compounds act on the heart?

The cardiac complications associated with organophosphate poisoning are not fully appreciated by many medical practitioners. Organophosphate poisoning may precipitate complex ventricular arrhythmias, a frequently overlooked and potentially lethal aspect of this condition [88]

The mechanism by which organophosphates induce cardiotoxicity is still uncertain. In 1982, Ludomirsky et al [8] described 3 phases of cardiac toxicity after organophosphate poisoning: phase 1, a brief period of increased sympathetic tone; phase 2, a prolonged period of parasympathetic activity; and phase 3, in which QT prolongation is followed by torsade de pointes ventricular tachycardia and ventricular fibrillation [8].

Hypertension and sinus tachycardia, which may be seen in organophosphate poisoning, are nicotinic effects, while hypotension and sinus bradycardia are cholinergic manifestations [89]. Although bradycardia is thought to dominate in the early cholinergic phase of the poisoning, sinus tachycardia was a more frequent finding in our study.

The same observation has also been made by others [90,91]. Bradycardia develops secondary to augmented vagal influence that shortens the effective refractive period of atrial myocytes and increases the refractive period and conduction time of

the SA and AV mode [68]. Some investigators consider the presence of hypertension and sinus tachycardia to be manifestations of severe poisoning [92].

Both sympathetic and parasympathetic overactivity have been shown to cause myocardial damage.(95-97) As early as 1974, Yasue et al postulated that parasympathetic overactivity plays a major role in coronary artery spasm, and later Horio et al (98) induced coronary artery spasm in adult humans with healthy coronary arteries after intracoronary injection of acetylcholine. In a series of 168 cases of organophosphate poisoning reported by Kiss and Fazekas (93) five had a transient picture of myocardial infarction. Diffuse myocardial damage was found at necropsy in two cases of malathion poisoning (an old generation organophosphate) (99) and diffuse myocarditis has been reported after carbamate poisoning.(94)

In a study of 13 Indian patients who died of organophosphate poisoning, Annad et al [100] found on autopsy, all 13 patients had myocardial interstitial edema and vascular congestion, while 8 had patchy interstitial inflammation, 2 had patchy myocarditis, and 6 had a mural thrombus.

What are the ecg changes noted commonly in op poisoning and their mechanism?

The ECG may display a variety of abnormalities in acute organosphate poisoning. [101-103]Classically, cardiac rhythm in organophosphate poisoning consists of two phases: a transient phase of intense sympathetic tone, causing sinus tachycardia, followed by a second phase of extreme parasympathetic tone, causing sinus bradycardia, atrioventricular block, and ST segment and T wave abnormalities. Prolongation of the QT interval has also been commonly noted. [8]

Qt prolongation

It has been well demonstrated that QT-interval prolongation affects mortality rate in the general population [101]. QT-interval prolongation also affects the mortality rates of patients with a variety of diseases, including end-stage renal disease [102], coronary artery disease, congestive heart failure [103], diabetes mellitus [104], acute ischemic stroke [105], chronic liver disease [106], and chronic obstructive pulmonary disease [107].

Sometimes, QT prolongation has been observed in cases of severe bradycardia or disease of the central nervous system. Therefore, it is not surprising to find a QT prolongation in a case of severe organophosphate poisoning presenting with extremely intense autonomic discharge and/or coma in consciousness. In 1975, Luzhnikov et a1(109) reported 183 cases of severe organophosphate poisoning. Various arrhythmias and conduction disturbances were observed in 34 patients (18.5%). All patients with arrhythmias had a prolonged QT interval, correlating with the severity of poisoning and decrease of cholinesterase activity in the blood.

It is clear that the phenomenon of long Q-T interval-torsades in OP poisoning is unrelated to serum electrolyte imbalance or the use of atropine. Although abnormalities like hypokalemia, hypomagnesemia and hypocalcemia can prolong the Qt interval, Serum electrolytes were normal in most of the patients of Kiss and Fazekas (93). Atropine has likewise never been reported to produce these electrocardiographic abnormalities, although it is sometimes implicated as the cause of classic ventricular arrhythmias, including tachycardia and fibrillation.(9,108) However, Ludomirsky et al. and Lyzhnikov et al. found no correlation between atropine therapy and ventricular arrhythmias in OP poisoning (8,109).

The long QT-interval syndrome is believed to originate from intense and unequal sympathetic stimulation of myocardial fibers. Both sympathetic and parasympathetic overactivity may cause QT-interval prolongation, and it is not surprising to find QTinterval prolongation in cases of severe organophosphate poisoning. Possible mechanisms include sympathetic and parasympathetic overactivity, hypoxaemia, acidosis, electrolyte derangements and a direct toxic effect of the compounds on the myocardium (10). Excessive cholinergic innervation of the heart results in both negative chronotropy and negative inotropy that slows myocardial conduction or repolarization [8].

It is well documented that QT prolongation in the rat is explained in terms of blockade of the Ito potassium channels and the Na+/Ca+2 exchanger [110]. Muscarinic action induced by OP intoxication causes vagotony and acetylcholine accumulation on nerve endings, which results in spastic contraction of the coronary artery has also been a proposed mechanism of qt prolongation [111]. Although the nonspecific ST-T change has generally been recognized as being not directly related to any cardiac diseases, it has been observed before starting the ST elevation caused by coronary spasm [112]

Predisposing factors for QT prolongation and development of TdP that requires meticulous care even in mild OP-poisoned patients include: older ages, female gender, low left ventricular ejection fraction, left ventricular hypertrophy,Acidosis, ischemia and electrolyte abnormalities including hypokalemia and hypomagnesemia (88).

<u>Does prolonged qtc have a role in prediciting the severity of OP poisoning?</u>

Prolonged Qtc may induce VT and even cause death if timely identification and intervention is not instituted. This cardiac effect was also the most common

finding in studies done in Nepal, Turkey and India though, its frequency was higher (37.8%, 55.5%, 62.5%, respectively). In overall, the frequency of QTc prolongation in several series of severe OP poisoning was shown to be 20 to 80% depending on the severity of the poisoning (10,104,122,128-129). This complication usually starts during the second to third day and may last up to two weeks post intoxication (109). The relationship between QTc interval prolongation and subsequent mortality after organophosphate poisoning remains uncertain.

In a preliminary study at Chang Gung Memorial Hospital, Chuang et al reported that patients with QTc prolongation had a higher mortality rate (19.6% vs. 4.8%, P,0.001) and a higher incidence of respiratory failure (56.7% vs. 20.6%, P,0.001) than patients without QTc prolongation. [15] Shadnia et al [115] reported that the mortality rate of Iranian patients with long QTc intervals was significantly higher than that of those with normal QTc intervals.

Another study done by Ravikumar et al [116] in Department of General Medicine, Silchar Medical College, Silchar, Assam, India In 2016 Majority of the patients with sinus tachycardia (76%), hypertension (92%) and hypotension (83%) were in the highest severity grade.. Among 28 patients with prolonged corrected QT interval, 86% were in the highest severity grade as per Poison Severity Score (IPCS PSS) developed by the International Program on Chemical Safety and the European Community.

In a subsequent study, Akdur et al [117] found that 26 of 54 (53.7%) Turkish patients had prolonged QTc intervals. However, no significant correlation was found between poisoning severity and QTc interval. More recently [49], Vijayakumar evaluated 20 Indian patients with organophosphate poisoning and discovered that 12

patients (60.0%) had prolonged QTc intervals. However, the predictive power of QTc interval prolongation on subsequent mortality was not explored.

In contrast, Baydin et al [118] reported that 35.4% of the 20 Turkish patients studied presented with prolonged QTc intervals. Further, there was a negative correlation between QTc interval and blood cholinesterase level. In a study of 13 Indian patients who died of organophosphate poisoning, Annad et al [100] found 4 with episodic tachycardia and ST-T changes, 3 with QT prolongation, and 2 with episodic bradycardia.

Yurumez et al [113] analyzed the records of 85 Turkish patients with organophosphate poisoning and found that 47 patients (55.5%) had a prolonged QTc interval. Only 2 patients died in this study (2.4%), and no QTc prolongation was found in either patient (QTc intervals: 0.44 s and 0.40 s).

The authors therefore concluded that QTc interval prolongation could not be used as a unique predictive factor in determining short-term mortality in their study [113].

METHODOLOGY

This study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2016 to December 2016.

Study design and duration

A one year hospital based longitudinal study.

Study period

The present study was conducted from January 2016 to December 2016.

Place

The present study was conducted in Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum a tertiary care teaching hospital attached to Jawaharlal Nehru Medical College, Belgaum.

Source of Data

The study comprised of patients with organophosphorus compound poisoning admitted in the wards and ICUs of the Department of General Medicine.

Sample size

A total of 102 patients with organophosphorus compound poisoning were studied.

Sampling procedure

The total number of organophosphorus poisoning cases in the last three years was 220 according to the Department of Medical Records, KLES Dr. Prabhakar Kore Hospital and Research Centre, Belgaum. Considering 80% of the annual average number of cases with organophosphorus compound poisoning during the last three years, the sample size was determined as a minimum of 100 cases. However during the study period 102 cases were admitted with organophosphorus compound poisoning all of whom were included in the study.

Selection criteria

INCLUSION CRITERIA

1.Ages above 18years

2.Patients with history of consumption of organophosphorous compounds based on history and have confirmed toxicology reports post admission.

EXCLUSION CRITERIA

1. Patients who have consumed other toxic compounds.
2. Patients who are known cases of cardiac diseases. Diabetes mellitus; respiratory, renal and/or hepatic failure; surgical operation in the past 7 days.
3. Patients on anti-arrhythmic agents, macrolides, fluoroquinolones, anti-malarials, pentamidine and azole anti-fungals, the anti-psychotics and antidepressants.

Ethical clearance

Prior to the commencement, the ethical clearance was obtained from Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belgaum.

Informed Consent

All the patients fulfilling selection criteria were explained about the nature of study and a written informed consent was obtained before enrollment (Annexure II). In case of patients with altered mental status, the relatives of the patients were briefed about the nature of study and written informed consent was obtained.

Method of collection of data

A predesigned and pretested proforma (Annexure I) was used to record and collect the necessary data. This began with collection of the Demographic data such as age and sex after which the patients / relatives were interviewed regarding the chief complaints and presenting history which included the time of exposure to organophosphorus compound, type of compound, quantity consumed, route of exposure, time delay to hospital arrival and were noted as reported

A thorough physical, clinical and systemic examination was carried out. Based on these findings, The Glasgow coma score and the Peradeniya Organophosphorus Poisoning (POP) Scale clinical criteria score was calculated.

Investigations

The selected patients underwent the following investigations.

- Complete blood count
- Urine routine
- Liver function tests
- Renal function tests
- Serum electrolyte levels
- Serum cholinesterase levels
- Electrocardiogram
- Toxicology evaluation of gastric lavage.

Study variables

The patients were evaluated for following study variables

Electrocadiogram

Standard Digital 12 lead Electrocardiograms will be taken and patient Only standard 12 lead ECG done at admission in Casualty or ICU and at the time of discharge will be studied and QTc interval will be calculated using Bazett's formula.

Bazett's formula =QTc = QT/√RR; Normal value (350-430ms)

Severity of organophosphate compound poisoning

The severity of organophosphate compound poisoning was determined by Peradeniya Organophosphorus Poisoning (POP) Scale clinical criteria score. The POP scale assesses the severity of the poisoning based on the symptoms at presentation and is simple to use. [119]

Peradeniya Organophosphorus Poisoning (POP) Scale Clinical criteria Score

Pupil size	**Respiratory rate**	**Heart rate**	**Fasciculation**	**Level of consciousness**	**Seizures**
>2 mm 0	<20/min 0	>60/min 0	None 0	Conscious and rationale 0	Absent 0
					Present 1
<2 mm 1	>20/min 1	41–60/min 1	Present, generalized or continuous 1	Impaired response to verbal commands 1	
Pin-point 2	>20/min with central cyanosis 2	<40/min 2	Both, generalized and continuous 2	No response to verbal commands 2	

Interpretation of POP score

• Mild poisoning - A score of 0 to 3

• Moderate poisoning - A score of 4 to 7

• Severe poisoning - A score of 8 to 11

Glasgow Comma Scale scoring at admission.

Best Eye Response. (4)	**Best Verbal Response. (5)**	**Best Motor Response. (6)**
1. No response	1. No verbal response	1. No motor response.
2. Eye opening to pain.	2. Incomprehensible sounds.	2. Extension to pain.
3. Eye opening to verbal command.	3. Inappropriate words.	3. Flexion to pain.
4. Eyes open spontaneously.	4. Confused	4. Withdrawal from pain.
	5. Orientated	5. Localising pain.
		6. Obeys Commands.

Serum cholinesterase levels

The estimation of serum cholinesterase levels was done by PCHE method using Flex reagent cartridge manufactured by Siemens Dimension clinical chemistry system. Pseudocholinesterase levels were estimated at the time of admission and to monitory therapy patient. The lowest pseudocholinesterase levels were also noted. The serum cholinesterase levels between 7000 to 19000 U/L were regarded as normal.

Ventilator support and duration of ventilation

Number of patients requiring ventilation and duration of ventilatory support in each patient were noted.

Complications

Patients were monitored for the complications during their stay in the hospital.

Length of stay in the hospital

The duration of hospital stay was recorded.

Duration of atropine use.

The duration of atropine in days use was noted in the study subjects.

Outcome

The eventual outcome of the patient was noted as a survivor or a non – survivor.

Statistical analysis

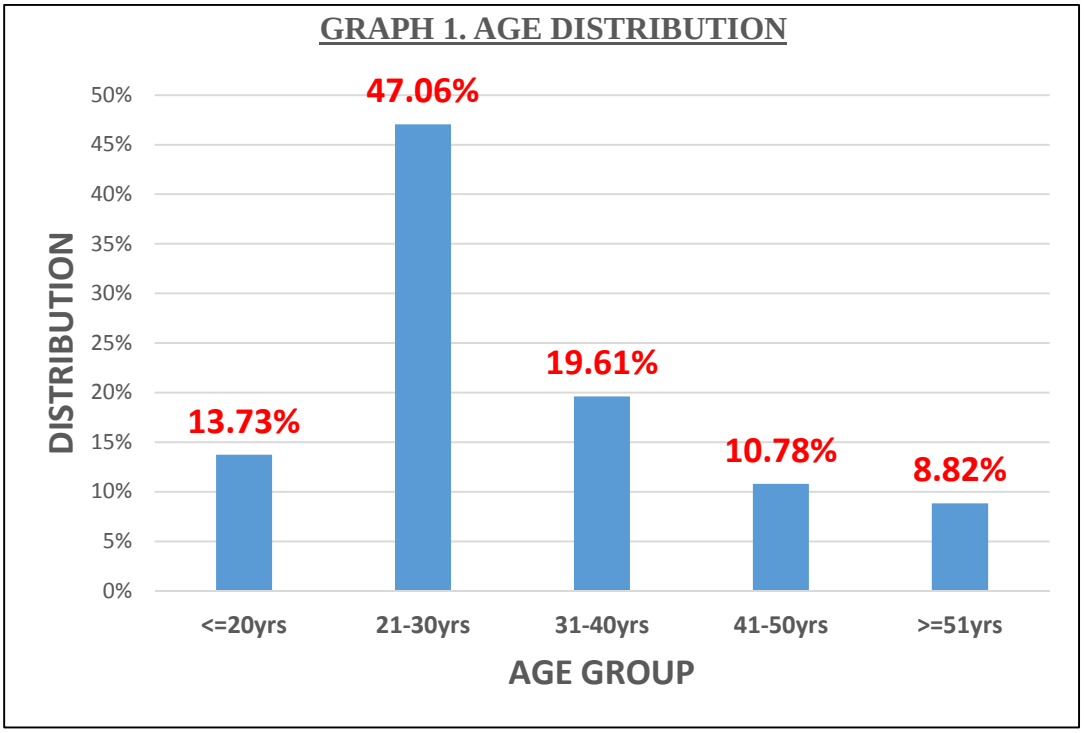
The data obtained was coded and entered into Microsoft Excel Worksheet (Annexure III). Data was analysed using SPSS statistical software version 20.0 The categorical data was expressed as rates, ratios and proportions and comparison was done using either chi-square test or Fisher.s exact test. The continuous data was expressed as mean ± standard deviation (SD) and comparison was done using independent sample .t. test. A probability value (.p. value) of less than or equal to 0.05 was considered as statistically significant.

RESULTS

The present one year hospital based longitudinal study titled " A hospital based longitudinal study on the electrocardiographic findings in acute organophosphorus poisoning (OPP) with special reference to corrected QT interval (QTc)" was carried out in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. During the study period from January 2016 to December 2016, a total of 102 patients admitted with organophosphorus compound poisoning were studied. The findings / observations and final results are tabulated as below.

TABLE 1. Age distribution

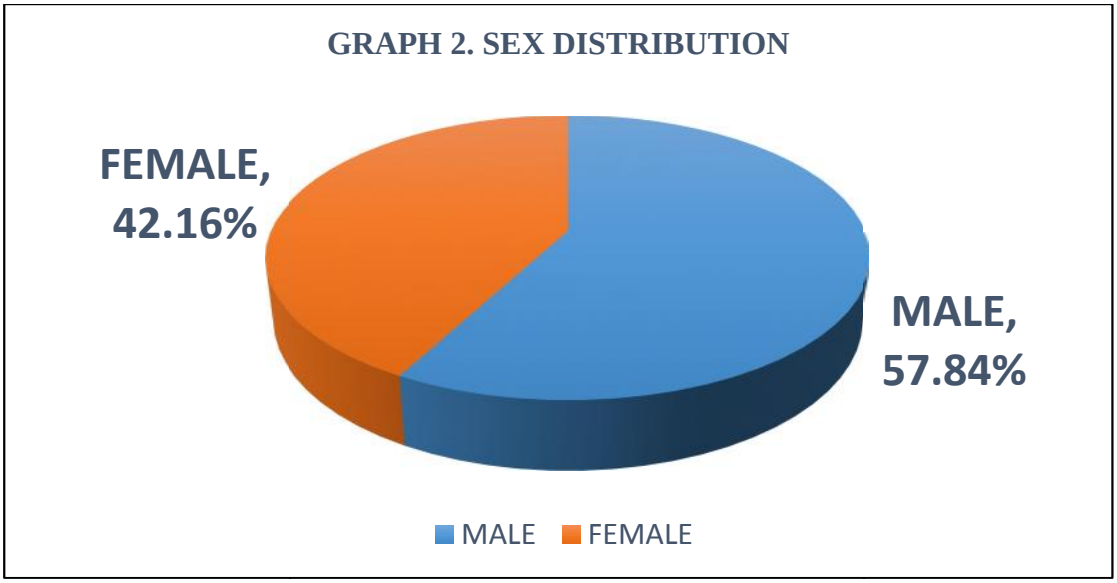
AGE GROUP (Years)	NUMBER (n=102)	PERCENTAGE
<=20yrs	14	13.73
21-30yrs	48	47.06
31-40yrs	20	19.61
41-50yrs	11	10.78
>=51yrs	9	8.82
TOTAL	**102**	**100**



Patients age ranged from 18 to 84 years, maximum number of cases were in the age group 21 to 30 years that is 48 patients (47.06%), between 31 to 40 years had 20 patients (19.61%), age group less than or equal to 20 years had 14 patients (13.70%) , between 41- 50 years had 11 patients (10.78%) and only 9 patients (8.82%) in the group of more than or equal to 51 years. The mean age was 31.51 ± 3.57years.

