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Original Research Article

Intensive care management of organophosphorus poisoning patients: an experience from tertiary care centre

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ABSTRACT

Background: Organophosphate (OP) insectide poisoning results from occupational, accidental and intentional exposure. The mortality rate of OP poisoning is high. Early diagnosis and appropriate treatment is often lifesaving.
Methods: This study "Intensive care management of organophosphorous poisoning in Govt. medical college Srinagar (Sgr) hospital was a prospective one and was conducted over a period of two. All the patients with a provisional diagnosis of Organophosphorous poisoning who reported to the medical casualty and intensive care unit of SMHS hospital Sgr were included in this study.
Results: Out of a total of 1258 Organophosphorous poisoning cases, males were (34.5%) and females were (65.5%). Suicidal mode of poisoning was most common in our patients and constituted 63.20%. Out of 254 Organophosphorous poisoned patients admitted in ICU, 184 survived and 70 expired. Therefore, mortality rate for Organophosphorous poisoned patients who needed mechanical ventilation was 27.55.
Conclusions: OP poisoning is a serious problem in Kashmir Valley. Efforts should be directed towards rapid diagnosis and management of this condition. Additionally, close intensive monitoring of these patients for early recognition of respiratory failure which is one of the serious complication of OP poisoning with intensive care support will help in decreasing the mortality rate in these patients.

Keywords: Intensive care, Management, Organophosphorus

INTRODUCTION

Organophosphate (OP) insectide poisoning results from occupational, accidental and intentional exposure. According to the World Health Organization, about 1 million accidental and 2 million suicidal poisonings with organophosphorus insecticides are reported per year, with more than 300 000 fatalities.[1] In Asia 4.42 lac metric tonnes of pesticides are used per year, out of which 72000 metric tonnes are consumed in India.[2] In India OP compounds are among the most commonly used agents for suicidal poisoning.[3] Hundreds of organophosphorous compounds are currently available as Pesticides.[4,5] Organophosphorous compounds were first developed by 'Dr. Gerhard Schrader, a German scientist' shortly before the second world war.[6] They were first used as an agricultural insecticide and later as potential warfare agents.[7] These compounds are normally esters, thiol

esters or acid anhydride derivatives of phosphorous containing acids. Of the more than hundred organophosphorous pesticides used worldwide,the majority are either dimethyl phosphoryl or diethyl phosphoryl compounds as under.[8]

Acute cholinergic crisis is a medical emergency that often requires treatment in Intensive Care Unit. It consists of muscarinic, nicotinic and central nervous system effects. Muscarinic features include bronchorrhoea, bronchoconstriction, miotic pupils, abdominal cramps, involuntary defecation and urination, bradycardia, QT prolongation, hypotension. Nicotinic features include twiching of fine muscles, fasciculation and hyperreflexia which may progressively lead to flaccid paralysis. The most prominent CNS symptoms are: headache, dizziness, drowsiness, nausea, confusion, anxiety, slurred speech, ataxia, tremor, psychosis, convulsions, coma and respiratory depression.[9-11] Clinical diagnosis is relatively simple and is based on medical history, circumstances of exposure, clinical presentation, and laboratory tests. Confirmation of diagnosis can be made by measurement of erythrocyte AChE or serum ChE levels. Many OP insecticides (e.g. chlorpyrifos, demethon, diazinon, dichlorvos, malathion, trichlorphon) appear to be more potent inhibitors of ChE than AChE and, as the consequence, ChE inhibition might occur to a greater extent than AChE inhibition. Erythrocyte AChE is identical to the enzyme present in the target synapses, thus, it is regarded as biomarker of toxicity of these compounds.[12]

Management of organophosphorus poisoning include:

General measures

Decontamination

Clothes are removed off the patient and the patient is thoroughly cleaned gently with soap and water. Eyes are irrigated with normal saline.

Gastric decontamination is done by giving a gastric lavage. Healthcare workers protect themselves by using gloves, gowns and eye/foot wear.

Medication

The mainstay of treatment is atropine, pralidoxime (2-PAM), and benzodiazepines (for anxiety, restlessness and seizure control). Atropine:1–3 mg of atropine is given intravenously as a bolus and repeated after every 5 minutes till the end points of atropinization are achieved.

