



PROFILE OF ORGANOPHOSPHORUS INSECTICIDES POISONING IN KASTURBA HOSPITAL, MANIPAL, SOUTH INDIA

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ABSTRACT

A prospective study was conducted from November 2007 to November 2009 in a tertiary care hospital at Manipal in Southern India, and a profile of the victims of pesticide poisoning was prepared. The objective of the study was to recognise the magnitude and pattern of poisoning in relation to the manner of consumption in this region of Southern India. In the present study, the young productive age group between 20-40 years were mostly affected. Males were predominantly affected. The most common compound was chlorpyrifos. Vomiting was the most common symptom. The most common sign was dilated pupils as against the usually observed sign of miosis which was attributed to atropine received from referral hospital. Among the complications, respiratory failure followed by pneumonia was commonly observed with severe pneumonia being the most common cause of mortality.

Keywords: pesticide poisoning; chlorpyrifos; mortality

INTRODUCTION

Organophosphorus pesticides are used widely for agriculture, vector control, and domestic purposes.¹ Pesticide self-poisoning is a major clinical problem in many parts of the world, probably killing about 300,000 people every year. Although most deaths occur in rural areas of the developing world, pesticide poisoning is also a problem in industrialized countries, where it may account for a significant proportion of the deaths from self-poisoning that do occur.² Ingestion of an organophosphorus pesticide results in inhibition of acetylcholinesterase. The resulting build-up of acetylcholine causes overstimulation of cholinergic synapses in the autonomic nervous system, central nervous system and neuromuscular junction, producing the acute cholinergic crisis. Patients die from respiratory failure during this crisis, or from a delayed respiratory failure called the intermediate syndrome.³ A history of acute exposure to an organophosphorus pesticide and development of characteristic clinical effects is diagnostic of organophosphorus poisoning and depression in plasma pseudocholinesterase value or erythrocyte cholinesterase value. This study looks into profile of OP poisoning in patients admitted to Kasturba Hospital, Manipal, South India.

