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Study the Clinical and Electrocardiographic Changes in Organophosphorus Poisoning

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Abstract: OP compound poisoning is a global problem and is most common medico-toxic emergency in India. Cardiac manifestations are also seen due to OP poisoning which may be serious and often fatal if not diagnosed early and treated adequately. We studied clinical and electrocardiographic changes in op poisoning. To study the clinical as well as electrocardiographic changes in patients with Organophosphorus compound poisoning. A total of 95 patients were included in the study. Patients admitted to tertiary care center with history of OP poisoning, with clinical symptoms and signs in less than 24hrs were included in the study. Electrocardiogram was done on day of admission, 3rd day and 7th day. In our study (51.6%) were female, 48.4 % were male. Incidence of OP poisoning was more common among age group 21-30 years. Farmers (35.8%) were more common among occupation. Monocrotophos (25.3%) was commonly used compound for poisoning. The commonest clinical findings noted were increased secretions (vomiting, urination, sweating) followed by signs such as mitosis and fasciculation's. Study also revealed significant electrocardiographic changes associated with OP poisoning during hospitalization and reverted during discharge by 7th day. Common electrocardiographic changes in patients with organophosphorus compound poisoning noticed were sinus tachycardia, sinus bradycardia, prolonged QTc, ST-T changes like ST inversion and T inversion and Tall T. Electrocardiogram changes can also be used as a prognostic tool.
Keywords: Organophosphorus, poisoning, Cardiac manifestations, Farmers

INTRODUCTION

During the past forty years, there have been fifteen thousand individual compounds and more than three lakh fifty thousand compounds with different formulas have been used as pesticides. But today the number actually used as insecticide doesn't exceed three dozen [1].

Organophosphorus (OP) compounds are the special group of chemicals which are aimed for the control of pest, weeds and other diseases of plant and have contributed significantly to increase good agricultural productivity and crop yields [2, 3]. Insecticides are the most commonly used as pesticides in the developing countries; herbicides are more commonly encountered in developed countries. As agriculture is major component of Indian economy, insecticides are widely used in our country. Incidence of poisoning by pesticides and consequent admission to the hospital has been increasing in recent decades [4].

Pesticides are easily available in lethal and concentrated forms. Self-poisoning with OP compounds is associated with thousands of deaths every year .Unemployment, failure in exam, increase in social and domestic as well as economic problems are ultimately leading to psychological problems such as stress may force a person to consume poisons such as OP compounds due it's low price, high toxicity and easily availability[5]. Accidental OP poisoning may occur due to inhalation while spraying the crops [6].

Some of the OP compounds which are grouped under esters are still being used for the treatment of glaucoma (Ecothiopate). Besides these favourable agricultural, veterinary, and medical uses, few of these are also used as "nerve gases" in the chemical warfare which are highly potent OP anticholinesterase compounds, includes Tabun, Sarin, Soman. These are also useful in the manufacturing of plastics (plasticizers), stabilizers used as lubricating and hydraulic liquids, in the prevention or inhibiting the outbreak of fire (flame retardants), and gasoline additives[7].

Organophosphorus compound inhibit the enzyme acetyl cholinesterase leading to accumulation of acetyl choline, which binds to muscarinic and

nicotinic receptors throughout nervous system. Signs and symptoms of OP poisoning are due to persistent acetylcholine hyperstimulation at muscarinic and nicotinic receptor sites [8].

There are two forms of cholinesterase's one is true cholinesterase or acetyl cholinesterase, distributed in all cholinergic sites, RBC, greywater. Hydrolysis of acetylcholine is very fast in less than 1 microsecond. Its function is termination of acetylcholine; another one is pseudo-cholinesterase or butyryl-cholinesterase, distributed in plasma, liver, intestine and white matter. Hydrolysis of acetylcholine is slow. Its function is hydrolysis of ingested esters. Both these types of enzymes are inhibited by insecticide poisoning [9].