TABLE 2 . Sex distribution

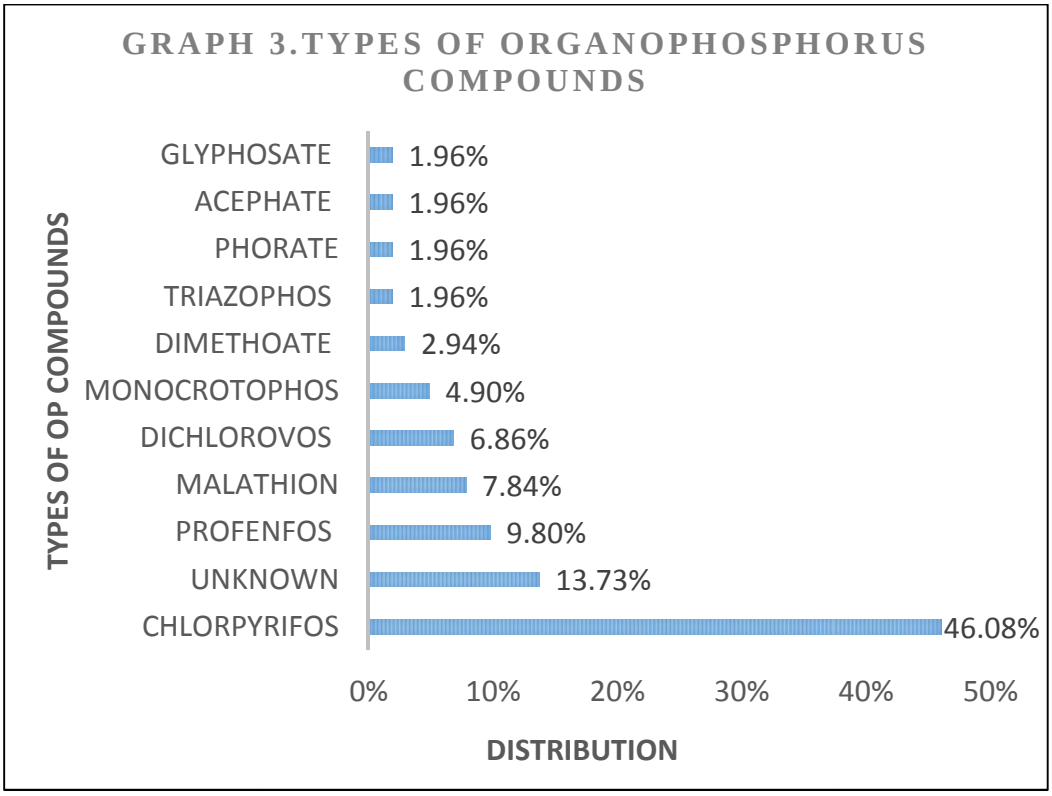
SEX	NUMBER (n=102)	PERCENTAGE
MALE	59	57.84
FEMALE	43	42.15
TOTAL	**102**	**100**



Out of 102 patients 59 (57.84%) were males and 43 patients (42.15%) were females, accounting a ratio of male to female of 1.4:1.

TABLE 3. Types of Organophosphorus compounds

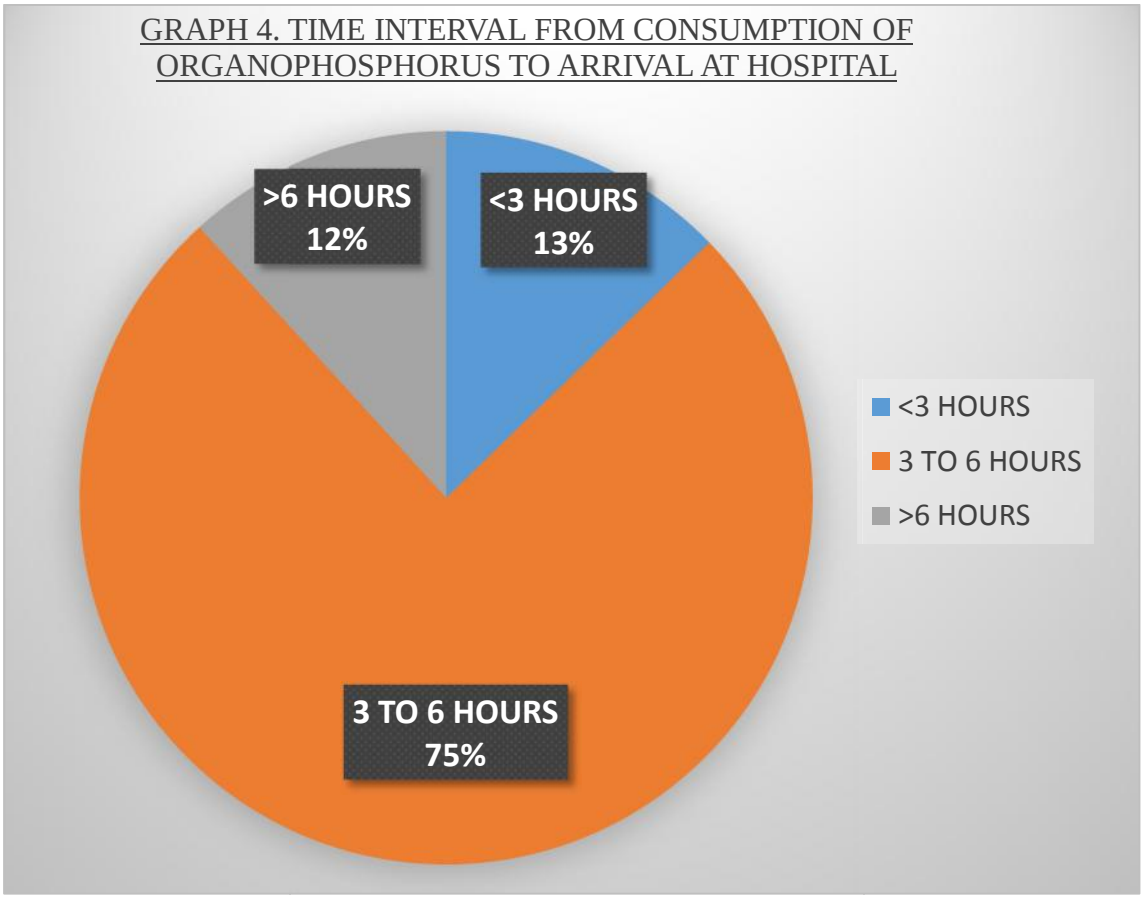
TYPE OF OP COMPOUND	NUMBER	PERCENTAGE
CHLORPYRIFOS	47	46.08
UNKNOWN	14	13.73
PROFENFOS	10	9.80
MALATHION	8	7.84
DICHLOROVOS	7	6.86
MONOCROTOPHOS	5	4.90
DIMETHOATE	3	2.94
TRIAZOPHOS	2	1.96
PHORATE	2	1.96
ACEPHATE	2	1.96
GLYPHOSATE	2	1.96
TOTAL	102	100



We observed that 47 patients (46.08%) had consumed Chlorpyrifos, 10 patients (9.80%) had consumed Profenfos, 8 patients (7.84%) Malathion, 7 patients (6.86%) Dichlorovos, 5 patients (4.90%) Monocrotophos, 3 patients (2.94%) Dimethoate, 2 patients (1.96%) Triazophos, 2 patients (1.96%) Phorate, 2 patients (1.96%) Acephate, 2 patients (1.96%) Glyphosate and in 14 patients (13.73%) the compound was unknown.

TABLE 4 .Time interval from consumption of organophosphorus compound to arrival at hospital.

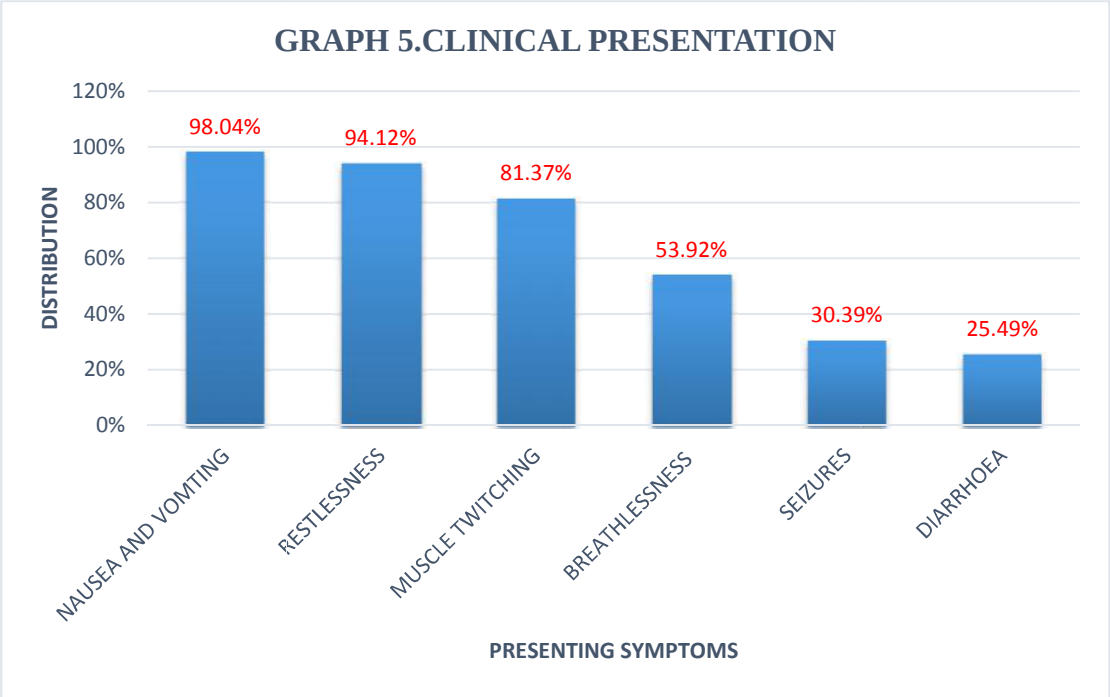
TIME (HOURS)	NUMBER	PERCENTAGE
<3 HOURS	13	12.75
3 TO 6 HOURS	77	75.49
>6 HOURS	12	11.76
TOTAL	102	100.00



We observed that most of the patients 77 (75.49%) arrived within 3 to 6 hours of consumption ,13 patients (12.75%) arrived in less than 3 hours and only 12 patients (11.76%) arrived after 6 hours.

TABLE 5. Clinical presentation.

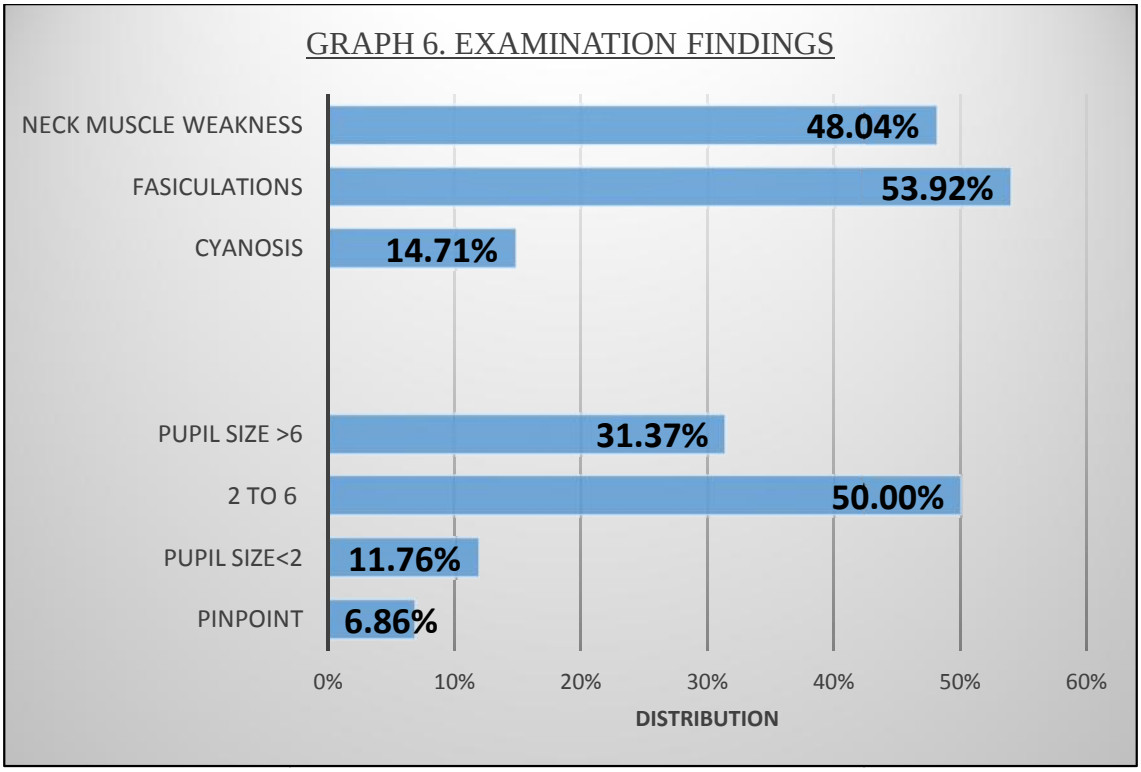
PRESENTING SYMPTOMS	NUMBER	PERCENTAGE
NAUSEA AND VOMTING	100	98.04%
RESTLESSNESS	96	94.12%
MUSCLE TWITCHING	83	81.37%
BREATHLESSNESS	55	53.92%
SEIZURES	31	30.39%
DIARRHOEA	26	25.49%



Majority of the patients were symptomatic. 100 patients (98.04%) had nausea and vomiting, restlessness in 96 patients (94.12%), sweating in 65 patients (63.73%), breathlessness in 55 patients (53.92%), seizures in 31 patients (30.39%) and diarrhoea in 26 patients (25.49%).

Table 6. Examination Findings

PUPIL SIZE (mm)	NUMBER	PERCENTAGE
PINPOINT	7	6.86%
PUPIL SIZE<2	12	11.76%
2 to 6	51	50.00%
PUPIL SIZE >6	32	31.37%
TOTAL	**102**	**100%**
NECK MUSCLE WEAKNESS	49	48.04%
CYANOSIS	15	14.71%
FASICULATIONS	55	53.92%



We observed that majority of the patients ,51 (50.00 %) ,presented with a normal pupil size of 2 to 6 mm. 32 patients (31.37%) had a pupil size of more than 6 mm and 19 patients (18.63%) had a pupil size of less than 2 mm (miotic).

Cyanosis was noted in 15 patients (14.71%), fasiculations in 84 patients (82.35%) and neck muscle weakness in 78 patients (76.47%)

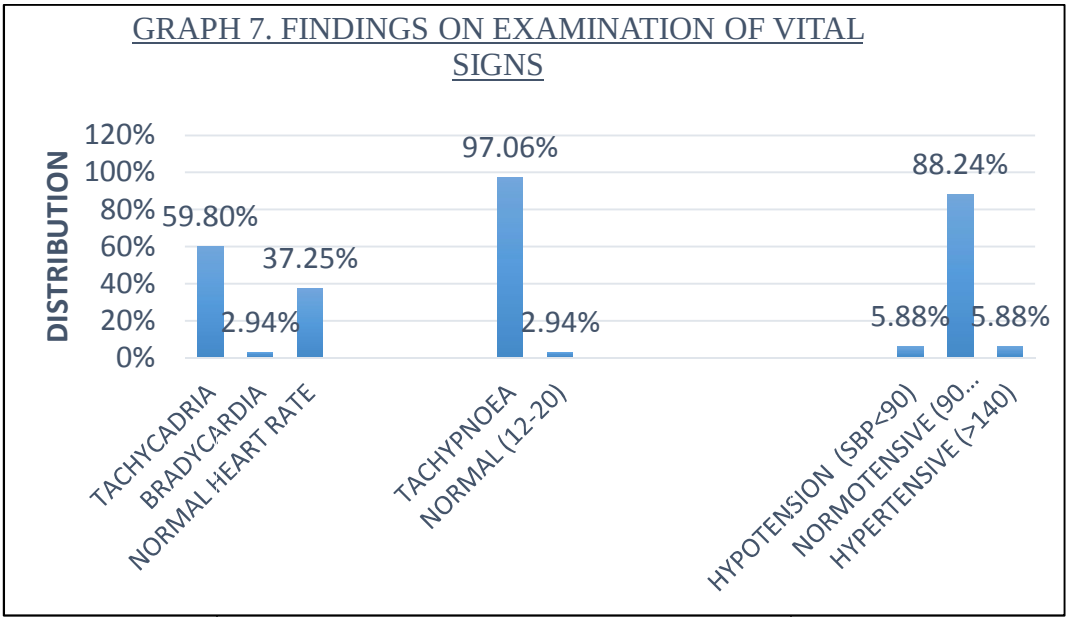
Table 7. Findings on examination of Vital Signs

HEART RATE	NUMBER	PERCENTAGE
TACHYCADRIA	61	59.80%
BRADYCARDIA	3	2.94%
NORMAL HEART RATE	38	37.25%
TOTAL	**102**	**100%**

RESPIRATORY RATE	NUMBER	PERCENTAGE
TACHYPNOEA	99	97.06%
NORMAL (12-20)	3	2.94%
TOTAL	**102**	**100%**

BLOOD PRESSURE	NUMBER	PERCENTAGE
HYPOTENSION (SBP<90)	6	5.88%
NORMOTENSIVE (SBP90 TO 140)	90	88.24%
HYPERTENSIVE (SBP>140)	6	5.88%
TOTAL	**102**	**100%**

GRAPH 7. FINDINGS ON EXAMINATION OF VITAL SIGNS

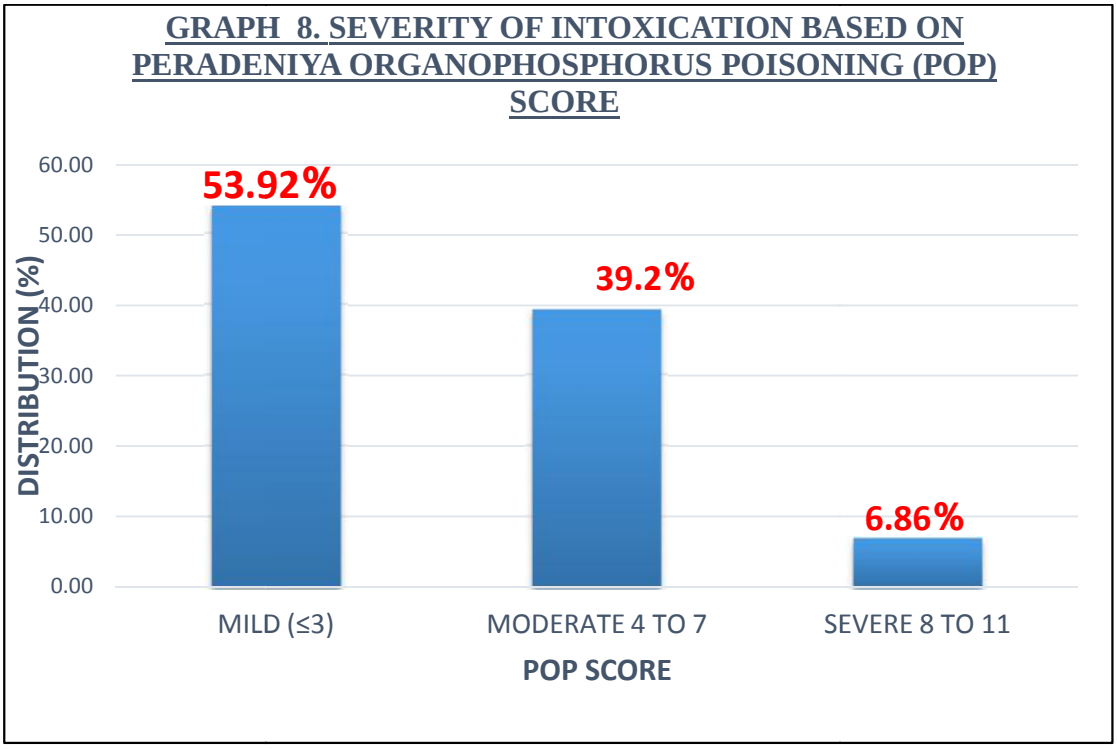


In our study, revealed 61 patients (59.80%) had tachycardia while 3 patients (2.94%) had bradycardia and in remaining 38 patients (37.25%) normal heart rate was noted. With respect to the respiratory rate, 99 patients (97.06%) had presented with tachypnoea while only 3 patients (2.94%) had a normal respiratory rate.

