Original Article

Lethality of Suicidal Organophosphorous Poisoning in Karachi in 2010

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ABSTRACT

Objective: The study was aimed to evaluate the cases of suicide using organophosphorous compounds as intoxicant with the objective to bring up possible preventive measures based upon modifiable factors associated with lethality.
Design: Cross-sectional analytical Study.
Materials and Methods: This retrospective study was based upon 66 patients of poisoning treated at intensive care unit of Ward No.5, JPMC Karachi during a period of one year from January 2010 to December 2010.
Results: Out of total 66 cases of poisoning 38 were of organophosphorous compounds (OPC) poisoning, 20 males and 18 females. Most of the cases (63%) of OPC poisoning were in age range of 20-40 years, 33 (86.84 %) were of suicidal poisoning while 5 (13.16%) had accidental poisoning and 57 % reported to treatment facility within 6 hours.
Conclusions & recommendations: All the cases of OPC poisoning had severe symptoms with fatal out come. Suicidal ratio was quite high. The period between the ingestion of poison and initiation of treatment plays vital role. In order to reduce fatality rate urgent intervention is required by government by improving the treatment facilities at local level hospitals i.e. primary health care centres and banning of highly toxic organophosphorous compounds. Additional measures which can help include improving the public awareness regarding recognition of toxic symptoms & importance of prompt referral to an appropriate facility.
Key words: organophosphorous compounds, poisoning, suicide.

INTRODUCTION

The organophosphorous compounds (OPC) are widely used in agriculture as pesticides and as chemical warfare. They are easily accessible, thus they are commonly associated with deliberate self poisoning. OPC poisoning is a major clinical health problem across much of the rural Asia & account for an estimated 500,000 deaths from self harm each year. These are common suicidal agents in Pakistan, India, Turkey, Sri Lanka and other South Asian countries.[1] Accidental poisoning may also occur especially when they are kept within the reach of children & farmers could get exposed while spraying crops if they are not well protected with masks or gloves.

Poisoning is a common method of suicide especially in developing countries.[5] In many reports the rates of poisoning with OPC as suicidal method range from 20.6%-56.3%.[6,7,8] It has remained so for almost a century, 44.2% in 1872-76 and 49.2% in1972.[9] The reported poisoning rates in the suicide attempters who reached hospital varies from 40%- 80%.[10-12] OPC available as pesticides are amongst the most common poisons used.[13-14] In hospital based studies mortality rates associated with pesticides have been reported as high as 50%-70%.

In order to understand the mechanism of action of OPC recall that the nervous system is made up of a large number of nerves & which are meant for transmission of signals. When a signal reaches the end of a nerve, it releases a substance called neurotransmitter that carries the signal to adjacent nerves of an organ such as muscle or gland. Many nerves release acetylcholine as the neurotransmitter. Once the signal passes to the next nerve an enzyme called cholinesterase destroys the acetylcholine. OPC blocks this enzyme thus preventing the breakdown of acetylcholine & which acts for an excessively long time causing symptoms like increased secretions of glands, muscles twitches and the muscles after sometime get fatigued leading to paralysis.

The mode of exposure of OPC insecticides varies including dermal, GIT, inhalation and intravenous routes.[2, 3, 4] The mortality rate of OPC is very high, fatality is often related to a delay in diagnosis or an improper management.

MATERIALS AND METHODS

It was a retrospective study based upon 66 patients of poisoning treated at intensive care unit of Ward No. 5 of JPMC Karachi during a period of one year from January 2010 to December 2010. The initial diagnosis was made from the history taken from the patient's relatives regarding exposure to poisoning agent and was confirmed from serum and red blood cell cholinesterase levels. Blood gases, electrolytes, pH, LFT, LDH and PT were also assessed. The outcome of patients was assessed after applying a standard protocol of management. This includes use of intravenous atropine and pralidoxime as soon as possible, along with other measures like gastric lavage and administration of activated charcoal via nasogastric tube and cleansing of the patient's body with soap and water. Ventilator

support was provided if they had excessive salivation, respiratory failure or a depressed level of consciousness or unresponsive to oxygen treatment.