Cardiac complications are also seen secondary to consumption or toxicity with these insecticide compounds, which may range from innocuous electrocardiographic manifestations to life-threatening complications, such as cardiogenic pulmonary edema and frequently causing death. These types of complications can be prevented and treated if recognised early. The pathogenesis, extent and the frequency of cardiac toxicity from these op compounds are not clearly defined. Because of the limited former literature many health care professionals may not be fully aware of the electrocardiographical changes of OP poisoning [10].

The electrocardiogram (ECG) in patients with OP poisoning may display a variety of abnormalities, such sinus tachycardia, sinus bradycardia, atrioventricular block, and ST segment and T-wave abnormalities, ventricular premature complexes, but extreme QT interval prolongation and ventricular tachydysrhythmia of the torsades de pointes type are not common. Thus early reorganization of abnormal rhythm in organophosphate poisoning protects the patients against acquiring life-threatening arrhythmias [11, 12].

Sympathetic and parasympathetic over-activity, hypoxemia, acidosis, electrolyte derangements, and a direct toxic effect of the compounds on the myocardium and vascular system are thought to be involved in myocardial damage associated with OP poisoning [12].

MATERIALS AND METHODS

The present study was carried out in department of Medicine, Shri B. M. Patil Hospital and Research Centre, Vijayapura, Karnataka. This study was conducted between November 2014 to June 2017.

Patient's admitted to hospital less than 24 hrs of history of OP poisoning with clinical symptoms and signs, Gastric lavage contents were sent for analysis to poison detection centre with confirmed PDC report were included in the study. Patients who have ingested non organophosphorus compound and with a history of cardiac diseases are excluded.

Patients ECG's are recorded at the time of presentation in casualty, on 3rd day and on 7th day. ECG's are recorded using BPL CARDIART 6108 T ECG Machine. Other investigations done were random blood glucose estimation, serum electrolytes (Sodium and Potassium), blood urea and serum creatinine, complete blood picture, chest radiograph, HIV, HBs Ag , liver function test and ABG (if necessary). Statistical data was analyzed by using Mean $\pm$ SD, diagrammatic present, correlation coefficient and chi- square test.
This study was approved by the ethical committee.

RESULTS

A total of 128 patients were recruited in the study.

Flow chart showing distribution of poisoning patients.