The blood pressure in 90 patients (88.24%) was normal, 6 patients (5.88%) were found to be hypertensive and 6 patients (5.88%) were hypotensive (SBP<90mmhg).

Table 8. Severity of intoxication based on Peradeniya organophosphorus poisoning (POP) score

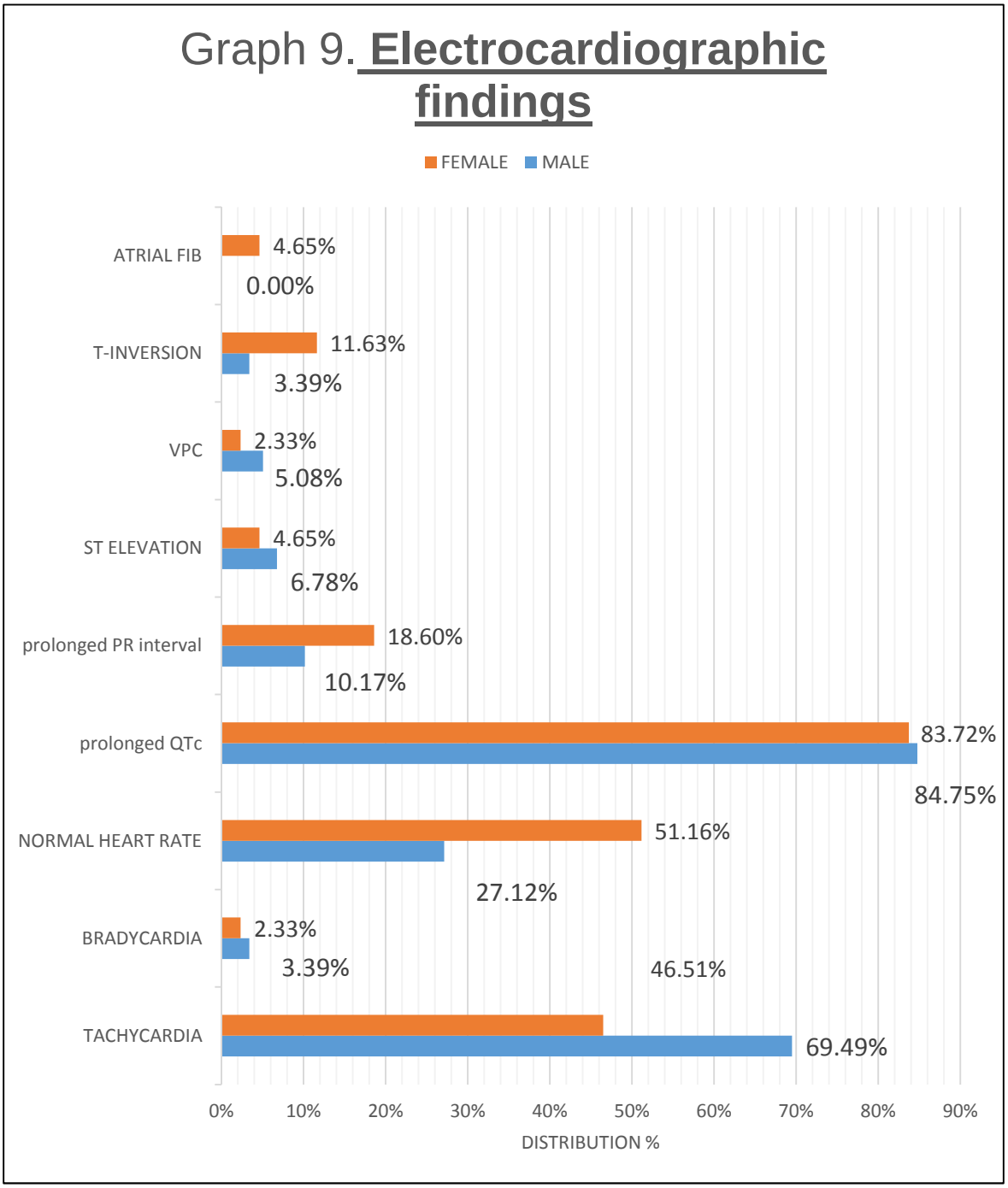
SEVERITY	POP SCORE	NUMBER	PERCENTAGE
MILD	≤3	55	53.92
MODERATE	4 TO 7	40	39.22
SEVERE	8 TO 11	7	6.86
	TOTAL	**102**	**100**



We observed that 55 patients (53.92%) had mild intoxication, 40 patients (39.33%) had moderate intoxication and 7 patients (6.86%) had severe intoxication.

Table 9. Electrocardiographic findings

	MALE	%	FEMALE	%	TOTAL	%
TACHYCARDIA	41	69.49	20	46.51	61	59.80%
BRADYCARDIA	2	3.38	1	2.32	3	2.94%
NORMAL HEART RATE	16	27.11	22	51.16	38	37.25%
TOTAL	59	100	43	100	102	100
ST ELEVATION	4	6.77	2	4.65	6	5.88%
T-INVERSION	2	3.38	5	11.62	7	6.86%
ATRIAL FIB	0	0	2	4.65	2	1.96%
VPC	3	5.08	1	2.33	4	3.92%
PROLONGED PR INTERVAL	6	10.16	8	18.60	14	13.72%
PROLONGED QTc	51	86.44	39	90.69	90	88.23%

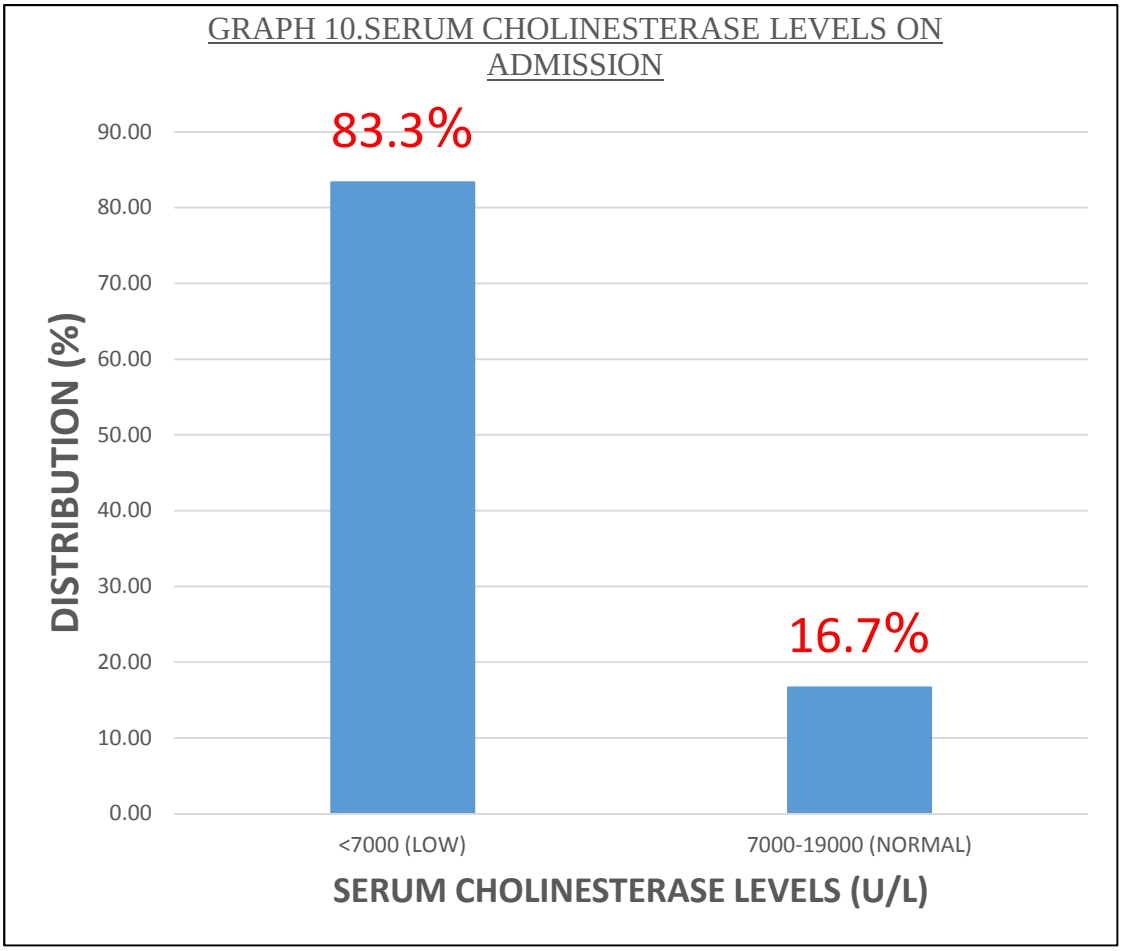


ECG Tracing revealed 61 patients (59.80%) had tachycardia while 3 patients (2.94%) had bradycardia and in remaining 38 patients (37.25%) normal heart rate was noted.

Prolonged Qtc was noted in 90 patients (88.23%) while a prolonged PR interval was noted in 14 patients (13.72%). T inversion in 7 patients (6.86%), ST elevations in 6 patients (5.88%), VPCs in 4 patients (3.92%) and atrial Fibrillation was seen in 2 patients (1.96%).

Table 10. Levels of Serum Cholinesterase on Admission

SERUM CHOLINESTERASE LEVELS(U/L)	NUMBER	PERCENTAGE
<7000 (LOW)	85	83.33
7000-19000 (NORMAL)	17	16.67
TOTAL	**102**	**100**



Serum cholinesterase estimation on admission revealed 85 patients (83.33%) with a low level (<7000) and 17 patients (16.67%) with a normal level.

Table 11. Renal Functions test

BLOOD UREA (mg/dl)	NUMBER	PERCENTAGE
Normal (≤36)	86	84.31%
Elevated (>36)	16	15.69%
TOTAL	102	100.00%
SERUM CREATININE (mg/dl)	NUMBER	PERCENTAGE
Normal (≤1.30)	94	92.16%
elevated (>1.30)	8	7.84%
TOTAL	102	100.00%

We observed that out of 102 patients 86 patients (84.31%) had normal Urea levels while 16 patients (15.69%) had elevated urea levels.

Creatinine was elevated in 8 patients (7.84%) and normal in 94 patients (92.16%).

Table 12. Electrolytes

POTASSIUM LEVELS (mEq/l)	NUMBER	PERCENTAGE
NORMAL 3.5-.5.10	71	69.61%
<3.50	29	28.43%
>5.10	2	1.96%
TOTAL	102	100.00%

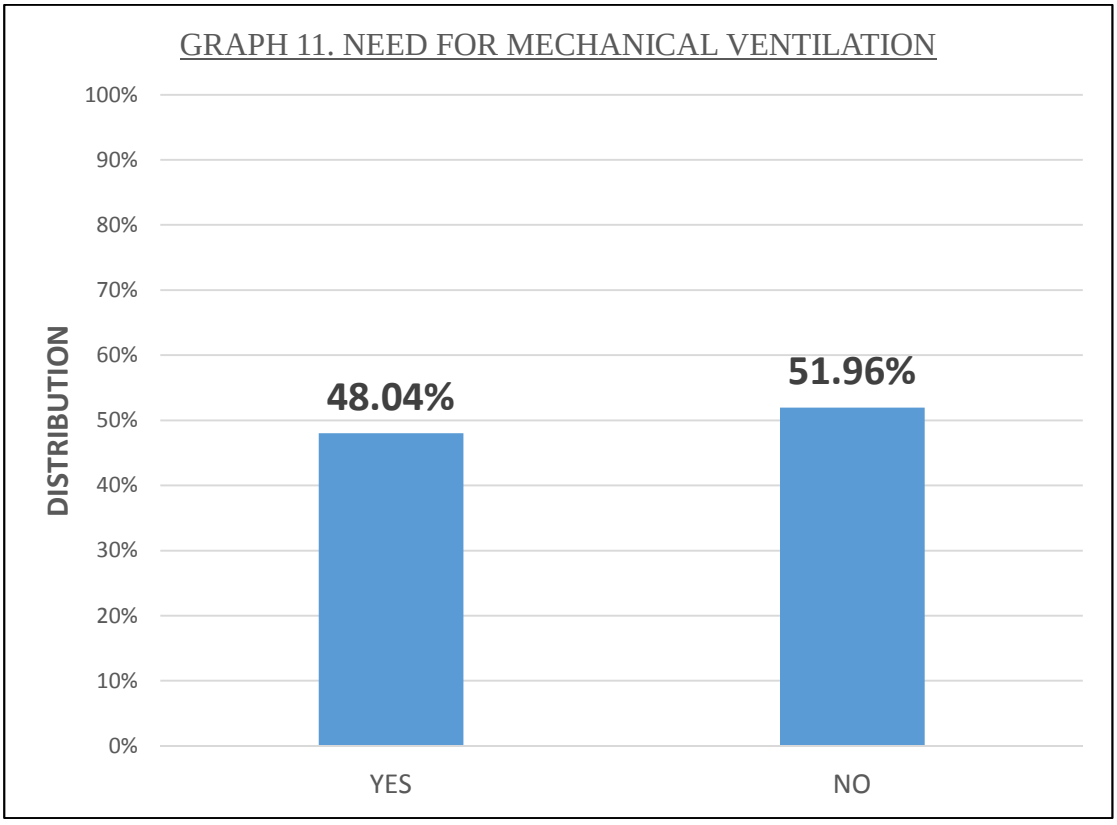
We observed that out of 102 patients 71 patients (69.61%) had normal potassium levels while 29 patients (28.43%) had low potassium and 2 patients (1.96%) had high levels.

SODIUM LEVELS (mEq/l)	NUMBER	PERCENTAGE
NORMAL 136-145	66	64.71%
<136	14	13.73%
>145	22	21.57%
TOTAL	102	100.00%

We observed that out of 102 patients 66 patients (64.671%) had normal sodium levels while 14 patients (13.73%) had low potassium and 22 patients (21.57%) had high levels.

Table 13. Need for Mechanical Ventilation

VENTILATOR USE	NUMBER	PERCENTAGE
YES	49	48.04%
NO	53	51.96%
TOTAL	**102**	**100.00%**



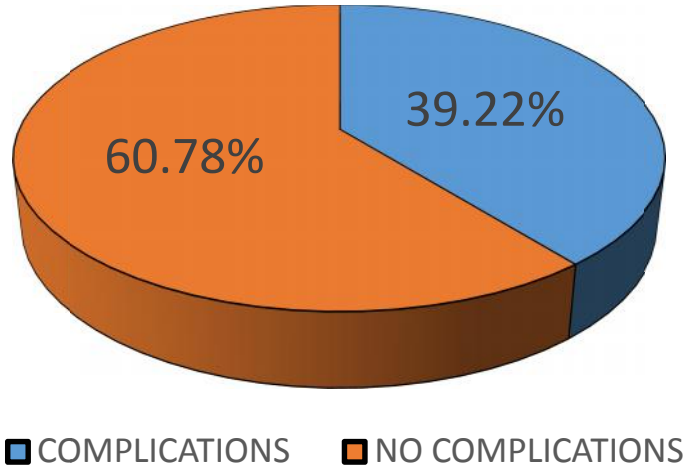
As a part of medical intervention, Mechanical Ventilator was needed in 49 patients (48.04%) and not required in the rest of the 53 patients (51.96%).

