Intermediate Syndrome in Organophosphorus Compound Poisoning - A Study in Rural Area

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ABSTRACT

BACKGROUND

Poisoning with organophosphorus compounds is one of the commonest forms of poisoning in our country. Patients with organophosphorus poisoning present with symptoms and signs of acetyl choline excess which is called as acute cholinergic crisis. Type II paralysis or intermediate syndrome (IS) is one of them. We wanted to study the clinical profile of patients of OPP, with a special reference to the neurological aspect of OPP, especially the intermediate syndrome.

METHODS

This study was conducted among 100 patients in the rural region, where the incidence of poisoning with OPCs is very high.

RESULTS

Most common poisoning was found to be organophosphorus compound (72%) 100 out of 140 patients, and higher incidence reported in age group (12 to 40 years). Maximum incidence of intermediate syndrome was seen in poisoning with Parathion (75%) followed by dimethoate poisoning with an incidence of 45.3%. The overall incidence of Intermediate syndrome in present study was 37%.

CONCLUSIONS

Insecticide poisoning was associated with wide clinical features ranging from vomiting, miosis to convulsions and intermediate syndrome. No correlation was found between dose ingested and clinical severity. But clinical severity did depend upon, to certain extent, the delay in admission.

KEYWORDS

Organophosphorus Compounds, Intermediate Syndrome, Serum Cholinesterase

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BACKGROUND

Poisoning with organophosphorus compounds (OPCs) is one of the commonest forms of poisoning in our country. It may occur due to suicidal ingestion, after an accidental exposure or a gradual cumulative poisoning as an occupational hazard, with repeated mild exposures in the working environment. In the rural Basin, sugar cane and grapes are commonly cultivated and organophosphorus compounds are used as pesticides. Because of easy availability, organophosphate pesticides are used commonly by the members of farming community with suicidal intent. Patients with OPP present with symptoms and signs of acetyl choline excess which is called as acute cholinergic crisis. There are well defined neurological abnormalities associated with OPP.[1,2] Type II paralysis or intermediate syndrome (IS) is one of them.[3,1] it is characterized by delayed onset of involvement of muscles in the territory of cranial nerves, flexors of neck, respiratory and proximal limb muscles. These neurotoxic effects appear after the acute cholinergic crisis and can persist for up to three weeks.[4,3]

This study has been undertaken in the rural region, to study the clinical profile of patients of OPP, with a special reference to the neurological aspect of OPP, especially the intermediate syndrome. This work is designed to try and correlate the presenting signs with subsequent development of IS. An attempt was also made to know the symptoms and signs associate with bad prognosis, in patients of acute OPP, in prospective manner.

METHODS

The present study was conducted at rural hospital in the Department of medicine, from January 1997 to July 1998. Hundred patients admitted and recovered with the history of organophosphorus poisoning were proven both by clinical symptoms, signs and laboratory findings. Information about the age, sex, type and amount of poison, time of ingestion, time interval between ingestion and arrival at the hospital, mode and route of poisoning, intent of poisoning was obtained from the patient or accompanying close relative of the patient. During their stay in intensive care unit, Particular attention was paid to cholinergic, nicotinic and intermediate syndrome-related symptoms and signs, with detailed assessment of motor function and fatigability of muscle groups innervated by cranial nerves and that neck muscles, limb muscles and respiratory muscles. When available, internationally accepted grading was used; the Glasgow coma scale for consciousness level; the Medical Research Council scale for muscle strength and deep tendon reflexes. All other symptoms were semi-quantitatively scored from 0 to 4+. For immediate management, patients were given first aid measures, clear airway if necessary and supported ventilation if necessary. After clinical assessment, blood samples were analysed for Haemoglobin level, Total Lymphocyte Count (TLC) and Differential Lymphocyte Count (DLC), ESR, Blood sugar, blood urea, serum creatinine, serum electrolytes and Serum Cholinesterase level. The laboratory reference range used in the present study for serum cholinesterase: 5100 to 11700 IU / Ltr. The serum cholinesterase activity was measured by kinetic/ DGKC calorimetric method, of Zydus Pathline Limited.