After achieving atropinization, the effect is maintained by atropine infusion (dose of 20% to 30% of the total amount initially required to atropinize), which is continued for the 2 to 3 days and then tapered off slowly.

Pralidoxime (2-PAM)

Oximes, also known as cholinesterase re-activators, are used as antidotes in the cases of OP poisoning. The recommended dose of PAM is 2 g (25-50 mg/kg in children) intravenously over 30 minutes, followed by infusion at 8 mg/kg/hour in adults (10 –20 mg/kg/hour in children) or 2g every 4-6 hours. PAM must not be given without concurrent atropine, as oximes can transiently induce cholinesterase inhibition and worsen the symptoms. Oximes are effective if administered early before the ageing (irreversible binding of OP with acetylcholinesterase) occurs (preferably given within 12 hours of intoxication, but can be given up to 48hrs after intoxication.

Supportive measures

These include oxygen support (ventilator support may be required in cases of severe intoxication causing bronchorrhea induced bronchospasm or respiratory muscle paralysis), intravenous fluids, and maintaining electrolyte balance. Psychiatry referral is required in cases of suicidal ingestion and also in cases involving neuropsychiatric side effects of the poisoning.

Aims and objectives of the study was to highlight the incidence and outcome of patients with organophosphorous poisoning with emphasis on role of intensive care management in these patients.

METHODS

This study i.e., “Intensive care management of organophosphorous poisoning in Govt. medical college Srinagar (Sgr) hospital was a prospective one and was conducted over a period of two years i.e., from August 2006 to August 2010.

All the patients with a provisional diagnosis of Organophosphorous poisoning who reported to the medical casualty and intensive care unit of SMHS hospital Sgr were included in this study.

Immediately after admission, patient’s socio-demographic profile was screened, and diagnosis was established by:

History and circumstantial evidence

The history of intake of organophosphorous compound was either communicated directly by the accompanying attendants of the patient (in many cases attendants would carry with them the bottles containing the residual poison), the nature of which was identified by the composition written on the bottles.

Signs and symptoms

Diagnosis was also made directly by the signs and symptoms of organophosphorous poisoning which included muscarinic, nicotinic and CNS effects.

Active resuscitation (including endotracheal intubation & mechanical ventilation) was instituted in all patients of grade 2 or 3 severity (Bardin PG et al (1987), and the overall management protocol of OP poisoned patients was as under.[13]

After thorough clinical examination, an intravenous line was established with isotonic saline. Patients were treated for parasympathetic over activity by i/v administration of Atropine sulphate in a dose of 2 mg i/v followed by 2 mg doses at 5-10 min interval until heart rate increased to >80bpm. After that atropine infusion in a dose of 0.02-0.08 mg/kg/hour was started till patient was fully atropinised.[14-18] Signs of atropinisation included clear lung fields, adequate heart rate (more than 80 bpm), blood pressure (more than 80 mmHg systolic with good urine output) dry skin and pupils no longer pinpoint. A uniform improvement in most of the five parameters was taken as the signs of atropinisation. However, the most important parameters were air entry on auscultation, heart rate, and blood pressure.

Dosage of atropine was titrated according to the improvement in signs and symptoms. When relapse occurred, the infusion rate was accelerated. In cases of atropine toxicity, the drug was withdrawn and the clinical response assessed. The infusion rate of atropine was decreased when clinical signs of organophosphorous poisoning had been absent for 24 hr, and the drug was gradually tapered off under constant observation until complete withdrawal.

Specific antidote pyridine-2-Aldoxime (P2AM) was given to all patients for 5 days in doses of: Adult=1 gm i/v 6 hrly, Children 25-50mg/kg.

Simultaneously patients were graded as per Bardin PG et al criteria (1987) as under Table 1.[13]

Table: 1: Bardin criteria for grading patients.

Grade 0	**Positive history and no signs of poisoning**
Grade I	Secretions and fasciculations present
Grade II	Marked secretions, gross fasciculations, ronchi and hypotension (systolic BP< 90mmHg) and altered sensorium
Grade III	Attempted suicide, PaO2 <10kPa (75.18mmHg) Abnormal chest radiograph

Two or more criteria were needed for specific grade. If there were less than 2 criteria, patient was placed in the preceding grade.