Table 14. Distribution of patients according to complications

	NUMBER	PERCENTAGE
COMPLICATIONS	40	39.22%
NO COMPLICATIONS	62	60.78%
TOTAL	102	100.00%

COMPLICATIONS	NUMBER	PERCENTAGE
ACUTE RENAL FAILURE	8	20.00%
INTERMEDIATE SYNDROME	18	45.00%
SEPSIS	21	52.50%
CARDIAC ARRHYTHMIAS	10	25.00%
SHOCK	6	15.00%
ASPIRATION PNEUMONIA	1	2.50%

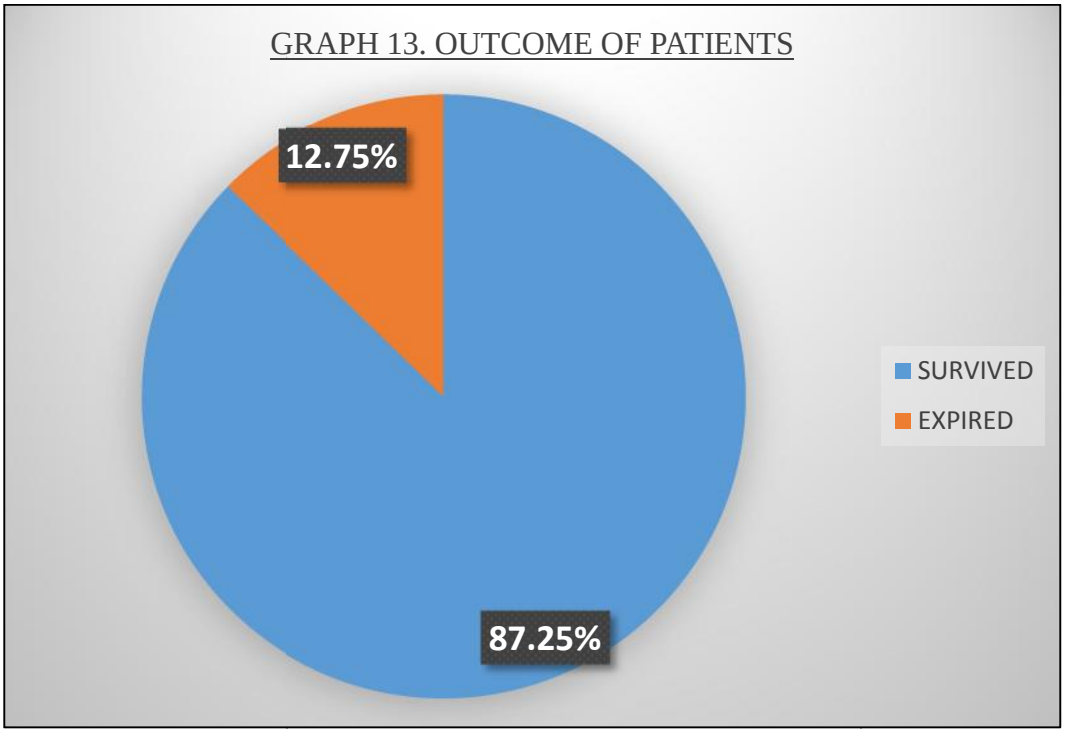
GRAPH 12. DISTRIBUTION OF PATIENTS ACCORDING TO COMPLICATIONS



A total of 40 Patients had various complications, in which sepsis was noted to be the commonest complication in the study i.e 21 patients (52.50%), intermediate syndrome in 18 patients (45.00%), cardiac arrhythmias in 10 patients (25.00%), acute renal failure in 8 patients (20.00%), shock in 6 patients (15.00 %) and aspiration pneumonia in 1 (2.50%).

Table 15. Outcome of Patients

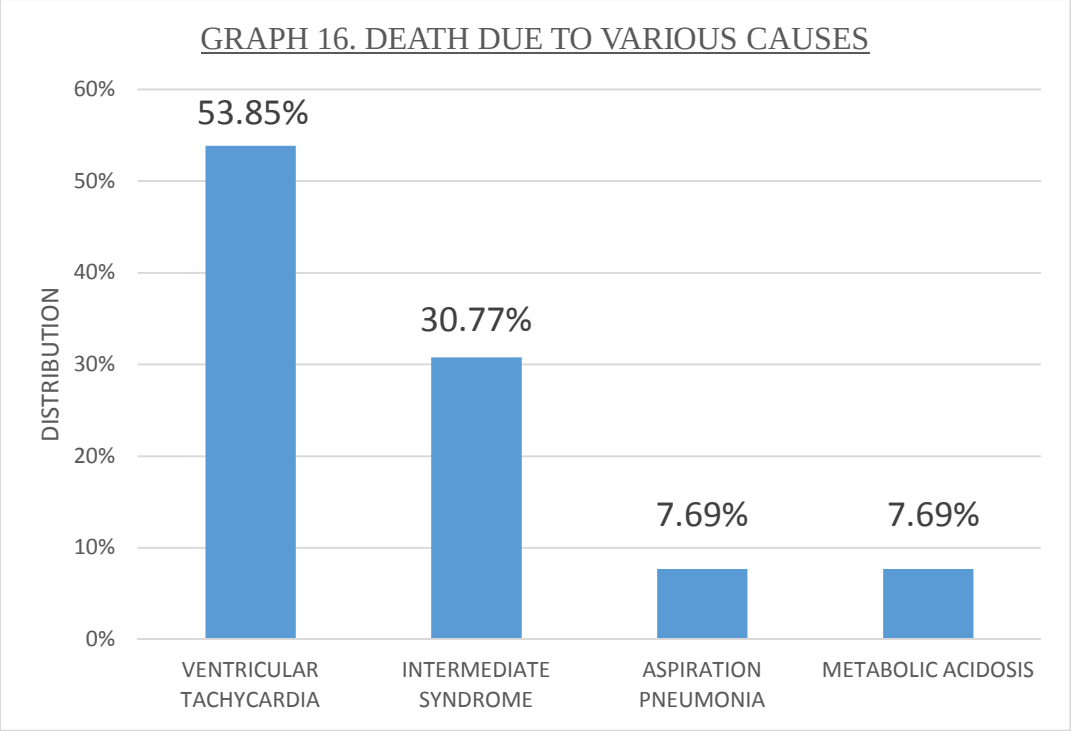
OUTCOME	NUMBER	PERCENTAGE
SURVIVED	89	87.25
EXPIRED	13	12.75
TOTAL	102	100



In our study 89 patients (87.25%) survived and the remaining 13 patients (12.75%) expired.

Table 16. Death due to various causes

DEATH	NUMBER	PERCENTAGE
VENTRICULAR TACHYCARDIA	7	53.85%
INTERMEDIATE SYNDROME	4	30.77%
ASPIRATION PNEUMONIA	1	7.69%
METABOLIC ACIDOSIS	1	7.69%
TOTAL	13	100.00%



We observed that out of 102 patients, 13 expired due to different causes. Ventricular tachycardia in 7 patients (53.85%), intermediate syndrome in 4 patients (30.77%), aspiration pneumonia in 1 patient (7.69%) and metabolic acidosis in 1 patient (7.69%).

Table 17. Association between age groups with status of QTC

Age groups	Normal QTC	%	Prolonged QTC	%	Total
<=20yrs	2	14.29	12	85.71	14
21-30yrs	7	14.58	41	85.42	48
31-40yrs	1	5.00	19	95.00	20
41-50yrs	1	9.09	10	90.91	11
>=51yrs	1	11.11	8	88.89	9
Total	12	11.76	90	88.24	102

In our study correlation between Age and Qtc is shown in the table above. P value being statistically insignificant.

P value = 0.8421

Table 18. Correlation between Qtc and duration of hospital stay (in days)

QTC	NUMBER	MEAN DURATION(DAYS)
Normal QTC	12	6.67
Prolonged QTC	90	8.60
TOTAL	**102**	

Correlation between Qtc and mean hospital stay is shown in the table above. Patients with a prolonged Qtc had a higher mean duration of hospital stay. However, P value was statistically insignificant.

P-VALUE- 0.3366

Table 19. Correlation between Qtc and GCS Score

QTC(n=102)	MEAN GCS
PROLONGED QTC(n=90)	10.93
NORMAL QTC(n=12)	13.43

Correlation between Qtc and mean GCS score is shown in the table above. Patients with a prolonged Qtc had a lower mean GCS score.

P value was statistically significant.

P VALUE- 0.0055*

Table 20. Correlation between Qtc and Mechanical ventilator support

VENTILATOR USE	NUMBER	PERCENTAGE
NORMAL QTC	1	2.04%
PROLONGED QTC	48	97.96%

In our study of 102 patients, 53 patients did not require ventilatory support and the remaining required ventilatory support in view of respiratory distress. The above table depicts the percentage of patients who required ventilatory support with respect to their Qtc intervals. Almost all patients who required the Ventilator had a prolonged Qtc interval.

P value is statistically significant.

P VALUE-0.0031*

Table 21. Correlation between Qtc and Mean POP score

QTC	Mean	SD
Normal QTC	2.75	0.97
Prolonged QTC	4.13	2.03

Correlation between Qtc and mean POP score is shown in the table above. Patients with a prolonged Qtc had a higher mean POP score indicating a higher severity of poisoning.

P value was statistically significant.

P-value-0.0266*

Table 22. Correlation between Qtc and time to Hospital arrival (hours)

	TIME DELAY <3 HRS	TIME DELAY 3 TO 6	TIME DELAY >6
MEAN QTC (ms)	476.45	489.76	498.97

Correlation of Qtc with time delay to hospital arrival revealed a longer Qtc with a longer time delay to hospital arrival.

P values were statistically insignificant.

P VALUE-0.0997

Table 23. Correlation between Qtc and Serum Cholinesterase (u/l)

QTC(n=102)	Mean LEVELS (U/L)
Normal QTC (n=12)	1599.17
Prolonged QTC (n=90)	2960.84

Correlation between Qtc and cholinesterase levels is shown in the table above. Patients with a prolonged Qtc had a higher mean cholinesterase level. And P value was not statistically significant.

P-value 0.3944

Table 24. Correlation between Qtc and duration of atropine use (days).

QTC (n=102)	Mean Duration (days)	SD
Normal QTC(n=12)	6.17	4.69
Prolonged QTC(n=90)	7.76	6.70

Correlation between Qtc and mean duration of atropine use is shown in the table above. Patients with a prolonged Qtc had a higher mean duration atropine use. However, P value was statistically insignificant.

P-value-0.4266

Table 25. Correlation between Qtc and complications.

QTC (n=102)	COMPLICATIONS	
	NUMBER	PERCENTAGE
PROLONGED QTC(n=90)	37	92.50%
NORMAL QTC(n=12)	3	7.50%
TOTAL	40	100.00%

The table above depicts the percentage of complications in the normal Qtc and prolonged Qtc subgroups.Patients with a prolonged Qtc developed more complications. However, P value was statistically insignificant.

P -VALUE = 0.4481

Table 26. Correlation between Qtc and Mortality.

QTC	NUMBER	PERCENTAGE
PROLONGED QTC	13	100.00%
NORMAL QTC	0	0.00%

The table above depicts the correlation of Qtc with mortality. All Non survivors were found to have a prolonged Qtc. However P value was statistically insignificant.

P VALUE= 0.3432

Table 27. Comparison of various parameters with type of organophosphorus compound

TYPE OF COMPOUND	NUMBER	MEAN QTC	MEAN POP SCORE	MEAN DURATION OF HOSPITAL STAY	MORTALITY
CHLORPYRIFOS	47	501.11	4.55	10.5	4
UNKNOWN	14	489.62	3.36	4.1	2
PROFENFOS	10	494.65	3.60	11.4	3
MALATHION	8	463.18	3.00	8.25	0
DICHLOROVOS	7	477.12	4.29	3.36	3
MONOCROTOPHOS	5	500.93	4.60	6	1
DIMETHOATE	3	498.73	3.67	6	0
TRIAZOPHOS	2	413.76	2.00	7	0
PHORATE	2	446.88	2.00	8	0
ACEPHATE	2	465.54	4.00	8.5	0
GLYPHOSATE	2	471.88	4.50	7	0
TOTAL	**102**				**13**

We observed that the highest mean Qtc was 501.11 seen with Chlorpyrifos, while the highest POP severity score was 4.60 seen with Monocrotophos. The mean duration of hospital stay is was longest with profenfos of 11.4 days and Mortality was highest with Chlorpyrifos of 4 cases.

Table 28. Correlation of various parameters between survivor and non-survivor groups with prolonged Qtc.

PRAMETERS	PROLONGED QTC		
	NON SURVIVORS	SURVIVORS	P VALUES
MEAN POP SCORE	6.08	3.81	**0.0001***
MEAN QTC	498.50	500.49	0.4588
MEAN CHOLINESTERASE (U/l)	3156.23	2927.86	0.7093
MEAN DURATION OF HOSPITAL STAY (DAYS)	4.73	9.25	**0.0302***
MEAN DAYS OF ATROPINE USE	4.63	8.29	0.0802
MEAN GCS	7.20	11.42	**0.0001***
MEAN DURATION OF VENTILATOR USE (DAYS)	3.73	6.80	0.0841
MEAN TIME DELAY TO HOSPITAL (HRS)	5.21	5.01	0.0997

We observed that on comparing patients with prolonged qtc who survived and who did not there was a statistically significant correlation with the POP score, the Mean duration of Hospital stay and the Glasgow coma score.

Table 29. Correlation of POP score with various parameters

POP SCORE	MILD POISONING (≤3)	MODERATE (4 TO 7)	SEVERE (8 TO 11)	P values
MEAN QTC	**477.11**	**501.99**	**510.29**	**0.0216***
MEAN CHOLINESTERASE (U/L)	**3427.38**	**1748.78**	**3887.00**	0.0611
MEAN LENGTH OF HOSPITAL STAY	**7.23**	**10.30**	**6.36**	**0.0500***
MEAN DAYS OF ATROPINE USE (DAYS)	**6.30**	**9.61**	**6.00**	**0.0384***
MEAN GCS	**13.09**	**9.46**	**5.17**	**0.0001***
MEAN DURATION OF VENTILATOR USE (DAYS)	**6.40**	**6.31**	**3.64**	0.4842
MEAN TIME DELAY TO HOSPITAL (HRS)	**4.89**	**4.49**	**7.14**	0.0905
NUMBER OF COMPLICATIONS	**10**	**24**	**6**	**0.0001***
MORTALITY NUMBER	**2**	**7**	**4**	0.0623
MORTALITY %	**3.64%**	**17.50%**	**57.14%**	**0.0001***

We observed that on comparing the POP score with various other parameters, there was a statistically significant correlation with the Mean QTC, duration of hospital stay, durations of atropine use, the Glasgow coma score, the number of complications observed and the mortality.

Table 30. Comparing QTc at admission with Qtc at discharge.

	ON ADMISSION	AT DISCHARGE
MEAN QTC (ms)	489.14	452.22

	QTC >430 (NUMBER)	PERCENTAGE	QTC<430 (NUMBER)	PERCENTAGE	TOTAL
AT ADMISSION	90	88.24%	12	11.76%	102
AT DISCHARGE	69	67.65%	33	2.94%	102

We observed that the Mean QTc interval at Admission was higher (489.14ms) that than the QTc interval at Discharge (452.22ms). The number of patients who continued to have a prolonged QTC interval at the time of discharge was less (67.5%) compared to those at admission (88.24%).

The P value was statistically significant.

P- VALUE is 0.000391

DISCUSSION

In the present study of 102 patients the Electrocardiographic findings in Acute organophosphrus poisoning was studied and the changes in corrected QT (QTc) interval were correlated with the severity of organophosphorous poisoning and various other parameters.

All the 102 patients who presented with organophosphorus compoundpoisoning were with a suicidal bid. This is similar to a study done in Hyderabad by Khazi MA et al.[120] and in Nepal by Karki P et al (10) found in their series of study that most common cause of OP poisoning was suicidal intentions (89% of patients) followed by accidental (11% of patients).

In our study patient age ranged from 18 to 84 years. maximum number of cases were in the age group 21 to 30 years that is 48 patients (47.06%),. This is similar to study done by Sen R et al [121] Rehiman et al. [122] and Patil G et al [123] Similar observations found by Karki P et al (10) that majority (65%) of the patients were in the 15 to 30 years age group, Shankar laudari et al (124) that most of the patients belonged to the population of active productive age group (86.9% were between 15 and 45 years of age).

When sex was taken into consideration 102 patients 59 (57.84%) were males and 43 patients (42.15%) were females. There was male preponderance with a ratio of male to female of 1.4:1.This observation is similar to study by Kang EJ et al.[125] and Patil G et al.[123] In contrast Rehiman et al.[122] and Sen R et al.[121] observed more number of females in their study. In present study, male: female ratio was 1.38: 1 which correlates with studies by Saadeh AM et al [112] (1.1:1), S Agarwal et al

(126) (1.8:1) whereas dissimilar observations were noticed by G. Someswar et al (127) (3.17:1), Gouda H.S et al (128) (male - 32; female – 18), Ghulam Hussain Balouch et al (129) (male - 60; female – 27) and Rahbar Taromsari et al (130) (male - 75; female – 25).

We tried to analyse our patients with the organophosphorus compounds they had consumed which is shown in table 3. The most commonly consumed compound was Chlorpyrifos 47 patients (46.08%) had, 10 patients (9.80%) had consumed Profenfos, 8 patients (7.84%) Malathion. In studies by Nouira et al.[131] and Rehiman et al.[122] the most commonly consumed compounds were parathion and dichlorvos. In contrast a study by Kumar CU et al. [132] found that the most commonly consumed compounds were monocrotophos and chlorpyrifos.

Similarly we tried to analyse the time of consumption to arrival at the hospital. We observed that most of the patients 77 (75.49%) arrived within 3 to 6 hours of consumption , 13 patients (12.75%) arrived in less than 3 hours and only 12 patients (11.76%) arrived after 6 hours. Nouira et al.[131] observed that in their study patients presented within a mean time interval of 2.5 hours (range, 30 minutes to 15.5 hours).

Patients presented with various symptoms of organophosphorus compound poisoning. Majority of the patients had nausea and vomiting 100 patients (98.04%), restlessness in 96 patients (94.12%), breathlessness in 55 patients (53.92%), seizures in 31 patients (30.39%) and diarrhoea in 26 patients (25.49%). We observed that majority of the patients ,51 (50.00 %) ,presented with a normal pupil size of 2 to 6 mm. 32 patients (31.37%) had a pupil size of more than 6 mm and 12 patients (12.76%) had a pupil size of less than 2 mm (miotic) and only 7 had pinpoint pupils

(6.86%). Cyanosis was noted in 15 patients (14.71%), fasciculation in 84 patients (82.35%) and neck muscle weakness in 78 patients (76.47%). Similarly studies by Nouira et al.[131] and Venkateshwarlu N et al.[133] observed combination and permutation of symptoms of nausea, vomiting, fasciculation, diarrhoea, etc. Ghulam Hussain Balouch et al (129) found the clinical manifestation as follows as salivation in 92%, lacrimation in 86%, urination in 55%, diarrhoea in 52%, GI upset in 71%, emesis in 92%, diaphoresis in 69%, meiosis in 90%, breathlessness in 40%, muscle fasciculation in 71%, restlessness in 80%, confusion in 83%, ataxia in 34%, tremors in 46%, dseizures in 43% and coma in 66% patients. Bardin P. G et al (91) revealed that clinical features in the following decreasing order: meiosis (82%), salivation and disturbed level of consciousness (61%), fasciculation (54%), tachycardia (49%), rhonchi or crepitations (48%), tachypnoea (39%), vomiting (38%), sweating (23%) and diarrhoea (21%).