RESULTS

Compound	No. of Patients	Incidence
1. Organo Phosphor Compounds	100	72%
2. Deltamethrin (Butox)	16	11%
3. Propoxur (Baygon)	7	5%
4. Organochlorus Compounds	4	3%
5. Miscellaneous	13	9%
Total No. of Patients	140	100%

Table 1. Incidence of Poisoning with Different Groups of Poisons

Sr. No.	Name of Compounds	No. of Cases
1.	Dimethoate	64
2.	Dichlorvos	8
3.	Parathion	7
4.	Monocrotophos	6
5.	Chlorpyriphos	5
6.	Malathion	4
7.	Methyl parathion	4
8.	Diazinon	1
9.	Phorate	1
	Total	100

Table 2. Organophosphorus Compounds Involved in Poisoning

Age Group (Yr.)	Male	Female	Total
12-21	21	15	36
22-31	24	14	38
32-41	13	2	15
42-51	4	2	6
52-61	2	0	2
62-71	2	1	3
Total	66	34	100

Table 3. Age and Sex Distribution

Time Interval	Clinical Severity		
	Mild	Mod	Severe
0-30 min	3	6	-
30-60 min	4	4	1
1 Hr. - 2 hrs	1	13	10
2 his - 4 hrs	4	7	11
4 hrs - 6 hi s	2	11	10
6 hrs - 8 hrs	-	3	2
8 hrs - 12 hrs	-	2	3
>12 hours	-	-	3
Total no. of Patients	14	46	40
Range	0 min – 6 hrs	30 min – 12 hr	1 hr > 12 hr.
Mean	2 hrs	3.423 hrs	4.99 hrs.

Table 4. Correlation of Delay in Hospitalization After Poisoning with Clinical Severity

Sr. No.	Symptom	No. of Patients
1.	Nausea	94
2.	Vomiting	86
3.	Diarrhoea	26
4.	Sweating	40
5.	ABD. Pain	28
6.	Headache	16
7.	Muscle Weak	16
8.	Dyspnoea	10

Table 5. Frequencies and Incidence of Presenting Symptoms

Presenting Sings	Clinical Severity			Total No. of Cases
	Mild	Mod	Severe	
Perspiration	5(7.5%)	17(42.5%)	: 20(50%)	40
Lacrimation Salivation	1(2.3%)	15(33.3%)	29(64.4%)	45
Froth at Mouth	-	-	22(100%)	22
Cyanosis	-	2(8.7%)	21(91.3%)	23
Bradycardia	4(28.6%)	3(21.4%)	7(50%)	14
Hypotension	-	-	-	-
Bronchospasm	2(9.1%)	12(54.5%)	8(36.4%)	22
Pulmonary Oedema	-	3(9.4%)	29(90.6%)	32
Apnoeic Spells	-	-	25(100%)	25
Hyperperistalsis	2(7.1%)	14(50%)	12(42.9%)	28
Incontinence of Urine & Stool	-	-	16(100%)	16

Table 6. Presenting Signs (Non-Neurological)

Presenting Sings	Clinical Severity			Total No. of Cases
	Mild	Mod	Severe	
Alert	13(27.7%)	32(68%)	2(4.3%)	47
Delirious	1(5.6%)	13(72.2%)	4(22.2%)	18
Stuporous	-	1(11.1%)	8(88.9%)	9
Coma	-	-	26(100%)	26

Table 7. Presenting Signs (Neurological) Level of Consciousness

Sings	Clinical Severity			Total No. of Cases
	Mild	Mod	Severe	
Fasciculations	-	39 (60%)	26 (40%)	65
Meiosis	3(5%)	33 (53%)	26 (42%)	62
Seizures	-	-	5 (100%)	5

Table 8. Presenting Signs (Other Neurological Signs)

Sings	Clinical Severity			Total No. of Cases
	Mild	Mod	Severe	
I. Flaccid Paralytic Signs	-	3 (9.4%)	29 (90.6%)	32
1. Hypotonia	-	2	29	31 (96.9%)
2. Weakness	-	3	28	31 (96.9%)
3. Cr. Nerve Palsy	-	3	16	19(59.4%)
4. Areflexia	-	1	26	27 (84.4)
II. Pyramidal Tract Signs	1 (6.6%)	7 (46.7%)	7(46.7%)	15
1. Hypertonia	1	7	7	15 (100%)
2. Hyperreflexia	1	7	5	13 (86.7%)
3. Weakness		2	6	8 (53.3%)
4. Ext. Planters	1	6	4	11 (73.3%)