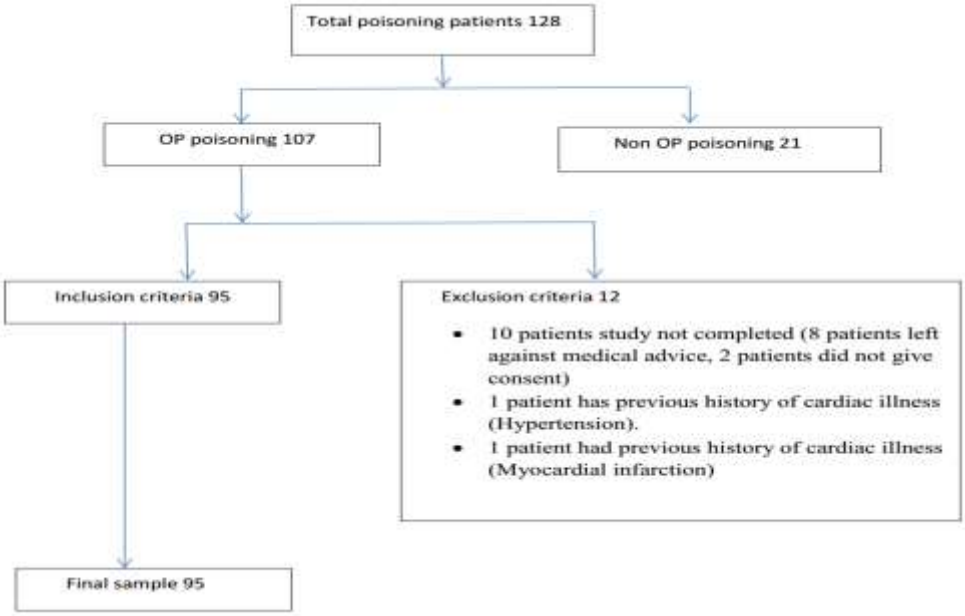

Table-1: Sex Distribution

SEX	N	%
Male	46	48.4
Female	49	51.6

Table-2: Age Distribution

Age (Yrs)	N	Percentage
15-20	18	18.9
20-25	21	22.1
25-30	19	20.0
30-35	10	10.5
35-40	12	12.6
>40	15	15.8
Total	95	100