In our study, revealed 61 patients (59.80%) had tachycardia while 3 patients (2.94%) had bradycardia and in remaining 38 patients (37.25%) normal heart rate was noted. With respect to the respiratory rate, 99 patients (97.06%) had presented with tachypnoea while only 3 patients (2.94%) had a normal respiratory rate. The blood pressure in 90 patients (88.24%) was normal, 6 patients (5.88%) were found to be hypertensive and 6 patients (5.88%) were hypotensive (SBP<90mmhg). In a relatively small series over a longer period of study Karki P et al (10) found sinus tachycardia in 40.5 % and sinus bradycardia in 18.9 %, hypertension in 13.5% and hypotension in 10.8%. Saadeh AM et al (112) observed that sinus tachycardia in 35% and sinus bradycardia in 28%, hypertension in 22% and hypotension in 17%. Shankar Laudari et al (124) revealed that 49.6% of cases developed cardiac effects, the most common abnormality was sinus tachycardia (49.6%). Other abnormalities were sinus

bradycardia in 2.6%, and hypertension in 20%. The predominant observation of tachycardia in patients can be explained by the intense sympathetic stimulation observed in the initial phase of cardiotoxicitiy of OP compounds as observed by ludomirsky [8] and also by the administration of Atropine antidote prior to hospital admission. The significant number of patients with a normal heart rate (37.25%) can be explained by the fact that majority of the patients had a 3-6 hours hospital delay and may have been in the transition of phase to the predominant parasympathetic phase of cardiotoxicity. The time interval to action to OP compounds depends on the type of compounds ingested and the amount and other factors such as concomitant consumption of food or alcohol. Majority of the Patients had consumed Compounds such as Chlorpyrifos which is a Inactivated Diethyl compound and hence has a slower onset of action than Dimethyl Oxon compounds like Monocrotophos.

In our study all 102 patients were subjected to ECG tracing at arrival and during their stay in the hospital which is shown in table 9. ECG Tracing revealed 61 patients (59.80%) had tachycardia while 3 patients (2.94%) had bradycardia and in remaining 38 patients (37.25%) normal heart rate was noted. Prolonged Qtc was the most common abnormality noted in 90 patients (88.23%) while a prolonged PR interval was noted in 14 patients (13.72%). T inversion in 7 patients (6.86%), ST elevations in 6 patients (5.88%), VPCs in 4 patients (3.92%) and atrial Fibrillation was seen in 2 patients (1.96%).

Ghulam Hussain Balouch et al (129) observed bradycardia in 14.9%, tachycardia in 12.6% and Morteza Rahbar Taromsari et al (130) observed sinus tachycardia as the most common ECG abnormality that was seen in 49.2% and sinus bradycardia 10 % which are comparable to the present series. In present series,

common ECG changes were found as follows as QTc prolongation (28%), T wave changes (26%), ST segment changes (4%) and ectopics in 2% of patients. Similar observations seen by Karki P et al (10) as QTc prolongation (37.8%), T wave changes (13.5%), ST segment changes (16.2%) and ectopics in 5.4% of patients. Vijayakumar S et al (49) study revealed that the most common ECG finding was QTc prolongation (60%) followed by T wave changes and ST segment changes (40%). Yun et al.[134] had made an attempt to study QTc prolongation and its correlation with mortality in their patients. A statistically insignificant difference was found between the mean QTc intervals of the patients who survived and those who expired.

An attempt to study clinical presentation based on POP score We observed that 55 patients (53.92%) had mild intoxication, 40 patients (39.33%) had moderate intoxication and 7 patients (6.86%) had severe intoxication. Same is depicted in table 8 (POP score). This is in line with study by Rehiman et al.[122] who observed 70% of their cases had mild intoxication, 26% cases had moderate intoxication and only 4% cases had severe intoxication.

Serum cholinesterase estimation on admission revealed 85 patients (83.33%) with a low level (<7000) and 17 patients (16.67%) with a normal level. Spontaneous Cholinesterase recovery occurs very slowly via denovo synthesis of enzyme and spontaneous dephosphorylation at the rate of approximately 1 % a day. This varies between plasma AChE and RBC AChE, taking upto 4 weeks for plasma levels and longer for the RBC AchE levels to normalize. [40-42] Moreover, In many of the poisoning cases, and the quantity consumed by the patient are unknown and in about 14 patients the type of OP compound was unknown necessitating the need of serial estimation of serum AChE.

However a limitation in our study was that serial estimation of Serum cholinesterase was not done. Studies by Kumar CU et al.[132] and Yun et al.[134] have revealed serial estimation and analysis of serum cholinesterase levels as a prognostic utility which would have also been a useful correlation with prolonged Qtc intervals.

We observed that out of 102 patients 86 patients (84.31%) had normal urea levels while 16 patients (15.69%) had elevated urea levels. Creatinine was elevated in 8 patients (7.84%) and normal in 94 patients (92.16%). In an observational study by Feng you lee [135] the overall incidence of AKI was higher in the patients with OP poisoning than in the controls (4.85 vs 3.47/1000 person-years). After adjustment for age, sex, comorbidity, and interaction terms, patients with OP poisoning were associated with a 6.17-fold higher risk of AKI compared with the comparison cohort.

Kang EJ et al. [125] had subjected their patients for hemoperfusion and found that it was ineffective in terms of survival and improvement of patients with organophosphorus compound poisoning. Study by Altintop et al. [136] reported hemoperfusion to be useful in severe cases. This is by a proposed mechanism that activated charcoal or resins used for hemoperfusion may help in purifying the blood thus allowing the patients to improve. We did not attempt hemoperfusion in any of our patients since there was no definite evidence of benefit in terms of morbidity or mortality.

We observed that out of 102 patients 71 patients (69.61%) had normal potassium levels while 29 patients (28.43%) had low potassium and 2 patients (1.96%) had high levels. We observed that out of 102 patients 66 patients (64.671%) had normal sodium levels while 14 patients (13.73%) had low sodium and 22 patients

(21.57%) had high levels. Among Patients who died in the study 38% was noted to have hypernatremia and 15% to have low sodium level. 38% were noted to have hypokalemia in the Mortality group. In 2002, Kara et al.[137] from Turkey found that hypokalemia followed by hyponatremia were most common electrolyte derangements in their study, however, Serum electrolytes were normal in most of the patients of Kiss and Fazekas et al.[93], Yurumez et al.[113] and in all patients of Ludomirsky et al.[8]. The mechanism of electrolyte derangement in Op poisoning has not been described and a study by G. Someswar[127] have shown Serum electrolytes (Na, K+, Ca2+) derangements were found statistically insignificant and are not helpful in assessing prognosis in OPC poisoning.

As depicted in table 13 Mechanical Ventilator was needed in 49 patients (48.04%) and not required in the rest of the 53 patients (51.96%). A study in Turkey by Okhan et al [138] revealed that 13% of the patients required Mechanical ventilator and another study done in Hyderabad by Kumar CU [132] et al revealed that 77% required Mechanical ventilator. In a study done in Taiwan by Shou-Hsuan Liu et al [139] 51% of patients required mechanical intubation and ventilation for 24 hours or more during Hospital stay which was similar to our results.

We observed a total of 40 patients had various complications of organophosphorus compound in which sepsis was noted to be the commonest complication in the study i.e 21 patients (52.50%), intermediate syndrome in 18 patients (45.00%), cardiac arrhythmias in 10 patients (25.00%), acute renal failure in 8 patients (20.00%), shock in 6 patients (15.00 %) and aspiration pneumonia in 1 (2.50%). Of the Cardiac Arrhythmias 1 patients were noted to have only VPCs , 3 patients had VPCs with Ventricular Tachycardias , 2 patients were noted to have Atrial

fibrillation and 4 patients with Fatal Ventricular Tachycardias. This is in contrast to a study by Venkateshwarlu N et al.[133] who observed that, pulmonary oedema was the most common complication seen in their patients. Sen R et al.100 found respiratory acidosis to be the most common complication followed by intermediate syndrome.

In our study the outcome of patients was evaluated , 89 patients (87.25%) survived and the remaining 13 patients (12.75%) expired. .A study by Kang EJ et al.[125] in Korea showed that 13 of the 68 (19.15%) patients died and a Study by Kumar CU [132] in hyderbad revealed mortality of 19.5% which was more similar to our study.

An attempt to find out the various causes of death in our patients were multi-factorial. The immediate cause of death was Ventricular tachycardia in 7 patients (53.85%), intermediate syndrome in 4 patients (30.77%), aspiration pneumonia in 1 patient (7.69%) and metabolic acidosis in 1 patient (7.69%). All patients had developed respiratory distress and required mechanical ventilation and there were 11 out of 13 patients who had sepsis as a complication and 5 out of 13 developed acute renal failure with one patient requiring hemodialysis. Studies (128-132) by different authors have not commented on the causes of death.

In our study correlation between Age and Qtc is shown in the table 17. P value being statistically insignificant. P value = 0.842.1 In a longitudinal study [140], Su et al reported that QTc interval increased significantly with age in a population of healthy elderly subjects. Elderly hearts tend to have relative mid myocardial myocyte hypertrophy and a distinct increase in connective tissue as compared to younger hearts [141]. Myocyte hypertrophy may be associated with a significant prolongation of the transmembrane action potential, explaining the prolongation of the QTc interval [142]

with age. There are also reports of an exaggerated shift towards sympathetic activity in the elderly [143], and such sympathetic over activity might be an important factor in the prolongation of the QT interval. Therefore, it is not surprising to find longer QT intervals among elderly patients with organophosphate poisoning than among younger organophosphate poisoning patients. However in our study no such finding was observed indicating a more direct role of the OP compound in Qtc prolongation.

Correlation of Qtc with time delay to hospital arrival revealed a longer Qtc with a longer time delay to hospital arrival is shown in table 22. P values were statistically insignificant. (P value-0.0997) In a study by Shou-Hsuan Liu [139] ,the average time from poisoning to hospital arrival in patients with normal qtc was 6.3 hours while it was 4.4 hours in those with a prolonged Qtc but this was statistically not significant.(P=0.413)

Correlation between Qtc and mean hospital stay is shown in the table 18. Patients with a prolonged Qtc had a higher mean duration of hospital stay. However, P value was statistically insignificant. In a study by Okhan et al [138] to investigate effectiveness of the poisoning severity score (PSS), Glasgow coma scale (GCS), and corrected QT (QTc) interval in predicting outcomes in acute organophosphates (OP) poisoning, There was an increased in hospital stay noted with increasing mean Qtc interval till Grade 3 severity of PSS score (severe poisonoing).

Correlation between Qtc and cholinesterase levels is shown in the table 23. Patients with a prolonged Qtc had a higher cholinesterase level. And P value was not statistically significant. (P-value 0.3944). Chuang et al. (15) and Baydin et al [118] have reported that, there is an inverse correlation between blood ChE level and clinical severity in acute OPP. However, this association was weak as there were

many other factors operating in determining the QTc in these patients. But it is expected that, in patients with acute OPP, a longer QTc interval than normal may be a point to a lower blood ChE level, thereby increasing in clinical severity. However, normal ranges of RBC and serum cholinesterase vary widely between individuals (and even in the same individual at different times). Because of this, a person who usually has a “high-normal” level of cholinesterase could be significantly toxic but his or her cholinesterase level could decrease only into the “low-normal” range. Thus, the toxic patient would have a falsely normal test result. [144] (One author indicates that for serum cholinesterase there is a 300% difference between the lower and upper normal values.) [145]

Correlation between Qtc and duration of atropine use is shown in the table 24. Patients with a prolonged Qtc had a higher mean duration atropine use of 7.76 days versus 6.17 days in those with a normal Qtc . However, P value was statistically insignificant. P-value-0.4266.

In a Study by Shadinia et al [115] revealed the average atropine required to control the muscarinic signs and symptoms such as salivation, bronchorrehea, and miosis in patients with prolonged QTC interval was higher than in patients with normal QTC interval.

In relation to this, We observed that the Mean QTc interval at Admission was higher (489.14ms) that than the QTc interval at discharge (452.22ms). The number of patients who continued to have a prolonged QTC interval at the time of discharge was less (67.5%) compared to those at admission (88.24%). The P value was statistically significant, and so there may be a possible role of atropine in reversing the initial prolongation of Qtc. However no studies were found to support this finding.

As shown in Table 29. We observed that on comparing the POP score with various other parameters, there was a statistically significant correlation with the Mean QTC, duration of hospital stay, durations of atropine use, the Glasgow coma score, the number of complications observed and the mortality. All Non survivors were found to have a prolonged Qtc. This is line with a preliminary study at Chang Gung Memorial Hospital, Chuang et al [15] reported that patients with QTc prolongation had a higher mortality rate (19.6% vs. 4.8%, P, 0.001) and a higher incidence of respiratory failure (56.7% vs. 20.6%, P,0.001) than patients without QTc prolongation. Shadnia et al [115] reported that the mortality rate of Iranian patients with long QTc intervals was significantly higher than that of those with normal QTc intervals. Grmec et al. [14] showed that in OPP, a longer QTC interval and a lower Glasgow Coma Scale (GCS) score accompany higher numbers of intubations and worse outcomes. Jang et al. [146] found significant elevations in mortality and respiratory rates among cases showing prolonged QTc intervals. Study by Ravikumar et al [116] revealed that out of 100 patients, 28 patients had a prolonged corrected QT interval of which 86% were in the highest grade (grade III) of Poison Severity Score (IPCS PSS).

CONCLUSION

In our present study of 102 patients with Organophosphorus compound poisoning,The commonest ECG abnormality noted was prolonged QTc followed by tachycardia and prolonged PR interval.

Patients were categorized based on their severity according to the POP severity scoring system. We found that patients with a higher severity of poisoning had a longer corrected Qtc interval.

Patients with longer corrected QT intervals had lower GCS scores on admission and were more likely to require Mechanical ventialation. They also had a longer duration of hospital stay, a longer duration of atropine use, with an increase in complications and a poorer outcome. This observation suggests that corrected Qtc interval is a useful prognostic utility in Organophosphorus poisoning.

SUMMARY

The present study of 102 patients with organophosphorus compound poisoning admitted in department of medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the study period from January 2016 to December 2016, was undertaken to study on the electrocardiographic findings in acute organophosphorus poisoning (OPP) with special reference to corrected QT interval (QTc).

The commonest ECG abnormality noted was prolonged QTc followed by tachycardia and prolonged PR interval.

The results obtained were significant when comparing patients with prolonged qtc who survived and who did not with respect to the POP score, the mean duration of hospital stay and the glasgow coma score.

We observed that on comparing the POP score with various other parameters, there was a statistically significant correlation with the Mean QTC, duration of hospital stay, durations of atropine use, the Glasgow coma score, the number of complications observed and the mortality.

Correlation of Qtc with Age, sex, cholinesterase levels, time delay to hospital arrival, were statistically insignificant.

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ANNEXURE 1

"A HOSPITAL BASED LONGITUDINAL STUDY ON THE ELECTROCARDIOGRAPHIC FINDINGS IN ACUTE ORGANOPHOSPHORUS POISONING (OPP) WITH SPECIAL REFERENCE TO CORRECTED QT INTERVAL (QTC)."

PROFORMA

Case No:

NAME:

AGE/SEX:

IP No.

ADDRESS:

OCCUPATION:

COMPLAINTS AT PRESENTATION:

Time-consumed : Place : Amount

Hospital Arrival delay: Name of compound:

SYMPTOMS

CENTRAL NERVOUS SYSTEM

TREMORS AND FASICULATIONS
ATAXIA (LOSS OF CORDINATED MOVEMENTS)
CONFUSION/ DELIRIUM
ANXIETY
RESTLESSNESS
EMOTIONAL LABILITY
SEIZURES
COMA

RESPIRATORY SYSTEM

RHINORRHEA
PRODUCTIVE COUGH (BRONCHORRHEA)
DIFFICULTY BREATHING (DYSPNOEA)

GASTROINTESTINAL SYSTEM

HYPERSALIVATION
NAUSEA AND VOMITING
ABDOMINAL PAIN
FECAL INCONTINENCE
DIARRHEA

CARDIOVASCULAR SYSTEM

PALPITATIONS
CHEST PAIN
CYANOSIS

OTHERS

BLURRING OF VISION
INCREASED LACRIMATION
INCREASED SWEATING (DIAPHORESIS)
URINARY INCONTINECE

PAST HISTORY:

Hypertension,
Old stroke,
Coronary artery disease
Structural heart disease
Chronic obstructive pulmonary disease
Malignancy,
Mental disorder
Smoking habit
Alcohol consumption
OTHERS-

TREATMENT HISTORY:

Use of medications that might be associated with QTcprolongation
Anti-arrhythmic agents,
Anti-psychotics and anti-depressants,
Anti-microbials
OTHERS-

GENERAL PHYSICAL EXAMINATION:

SWEATING
CYANOSIS
PUPIL SIZE –
PUPILLARY REFLEX-
SMELL OF OP COMPOUND-
FASICULATION
NECK MUSCLE WEAKNESS
PALLOR
ICTERUS
PEDAL EDEMA

VITALS:
TEMPERATURE
PULSE RATE / MIN
RESPIRATORY RATE
SYSTOLIC BLOOD PRESSURE MMHG
DISTOLIC BLOOD PRESSURE MMHG
PULSE OXIMETRY (SPO2)

SYSTEMIC EXAMINATION-

CENTRAL NERVOUS SYSTEM;

Glasgow Coma Score

Best Eye Response. (4)	Best Verbal Response. (5)	Best Motor Response. (6)
1. NO response	1. No verbal response	1. No motor response.
2. Eye opening to pain.	2. Incomprehensible sounds.	2. Extension to pain.
3. Eye opening to verbal command.	3. Inappropriate words.	3. Flexion to pain.
4. Eyes open spontaneously.	4. Confused	4. Withdrawal from pain.
	5. Orientated	5. Localising pain.
		6. Obeys Commands.

TOTAL SCORE-

OTHER RELEVANT FINDINGS-

RESPIRATORY SYSTEM-

SINGLE BREATHE COUNT –

BREATH SOUNDS-

OTHER RELEVANT FINDINGS-

CARDIOVASCULAR SYSTEM

HEART SOUNDS-

OTHER RELEVANT FINDINGS-

GASTROINTESTINAL SYSTEM

Peradeniya Organophosphorous Poisoning (POP) Scale

- Pupil size	Respiratory rate	Heart rate	Fasciculation	Level of consciousness	Seizures
>2 mm 0	<20/min 0	>60/min 0	None 0	Conscious and rationale 0	Absent 0
					Present 1
<2 mm 1	>20/min 1	41–60/min 1	Present, generalized or continuous 1	Impaired response to verbal commands 1	
Pin-point 2	>20/min with central cyanosis 2	<40/min 2	Both, generalized and continuous 2	No response to verbal commands 2	

TOTAL SCORE-

0–3 (mild)
4–7(moderate)
8–11 (severe)

INVESTIGATIONS
Hemoglobin, g/dL
Blood urea nitrogen, mg/dL
Creatinine, mg/dL
Sodium, mEq/L
Potassium, mEq/L
SERUM Cholinesterase, initial, U/mL (NORMAL- 7000-19000)
SERUM Cholinesterase, lowest, U/mL
RBS
LIVER FUNCTION TEST
ABG STUDY
OTHERS RELEVANT-

TREATMENT GIVEN
Gastric lavage,
Active charcoal
Atropine STARTING DOSE- TOTAL DOSE DURATION-
Pralidoxime,

FINAL DIAGNOSIS-

RESULTS
DURATION OF HOSPITAL STAY
VENTILATOR SUPPORT NEEDED- YES/NO DURATION ON VENTILATOR SUPPORT-
SURVIVOR/NON SURVIVOR
CAUSE OF DEATH

ECG on admission

ECG- on discharge

ANNEXURE 2

INFORMED CONSENT

Title Of Research Study: A HOSPITAL BASED LONGITUDINAL STUDY ON THE ELECTROCARDIOGRAPHIC FINDINGS IN ACUTE ORGANOPHOSPHORUS POISONING (OPP) WITH SPECIAL REFERENCE TO CORRECTED QT INTERVAL (QTC).