RESULTS

During the study period total 66 cases of poisoning were admitted in the unit. Out of these 38 were of organophosphorous compounds poisoning (used Typhon), 8 were of alcohol intoxication, 6 of heroin intoxication, 3 of corrosive acid ingestion and 11 of undetermined poisoning. (Table 1)

Table No.1: Distribution of cases of poisoning (n=66)

Sr. No.	Type of poisoning	No. of cases	Percentage
1	Organophosphorous poisoning	38	57.58
2	Alcohol poisoning	08	12.12
3	Heroin poisoning	06	9.09
4	Acid ingestion	03	4.54
5	Unknown poisoning	11	16.67
	Total	66	100.00

Table No.2: Gender distribution of cases of organophosphorous poisoning (n= 66)

Sr. No.	Sex	No. of cases	Percentage
1	Males	20	52.63
2	Females	18	47.37
	Total	38	100.00

Gender distribution revealed 52.63% males and 47.37% females (Table 2). Most of the cases were between the ages of 20-40 years. All cases proved fatal. Regarding manner 33 cases were of suicide and 5 were intoxicated accidentally. In suicidal cases the route of administration of poison was through GIT while in others it was inhaled accidentally.

All of the patients received atropine which was administered during 2-5 days as a continuous infusion or intermittent dosing. Mechanical ventilatory support was needed for 25 patients. The mean arterial blood gas values of these patients were, PCO_2 ranged between 17-48.3mmHg with mean 32.64+15.65, PO_2 ranged between 56 – 90 mm Hg with mean 73 $\pm$ 17mm Hg, HCO_3 ranged between11 – 25 mmol/l with mean 18 $\pm$ 7, SaO_2 ranged between 78 – 95% with mean 86.5 $\pm$ 8.5 & pH ranged between 78 – 95% with mean 7.23$\pm$0.29.

The duration of mechanical ventilation was 4-7 days. The mortality rate for the mechanically ventilated patients was not statistically different from patients not mechanically ventilated.

Liver function profile of these patients showed Total bilirubin 0.9-1.6mg/dl with mean 1.25$\pm$0.35 (Normal = 0.1-1), direct bilirubin 0.5-0.6mg/dl with mean 0.55 $\pm$ 0.05 (Normal = upto 0.3mg/dl), indirect bilirubin 0.8-0.9 with mean 0.85 $\pm$ 0.05mg/dl(Normal = upto0.7mg/dl), alkaline phophatase 102-107u/l with mean 104.5 $\pm$ 2.5 (Normal =50-135),Gamma GT 28-50u/l with mean 39$\pm$11 (Normal =10-50).

The other biochemical tests showed Lactic dehydrogenase (LDH) ranged between 321-325 u/l with mean 323$\pm$2 (Normal = 100-190 u/l), Prothrombin time ranged between 14-18 with mean 6$\pm$2 (Normal = 12-14), Serum Electrolytes, Na ranged between 146-149mEq/l with mean 147.5 $\pm$ 1.5 (Normal = 136-146), K ranged between 2.6-3.2 mEq/l with mean 2.9$\pm$0.3 , (Normal = 3.5-5.5), Cl ranged between 108-118mEq/l with mean 113 $\pm$ 5 (Normal = 98-106), blood urea nitrogen ranged between 7-9mg /dl with mean 8 $\pm$1 (Normal = 10-20), creatinine ranged between 0.6-1.3mg /dl with mean 0.95$\pm$0.35, serum cholinesterase level ranged between 0.2-6.5u/ml with mean 3.35 $\pm$ 3.15 (Normal = 7.0-190).

Intermediate syndrome was observed in most of the patients. The most frequent signs were meiosis, change in mental status, hyper-salivation, abdominal pain, diarrhea, respiratory distress, fasciculations & depressed level of consciousness.

Fatal outcome was significantly associated with higher mean age, lower mean pseudocholinesterase level, longer duration between organophosphorous compound ingestion and specific intervention.

Table No.3: Interval between OPC ingestion and specific intervention

Sr. No.	Interval	No. of cases
1	1 – 6 hours	22
2	7 – 12 hours	03
3	13 – 18 hours	03
4	18 – 24 hours	09

Table No.4: Blood chemistry in OPC poisoning cases.