Table 9. Presenting Signs (Neuroparalytic Signs: Type I) and Their Distribution According to Clinical Severity

Sings	No. of Patients	Clinical Severity			Total No. of Patients with IS
		Mild	Mod	Severe	
Dimethoate	64	2	17	10	29 (78.3%)
Dichlorvos	8	-	-	-	-
Parathion	7	-	1	2	3(18%)
Monocrotophos	6	-	2	-	2 (5.4%)
Chlorpyriphos	5	-	-	-	-
Malathion	4	-	-	-	-
M, parathion	4	-	-	3	3 (8%)
Diazinon	1	-	-	-	-
Phorate	1	-	-	-	-
Total (Incidence)	100	2(14.3%)	20(43.5%)	15(37.5%)	37%

Table 10. Distribution of Patients of Intermediate Syndrome with Regard to Organophosphorus Compound and Severity of Poisoning

Neurological Sings	No. of Patients	Patients Without IS	Patients with IS
1. Sensorium			
A. Normal	65	40 (61.5%):	25 (38.5%)
B. Altered	35	23 (65.7%)	12(34.3%)
2. Paralytic Signs (Type I)			
Absent	53	33 (62.2%)	20 (37.8%)
Present	47	30 (63.8%)	17 (36.2%)
A. Flaccid Paralysis	32	22 (68.8%)	10 (31.2%)
B. Pyramidal Tract Signs	15	8 (53.3%)	7 (46.7%)
3. Fasciculations			
Absent	35	29 (82.9%)	1 6(17.1%)
Present	65	34 (52%)	30 (48%)
4. Meiosis			1
Absent	38	31 (81.6%)	7 (18.4%)
Present	62	32(51.6%)	30 (48.4%)

Table 11. Comparison of Clinical Parameters at the Time of Admission (Neurological) in Patients with or without IS

Poisoning with organophosphates is most common form of poisoning encountered in this region. Poisoning with dimethoate was most commonly encountered poisoning in this study. It is clear from the table that the incidence of patients presenting with significant disturbance of consciousness was 35% (35 patients). 34 patients (97%) were from severe group. 85% of patients in severe group had significant disturbance of sensorium whereas only 2.1% from moderate grade and none from mild grade poisoning had significantly altered sensorium. Incidence of neuroparalytic signs (type I sings) in present study was 47%. Flaccid paralysis was seen in 32 patients and in these patients the most commonly observed signs were hypotonia and profound weakness. Pyramidal signs were seen in 15 patients, hypertonia was most commonly encountered signs, (seen in all patients), again the incidence of this type of sign was noted to be higher in severe grade of poisoning (17.5%) compared to moderate grade of poisoning (15%).

DISCUSSION

Incidence of Poisoning with OPCs and Different OPCs Involved

From table 1 it is clear that commonest mode of poisoning is ingestion of commercially available pesticides, because of the easy availability of these to the agricultural population. Poisoning with organophosphorus compounds formed 71.5% of this series of 140; cases of acute poisoning followed by Baygon (propoxur) which constituted 8.5% of total cases. Out of 100 patients of OPP, 64 had poisoning with dimethoate which is marketed in liquid form in a concentration of 30% to 40% (Table 2). In study by Wadia et al the different compounds reported were, diazinon, malathion, Sumithion, fenthion. Tick-20 (diazinon) a household insecticide was commonly used by low condition in urban areas (S.C. income, illiterate people living in bad sanitary et al). [1,2,5]

Age and Sex Distribution

The incidence of poisoning was higher in males as compared to females with M: F ratio of 1.9:1 (Table 3). The incidence of poisoning was maximum in the group 22-31 (38 cases); followed by 36 cases in the age group 12-21 i.e. 74 patients were below 31 years of age while only 11 patients were of 40 years or above.