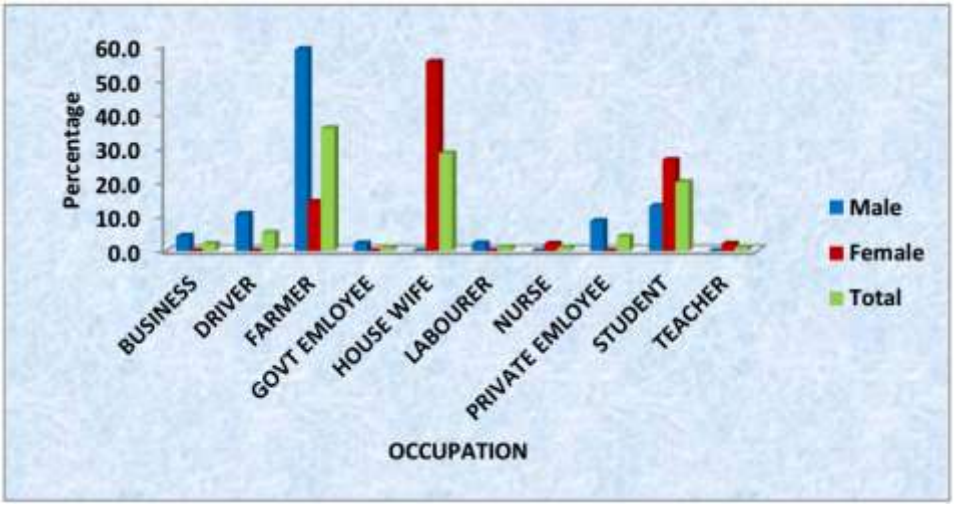

Fig-1: distribution of cases according to sex and occupation

Table-3: Nature of Poisoning

Nature of Poisoning	N	Percentage
Suicidal	95	100
Total	**95**	**100**

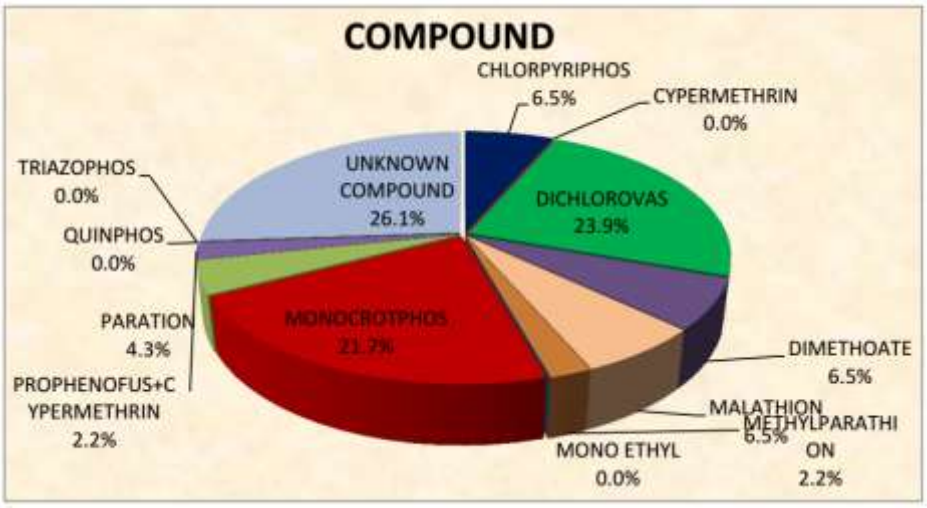

Fig-2: Distribution of compound in males

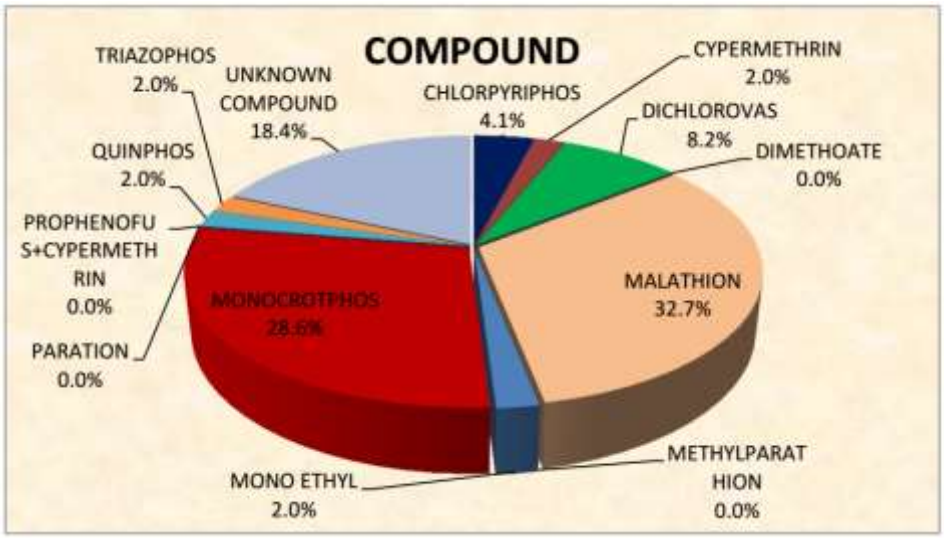

Fig-3: Distribution of compound in females

Table-4: Distribution of ECG changes

Parameters	ECG 1 DAY 1		ECG 2 DAY 3		ECG 3 DAY 7	
	No. of cases	%	No. of cases	%	No. of cases	%
SINUS TACHYCARDIA HR >100b/MIN	41	43.2	25	26.3	17	17.9
SINUS BRADYCARDIA HR<60b/MIN	7	7.4	9	9.5	10	10.5
ST-T CHANGES	34	35.8	34	35.8	29	30.5
PROLONGED QT >0.43 SECS	18	18.9	9	9.5	10	10.5
OTHER CHANGES	0	0.0	0	0.0	0	0.0

In our study out of 95 patients, 46 (48.4%) were male, 49(51.6%) were females. Male: female ratio is 1:1.47 (Table 1). In our study, the incidence of organophosphorus poisoning was more among the age group 21-30 years (42 %), (Table 2). In our study study the nature of poisoning was suicidal in 95 patients (100 %), (Table 3). In our study most. Of the patients were Farmers (35.8%), Housewives (28.4%) and Students (20%) (Figure1). Among the symptoms vomiting was most common symptom. Out of 95 cases, 95 (100%) cases had vomiting.

In our study most commonly used op compounds were Monocrotophos (25.3%), Malathion (20%), (Figure 2&3). Total 5 patients required ventilator support, out of them 3 patients had Monocrotophos poisoning, 1 patient had chlorpyrifos poisoning and 1 patient had unknown compound popisoning.