Care of airway and respiration

Proper and thorough suctioning of secretions was done. Care of adequate ventilation and high flow oxygen was immediately started.

Decontamination After securing airway all the soiled clothing was removed from the patient and a thorough body wash was given with soap and water.

Stomach wash with isotonic saline through a Ryle's tube was given intermittently till the gastric aspirate became clear. All the baseline investigations, ABG and chest radiography, ECG were performed in all the cases.

Subsequent management in the form of ventilatory support was instituted if there was deterioration of blood gases (PaO2 < 50mmHg, PCO2 > 50mmHg, pH < 7.30) at any given stage. During the course of therapy, patients were continuously monitored, treated symptomatically and their nutrition was well maintained. Supportive treatment was given to all the patients as per standard ICU protocol and antibiotics were started where ever necessary.

Complications which we encountered e.g. hyperthermia, acute renal failure, aspiration pneumonia, pulmonary edema, etc. were managed appropriately.

Following the recovery, counseling with the patient and attendants was done and the consultation of a psychiatrist for this purpose was also sought.

RESULTS

This study "Intensive care management of Organophosphorous poisoning in Government medical college Srinagar Hospital: A prospective study over a period of five years" was designed to evaluate the present incidence, age and sex distribution, socio-demographic profile, mode of poisoning, need for mechanical ventilation and the outcome of these patients in this part of the country. Following parameters were recorded and evaluated statistically.

Total number of patients

Total hospital admissions during the study period were 89,580. Out of these 2057 were the total poisoning admissions in our hospital, among them 1258 being Organophosphorous poisoning cases which constituted 61.16% of total poisoning admissions. Rodenticide poisoning cases were 382 which constituted 18.57% of total poisoning admissions. Other poisoning cases which included Benzodiazepine poisoning, poisoning with anti-depressants, analgesics, kerosene and alcohol were 417 in number constituting 20.27% of total poisoning admission (Table 2).

Table 2: Patient characteristics.

Patient Characteristics	Number
Total hospital admissions during study period	89,580
Total poisoning patients	2057 (2.30)
Organophosphorous (OP)	1258 (61.16)
Rodenticide	382 (18.57)
Others like benzodiazepines, anti-depressants, analgesics, kerosene, and alcohol.	417 (20.27)

Mode of poisoning

Mode of poisoning varied in different patients. Suicidal mode of poisoning was most common in our patients and constituted 63.20%.

Accidental mode of poisoning constituted 36.41%. In 5 patients (0.40%) mode of poisoning was homicidal (Table 3).

Table 3: Mode of OP poisoning subjects.

Mode of OP Poisoning Subjects		
Mode	**n**	**%**
Suicidal	795	63.20
Accidental	458	36.41
Homicidal	5	0.40

Topographical distribution

Majority (28.78%) of cases were from Pulwama district. Other districts in the order of descending frequency were Baramulla, Bandipora, Srinagar, Budgam, Kupwara, Shopian, Anantnag, Kulgam and Ganderbal (Table 4).

Table 4: District wise distribution of OP poisoning subjects.

District wise distribution of OP poisoning subjects		
District	**n**	**%**
Pulwama	362	28.78
Baramulla	274	21.78
Bandipora	224	17.81
Srinagar	187	14.86
Budgam	87	6.92
Kupwara	49	3.90
Shopian	29	2.31
Anantnag	24	1.91
Kulgam	14	1.11
Ganderbal	8	0.64

Poisoning admission in ICU

Out of a total of 2057 poisoning admission in the hospital, 281 (13.66%) patients were admitted I n the ICU. 254 (20.19%) were Organophosphorous poisoned patients who were admitted in the ICU for mechanical ventilation and 27 were Benzodiazepine poisoning, Rodenticide poisoning and Anti-depressant poisoning who were admitted in the ICU for mechanical ventilation. This is shown in Table 5.

Table 5: Poisoning admission in ICU during study period.