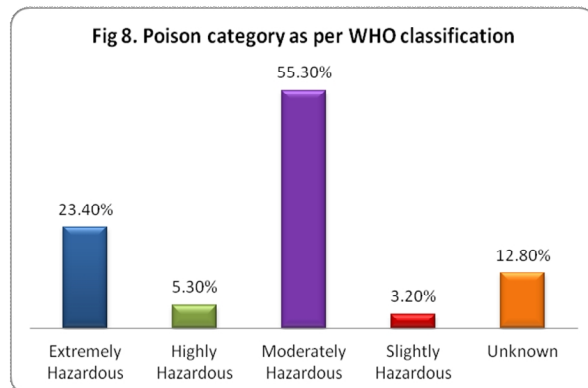
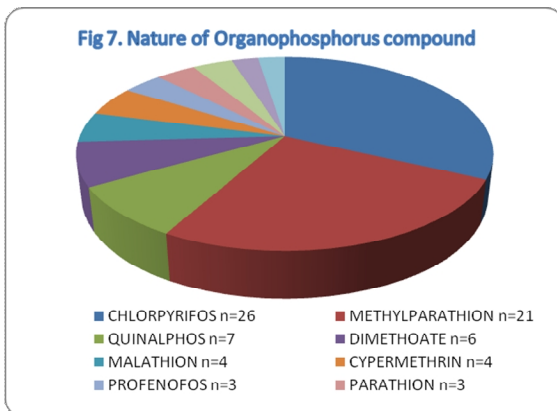
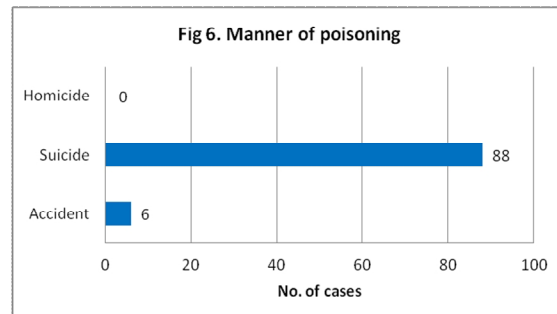
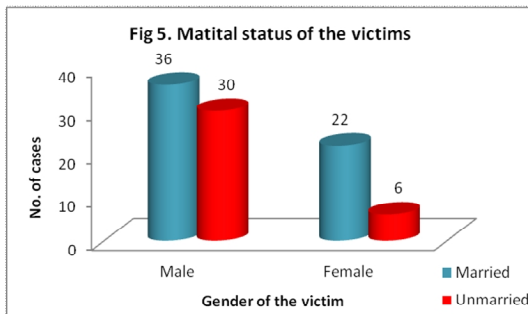
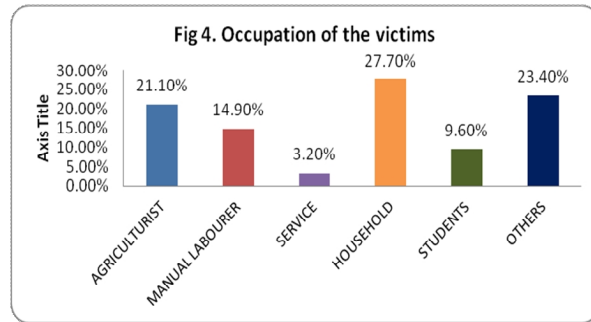
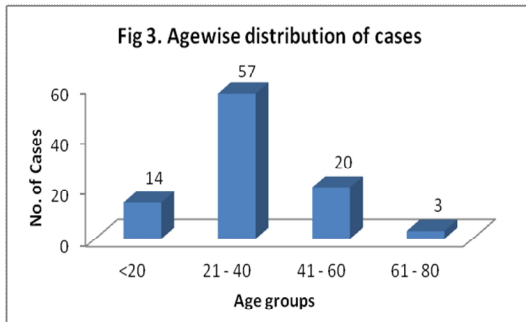
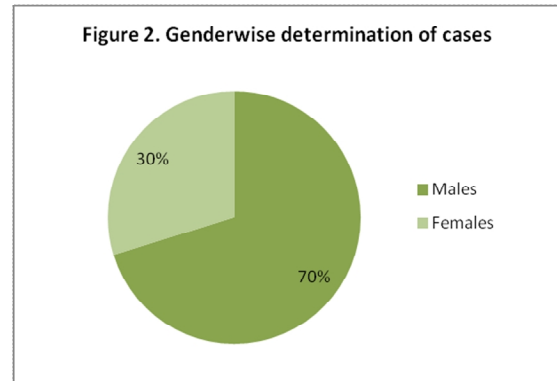
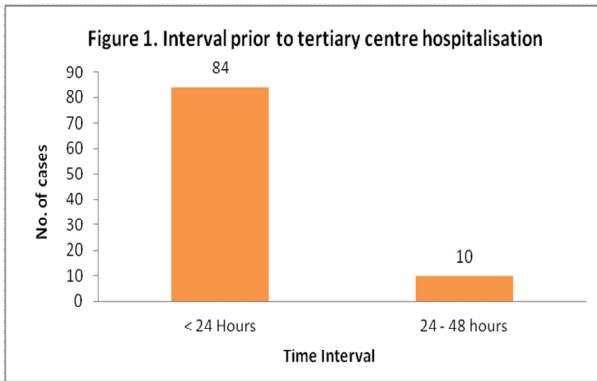
MATERIALS AND METHODS

This prospective cross-sectional open-labelled observational study was conducted in the emergency wards, Intensive care units and wards of Medicine department of Kasturba Hospital, Manipal, South India, for the period of two years from November 2007-November 2009. IEC clearance no: UEC/35/2009 dated 20.08.2009

The study included 94 cases of organophosphorus compound poisoned patients admitted in emergency wards of Kasturba Hospital, Manipal, India. The patients with following criteria were included in the study.