Principal Investigator:-

DR. _____
DEPARTMENT OF GENERAL MEDICINE
JAWAHARLAL NEHRU MEDICAL COLLEGE, BELGAUM 590010
KARNATAKA

Guide:-

DR. _____
MD MEDICINE, DNB, MNAMS, FGSI
ASSOCIATE PROFESSOR
DEPARTMENT OF GENERAL MEDICINE
J.N.M.C NEHRU NAGAR, BELGAUM 500910

Introduction and Purpose:-

Suicidal poisoning with organophosphorus (OP) pesticides is common, particularly from rural areas in India.

Recent data from National crime bureau of India shows suicide by consumption of OP pesticides account for 19.4% and 19.7% of all cases of suicidal poisoning in the year 2006 and 2007 respectively.

Cardiac arrhythmias, including heart block and QTc prolongation, are observed in organophosphorous agent poisoning. There are reports suggesting a relationship between prolonged corrected QT (QTC) interval and the severity of poisoning. There are reports suggesting a relationship between prolonged corrected QT (QTC) interval and the severity of poisoning.Several studies have shown that QTc intervals have been used to assess the prognosis and severity of organophosphorous poisoning patients.

The study is intended to investigate these Electrocardiographic changes and the relationship between the QTc interval and the severity of organophosphorous poisoning in patients admitted to Dr. Prabhakar Kore Hospital ,Belgaum.

Procedure:

If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood samples for the necessary investigations.

Risk and Benefits:

The only risk and possible discomfort you might get is while taking blood from arm for the investigations. It may cause swelling, pain, redness, bruising or infection (rarely happens) at the site from where the blood is drawn.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part you can later change my mind and withdraw from

the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy:

Does not apply to this research

Financial incentives for participation:

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results:

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.
In case of the queries during study or in future you may contact following persons,

1. **Dr.** _____ ,
J.N.M.C Ethical Committee for
Human Research,

2. **Dr.** _____**,**
Professor & HOD,
Department of Medicine,
JNMC, Belgaum

3. **DR.** _____
DEPARTMENT OF GENERAL
MEDICINE
JAWAHARLAL NEHRU
MEDICAL COLLEGE, BELGAUM
590010
KARNATAKA

Consent Statement

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

Name of the Participant: _____

Signature / Thumb print _____

Name of the Witness _____

Signature/ Thumb print _____

Investigator Name: _____

Signature: _____

Date: Place

KEY TO MASTER CHART

M-Male

F- Female

NS- Nothing Significant

N/A- Not available

VPCs- Ventricular premature complexes

ECG- Electrocardiography

ST- ST segment on Electrocardiography

QT- QT interval on Electrocardiography

RR- RR interval on Electrocardiography

ABG- Arterial Blood gas Analysis

PCo2- Partial pressure of Carbon dioxide

HCO3- Bicarbonate Levels

SGOT-Serum glutamic oxaloacetic transaminase

SGPT-Serum glutamate-pyruvate transaminase

Mg/dl- Milligrams per decilitre

Meq/L- Milliequivalents per litre

U/l- Units per litre

Spo2- Peripheral capillary oxygen saturation

F- Fahrenheit

CNS- Central Nervous system

RS- Respiratory system

CVS- Cardiovascular system

GIT- Gastrointestinal system

PATIENT DEMOGRAPHICS						HISTORY		SYMPTOMS OF CNS								SYMPTOMS OF RS			SYMPTOMS OF CVS			SYMPTOMS OF GIT					OTHER SYMPTOMS			
CASE NUMBER	OUTPATIENT / INPATIENT NUMBER	AGE (years)	GENDER	OCCUPATION	DATE OF ADMISSION	Hospital Arrival Delay (HOURS)	Name of compound	TREMORS AND FASICULATIONS	ATAXIA	CONFUSION	ANXIETY	RESTLESSNESS	EMOTIONAL LABILITY	SEIZURES	COMA	RHINORRHEA	BRONCHORRHEA	DYSPNOEA	PALPITATIONS	CHEST PAIN	CYANOSIS	HYPERSALIVATION	NAUSEA AND VOMITING	ABDOMINAL PAIN	FECAL INCONTINENCE	DIARRHEA	BLURRING OF VISION	INCREASED LACRIMATION	DIAPHORESIS	URINARY INCONTINENCE
1	714555	18	F	STUDENT	1/20/2016	7	CHLORPYRIFOS 50%	1		1	1	1	1	1			1	1				1	1	1	1			1	1	1
2	715145	55	M	X-SERVICEMA	1/22/2016	5	CHLORPYRIFOS 50%	2		2	2	2	2	2			2						2	2				2	2	
3	717660	36	M	COOLI	2/6/2016	5	CHLORPYRIFOS 20%	3			3	3	3				3					2	3	3					3	
4	718073	27	M	MASON	2/9/2016	5	PROFENFOS 40%	4		3	4	4	4	3			4	2				3	4	4					4	2
5	718731	65	F	COOLI	2/12/2016	5	CHLORPYRIFOS 20%	5		4		5	5		1		5					4	5	5				3	5	
6	718682	36	M	COOLI	2/12/2016	5	CHLORPYRIFOS 20%			5	5	6	6				6					5	6	6				4	6	
7	718687	31	F	HOUSEWIFE	2/12/2016	5	UNKNOWN	6		6	6	7	7				7	3				6	7	7				5	7	
8	719824	35	M	COOLI	2/18/2016	4	MALATHION	7		7	7	8	8	4			8	4				7	8	8				6	8	
9	720571	23	M	FARMER	2/22/2016	2	UNKNOWN	8		8	8	9	9				9	5					9	9						
10	722372	24	M	FAMRER	3/1/2016	5	CHLORPYRIFOS 20%	9		9	9	10	10									9	10	10				7	9	
11	723006	18	F	STUDENT	3/5/2016	5	UNKNOWN			10	10	11	11									10	11	11						
12	726686	24	M	SERVICEMAN	3/25/2016	4	CHLORPYRIFOS 50%	10		11	11	12	12				10	6				11	12	12				8	10	3
13	726944	28	F	HOUSEWIFE	3/27/2016	7	UNKNOWN	11			12			5	2			7					13	13				9	11	
14	727738	35	M	FARMER	3/31/2016	7	DICHLOROVOS 75%	12		12	13	13	13	6	3		11	8				12	14	14	2				12	4
15	728609	24	F	COOLI	4/5/2016	1	CHLORPYRIFOS 50%	13	1	13	14	14	14				12					13	15	15			1	10	13	
16	729552	18	F	UNEMPLOYED	4/10/2016	5	DIMETHOATE 75%				15	15	15									14	16	16				11	14	
17	730865	44	M	FARMER	4/17/2016	4	DIMETHOATE 75%	14		14		16											17							
18	731877	48	M	FARMER	4/21/2016	5	PROFENFOS 40%	15		15		17		7	4		13	9				15	18		3	1		12	15	5
19	734908	25	F	HOUSEWIFE	5/7/2016	5	CHLORPYRIFOS 20%	16		16		18					14	10					19	17	4	2			16	6
20	736178	22	M	COOLI	5/14/2016	5	PHORATE						16										20	18						
21	740119	30	F	UNEMPLOYED	6/2/2016	7	MONOCROTOPHOS 36%	17		17	16	19		8	5			11				16	21	19	5			13	17	7
22	740420	18	F	UNEMPLOYED	6/3/2016	5	CHLORPYRIFOS 20%	18		18		20					15	12					22	20		3			18	8
23	741201	27	F	HOUSEWIFE	6/8/2016	7	CHLORPYRIFOS 20%	19		19		21											23							
24	742630	20	F	UNEMPLOYED	6/15/2016	2	DICHLOROVOS 75%	20		20	17	22						13					24							
25	743045	22	M	UNEMPLOYED	6/16/2016	2	MALATHION	21		21	18	23											25		6	4				
26	743915	50	M	TAILOR	6/21/2016	2	MALATHION	22	2	22	19	24										17	26	21				14	19	
27	744137	21	F	COOLI	6/22/2016	4	CHLORPYRIFOS 20%	23		23	20	25	17					14				18	27	22					20	
28	745535	18	M	STUDENT	6/28/2016	5	CHLORPYRIFOS 20%	24		24	21	26	18	9			16	15				19	28	23	7			15	21	9
29	746991	21	M	COOLI	7/6/2016	6	DICHLOROVOS 75%	25	3	25	22	27											29	24						
30	746924	25	M	FARMER	7/6/2016	4	PROFENFOS 40%	26		26	23	28	19	10			17	16				20	30	25			2	16	22	
31	747068	44	M	COOLI	7/7/2016	5	CHLORPYRIFOS 20%	27		27	24	29											31	26						
32	748054	18	F	STUDENT	7/12/2016	4	DICHLOROVOS 75%	28		28	25	30	20										32	27						
33	748702	25	M	UNEMPLOYED	7/15/2016	7	UNKNOWN	29	4	29	26	31						17					33	28						
34	748877	18	F	STUDENT	7/15/2016	6	UNKNOWN			30	27	32	21										34	29			3			
35	749004	22	M	FARMER	7/16/2016	5	UNKNOWN			31		33											35	30						
36	749051	62	M	FARMER	7/16/2016	5	MONOCROTOPHOS 36%	30		32	28	34										21	36	31					23	
37	751480	21	M	FARMER	7/28/2016	4	UNKNOWN	31		33	29	35										22	37	32						
38	753788	26	F	HOUSEWIFE	8/9/2016	5	DICHLOROVOS 75%	32		34	30	36	22										38	33						
39	754734	84	M	FARMER	8/14/2016	4	TRIAZOPHOS 35%	33		35		37						18	1				39				4			10
40	754760	45	M	FARMER	8/14/2016	5	CHLORPYRIFOS 50%	34		36	31	38	23				18	19				23	40	34				17	24	
41	755661	29	M	FARMER	8/19/2016	6	MONOCROTOPHOS 36%	35			32	39	24					20				24	41	35						
42	756081	43	M	X-SERVICEMA	8/21/2016	6	MALATHION	36		37																	5			
43	756276	31	M	FARMER	8/22/2016	6	PHORATE	37				40											42	36						
44	757329	24	M	FARMER	8/27/2016	6	CHLORPYRIFOS 20%			38	33	41					19	21				25	43	37						
45	758310	28	F	HOUSEWIFE	8/31/2016	5	TRIAZOPHOS 35%	38		39	34	42	25				20		2				44				6	18		11
46	758983	24	M	COOLI	9/5/2016	5	CHLORPYRIFOS 20%															26	45	38						
47	759355	30	M	BUSINESSMAN	9/7/2016	10	CHLORPYRIFOS 20%	39						11	6	1		22				27	46	39	8	5		19	25	12
48	759954	52	M	FARMER	9/9/2016	6	PROFENFOS 40%			40	35	43						23				28	47	40			7	20		
49	759908	42	F	HOUSEWIFE	9/9/2016	6	CHLORPYRIFOS 20%										21	24												
50	759944	22	F	HOUSEWIFE	9/9/2016	5	UNKNOWN				36	44	26									29	48	41				21	26	
51	760224	23	M	UNEMPLOYED	9/11/2016	5	UNKNOWN				37	45	27									30	49	42						
52	760717	18	F	STUDENT	9/13/2016	5	ACEPHATE 75%	40		41	38	46	28									31	50	43	9	6	8	22		
53	762686	37	M	FARMER	9/22/2016	6	CHLORPYRIFOS 20%	41		42	39	47						25					51	44	10		9		27	
54	762824	58	M	FARMER	9/23/2016	5	MONOCROTOPHOS 36%	42		43	40	48	29									32	52	45				23	28	
55	763700	32	F	COOLI	9/27/2016	1	DICHLOROVOS 75%	43	5	44	41	49	30		7			26					53	46						
56	764344	30	F	HOUSEWIFE	10/1/2016	5	CHLORPYRIFOS 20%	44			42	50				2						33	54	47					29	
57	764774	27	F	HOUSEWIFE	10/4/2016	6	CHLORPYRIFOS 20%	45		45	43	51	31					27					55	48					30	
58	765686	48	F	HOUSEWIFE	10/8/2016	0.75	CHLORPYRIFOS 20%	46		46	44	52		12			22	28				34	56	49				24	31	13
59	765736	35	F	HOUSEWIFE	10/8/2016	5	PROFENFOS 40%	47		47	45	53	32	13	8		23	29				35	57		11			25	32	14
60	766014	28	M	UNEMPLOYED	10/11/2016	5	CHLORPYRIFOS 20%			48		54					24	30				36	58					26	33	15
61	766478	24	F	HOUSEWIFE	10/13/2016	6	GLYPHOSATE 41%	48		49	46	55	33			3	25					37	59	50			10	27		
62	767452	30	F	FARMER	10/18/2016	6	CHLORPYRIFOS 20%	49		50	47	56	34	14			26	31				38	60	51				28	34	
63	770014	26	M	FARMER	11/2/2016	5	CHLORPYRIFOS 50%				48	57				4							61	52					35	
64	770053	32	F	COOLI	11/3/2016	6	CHLORPYRIFOS 20%	50		51	49	58					27	32				39	62	53				29		
65	771538	40	M	FARMER	11/9/2016	6	CHLORPYRIFOS 20%	51		52	50	59										40	63	54						
66	772186	40	M	CONTRACTER	11/13/2016	4	CHLORPYRIFOS 50%	52		53	51	60					28						64	55						
67	772690	75	M	FARMER	11/16/2016	5	MALATHION	53		54	52	61					29	33					65	56						
68	772838	19	F	COOLI	11/16/2016	7	CHLORPYRIFOS 50%	54		55		62	35	15			30	34				41	66	57		7	11	30	36	16
69	773040	24	M	FARMER	11/17/2016	5	CHLORPYRIFOS 50%	55		56	53	63					31	35				42	67	58	12	8	12	31		
70	774629	31	M	CLERK	11/27/2016	6	PROFENFOS 40%	56		57	54	64	36	16			32					43	68	59					37	
71	775185	40	M	FARMER	11/30/2016	6	DIMETHOATE 75%	57		58	55	65	37	17		5	33	36			1	44	69	60	13	9		32	38	17
72	777171	35	F	FARMER	12/10/2016	5	UNKNOWN	58		59	56	66					34	37				45	70	61				33	39	18
73	779793	24	M	FARMER	12/26/2016	5	PROFENFOS 40%	59		60	57	67										46	71	62						
74	780520	21	F	HOUSEWIFE	12/30/2016	4	CHLORPYRIFOS 20%	60		61	58	68				6	35	38				47	72	63	14	10		34	40	19
75	780764	30	M	BUSINESSMAN	1/1/2017	5	MALATHION				59	69	38							1			73	64			13			
76	781791	23	M	UNEMPLOYED	1/6/2017	7	CHLORPYRIFOS 50%	61				70	39	18	9	7	36	39					74	65	15	11	14		41	20
77	782468	24	F	HOUSEWIFE	1/10/2017	5	UNKNOWN	62		62	60	71	40	19			37	40				48	75	66	16			35	42	21
78	783189	18	F	STUDENT	1/15/2017	5	CHLORPYRIFOS 50%	63		63	61	72	41	20		8	38	41				49	76	67	17	12	15	36	43	22
79	783401	22	M	FARMER	1/16/2017	7	CHLORPYRIFOS 20%	64		64	62	73	42	21			39	42				50	77	68	18		16	37	44	23
80	783523	35	M	COOLI	1/17/2017	5	DICHLOROVOS 75%	65		65	63	74	43	22	10	9	40	43				51	78	69	19	13		38	45	24
81	784564	24	M	COOLI	1/23/2017	6	CHLORPYRIFOS 20%	66		66	64	75	44	23			41	44					79	70	20	14		39	46	25
82	785343	33	M	COOLI	1/27/2017	1	CHLORPYRIFOS 20%	67	6	67	65	76	45	24		10	42	45				52	80	71	21			40	47	26
83	787596	50	M	BUSINESSMAN	2/8/2017	8	CHLORPYRIFOS 50%	68		68	66	77	46	25			43	46				53	81	72	22	15		41	48	27
84	788085	25	F	HOUSEWIFE	2/11/2017	5	PROFENFOS 40%			69	67	78	47									54	82	73						
85	789082	35	M	FARMER	2/16/2017	5	PROFENFOS 40%	69		70	68	79	48				44	47				55	83	74	23			42	49	28
86	789283	34	M	FARMER	2/17/2017	5	CHLORPYRIFOS 50%	70		71	69	80		26			45	48				56	84	75	24			43	50	29
87	792571	19	M	UNEMPLOYED	3/6/2017	5	CHLORPYRIFOS 50%	71		72	70	81	49									57	85	76				44	51	30
88	792593	52	M	BUSINESSMAN	3/7/2017	5	CHLORPYRIFOS 50%	72		73	71	82	50									58	86	77			17	45	52	