Correlation between Delay in Admission and Clinical Severity

Patients with milder grades of OPP had presented earlier in our study with a range of interval being 30 min to 6 hrs (mean 2 hrs.) While the patients, who presented later, range 1 hr. to more than 12 hours (mean 4.99 hrs) had severe grades of poisoning. This observation suggests that clinical severity does depend upon to certain extent, on the interval between the time of ingestion and hospitalization (Table 4). Adlakha et al have shown that greater the delay in hospitalization, worse are the symptoms and while the patients who delayed admission had more severe symptoms, those who presented earlier with severe symptoms had a higher risk of mortality.[6] Similarly Wadia el al found no correlation between this time interval and clinical severity or mortality, they concluded that the different factors of absorption of different OPC being responsible for this clinical observation.[1]

Presenting Symptoms

Nausea and vomiting appealed to be universal symptoms with incidence of 95% and 85% respectively. Similar finding was reported by other workers.[4,7,8] Only 14 patients having dyspnoea, while on clinical examination 39 patients had respiratory distress, but the remaining patients could not

complain about difficulty in breathing due to their altered sensorium. Reported incidence of diarrhoea is 16%, 11% and 30% in studies by Adlakha et al, Sing S. et al and Balani et al respectively.[6,4,9] 16 patients complained of muscle weakness with related symptoms. Muscle weakness is due to nicotinic effect of OPC's (on N-M junction) and responds to PAM and not to atropine, as was seen during this study. This is a muscarinic effect of OPC's on sweat glands and was observed in 40% of patients. Higher incidence of sweating (95%) was observed by Singh S. et al who studied 20 cases of parathion and a lesser incidence (15%) of sweating was observed by Adlakha et al, in their study of 100 cases of OPP, mostly due to accidental exposure.[6,9]

Presenting Signs

Evidence of increased secretion by salivary and lacrimal glands was observed in 45 patients and is a sign of moderate to severe grade of poisoning, most commonly seen in severely poisoned patients (29%). Salivation was seen in 36% cases by Adlakha and in 80% cases by Singh S et al.[9] Bradycardia was seen in 14% of cases at its a muscarinic effect of OPC and considered to be a constant feature of OPP, irrespective of route of administration, abolished by atropine. Hypotension was observed in 11 patients at the time of admission, does suggest presence: of severe cholinergic crisis arid usually in most of cases it was associated with cardio-respiratory collapse, altered level of consciousness and cyanosis. K. Kanagastam et al had observed similar phenomena and has related it to severe cerebral depression (respiratory and vasomotor center) by OPCs. Bronchospasm was observed in 22 patients, detected clinically by the presence of rhonchi. Gupta et al and Balani et al have reported bronchospasm in 25% and 10% cases respectively.[4,7] Lesser degree of incidence has been noted by Adlakha et al (8%).[6] late onset of pulmonary oedema was observed by S.C. De and Chatterjee, after 12 hours, which was not observed in this study. However, we could not find statistically significant difference in mortality of patients with or without pulmonary oedema at the time of admission.

Respiratory Insufficiency

Respiratory failure was present in 39 patients at the time of admission. 33 patients (84.6%) were from severely poisoned group and 6 patients (13.4%) had poisoning of moderate severity none of the patients from mild grade j of poisoning had respiratory insufficiency. 11 of these 39 patients went on to develop intermediate syndrome. All of these patients showed improvement in their respiratory status after treatment, with reduction noted in their ventilatory requirement over a period of 24 to 48 hours, but in those patients who developed intermediate syndrome re-worsening of the respiratory status was noted, with subsequent prolongation of ventilatory support till their recovery from intermediate syndrome.

Neurotoxicity

Prior to 1974 unconsciousness and fasciculation's were described as a part of neurotoxicity of OPCs by various studies Mutalik et al (1962), Shankar (1967), Namba et al (1971).[10,11,12] Namba et al in 1971, also described weakness, difficulty in walking, speech disturbance in his patients. First delineation and classification of neurotoxicity j was done in 1974 by Wadia et al.[9] They divided these signs into type I and type II signs. The type II sings were latter rechristened by Senanaykae as "Intermediate Syndrome".[9]