Admission	N	%
Total poisoning admission in ICU (of 2057)	281	13.66
Op poisoning patients who needed Mechanical ventilation (of 1258)	254	20.19
Benzodiazepine, anti-depressant, rodenticide poisoning who needed mechanical ventilation (of 2057)	27	1.31

Table 6: Characteristics of the poisoning patients admitted in ICU.

Characteristics of the poisoning patients admitted in ICU			
	n	**%**	
Residence	Rural	54	96.4
	Urban	2	3.6
Gender	Male	17	30.4
	Female	39	69.6
Socioeconomic status	Lower middle class	38	67.9
	Average middle class	17	30.4
	Upper class	1	1.8
Marital status	Married	20	35.7
	Unmarried	36	64.3
History of poisoning	Yes	53	94.6
	No	3	5.4
Type of poison	Organophosphorous	51	91.1
	Benzodiazipine	2	3.6
	Anti-depressants	2	3.6
	Rodenticides	1	1.8
Mode	Suicide	41	73.2
	Accidental	14	25.0
	Homicidal	1	1.8
Route of poisoning	Oral	56	100.0
Age (mean ± sd)	25.5 ± 12.4 (10, 69)		

GCS

Organophosphorous poisoned patients who were admitted in the ICU had a mean GCS of 5.8±1.7. This is shown in Table 7.

Table 7: Glassgow coma scale.

Glassgow coma scale	
(Mean ± SD)	**Median**
5.8 ± 1.7 (3,9)	6

Table 8: Clinical presentation.

Clinical Presentation			
		n	%
Bardin PG Grading	2	25	44.6
	3	31	55.4
Pupils	Miotic	42	75.0
	Normal Size	14	25.0
Cyanosis		56	100.0
Salivation		56	100.0
Sweating		46	82.1
Fasciculation		48	85.7
Disturbed level of conciousness		56	100.0
Pulse (mean ± SD)		54.8 ± 15.0 (24, 84)	
Systolic Blood Pressure (mean ± SD)		76.0 ± 8.8 (60, 90)	
Diastolic Blood Pressure (mean ± SD)		42.0 ± 8.3 (25, 62)	
Respiratory Rate (mean ± SD)		26.5 ± 10.3 (6, 40)	

Arterial blood gases (ABG)

The mean pH of OP patients on admission to ICU was 7.1±0.3. PaO2 was < 75mmHg in 140 (54%) patients, PaCO2 was > 56.2mmHg in 125 (49.21%) patients and pH was < 7.1 in 89 (35.05%) patients (Table 9).

Table 9: pH monitored in the studied subjects.

pH monitored in the Studied Subjects	
(Mean ± SD)	7.1 ± 0.3 (6.4, 7.9)

Table 10: ICU stay: The mean duration of stay in the ICU was 4.6±2.7 days.

ICU stay: The mean duration of stay in the ICU was 4.6±2.7 days	
Duration of Stay in ICU (mean ± SD)	4.6 ± 2.7 (1, 13)

Table 11: Complications of OP poisoned patients in ICU.

Complications of op poisoned patients in ICU			
		n	%
Complication	No complication	167	65.75
	Aspiration pneumonia	42	16.53
	Pulmonary edema	21	8.27
	Acute renal failure	12	4.72
	Hyperthermia	10	3.93
	Fetal demise	2	0.78
	Total	254	100.00

Complications in ICU

Out of 254 OP patients in ICU, complications were observed in 87(34.25%) patients whereas 167 (65.75%) patients had no complication. (Table 11).

Outcome

Out of 254 Organophosphorous poisoned patients admitted in ICU,184 survived and 70 expired. Therefore, mortality rate for Organophosphorous Poisoned patients who needed mechanical ventilation was 27.55 (Table 12).

Table 12: Final outcome in relation with type of poison consumed.