- All patients with history, symptoms and clinical signs of OP poisoning who present within 48 hours of consumption or
- Decrease in serum pseudocholinesterase level whereas patients with following criteria were excluded from the study.
- Presentation after 48 hours of consumption of organophosphorus poison
- Carbamate poisoning
- Double poisoning with opioids, barbiturates
- Known cases of Systemic diseases like malignancies, COPD, cardiac disease, neuromuscular diseases, renal failure, Diabetes mellitus and hypertension.

Patients who present with OP poisoning were selected based on the history of consumption of OP compound and clinical symptoms of OP poisoning. All ages other than the paediatric group (i.e. age 17 and above) both males and females who satisfy the above mentioned inclusion and exclusion criteria was included in the study. A detailed victimologic profile was prepared based on hospital records and information furnished by the relatives. The type of OP compound, quantity, time interval between consumption and arrival at hospital, treatment received elsewhere were assessed. Different variables like diurnal and seasonal variations along with the manner of death were studied. The outcome measures of this observational study were assessed in terms of the percentage of recovery, related morbidity and mortality. The data were registered in a database and analysed using Statistical Package for Social Sciences (SPSS) version 10.0. Chi square (χ^2) test was performed to see the significance of each group. p-value < 0.05 was considered significant.



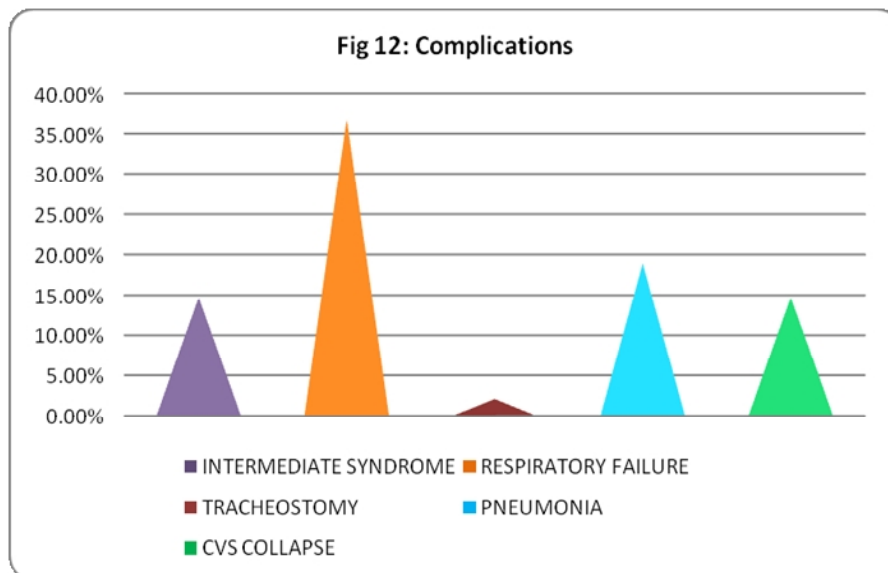
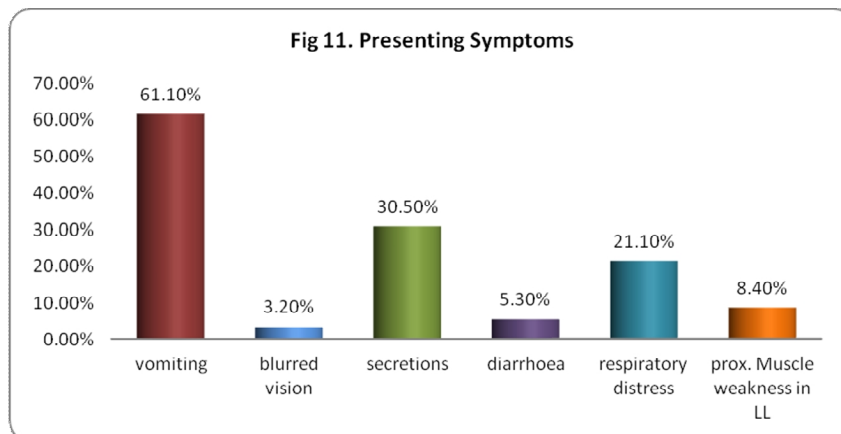
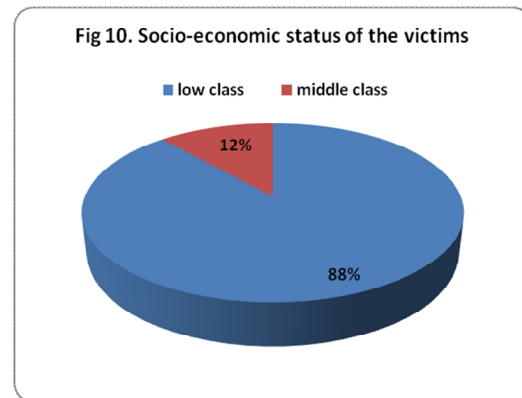
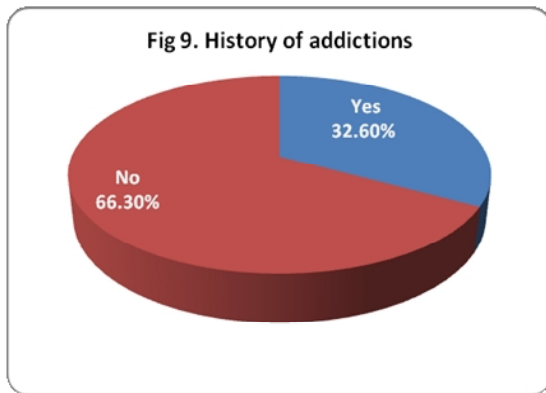