ECG changes seen in our study on 3 consecutive days (day 1, day 3 and day 7) are shown in Table 4).In our study of 95 patients the most common ECG finding was sinus tachycardia which is seen in 41(43%) patients. Similarly P Karki et al studied cardiac manifestations of OP manifestations and stated that cardiac complications usually occur during the first few hours after exposure and Sinus tachycardia was seen in 15 patients (40%) out of 37 patients. They explained that sinus tachycardia is due to nicotinic receptor stimulation on the heart and increased sympathetic tone [13, 14]

Sinus bradycardia is also seen. In our study of 95 patients with OP poisoning 10 (10.5%) patients developed sinus bradycardia. In a similar study done by Agarwal S *et al*. Out of 121 patients of Organophosphorus poisoning Sinus Bradycardia is seen in 8 patients (6.6%). The mechanism being behind sinus bradycardia explained by Agarwal S *et al*. is that it's caused due to muscarinic receptor stimulation on the heart and also due to parasympathetic over activity [15].

The second most common ecg changes seen our study are ST-T changes ST-T changes seen in 34 (35.8 %), 34 (35.8 %) and 29 (30.5%) patients on three consecutive days 1, 3 and 7 simultaneously. Out of 95 patients among both males and females of OP poisoning the common ST-T changes noticed were T inversions (leads II, III,aVF) (III,V1) (V1-V3) , Tall T (V2-V3), ST elevation (I,aVL), ST depression. In a similar study done by Agarwal. S *et al.* out of 121 patients 9 (7.4 %) patients developed ST depression, 9 (7.4 %) patients developed T inversions in leads II, III, and aVF. In a study done by Morteza Rahbar Taromsari et al studied 100 patients in which 24 patients showed ST elevation [15, 17].

In another study done by Gouda HS *et al.* showed out of 50 patients ,elevated ST segment is seen in 2 (4%),inverted T waves in 13 (26%), and conduction defects in 1 (2%) [12]

The mechanisms behind ST-T changes explained by both Agarwal .S et.al and Gouda HS et al are T inversions may be due to ischemia in sub endocardial myocardial tissue and Tall T may be due to prolonged period of parasympathetic activity, electrolyte abnormalities such as hyperkalaemia, hypokalaemia, hypomagnesemia and hypocalcaemia. ST elevation may be due to multiple mechanisms such as transient or transmural myocardial infarction, coronary vasospasm, hypoxemia, due to raised cardiac enzymes due to injury to myocardial tissue by organophosphorus poisoning [12,15].

Other ecg changes observed in our study is QT prolongation. Out of 95 patients 18 (18.9%) patients developed QT prolongation on day 1 followed by 9 (9.5%) patients on day 3 and 10 (10.5%) patients on 7th day. In a similar study done by P Karki *et al.* out of 37 patients with OP poisoning described prolonged QTc interval in 14 (37.8%) patients. QTc interval value range in between (>0.43-0.46 seconds) using Bazett's formula [13]. In a similar study done by P. Ravikumar *et al*. out of 100 patients QT prolongation was observed among 28 patients [17]. QT prolongation occurs a few hours after the intoxication and sometimes it may occur 1-15 days after Organophosphorous compound exposure. It's possible that with nerve agents this phase even prolongs [11].

According to P Karki *et al.* P. Ravikumar *et al.* H.S Gouda *et al.*, Lausdari .S *et al.* multiple mechanisms play role in QT prolongation in op poisoning.Malfunction of ion channels which leads to an intracellular excess of positively charged ions extends ventricular repolarisation and results QT interval prolongation.

Large QT dispersion (longest-shortest QT interval on any of the 12 lead ecg) is due to ischemic changes which may conceal the QT prolongation in affecting vascular area.Predisposing factors for QT prolongation and development of Torsade de pointes requires special care even in mild op poisoned patients of older age group, females, and patients with low LVEF, LVH, ischemia and dyselectrolytemia (hypomagnesemia and hypokalemia). QT prolongation also shows correlation with serum cholinesterase levels.

QTc prolongation depends on severity of op poisoning and the nature of the toxic agent. This complication usually starts during second to third day and may last up to 2 weeks post intoxication. QTc prolongation may also be due to unequal sympathetic stimulation of myocardial cells, interaction with potassium channels and sodium and calcium exchanger in myocardial cell membrane are probably responsible for occasional prolonged QTc interval [12, 17, 18, 19].

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