	PAST HISTORY	TREATMENT HISTORY	GENERAL PHYSICAL EXAMINATION									VITALS							EXAMINATION			INVESTIGATIONS														
CASE NUMBER	ANY SIGNIFICANT	ANY SIGNIFICANT	SWEATING	CYANOSIS	PUPILLARY SIZE (MM)	SMELL OF COMPOUND	FASICULATIONS	NECK MUSCLE WEAKNESS	PALLOR	ICTERUS	PEDAL EDEMA	TEMPERATURE (F)	PULSE RATE/MIN	RESPIRATORY RATE	SYSTOLIC BLOOD PRESSURE MMHG	DIASTOLIC BLOOD PRESSURE	PULSE OXIMETRY (%SPO2)	OXYGEN SUPPLEMENTATION	GLASGOW COMA SCALE (/15)	SINGLE BREATH COUNT	POP SCALE	HEMOGLOBIN	TOTAL COUNTS	BLOOD UREA NITROGEN (MG/DL)	CREATININE (MG/DL)	SODIUM (Meq/L)	POTASSIUM (Meq/l)	SERUM CHOLINESTERASE ON ADMISSION (u/l)(7000-19000)	LOWEST SERUM CHOLINESTERASE	RANDOM BLOOD SUGAR	TOTAL BILIRUBIN	SGOT (15-37)	SGPT (30-65)	ABG- PH	ABG-PCO2	ABG-HCO3
1	NS	NS	1	1	PINPOINT	1	1	1				99	140	40	100	50	80	48	7	N/A	8	15.7	34.7	27	1.04	145	3.45	1317	1317	322	0.66	44	40	7.31	48	15.2
2	NS	NS	2		1	2						98.6	120	24	120	60	98	50	13	N/A	5	16.5	10.7	25	0.86	135	3.69	1208	595	129	0.39	33	48	7.39	40	21.1
3	NS	NS	3		7			2				99	110	40	100	50	96	73	10	N/A	3	15	24.6	26	1.27	156	3.68	9930	7928	142	12.2	37	32	7.29	40	15.6
4	NS	NS	4		6		2	3				99	130	40	100	50	79	72	8	N/A	4	15.3	24	28	1.62	139	3.33	656	656	256	0.47	42	34	7.21	47	17.2
5	NS	NS	5		7							100	120	24	110	90	98	47	13	N/A	3	13.8	18.1	20	0.67	138	3.43	773	773	122	0.7	14	17	7.36	32	23.2
6	NS	NS	6		7							99	120	38	120	70	96	70	9	N/A	3	13.2	11.2	55	1.2	131	3.83	4804	4381	126	1.37	132	42	7.36	39	23.8
7	NS	NS	7		5		3					99	120	24	110	80	96	71	13	N/A	2	11.2	30.4	25	1.05	140	4.09	1125	1125	155	0.64	18	17	7.43	26.7	13.4
8	NS	NS	8		7	3	4	4				99	100	32	120	70	80	61	7	N/A	5	16.6	21.2	20	1.53	162	2.49	878	878	165	0.4	74	37	7.2	42	10.1
9	NS	NS	9		7		5					99	120	24	110	80	96	63	12	N/A	3	13.8	14,3	31	0.94	141	3.68	723	723	119	0.78	24	30	7.45	29	22.7
10	OCCASIONAL ALCOH	NS	10		7							99	80	24	100	60	98	62	13	NA	3	15.2	13.5	16	0.79	139	3.58	3803	3803	97	0.28	43	27	7.43	31.2	26
11	NS	NS	11		4							98.6	100	26	120	80	98	66	13	22	0	12.9	10.5	26	0.545	135	4.92	6627	6627	113	0.95	23	20	7.33	42	24.1
12	NS	NS	12		PINPOINT	4	6	5				99	130	90	100	50	90	67	9	N/A	5	14.5	20.5	24	0.7	140	3.4	3098	3098	96	1.06	71	96	7.34	35	22
13	NS	NS	13	2	PINPOINT	5	7	6				99	110	40	90	60	99	68	2T	N/A	8	14.7	12.2	35	1.29	173	2.43	861	861	242	0.33	49	20	7.2	44	15
14	NS	NS	14	3	PINPOINT		8	7				99	130	40	90	50	80	69	3	N/A	8	16.8	16.5	35	2.67	148	3.64	16587	16587	65	1.13	259	158	7.1	46	13.9
15	NS	NS	15		PINPOINT	6						99	120	30	100	50	90	59	12	N/A	5	12.4	5.6	11	0.6	140	3.4	10513	10513	83	0.15	21	18	7.45	29	26.6
16	NS	NS	16		4							98	80	24	120	70	98		15	24	1	11.4	14.3	23	0.72	138	4.55	12291	12244	292	1.05	30	19	7.3	39	17.5
17	NS	NS			7					1		98.4	120	30	140	90	96	1	13	4	3	13.9	12.1	13	0.72	141	3.53	4544	801	135	2.79	21	25	7.4	38	26.1
18	NS	NS	17		7		9	8	1			100	140	44	90	50	78	2	4	N/A	5	10.9	13.3	46	2.27	147	4.54	1033	1033	283	0.96	77	36	7.2	24.1	10
19	NS	NS	18		6		10	9	2			100	88	40	100	50	80	3	10	N/A	3	9.5	35.2	35	1.3	145	3.61	795	795	150	0.16	90	60	7.1	23	12.3
20	NS	NS			4							98	110	22	120	70	98		15	28	1	14.7	19.5	25	0.8	139	3.96	10697	10697	107	0.66	13	22	7.38	39.9	23
21	NS	NS	19	4	1	7	11	10	3			101	144	48	80	40	78	35	4	N/A	9	9.4	6.7	37	0.59	135	4.89	1386	1386	108	1.94	117	29	7.2	24	13
22	NS	NS			4		12	11	4			99	110	38	100	50	88	4	13	N/A	3	11.2	18.4	34	0.75	143	3.65	1919	1919	88	0.98	86	48	7.35	32	23
23	NS	NS			6			12				99	130	36	120	90	95	5	13	16	3	13.6	9.9	12	0.57	139	4.2	711	631	136	0.76	19	22	7.4	39	26.6
24	NS	NS			3		13					99	90	20	140	90	96		14	20	3	13.3	10.4	15	0.49	140	3.66	1865	1865	138	1.01	26	15	7.34	34	23.1
25	NS	NS			2							99	104	24	140	90	95		14	18	3	14.6	10.6	27	1.27	142	4.05	963	667	98	0.4	37	37	7.36	39	24.4
26	NS	NS	20		5							99	140	28	140	100	96		14	13	3	15.1	15.7	16	0.75	138	3.19	851	818	95	0.29	28	29	7.2	28	15
27	NS	NS	21		6		14					99	80	32	130	90	95	6	14	18	3	13.6	20.3	27	0.48	134	4.46	646	539	110	0.3	40	22	7.2	28	18
28	NS	NS	22	5	8	8	15	13				100	142	40	100	50	80	51	9	N/A	5	14.1	16.9	51	1.21	143	3.16	13928	13928	127	0.14	61	26	7.28	42	16.8
29	NS	NS	23		4			14				99.2	144	24	150	90	95		11	N/A	3	18.4	28.9	28	0.99	164	4.2	1131	867	112	0.87	33	18	7.3	30	20
30	NS	NS	24		7		16					99	106	30	120	80	98	59	13	N/A	3	13.9	7.8	18	0.58	140	4.39	13276	13276	95	0.3	20	22	7.48	28	26
31	SMOKER	NS			2							98.6	80	26	140	90	94		13	19	4	12.9	13.9	14	1.18	137	3.85	11353	58	152	0.67	40	35	7.33	33	22.3
32	NS	NS			4				5			99	75	26	130	80	94		14	15	3	9.4	19	16	0.47	143	3.71	6992	6992	103	0.24	29	14	7.4	38	25
33	NS	NS			2	9	17	15				98	78	30	130	90	100	7	4	N/A	5	17.6	27.4	26	0.83	145	4.29	991	991	204	0.97	35	24	6.825	166	26.8
34	NS	NS	25		5							98	150	38	140	90	92		13	10	3	11.2	13.7	10	0.48	141	4.14	700	689	127	0.89	22	15	7.44	36	28
35	NS	NS			6							99	130	30	120	70	97		10	N/A	2	13.5	7.3	73	1	140	3	1617	1617	100	0.77	273	73	N/A	N/A	N/A
36	NS	NS	26		4							98	130	22	140	70	96		12	16	3	11.3	17.6	17	1	148	3.89	826	826	146	0.36	80	30	7.38	38	26
37	NS	NS			3							89.2	140	24	126	80	100		12	N/A	3	13.8	18	38	0.96	140	3.4	1696	807	157	0.5	35	22	7.41	39	20
38	NS	NS			4							98.4	88	22	130	70	92		14	18	3	12.6	12.7	17	0.64	141	3.84	771	771	90	1.11	24	10	7.35	36	18
39	NS	NS	27		6		18					100	120	26	140	90	96		14	14	2	12.3	17.8	53	1.18	137	3.38	224	224	199	1.18	38	26	7.463	24.5	17.1
40	NS	NS	28		7		19					100	110	30	100	60	98	58	13	N/A	3	15.3	13.1	28	0.63	143	3.95	9069	7307	107	0.64	22	19	7.39	33	20
41	NS	NS	29		7		20					99	110	28	120	80	98	60	13	N/A	3	16.2	13.3	13	0.7	142	4.3	9545	7822	98	1.01	17	22	7.38	38	21
42	ALCOHOL	NS			4							99	90	18	140	90	100		15	26	2	12.6	10.6	13	0.79	131	3.5	10411	6411	136	1.03	78	65	7.4	38.5	24.1
43	NS	NS			6							99.6	130	24	140	100	96		13	18	3	11.2	10.7	18	0.63	143	4.37	232	96	91	1.44	18	13	7.46	29.7	20.5
44	NS	NS	30		2		21	16				98.4	98	38	110	70	86	8	11	8	3	10	34.6	43	0.51	137	4.69	173	100	168	0.48	46	21	7.318	43.9	22
45	NS	NS	31		4							99.4	120	28	160	90	94	9	14	12	2	14.6	18.5	18	0.51	135	3.4	931	77	211	0.36	10	11	7.3	30	15
46	NS	NS			4							99	90	17	110	70	98		15	26	0	15.7	10.7	10	0.47	140	4.15	6215	6215	102	1.52	25	11	N/A	N/A	N/A
47	ONAL SMOKER, AL	NS	32		2	10	22	17				98.4	77	34	120	80	80	10	8	N/A	7	17.4	22.1	25	1.7	135	5	139	139	151	0.56	72	74	7.27	51.6	23.3
48	TENSION TYPE 2 DM, OCCASI	NS	33		8		23	18				99	120	24	150	80	96		14	24	1	15.3	14.9	29	0.94	149	4.42	145	145	99	1.57	40	27	7.28	29	14
49	TPE 2 DM	NS			3		24	19				98.4	80	28	100	60	94	11	15	N/A	1	10.1	13.3	44	0.47	147	3.08	1502	1502	161	0.68	25	16	7,56	29.4	25.6
50	NS	NS	34		6							99	100	26	120	60	98		13	22	3	13	9.9	13	0.53	144	3.93	5973	5940	91	0.71	14	8	7.41	33	24
51	NS	NS	35		7							98.6	120	24	140	60	98	65	13	18	1	14.2	14.2	58	1.13	146	5.3	7501	6804	173	0.63	20	12	7.34	37	23
52	NS	NS	36		2							98.4	110	22	110	80	96		14	20	3	12.5	22.5	23	0.52	140	3.63	2860	2860	122	0.44	18	11	7.404	24.2	14.8
53	OCCASIONAL ALCOH	NS			3		25	20				98.4	120	38	140	100	80	12	8	N/A	4	12.1	24.3	21	1.03	141	3.81	121	87	141	0.18	26	41	7.1	24	13
54	NS	NS	37		7	11						100	140	28	110	80	98	34	13	N/A	3	12.1	8.9	28	0.96	141	4.38	211	73	114	0.96	22	13	7.33	33	21
55	NS	NS			2		26	21				99	110	40	140	100	80	13	8	N/A	5	15.9	31.1	37	0.79	129	3.41	425	425	127	0.43	25	11	7.1	22	10
56	NS	NS	38		7							98	120	24	110	70	98	57	13	18	3	12.4	11	14	0.51	137	4.08	4093	3436	105	0.62	19	15	7.34	39	17
57	NS	NS			6		27	22	6			99	140	30	150	100	98	14	13	10	3	9.2	13.8	50	0.55	137	3.49	74	74	108	0.73	22	10	7.3	29	19
58	PERTENSION TYPE 2	NS			2	12	28	23				99	125	40	130	90	99	15	7T	N/A	6	13.8	13.6	15	0.54	132	3.27	128	117	278	0.14	36	47	7.3	25.6	14.7
59	NS	NS	39		2	13	29	24	7			99	130	40	110	80	70	16	6T	N/A	7	9.5	25.5	32	0.56	137	3.4	88	59	193	1	34	18	7.46	32.1	24
60	NS	NS	40		7	14	30	25				99	120	38	140	70	70	17	6T	N/A	4	12.6	17.9	18	0.92	135	3.92	236	236	107	0.37	27	18	7.49	28.2	28
61	NS	NS	41		6							99	100	26	120	80	96	46	13	21	3	11.1	8.9	22	0.64	141	4.22	8109	8109	128	0.3	19	22	7.4	38	17
62	NS	NS	42		7		31	26				98.4	100	48	140	90	100	18	13	N/A	4	10.8	16.7	10	0.7	153	3.4	5443	4927	150	0.17	30	2664	7.27	28	14
63	NS	NS	43		7							98.8	94	24	130	70	96		14	22	2	14.3	11.3	21	0.42	154	4.64	6390	6758	243	0.34	18	14	7.34	34	18
64	NS	NS	44		2	15	32	27				98.6	120	30	130	90	96	19	14	13	5	12.2	18.5	8	0.58	132	3.47	240	240	323	0.69	19	16	7.36	28.1	15.4
65	NS	NS			2				8			98.6	150	30	150	90	96	20	13	N/A	4	8.6	13.6	26	1.01	140	3.87	217	106	91	0.55	335	38	7.3	36	24
66	NS	NS			3							99	74	30	110	60	90	21	13	16	3	17	15.1	25	0.93	136	3.93	223	91	79	0.12	29	32	7.28	26	16
67	HYPERTNSION SMOKER OCCASIO	NS			2		33	28				98.4	130	38	160	90	86	22	11	N/A	4	17.2	11.1	67	2.61	147	5.1	63	37	310	1.03	26	17	7.2	30	20
68	NS	NS	45	6	1	16	34	29				100	120	40	90	50	78	24	7	N/A	8	11.1	8.5	18	0.47	141	3.7	129	129	222	0.4	19	14	7.2	23	13
69	NS	NS	46		1	17	35	30	9			99.6	55	28	100	60	94	23	12	N/A	6	8.5	15.2	17	0.65	139	3.05	120	77	142	0.69	25	17	7.2	70	15
70	NS	NS	47		7			31				99	124	26	110	70	96	49	11	N/A	4	15.2	12.6	17	0.94	137	4.2	6977	1935	88	1.04	15	15	7.4	40	24.8
71	NS	NS	48	7	1	18	36	32				99.6	120	44	100	60	80	25	7	N/A	7	9.9	18	24	1.29	154	3.05	116	116	153	0.43	51	19	7.35	34	22
72	NS	NS	49		1	19	37	33				98.9	160	28	80	60	88	26	8	N/A	5	12.6	12.9	69	2	151	3.2	7539	7539	191	0.1	720	131	7.119	51.4	16.3
73	NS	NS			7							98.4	116	28	120	70	99		14	15	3	11.6	22.5	10	0.68	147	4.21	127	127	136	0.11	19	18	7.347	39.9	21.2
74	NS	NS	50		3		38					99.4	126	28	80	40	48	27	12	12	3	11.2	8.4	26	0.55	141	4.55	303	303	91	0.27	19	12	7.4	38	21
75	NS	NS			3							98	130	28	110	80	98	29	15	20	1	16.4	9.3	10	0.94	141	4.17	8040	7803	92	0.42	19	17	7.4	38	24
76	NS	NS	51	8	1	20	39	34		2	1	100	130	50	80	40	78	28	4	N/A	8	16.7	12.6	74	6.03	144	4.98	6801	6801	84	7.34	256	104	7	22	12
77	NS	NS	52	9	PINPOINT		40	35				100	100	40	100	50	80	64	8	N/A	7	15.1	23.8	27	0.66	175	4.51	5643	1874	142	0.42	24	20	7.18	44	12
78	NS	NS	53		1	21	41	36				100	120	30	100	80	98	30	11	N/A	6	12.1	7.1	30	0.59	142	3.45	98	92	113	0.23	34	10	7.3	46.5	23
79	NS	NS	54	10	1	22	42	37				94.6	110	44	100	50	80	31	8	N/A	7	16.1	16.7	27	0.95	141	2.65	1015	238	295	1.52	26	25	7.1	28	12
80	NS	NS	55	11	5	23	43	38				100	100	44	80	40	80	32	6	N/A	5	16.9	22.1	48	0.86	143	4.96	569	464	245	0.52	50	34	7.21	30	16
81	NS	NS			7	24	44	39				99.6	140	44	110	60	82	33	8	N/A	6	12.9	26.3	18	0.98	144	3.54	6751	6306	210	0.37	31	23	7.1	68	10
82	NS	NS	56	12	2	25	45	40				100	150	28	100	50	80	55	8	N/A	7	16.3	8.6	15	0.69	143	3.69	5072	5072	65	0.3	37	19	7.34	43.3	23.1
83	NS	NS	57	13	1	26	46	41				100	110	44	100	70	84	36	6	N/A	8	12.2	9.7	13	0.74	137	4.3	128	128	196	0.78	16	13	7.2	44	18
84	NS	NS	58		6							100	104	22	120	80	98	54	13	N/A	1	13.7	14.2	20	0.76	143	4.56	8898	7770	125	0.43	29	12	7.45	29	19.7
85	NS	NS	59		8		47					100	110	28	120	80	94	37	13	10	3	16	20.6	32	0.84	144	5.23	110	84	150	0.28	39	74	7.2	35.5	16.8
86	NS	NS			7	27	48	42		3		100	140	44	90	60	84	38	8	N/A	5	16.7	22.5	18	0.6	145	2.9	273	273	95	8.63	32	18	7	108.8	27.6
87	NS	NS	60		6							99	110	24	140	80	98	52	13	20	3	15.5	15.7	28	0.95	154	3.8	6425	6425	150	0.52	31	24	7.4	38	23
88	OCCASIONAL ALCOH	NS	61		7			43				98	140	30	110	70	96	39	12	N/A	4	15.5	22.7	19	1.24	146	4.12	141	23	164	0.78	37	21	7.38	36	22