Type of Poison	Total no. of patients	Expired		Recovered		P value
		n	%	n	%	
Organophosphorous	254	70	27.5	184	72.5	0.735 (NS)
Benzodiazipine	12	2	16.6	10	83.4	
Anti-depressants	8	2	25.0	6	75.0	
Rodenticides	7	1	14.3	6	85.7	
Total	281	75	26.7	206	73.3	

DISCUSSION

Organophosphorous compounds are used worldwide in agriculture as well as household gardens.[18] Their easy availability has resulted in a gradual increase in accidental and suicidal poisoning mainly in developing countries.[18,19] Acute organophosphorous poisoning is a significant causeof morbidity and mortality in developing countries including India.[20] In India, OP poisoning has been steadily increasing since 1963, and has become the second commonest poisoning in northern India.[21] A high proportion of pesticide intoxications appear to be due tolack of knowledge, unsafe attitudes and dangerous practices. The technology available to small farmers for pesticide application is often inappropriate i.e., faulty sprayers, lack of protective equipment, non-existent first-aid provision.

The total number of hospital admission during the study period i.e., from 1st August 2007 to 31st August 2009 was

89,580. Out of this, 2057 were the total poisoning admission which constituted 2.30% of the total hospital admission. A total of 1258 cases of Organophosphorous poisoning were admitted, contributing to 61.16% of the total poisoning patients. 382 cases were of rodenticide poisoning, making 18.57% of the total poisoning patients. The rest of the poisoning cases which were 417 in number included benzodiazepines, alcohol, anti-depressants, analgesics etc. contributed 20.27% of the total poisoning patients. Singh and Sharma (2000), suggest that nearly half of the admission to the emergency with acute poisoning are due to organophosphates.[20]

Singh and Unnikrishnan (2003) observed that acute poisoning constituted 1% of all emergency admissions which is slightly lower than the results which we observed in our study.[22] The reason for Organophosphorus compounds being the most common agent used for poisoning is because organophosphorous pesticides are extensively used in agriculture and remain freely available to almost everybody. The widespread use in agriculture contributes to poisoning due to lack of control during spraying and especially storage of these pesticides. The practice of storing

these insecticides in empty cough syrup bottles, cold drink bottles lead to the accidental ingestion of these agrochemicals. Age groups involved were different. The majority of our cases, 487 (38.71%) were in the age range of 20-29 years i.e 3rd decade, 290 (23.05%) cases were in 2nd decade, 287 (22.41%) cases were in the 4th decade, 127 (10.10%) cases were in the fourth decade, 46 (3.66%) cases were in the 5th decade and 21 (1.67%) cases were in the 6th decade. The youngest patient in our study was 10 yr old and the eldest was a 69 yr old patient. The mode of poisoning in 10 yr old patient was accidental. He had consumed many unwashed apples which had been recently sprayed with pesticide. Dash et al (2005) also observed the highest incidence in the age group of 21-30 yrs (3rd decade) which is in conformity with our results.[23] Bardin PG et al (1987) also observed 75% of their patients under the age of 40 yrs.[13] The highest number of patients were recorded in the age range of 20-29 years. This age group constitutes the 'youth of the society'. Vulnerability to suicide in young is strongly associated with unemployment, poverty, interpersonal problems, inability to cope up with the hardships of life and lack of tolerance among young population which make them resort to suicides. Both sexes were involved. 824 (65.5%) cases were females and 434 (34.5%) cases were males with a male: female ratio of 1: 1.89. Our results are in conformity with the results of Sunder Ram et al (1991).[24] A review of world literature shows that attempted suicide rates vary from 100 and 300 per 100,000 with a preponderance of females.[25] This higher incidence of poisoning in females is attributed to different reasons like low literacy rates among women, social problems like unwanted pregnancies, maltreatment from their husbands and in-laws and different kinds of psychological trauma. Most common mode of poisoning in our study was suicidal 63.20% cases followed by accidental cases 36.41% and 0.4% of homicidal cases.