Table 1: Severity of poisoning as per Glasgow Coma Scale

Glasgow Coma Scale	Frequency	Valid Percent
Minor (≥ 13)	52	55.4
Moderate (9-12)	32	34.0
Severe (≤ 8)	10	10.6
Total	94	100.0

RESULTS

Out of total of 94 patients of organo-phosphorus (OP) poisoning included in the study, 89.4 % (n = 84) of cases reached hospital in 24 hours and the rest 10.6 % reached within 24-48 hours (Figure 1). Majority of patients (n = 73, 77.7 %) received treatment from local hospital before reaching Tertiary care centre in the form of Gastric lavage, Atropine, Pralidoxime (PAM) and other conservative management. Hence presentation of symptoms were modified by the treatment given from outside.

It was observed that 66 patients were males (70.2 %) and 28 were females (29.8 %), (Figure 2) with age ranging between 17-70 years. The majority are between the age groups 21-40 years (60.6 %) (Figure 3)

It was seen that majority of patients were doing household jobs (n = 26, 27.7 %), this signifies the ease with which the compounds are available to the common man. This was followed by agriculturists (n = 20, 21.3 %), manual labourers (14.9 %), students (9.6 %) (Figure 4)

58 patients were married (61.7 %) and remaining 36 patients (38.3 %) were unmarried (Figure 5). Among the 28 females, 22 were married and 6 were unmarried. Among the 66 males, 36 were married and 30 were unmarried.

Intent

There were 6 accidental cases (6.4 %), rest of the 88 cases were intentional (suicide) consumption cases (93.6 %) (Figure 6)

Route of Consumption

All patients except one, consumed poison orally. There was 1 patient who used the poison intravenously.

Nature of Compound

The compound consumed were sorted as per WHO classification and 52 patients (55.3 %) consumed compounds belonging to Category II (moderately hazardous) group. 22 patients (23.4 %) consumed poison belonging to Category Ia (extremely hazardous) group. There were 12 patients in whom the compounds were not known. The most common compound consumed among OP was Chlorpyrifos (category II) followed by Methylparathion (category Ia- extremely hazardous) followed by Quinalphos (category II) (Figure 7, Figure 8).

Socioeconomic Status and Addictions

87.4 % of people who consumed poison belonged to low socioeconomic class with 11.6 % belonging to middle class. 32.6 % of people had addictions in the form of chronic alcohol abuse and cigarette smoking (Figure 9, Figure 10).