	TREATMENT					RESULTS					ECG CHANGES AT ADMISSION									ECG CHANGES AT DISCHARGE				COMPLCATIONS							CAUSE OF DEATH			
CASE NUMBER	GASTRIC LAVAGE	DURATION OF ATROPINE USE (DAYS)	ATROPINE TOTAL DOSE (MG)	DURATION OF PRALIDOXIME (DAYS)	PRALIDOXIME TOTAL DOSE (GM)	DURATION OF HOSPITAL STAY	VENTILATOR SUPPORT NEEDED	DURATION ON VENTILATOR SUPPORT (DAYS)	SURVIVOR	NON SURVIVOR	QT INTERVAL	RR INTERVAL	CORRECTED QT INTERVAL (QTC)	HEART RATE	ST ELEVATIONS	T INVERSIONS	PR INTERVAL	VPCs	ATRIAL FIBRILLATION	QT INTERVAL	RR INTERVAL	CORRECTED QT INTERVAL (QTC)	YES	ACUTE RENAL FAILURE	INTERMEDIATE SYNDROME	SEPSIS	CARDIAC ARRHYTHMIAS	SHOCK	ASPIRATION PNEUMONIA	METABOLIC ACIDODIS	VENTRICULAR TACHYCARDIA	INTERMEDIATE SYNDROME	ASPIRATION PNEUMONIA	METABOLIC ACIDOSIS
1	1	10	444	5	8.5	10	1	5	1		0.36	0.44	542.72042	136.363636			0.16			0.32	0.52	443.760157												
2	2	11	343.2	0	1	11			2		0.32	0.44	482.418151	136.363636			0.12			0.28	0.44	422.115882												
3	3	3	168	2	4	3	2	1	3		0.4	0.64	500	93.75			0.16	1		0.44	0.84	480.079359					1							
4	4	12	869	5	8.5	13	3	10	4		0.36	0.44	542.72042	136.363636			0.12			0.36	0.52	499.230177	1	1	1	1								
5	5	4	63.6	5	8.5	6			5		0.36	0.52	499.230177	115.384615			0.2			0.36	0.64	450												
6	6	5	99	5	8.5	6			6		0.32	0.48	461.880215	125			0.12			0.32	0.48	461.880215												
7	7	6	288	5	8.5	6			7		0.36	0.4	569.209979	150			0.12			0.32	0.52	443.760157												
8	8	4	243	3	5.5	4	4	4	8		0.4	0.68	485.07125	88.2352941			0.16			0.32	0.6	413.118224	2	2										
9	9	1	15	1	1	1			9		0.28	0.48	404.145188	125			0.16			0.28	0.48	404.145188												
10	10	7	154.2	5	8.5	8			10		0.44	0.76	504.714615	78.9473684			0.16			0.4	0.64	500												
11	11	0.5	11	1	1	0.5			11		0.48	0.68	582.0855	88.2352941			0.08			0.48	0.68	582.0855												
12	12	20	766.8	7	11.5	20	5	11	12		0.4	0.6	516.39778	100			0.24			0.36	0.64	450	3		2	2								
13	13	1	10	1	1	1	6	1		1	0.4	0.72	471.404521	83.3333333			0.24			0.4	0.72	471.404521	4				2				1			
14	14	1	13	1	1	0.5	7	0.5		2	0.36	0.48	519.615242	125			0.16	2		0.36	0.48	519.615242	5	3			3				2			
15	15	1	33	1	1	1			13		0.36	0.48	519.615242	125			N/A		1	0.36	0.48	519.615242	6				4							
16	16	4	54.6	4	7	5			14		0.52	0.84	567.366515	71.4285714			0.2			0.4	0.56	534.522484												
17	17	9	510	5	8.5	10			15		0.32	0.52	443.760157	115.384615			0.08			0.28	0.44	422.115882												
18	18	2	140	2	4	2	8	2		3	0.28	0.36	466.666667	166.666667			0.12			0.28	0.44	422.115882	7	4			5				3			
19	19	3	180	2	4	3	9	3	16		0.4	0.68	485.07125	88.2352941			0.22			0.4	0.68	485.07125												
20	20	1	3.6	0	1	1			17		0.36	0.64	450	93.75			0.2			0.36	0.64	450												
21	21	3	456	3	5.5	3	10	3		4	0.36	0.52	499.230177	115.384615		1	0.16			0.32	0.48	461.880215	8					1				1		
22	22	9	540	7	11.5	9	11	5	18		0.44	0.72	518.544973	83.3333333			0.24			0.36	0.48	519.615242												
23	23	17	912	7	11.5	20	12	2	19		0.32	0.52	443.760157	115.384615			0.16			0.32	0.52	443.760157												
24	24	7	264	7	11.5	8			20		0.32	0.68	388.057	88.2352941			0.16			0.32	0.68	388.057												
25	25	7	252	7	11.5	8			21		0.4	0.88	426.401433	68.1818182			0.12			0.32	0.64	400												
26	26	16	916	7	11.5	16			22		0.28	0.52	388.290137	115.384615			0.16			0.48	0.68	582.0855	9			3								
27	27	3	156	3	5.5	3			23		0.4	0.76	458.831468	78.9473684		2	0.16			0.36	0.64	450												
28	28	12	519.6	7	11.5	14	13	5	24		0.32	0.44	482.418151	136.363636			0.12			0.32	0.44	482.418151	10		3	4								
29	29	2	144	2	4	2	14	1		5	0.32	0.52	443.760157	115.384615			0.16			0.28	0.48	404.145188	11				6				4			
30	30	1	48	0	1	1			25		0.36	0.52	499.230177	115.384615			0.12			0.36	0.52	499.230177												
31	31	8	384	7	11.5	8			26		0.36	0.52	499.230177	115.384615		3	0.12			0.4	0.8	447.213596												
32	32	2	96	1	2.5	2			27		0.36	0.72	424.264069	83.3333333		4	0.16			0.4	0.92	417.028828												
33	33	7	528	7	11.5	7	15	7	28		0.32	0.48	461.880215	125			0.13			0.32	0.48	461.880215												
34	34	3	84	1	2.5	3			29		0.4	0.72	471.404521	83.3333333			0.22			0.36	0.6	464.758002												
35	35	10	448.8	7	11.5	13			30		0.4	0.6	516.39778	100	1		0.16			0.32	0.56	427.617987												
36	36	4	9.6	7	11.5	7			31		0.36	0.44	542.72042	136.363636			0.16			0.32	0.6	413.118224												
37	37	4	34.4	7	11.5	7			32		0.4	1.08	384.90018	55.5555556			0.22			0.32	0.68	388.057												
38	38	3	72	2	4	3			33		0.36	0.76	412.948321	78.9473684	2		0.12			0.32	0.64	400												
39	39	4	5.5	3	5.5	10			34		0.32	0.52	443.760157	115.384615			0.22			0.36	0.68	436.564125												
40	40	5	372	5	8.5	6			35		0.4	0.8	447.213596	75			0.16			0.4	0.8	447.213596												
41	41	4	37.8	3	5.5	5			36		0.36	0.56	481.070235	107.142857			0.2			0.44	1.16	408.529744												
42	42	3	32	3	5.5	5			37		0.36	0.56	481.070235	107.142857			0.22			0.36	0.52	499.230177												
43	43	13	780	7	11.5	15			38		0.32	0.52	443.760157	115.384615			0.16			0.32	0.72	377.123617												
44	44	11	828	7	11.5	11	16	11	39		0.36	0.68	436.564125	88.2352941			0.22			0.36	0.72	424.264069	12		4	5								
45	45	3	16.8	3	5.5	4			40		0.36	0.88	383.761289	68.1818182			0.16			0.28	0.52	388.290137												
46	46	3	60	3	5.5	3			41		0.36	0.76	412.948321	78.9473684	3		0.16			0.28	0.48	404.145188												
47	47	11	1368	7	11.5	11	17	11		6	0.32	0.4	505.964426	150			0.16			0.36	0.76	412.948321	13	5	5	6						2		
48	48	7	300	7	11.5	7	18	1		7	0.36	0.52	499.230177	115.384615			0.16			0.28	0.48	404.145188	14						1				1	
49	49	10	228	0	0	24	19	12	42		0.36	0.52	499.230177	115.384615			0.22			0.28	0.44	422.115882	15		6	7								
50	50	4	46.2	3	5.5	5			43		0.44	0.64	550	93.75			0.16			0.4	0.84	436.435781												
51	51	4	46.2	3	5.5	4			44		0.32	0.44	482.418151	136.363636			0.16			0.36	0.52	499.230177												
52	52	10	819	7	11.5	10			45		0.36	0.64	450	93.75			0.16			0.28	0.52	388.290137												
53	53	10	516	7	11.5	10	20	5	46		0.4	0.56	534.522484	107.142857			0.16			0.36	0.56	481.070235												
54	54	7	624	5	8.5	7			47		0.36	0.52	499.230177	115.384615			0.2			0.4	0.76	458.831468												
55	55	1	67	0	1	1	21	1		8	0.4	0.52	554.700196	115.384615			0.16	3		0.36	0.64	450	16				7				5			
56	56	4	134.4	4	7	5			48		0.4	0.68	485.07125	88.2352941			0.2			0.36	0.56	481.070235												
57	57	8	288	6.67	11	10	22	3	49		0.4	0.64	500	93.75			0.15			0.36	0.6	464.758002												
58	58	4	552	4	7	4	23	4		9	0.32	0.48	461.880215	125			0.16			0.2	0.4	316.227766	17									3		
59	59	26	1096	7	11.5	26	24	20		10	0.36	0.48	519.615242	125			0.22			0.32	0.48	461.880215	18		7	8						4		
60	60	11	204	7	11.5	11	25	6	50		0.4	0.56	534.522484	107.142857			0.16			0.36	0.72	424.264069	19		8	9								
61	61	4	79.2	3	5.5	4			51		0.4	0.64	500	93.75			0.16			0.36	0.68	436.564125												
62	62	6	132	7	11.5	6	26	6	52		0.32	0.44	482.418151	136.363636		5	0.22			0.32	0.48	461.880215												
63	63	9	177	7	11.5	11			53		0.44	0.48	635.085296	125			0.16			0.4	0.56	534.522484	20			10								
64	64	27	2172	7	11.5	29	27	17	54		0.36	0.64	450	93.75			0.22			0.36	0.52	499.230177	21		9	11								
65	65	6	183	7	11.5	8			55		0.36	0.48	519.615242	125			0.16			0.32	0.48	461.880215												
66	66	14	1176	7	11.5	14			56		0.4	0.92	417.028828	65.2173913			0.12			0.36	0.72	424.264069	22			12								
67	67	6	336	7	11.5	6	28	5	57		0.32	0.56	427.617987	107.142857			0.12			0.28	0.52	388.290137	23	6										
68	68	15	2016	5	8.5	15	29	10	58		0.4	0.52	554.700196	115.384615			0.16			0.36	0.68	436.564125	24		10	13								
69	69	22	1768	7	11.5	22	30	13	59		0.48	1.08	461.880215	55.5555556			0.16			0.36	0.8	402.492236	25		11	14								
70	70	22	674.4	5	8.5	23	31	5	60		0.36	0.52	499.230177	115.384615			0.16			0.28	0.4	442.718872	26		12	15								
71	71	3	384	3	5.5	3	32	3	61		0.4	0.68	485.07125	88.2352941	4		0.2			0.36	0.6	464.758002												
72	72	0.21	33	1	2	1	33	1		11	0.36	0.48	519.615242	125	5	6	0.16	4		0.28	0.36	466.666667	27	7			8	2			6			
73	73	8	2446	5	8.5	8			62		0.28	0.48	404.145188	125			0.16			0.28	0.6	361.478446												
74	74	8	420	5	8.5	8			63		0.36	0.52	499.230177	115.384615			0.2			0.32	0.52	443.760157	28					3						
75	75	3	132	4	7	4			64		0.32	0.44	482.418151	136.363636			0.16			0.4	0.92	417.028828												
76	76	1	144	1	2.5	1	34	1		12	0.36	0.48	519.615242	125			0.24			0.36	0.48	519.615242	29	8				4		1				1
77	77	2	75	2	4	3	35	3	65		0.36	0.64	450	93.75			0.12			0.36	0.64	450												
78	78	14	1848	7	11.5	16	36	5	66		0.4	0.48	577.350269	125			0.16			0.36	0.64	450	30		13									
79	79	13	1260	7	11.5	15	37	5	67		0.36	0.52	499.230177	115.384615			0.12			0.36	0.76	412.948321	31		14	16								
80	80	7	984	5	8.5	7	38	3	68		0.52	0.76	596.480908	78.9473684		7	0.16			0.44	0.72	518.544973	32					5						
81	81	7	624	5	8.5	9	39	1	69		0.32	0.4	505.964426	150			N/A			0.4	0.76	458.831468												
82	82	1	27	1	2.5	2	40	2		13	0.36	0.52	499.230177	115.384615			0.2			0.36	0.52	499.230177	33				9				7			
83	83	11	1252	5	8.5	14	41	5	70		0.36	0.6	464.758002	100			0.16			0.36	0.68	436.564125	34		15									
84	84	2	27.6	1	2.5	3			71		0.36	0.6	464.758002	100			0.2			0.36	0.8	402.492236												
85	85	10	966	2.33	4.5	12			72		0.4	0.56	534.522484	107.142857			0.12			0.36	0.56	481.070235	35			17								
86	86	7	1128	6	10	7	42	5	73		0.36	0.48	519.615242	125			0.12			0.36	0.52	499.230177												
87	87	2	26.4	2	4	7			74		0.4	0.56	534.522484	107.142857			0.16			0.4	0.56	534.522484												
88	88	24	2150	7	11.5	27	43	14	75		0.36	0.48	519.615242	125			0.12			0.4	0.64	500	36		16	18								

89	793251	23	M	FARMER	3/10/2017	5	MONOCROTOPHOS 36%	73		74	72	83	51	27			46	49				59	87	78	25		18	46	53	31	NS	NS	62		7		49	44				100	144	40	100	60	90	40	9	N/A	5	13.7	17.9	26	1.14	142	4.55	867	867	339	0.42	24	19	7.28	44	21	89	8	638.4	4	7	8	44	2	76		0.32	0.44	482.418151	136.363636			0.2			0.44	0.92	458.731711												
90	794090	20	F	COOLI	3/15/2017	1	ACEPHATE 75%	74		75	73	84	52									60	88	79				47	54		NS	NS	63		2	28						100	130	24	100	60	96	41	12	15	5	12.6	13.6	19	0.62	141	4.18	750	750	96	0.62	23	11	7.4	38	23	90	6	179	5	8.5	7			77		0.36	0.56	481.070235	107.142857			0.2			0.32	0.56	427.617987												
91	794563	21	F	HOUSEWIFE	3/17/2017	1	CHLORPYRIFOS 50%			76	74	85	53			11						61	89	80				48	55		NS	NS	64		1	29						98.6	126	28	100	60	98	42	13	n/A	6	9.8	18.5	18	0.51	143	3.68	129	129	96	0.6	19	20	7.39	35.9	21	91	4	384	4	7	4			78		0.32	0.44	482.418151	136.363636			0.16			0.32	0.48	461.880215												
92	794774	42	F	BUSINESSMAN	3/17/2017	1	CHLORPYRIFOS 50%	75		77	75	86	54	28			47	50				62	90	81		16		49	56	32	NS	NS	65		2	30	50	45				98	130	40	80	60	78	43	6	N/A	7	11.1	21.2	16	0.75	135	3.55	513	98	188	0.3	18	16	7.34	33.3	17.4	92	9	391	7	11.5	11	45	3	79		0.36	0.36	600	166.666667			N/A		2	0.36	0.56	481.070235	37			19	10	6						
93	796825	55	M	UNEMPLOYED	3/30/2017	7	CHLORPYRIFOS 50%	76		78	76	87	55	29		12	48	51				63	91	82	26	17		50	57	33	NS	NS	66	14	1	31	51	46				100	100	44	90	50	80	44	8	N/A	7	14.6	14.4	25	1	141	3.42	575	575	180	0.37	28	18	7.2	24	14	93	9	463.2	7	11.5	9	46	5	80		0.44	0.76	504.714615	78.9473684			0.16			0.36	0.56	481.070235												
94	797261	45	M	LABOURER	3/31/2017	5	MALATHION			79	77	88				13	49	52				64	92	83			19	51	58		NS	NS	67		7		52					99	90	24	120	80	96	45	15	16	2	14.2	11.2	19	1.3	138	3.64	2876	2876	104	1.55	27	19	7.41	37	21	94	7	244.8	5	8.5	7			81		0.44	0.84	480.079359	71.4285714	6		0.2			0.4	0.84	436.435781												
95	797359	35	M	UNEMPLOYED	4/1/2017	4	GLYPHOSATE 41%	77	7	80	78	89	56	30			50	53				65	93	84				52	59		NS	NS	68	15	7		53	47				100	100	40	100	50	79	55	7	N/A	6	13.1	23.6	18	0.9	140	4.65	250	250	94	0.93	26	23	7.4	41	25.4	95	9	202.8	5	8.5	10	47	3	82		0.32	0.52	443.760157	115.384615			0.16			0.36	0.68	436.564125												
96	802615	26	F	HOUSEWIFE	4/30/2017	6	CHLORPYRIFOS 50%	78	8	81	79	90	57				51	54				66	94	85				53	60		NS	NS	69		7		54	48				100	110	26	110	80	96	56	11	N/A	3	8.7	4.8	12	0.44	137	1.98	693	693	145	0.41	32	28	7.55	29.6	25.6	96	37	2530	7	11.5	25	48	25	83		0.44	0.68	533.578375	88.2352941			0.2			0.32	0.6	413.118224	38		17									
97	803201	25	F	HOUSEWIFE	5/4/2017	6	UNKNOWN			82	80	91	58										95	86			20	54	61		NS	NS	70		7							99	120	24	120	80	98	57	13	18	2	14.6	2.9	19	0.72	150	3.64	1969	1969	104	1.21	23	18	7.41	38	21	97	4	63.6	3	5.5	5			84		0.36	0.52	499.230177	115.384615			0.16			0.36	0.6	464.758002												
98	803858	20	F	STUDENT	5/7/2017	1	CHLORPYRIFOS 20%	79		83	81	92	59									67	96	87				55	62		NS	NS	71		PINPOINT	32						99	70	24	140	70	98	59	13	N/A	4	10	12.6	22	0.55	147	4.14	3663	3663	125	0.49	16	11	N/A	N/A	N/A	98	1	23	1	1	1			85		0.48	1.12	453.557368	53.5714286			0.16			0.48	1.12	453.557368												
99	804615	30	F	HOUSEWIFE	5/11/2017	5	UNKNOWN	80		84	82	93	60				52						97	88				56	63		NS	NS	72		7							99	120	24	100	70	98	58	13	N/A	3	12.8	19.5	28	0.94	138	4.14	7294	7294	100	0.43	16	11	7.3	40	18	99	1	17	1	1.5	1			86		0.44	0.8	491.934955	75			0.2			0.44	0.8	491.934955												
100	805090	21	F	STUDENT	5/13/2017	6	PROFENFOS 40%	81		85	83	94	61	31			53	55				68	98	89			21	57	64		NS	NS	73		7		55	49				100	110	24	120	80	80	54	8	N/A	5	12.9	16.6	35	0.73	145	4.16	95	95	93	0.79	40	31	7.26	54	13	100	17	433.2	5	8.5	19	49	15	87		0.4	0.6	516.39778	100			0.16			0.32	0.52	443.760157	39		18	20								
101	806761	25	F	HOUSEWIFE	5/22/2017	1	MALATHION	82			84	95	62									69	99	90				58	65		NS	NS	74		2	33						98.6	110	24	120	80	98	56	13	15	4	12.6	14.2	11	0.58	135	4.19	1392	1392	124	0.44	19	13	7.43	33	15	101	14	626.4	7	11.5	16			88		0.4	0.56	534.522484	107.142857			0.12			0.4	0.64	500	40			21								
102	807258	27	M	FARMER	5/24/2017	5	CHLORPYRIFOS 50%	83				96											100	91			22				NS	NS	75		6							99	120	24	120	80	98	53	15	22	3	14	8.9	12	1	151	3.48	6937	6937	154	0.49	31	10	7.4	36	19	102	6	79.2	5	8.5	6			89		0.32	0.44	482.418151	136.363636			0.2			0.44	0.92	458.731711												