Altered Consciousness

Impairment of consciousness was seen in 53 patients (53%) at the time of admission (Table 7). Higher incidence of coma (65%) and stupor (20%) was seen in most severe form of poisoning and only one patient was stupors in moderately server group. A lesser incidence of altered sensorium (coma) was noted by Wadia et al,[9] 20 of their 200 patients (10%) had coma at the time of admission. But they had also noted association of this with severe form of cholinergic crisis with cardio-respiratory collapse - 11/200 cases Paralytic signs - Flaccid 6%, Pyramidal 4%. The incidence of coma in other studies was 45% and 30% by Sing S et al and Balani et al respectively.[4, 9] Meiosis was observed in 62 cases; 33 cases were from moderate group, 26 were from severe group while only 3 from mild group of poisoning had meiosis. Adlakha et al and Balani et al had reported incidence of meiosis of 65% and Sing et al have reported meiosis in 20% of their cases.[6, 13,9] Maximum reported incidence of meiosis is of 95% in a study by S.C. De ET al.[13] Balani et al also observed that pupil dilatation was seen in 2% of cases with mild poisoning probably due to sympathetic stimulation from fear or other emotional stimuli.[4] 65 patients had fasciculations. Not all of these had fasciculation at the time of admission (25 patients). Commonest sites of occurrence, in descending order of frequency were, quadriceps, calf muscles, anterior chest wall, forearms, and deltoids. Much lesser incidence has been recorded in previous studies with incidence of 26%, 31%, and 10% by Wadia et al, Adlakha et al and Balani et al respectively.[6,4,9]

Neuroparalytic Signs

From table 9 it is clear that 32 patients had flaccid weakness with maximum incidence seen in most severe grade of poisoning where 29 patients (72.5%) had flaccid paralysis at the time of admission and remaining 3 patients were from moderate grade, Hypotonia was the most commonly encountered sign in all of these patients. Respiratory insufficiency due to severe respiratory muscle paralysis was seen in almost all of these cases and all of them required ventilatory support.

Pyramidal Tract Signs

These were observed in 15 patients with maximum incidence in severe group (17.5%) followed by incidence of 15% in moderate group (7 patients). Commonest sign observed was hypertonia which was more marked in lower limbs and was present in all patients. Mild weakness of limb muscle was observed in 8 patients (53.3%). Hyperreflexia was seen in 13 patients (86.6%). A much lesser incidence of type I paralytic signs (10.5%) was noted in the study by Wadia et

al (1974) which the OPC involved was diazinon but the same in group when studied poisoning due to three different OPCs, namely fenthion, malathion and Sumithion, they noted much higher incidence of paralytic signs (1977).[1,2] In other studies, Adlakha et al noted hypotonia in 15% of their patients where as it was 8% in a study by Mutalik ET al.[6,10] As seen in table 8, areflexia was seen in 27 patients; 26 patients were from severe grade of poisoning and only one from moderate group. Wadia et al have reported the incidence of areflexia to be 6% while Adlakha et al had observed areflexia in 15% of their patients. Similar observations have been made by Wadia et al with incidence of 1% in their first study (1974), of 3 .3% in their second study (1977) of poisoning with OPCs.[1,2]

Intermediate Syndrome (IS)

A. Incidence and Onset of IS

The neurotoxic effects characteristic of IS appeared after a relative symptom-free interval after reversal of initial cholinergic crisis with treatment. Total no. of 37 patients developed intermediate syndrome i.e. an incidence of IS in our study was 37%. The earliest development of IS was noted to be 16 hours after ingestion of poison, dimethoate presenting with poisoning of moderate severity.

B. Correlation with Clinical Severity and Different OPCs Involved

Maximum incidence of 43% was observed in patients from moderate group followed by an incidence of 38% in patients with poisoning of severe grade. Least incidence of IS observed in patient with mild grade of poisoning; only 2 patients from this group (14%) went on to develop IS. The Slightly higher incidence of IS in the moderate group as compared to served group was statistically not significant. But when mild group was compared to other two groups combined together, the difference almost reached statistical significance. (X^2 = 3.68, p<0.1). This may suggest that a correlation between clinical severity and development of IS with more server from of poisoning having higher incidence. Wadia et al in their study of 350 cases of poisoning with diazinon, Sumithion, fenthion, Malathion noted an incidence of 49%. They noted duration of onset of 9-85 horns.[1,2]

C. Total Duration of Intermediate Syndrome

The duration of time required for improvement of signs of IS varied from 2 days to 18 days with a mean duration of 9 days. It was observed that most of the patients (24/37 i.e. 64.9%) recovered within 7 days. The maximum duration of IS was of 18 days and seen in a patient of dimethoate poisoning with onset of IS, 36 hours after ingestion. The duration of IS described by Wadia et al was of 72 hours (or till death)[1] But observations similar to our study were made by Senanayake et al (Duration 4-18 days), Jan De Bleecker (5 - 33 days), K. K. Shailesh (2-18 days; mean 9 days).