Psychiatric Evaluation

Most of the patients have taken the poison as result of Impulsive act due to several familial/socioeconomic concerns. Three patients had past h/o Intentional Self-harm. Two others had chronic depressive disorders and were on medications for the same.

Severity Scale

As per the Glasgow Coma Scale (GCS) there were 52 patients in the minor category (GCS \geq 13), 32 patients in moderate category (GCS 9-12) and 10 patients in severe group (GCS \leq 8) (Table 1)

Symptoms

Fifty eight patients (61.1 %) had vomiting as the predominant complaint, 29 patients (30.5 %) had secretions as the next predominant complaint. 20 of them had breathlessness (21.1 %), 5 of them had diarrhoea (5.1 %) and 3 complained of blurred vision (Figure 11).

Outcome

Seventy four patients (78.7 %) recovered completely from the poison effect. Nine of them (9.6 %) had sequelae in the form of non-resolving pneumonia with respiratory failure at time of discharge, there was 1 patient who developed blindness probably due to prolonged hypoxic ischemic encephalopathy, 3 others had features of hypoxic encephalopathy, 1 patient who had decubitus ulcer at time of discharge due to prolonged recumbency. Eleven of them (11.7 %) expired. The most common cause of death was severe pneumonia and sepsis. Three of them had sepsis and other 3 had severe pneumonia. One patient had ARDS. Refractory shock, renal failure and rhabdomyolysis were seen each in one person.

Complication

Most common complication noted was respiratory failure in 35 patients (36.8%), followed by Intermediate syndrome in 14 patients (14.7%) and CVS collapse like cardiac arrest, hypotension in 14 patients (14.7%) as shown in Figure 12. Pneumonia was seen in 18 patients (18.9 %) and 2 patients (2.1 %) required tracheostomy due to prolonged ventilation.

Secondary Complications

In 14 patients other complications like sepsis (35 %), hypoxic ischemic encephalopathy (28.5 %), and seizure disorder (14.2 %) were seen. In 1 patient there was renal failure, 1 patient had rhabdomyolysis, 1 patient had left forearm abscess following I.V injection of poison, 1 had paralytic ileus and gastroenteritis, 1 had severe hyperpyrexia, 1 had decubitus ulcer, 1 had pneumothorax following central I.V line insertion.

DISCUSSION

Among 94 patients studied majority are between the age groups 21-40 years (60.6 %). This was in concordance with study done by Mutalik and Wadia⁴. The maximum number of cases fell in age group 20-30 years in many other studies⁵⁻⁷. Sixty-six were males and 28 were females, hence male predominated in this study which correlated well with the findings of previous studies^{4,7}. However in other studies like Vishwanathan M *et al* female showed a preponderance⁵. The most commonly consumed among OP was Chlorpyrifos (category II) followed by Methyl parathion. However in other studies methyl parathion was most common⁵. In some other studies monocrotophos, quinalphos and fenthion were commonly used^{8,9}. The intent of Poisoning was allegedly suicidal in most of the cases with very few accidental. Most of them were married. Most of them belonged to the low socioeconomic group. Majority of patients were doing household jobs. Most common symptom was vomiting followed by secretions which were muscarinic symptoms and of the nicotinic symptoms fasciculation was seen. These findings were similar to Vishwanathan *et al*⁵. Most common complication noted was respiratory failure. This was in concordance with 2 studies Namba *et al*¹⁰ and Philip *et al*¹¹. All 36.8 % patients who developed respiratory failure required mechanical ventilation¹²⁻¹⁴. The pathogenesis is

multifactorial and related to aspiration of gastric contents, excessive secretion in the airways, intermediate syndrome, pulmonary infection, septicaemia and the development of adult respiratory distress syndrome¹². It was observed that 22 patients (23.4 %) developed respiratory failure before 24 hours, 11 (11.7 %) of them developed between 24-96 hours and 2 (2.1 %) developed after 96 hours. Majority of patients developed respiratory failure during the acute cholinergic phase. Acute pneumonia¹²⁻¹⁴ and central depression of respiration which occurs following cholinergic overstimulation of synapses in the brain stem seems to be a major factor contributing for respiratory failure¹⁵. Late onset respiratory failure was because of the intermediate syndrome.