These results are in similarity with the results of Bardin PG et al (1987) who observed the suicidal mode as the commonest cause of organophosphorous poisoning.[13] Singh & Unnikrishnan observed in their study 72% cases were suicidal.[22] The precipitating factors for an increased rate of suicidal poisoning were poverty, unstable emotional relationships, psychiatric disorders, loss of property and lives, disagreement, quarrel among family members, marital discord. Accidental mode of poisoning was during spraying their fields with pesticides through inhalational route or consumed the poison accidently by mistaking it for some medication or applying it as a hair oil (anti-lice). OP poisoned patients reporting to our hospital

showed that the majority of patients i.e, 362 (28.78%) were from Pulwama district. Other districts in the descending order of frequency were Baramullah, Bandipora, Srinagar, Budgam, Kupwara, Shopian, Anantnag, Kulgam and Ganderbal.Pulwama district topped the list because apple orchards are commonly found in that area, followed by Baramullah (Sopore) where again apple orchards are the main source of income. Most of our patients took almost 3 hr in reaching the hospital. The reason for this was that most of our patients were from rural areas which took them a considerable time in reaching the hospital. We classified Organophosphorous poisoning patients into four grades as per Grading of Bardin PG et al (1987).[13] We observed 49.28%patients belonged to Grade 0, 30.52% patients belonged to Grade I, 8.27% patients belonged to Grade II and 11.93% patients belonged to Grade III. Out of 2057 poisoning patients admitted to our hospital during the study period, 281(13.66%) patients got admitted in the surgical ICU, while others were managed in casualty ward. Out of 1258 organophosphorous patients admitted to the hospital, 254 patients were admitted to the surgical ICU for mechanical ventilation which constituted 20.2% of the total OP poisoned patients. Our results coincide with the findings of Murat Sungur and Mohammed Guven (2001) who observed 21.2% of Organophosphorous patients (10 out of 47) required mechanical ventilation.[4] In contrast to our observations Bardin PG et al (1987) observed that 56% of their cases needed artificial ventilation.[13] The possible explanation for a lower percentage of patients in our study needing ventilatory support could be explained on the basis that majority of our patients belonged to Bardin PG Grade 0 and Grade I who did not demand intensive management and were therefore managed in the casualty ward of the hospital. Only 254 (20.2%) Organophosphorous patients belonged to Grade II or Grade III who needed intensive management in the form of ventilatory support.

The mean Glasgow coma score (GCS) of the organophosphorous poisoning patients admitted to the

surgical ICU was 5.8. This was because the patients who were admitted to the surgical ICU were severe poisoning cases (Grade II or Grade III). JOJ Davies et al (2008) found in their study that patients who present with a GCS of <13 need intensive monitoring and treatment.[26]

Dosage of atropine required in our patients was titrated according to the improvement in signs and symptoms. The minimum dose of atropine required in 24 hours in our study as 150 mg which is in similarity with the results of Du Dutoit et al (1981).[27] The maximum dose required in 24 hours was 1100 mg which is in similarity with the observations of Du Dutoit et el (1981) and Bardin PG (1987).[27,13] Atropine being a tertiary amine readily crosses the blood-brain barrier and is responsible for atropine toxicity with large doses of atropine causing agitation, confusion, urinary retention, hyperthermia, bowel ileus and tachycardia. We continued PAM (pyridine-2-aldoxime chloride) for a period of 3-5 days according to the severity of poisoning. PAM, is one of a class of nucleophilic oximes that regenerate AchE by removing the phosphate moiety from the acyl pocket. Complications were observed in 34.25% (87 out of 254 patients in ICU) patients. Aspiration pneumonia was observed in 42 (16.53%) patients, Pulmonary edema in 21 (8.27%), patients, Acute renal failure in 12 (4.72%) patients, hyperthermiain 10 (3.93%) patients and fetal demise in 2 (0.78%) patients who were four months pregnant at the time of poisoning.

The mortality rate for organophosphorous poisoned patients who required mechanical ventilation was 27.5% and the overall mortality was 6.8%.Our result coincide the results of Malik G M et al (1998) who observed the overall mortality rate for organophosphorous patients to be 5.49%.[28]

However, Murat Sungur and Mohammed Guven (2001) found mortality rate for patients who required mechanical ventilation as 50% which is much higher than our results.[4] Bardin PG et al (1987) observed a higher mortality rate of 16% in their study.[13]

CONCLUSION

OP poisoning is a serious problem in Kashmir Valley. Efforts should be directed towards rapid diagnosis and management of this condition. Additionally close intensive monitoring of these patients for early recognition of respiratory failure which is one of the serious complication of OP poisoning with intensive care support will help in decreasing the mortality rate in these patients.

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