D. Correlation between the Neurological Signs Present at Admission with Development of Intermediate Syndrome.

Altered Sensorium- The incidence of 38.46 for IS in patients with normal sensorium which was slightly higher than for the patients with altered sensorium (34.29%). This difference did not reach statistical significance (p>0.5)

Paralytic Signs- From the table 11 it is obvious that no difference in the incidence of IS was noted for patients who had type I paralysis on admission and for those who did not have paralytic signs. But here type I paralysis was studied without taking into account the two distinct types of sings viz. Flaccid paralytic signs and pyramidal tract signs. When these two types of neurotoxicity's were studied separately, it was noted that 7 out of 15 patients (46.7%) with predominant pyramidal tract signs developed IS as against 10 patients out of 32 with flaccid paralytic signs who developed IS (31.25%), But from statistical point of view this difference was not significant (p >0.5, NS). In the study by Wadia et al 5 out of 11 patients (45.45%) who had flaccid type of paralysis developed IS but none with pyramidal tract signs developed IS.[1]

E. Pattern of Muscle Weakness In IS

In the territory of motor cranial nerves: Most of the patients had weakness of muscles in the territory of cranial nerves including III, IV, VI, VII and lower cranial nerves (IX, X). Weakness was more marked proximally than in distal muscle groups. The severity of weakness varied from grade 0 to Gr II in different patients and when the power loss was more severe, involvement of both proximal and distal group of muscles was seen. Weakness was associated with depressed or absent DTRs, especially in upper limbs. Respiratory muscle weakness: Respiratory muscle weakness was seen in all patients with flaccid type of paralysis.

F. Course of Neuroparalytic Signs

The weakness was seen first in neck flexors and then the weakness of other groups of muscles was noted. At the time of recovery depression of tendon reflexes was first to recover; limb and respiratory weakness were the last to recover. Similar pattern of weakness was described by Wadia et al and also noted by Senanayake et al, K. K. Shailesh et al and Jan De. Bleecker ET [3,13,14]

G. Respiratory Distress in Intermediate Syndrome

Of the 37 patients who developed IS, 35 had respiratory muscle weakness severe enough to warrant artificial ventilator support; i.e. 94.6% of patients developing IS required ventilator support as against only 50.8% of patients who did not develop IS, required artificial ventilator support. 11 of these 35 patients! Had respiratory distress at the time of admission because of various factors like pulmonary oedema respiratory muscle weakness, prior to development of IS. Few of these patients showed improvement in their spontaneous respiratory status, but again deteriorated, as they developed IS subsequently. The duration of ventilation for these patients varied from 4 days to 20 days with a mean of 221.2 hours as Compared to patients who did not develop IS (Mean duration 102.5 hours). It shows that IS was the

cause of prolongation of ventilator support in cases of OPP. In previous studies by Senanayake et al, they noted respiratory distress in 7 out of 10 patients and 4 of them required ventilatory support (40%).[3] In the study by K. K. Shailesh et al, all of the patients developing IS had respiratory failure and all of them needed ventilatory support.[14]

CONCLUSIONS

Insecticide poisoning was associated with wide clinical features ranging from vomiting, miosis to convulsions and intermediate syndrome. No correlation between dose ingested and clinical severity was noted but clinical severity did depend upon, to certain extent, the delay in admission. Onset of IS was noted to be from 16 hours to 96 hours. Majority of patients improved within one week, while some patients had prolonged IS with duration of more than 2 weeks. The range of duration of IS as seen in this study was from 2 days to 18 days. Complications associated with artificial ventilation were commonly seen in patients who required respiratory support for prolonged period, especially in patients developing intermediate syndrome.

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