CONCLUSION


Pesticide self-poisoning is a major health problem in India. Due to its wide and easy availability it is one of the most common causes among poisoning. The study signifies the pattern of poisoning in Manipal region, India. Majority of the victims were males in the 3rd to 4th decades, which is the most active and productive phase of life. Intentional self-harm was the predominant manner and chlorpyrifos was responsible for majority of the cases. The route of administration was oral in most number of cases. Only one case was observed where the victim used intravenous route to administer the poison.

REFERENCES

1. Roberts MD, Aaron CK. Managing acute organophosphorus poisoning. *Br Med J* 2007; 334: 629-635. <http://dx.doi.org/10.1136/bmj.39134.566979.BE>
2. Eddleston M. Early management after self-poisoning with an organophosphorus or carbamate pesticide – a treatment protocol for

- junior doctors. *Crit Care* 2004; 8(6): 391-397. <http://dx.doi.org/10.1186/cc2953>
3. Eddleston M. Pathophysiology of organophosphorus pesticide self-poisoning is not so simple. *Netherlands J Med* 2008; 66(4): 146-148.
4. Mutalik GS, Wadia RS and Pai VR. Poisoning by diazinon, an organophosphorus insecticide. *J Indian Med Assoc* 1962; 38: 67-71.
5. Vishwanathan M, Srinivasan K. Poisoning by bug poisoning. *J Indian Med Assoc* 1962; 39: 345-349.
6. De SC, Chatterjee SC. Poisoning with organophosphorus insecticides. *J Assoc Phys India* 1967; 48: 153.
7. Singh S Sharma BK, Wahi PI, Anand BS, Chugh KS. Spectrum of acute poisoning in adults. *J Assoc Phys India* 1984; 32: 561-563.
8. Senanayake N and Karalliedde L. Neurotoxic effects of organophosphorus insecticides. An intermediate syndrome. *New Engl J Med* 1987; 316: 761-763. <http://dx.doi.org/10.1056/NEJM198703263161301>
9. Johnson S, Thomas K, Jeyaselan L, Peter JV, Cherian AM. Incidence of intermediate syndrome in organophosphorus poisoning. *J Assoc Phys India* 1995; 43: 321-323.
10. Namba T, Carl TN, Jerald J, David G. Poisoning due to organophosphorus insecticide, acute and chronic manifestations. *Am J Med* 1971; 50: 475-492. [http://dx.doi.org/10.1016/0002-9343\(71\)90337-8](http://dx.doi.org/10.1016/0002-9343(71)90337-8)
11. Philip G Bardin, Stephen F, Johan AM, Alwyan P, James RJ. Review article: Organophosphate and Carbamate poisoning. *Arch Intern Med* 1994; 154: 1433-1441. <http://dx.doi.org/10.1001/archinte.1994.00420130020005>
12. Tsao TC, Juang Y, Lan R, Shieh W, Lee C. Respiratory failure of acute organophosphate and carbamate poisoning. *Chest* 1990; 98: 631-636. <http://dx.doi.org/10.1378/chest.98.3.631>
13. Zwiene RJ, Ginsburg CM. Organophosphate and carbamate poisoning in Infants and children. *Pediatrics* 1988; 81: 121-120.
14. Bardin PG, Van Eden SF. Organophosphate poisoning grading the severity and comparing treatment between atropine and glycopyrrolate. *Crit Care Med* 1990; 18: 956-960. <http://dx.doi.org/10.1097/00003246-199009000-00010>
15. Steward WC, Anderson EA. Effect of a cholinesterase inhibitor when injected into the medulla of the rabbit. *J Pharmacol Exp Ther* 1968; 163: 309-317.

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