Parameter	Range	Mean
PH	7.43 – 7.52	7.23$\pm$0.29
PCO_2	17 – 48.3 mm Hg	32.65$\pm$15.65
PO_2	55 – 72.2 mm Hg	73.0$\pm$17.0
SaO_2	89% - 97%	86.5$\pm$ 8.5
HCO_3	11.2 – 25 mmol / l	18.0$\pm$ 7.0
Sodium	146 – 149 mEq/L	147.5 $\pm$ 1.5
Potassium	2.6 – 3.2 mEq/L	2.9$\pm$ 0.3
Chloride	108 – 118 mEq/L	113$\pm$ 5
Blood urea nitrogen	7 – 9 mg/dl	8$\pm$1
Creatinine	0.6 mg/dl- 1.3 mp /04	0.95$\pm$0.35
Serum cholinesterase level	0.2 – 6.5 u/ML	3.35$\pm$3.15
Total bilirubin	0.9 – 1.6 mg/dl	1.25$\pm$0.35
Direct bilirubin	0.5 – 0.6 mg/dl	0.55$\pm$0.05
Indirect bilirubin	0.8 – 0.9 mg/dl	0.85$\pm$0.05
Alkaline phosphatase	102 – 107 u/l	104.5$\pm$2.5
Gamma GT	28 – 50 u/l	39.0$\pm$11.0
Lactate dehydrogenase	321 – 325 u/l	323$\pm$2.0
Prothrombin time	14 – 18	16$\pm$2.0

DISCUSSION

0rganophosphorous compounds are used worldwide in agriculture as well as in household gardens.[16] The easy availability of these compounds has resulted into a gradual increase in suicidal poisoning mainly in developing countries.[17]

In our study of 66 patients of poisoning, 38 patients were of OPC poisoning, among them 33 cases were suicidal and 5 cases were reported as accidental. Majority of cases in this study died due to delay in admission but some were misdiagnosed. They presented with history of headache for 3-4 days along with recent history of inhaling pesticides during spray over fields. Before ruling out the ruptured brain aneurysm by CT scan they were given atropine with fatal outcome.

In our study the rate of suicidal poisoning was 90%, probably because of uncontrolled sale and use of these agents all over the country. Most of the patients died due to intermediate syndrome, despite of endotracheal intubation deaths were due to respiratory failure. The intermediate syndrome is a state of muscle paralysis that occurs after recovery from cholinergic crisis but before the expected onset of the delayed polyneuropathy and it probably results from post-synaptic junction dysfunction.[18]

The increase in respiratory rate (24-46/min) is an obvious sign of respiratory failure which is the most troublesome complication which was observed in almost all patients. Respiratory failure may be due to many other reasons like aspiration of gastric contents, excessive secretions, pneumonia and septicemia complicating acute respiratory distress syndrome. Exposure to OPC will interfere with synaptic transmission peripherally at muscarinic and nicotinic receptors which cause decreased muscle power, skeletal muscle fasciculations and excessive salivary secretions, meiosis, diarrhea, abdominal pain, depressed level of consciousness and respiratory distress [17] which was observed in almost all patients in our study.

In the present study we observed that mortality rate was not significantly different whether or not the patients were treated with pralidoxime sulphate. This observation is in consensus with that of De Silva.[19]

Our mortality rate is higher compared with other reported series of OPC poisoning and intensive care management.[19,20] The reason for this is that our patients were admitted to the hospital quite late after exposure as they were referred from small rural hospitals where the facilities were not sufficient. The lethality can be reduced by improving treatment facilities at periphery i.e. primary health care centres. This will save the time which is presently being wasted in moving the patient to a distantly placed tertiary care hospital.

Lethality is an important clinical variable for both medical and psychiatric evaluation and management. In contrast to intent-to-die which is subjective measure lethality is objective, more descriptive of the behavior and often co-relate with the degree of intent. [21]

Banning of extremely toxic pesticides and the restriction of their use have been urged by WHO.[15]

CONCLUSION

All the cases of OPC poisoning had severe symptoms with fatal out come. Suicidal ratio was significant. The period between the ingestion of poison and initiation of treatment plays vital role in survival. In order to reduce fatality rate, urgent intervention is required from government by improving the treatment facilities at local level hospitals i.e. primary health care centres and banning of highly toxic compounds. Additional measures which can help include improving the public awareness regarding recognition of toxic symptoms & importance of prompt referral to an appropriate facility.

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