CLINICAL SPECTRUM OF ORGANOPHOSPHORUS POISONING IN ICU

Ilyas Ahmed1, Abdul Razaak2, Srinivas S3, Ramakrishna Reddy4, Anil Kumar R5 and Feroz Khan6

1,2,3,4 FIMS. Kadapa
5,6 SRMC, Nandyal

DOI: http://dx.doi.org/10.24327/ijrsr.2017.0805.0273

ABSTRACT

Organo -phosphorus compounds have been imported to India since 1952 but we got awareness of the nature of these compounds when the Kerala food poisoning tragedy occurred in 1958. Organophosphorus compounds with effective control and eradication of the pets and insects affecting the plants and crops became a boon to agriculturist and horticulturists and they revolutionized agriculture and horticulture. In our country where agriculture is the main occupation of majority of the people in rural area, the organo-phosphorous pesticides have contributed their own share in bringing the green revolution. The agriculturists who use these pesticides, commonly do not follow all the precautions during spraying and become victims of the accidental acute poisoning and sometimes of chronic poisoning. Population is also at risk of chronic poisoning from those agents by ingestion of vegetables etc when they are brought to the market. The number of people who consume these pesticides for suicidal purpose is in increasingly rapidly.

Aim and Objectives: To study the clinical features of the organo-phosphorus poisoning in patients coming to emergency ward.

INTRODUCTION

MATERIALS AND METHODS

30 cases of the acute poisoning due to organophosphorus Pesticides were studied in detail, once the diagnosis of organophosphorus poisoning was made. Blood sample were collected for estimation of blood sugar, urea, SGOT and electrolytes. Gastric lavage was done at the earliest and specific therapy was started. ECG was obtained both at the time of admission and at the time of discharge.

RESULTS

Analysis of studied cases- In this study incidence among the age group 21-30 yrs of age (53.3%).

Sex wise distribution: In this study incidence among the male sex (53.3%) to female ratio (46.6%).

Analysis of symptoms in studied cases: In the present study Nausea (93.3%); Vomitting (60%); Abdominal pain (46.6%); Salivation (33.3%); Sweating and Lacrimation (26.6%); Weakness (20%); Restlessness (13.3%); Headache, Blurred

*Corresponding author: Ilyas Ahmed
FIMS. Kadapa
vision (10%) and Diarrhoea (6.6%) were comparable to the pattern reported by APN Kumar et al and Goel et al.

**Observed signs in studied cases:** In this study the clinical signs observed were: Offensive odour (100%); Altered sensorium (72%); Pin point pupil (70%); Fasciculations (50%); Oronasal froth (33.3%); Bradycardia (21%); Tachypnoea (20%); Bronchospasm (20%); Miosis (16.7%) and Coma (6.6%). These observations were comparable to the studies of Goel et al.

OP poisoning was common in young adults of either sex belonging to low socio-economic group; Nausea with or without vomiting; miosis, abdominal pain, cardiovascular defects of rhythm abnormalities as well as bronchospasm and altered sensorium are the commonest clinical features.

**DISCUSSION**

This high incidence of organo-phosphorus, insecticide poisoning is due to the fact that the surrounding of rural areas are the mainly of agricultural occupation with the main crops cultivated being paddy, maize, chilly and cotton which frequently require the use of pesticide for eradication of pests and insects. The incidences of the poisoning is maximum during the period of maximum agriculture activity i.e. between July and April but cases tend to occur throughout the year.

Nausea and vomiting are earliest and commonest symptoms observed in our study. Nausea was present in 93% and vomiting in 60% of cases. The gastrointestinal system gets the brunt of poisoning initially, as the poisoning is taken orally. The pungent smell and bitter taste provokes nausea and vomiting. These symptoms persist owing to muscarinic effect of acetylcholine. Shankar et al observed around 56% whereas Gupta, Patel et al observed 76% of these symptoms in their study.

In our study abdominal pain was observed in 46-66% of the patient, mostly on the 1st and 2nd day. This is due to increased tone and rhythmicity of duodenum and appears over when there is no other systemic effect. Stomach wash procedure may contribute partially to it. Duodenum appears to be very susceptible to the effect of cholinesterase inhibitor and the fall in cholinesterase level in the intestine is likely to cause sever spasm and gripping pain in the upper abdomen. The pain was self limiting in all the patients. Shankar et al observed around 32% whereas Gupta, Patel et al observed 36% of these symptoms in their study.

In our study, oronasal froth was observed in 33.33% of cases name and name reported oronasal froth in 30% and 40% of the patient respectively. The froth in the mouth due to increased secretion from bronchi and increased production in saliva. It is highly sensitive to atropine therapy. Shankar et al observed around 30% whereas Vishwanathan et al observed 40% of these symptoms in their study.

Breathlessness was observed in 33.33% of cases this was mainly due to bronchospasm and excessive secretion due to muscarinic nicotinic and CNS effect singly or in combination may cause breathlessness. Shankar et al observed around 28% whereas Vishwanathan and Srinivasan et al observed 42% of these symptoms in their study.

Diarrhoea was observed in 6.67 % of cases mainly on 1st and 2nd day.

---

### Table: Clinical Signs Observed

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Present Study Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea</td>
<td>28</td>
<td>93.33</td>
</tr>
<tr>
<td>Vomiting</td>
<td>18</td>
<td>60.00</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>14</td>
<td>46.66</td>
</tr>
<tr>
<td>Pin point pupil</td>
<td>10</td>
<td>33.33</td>
</tr>
<tr>
<td>Salivation</td>
<td>8</td>
<td>26.67</td>
</tr>
<tr>
<td>Lacrimation</td>
<td>8</td>
<td>26.67</td>
</tr>
<tr>
<td>Weakness</td>
<td>6</td>
<td>20.00</td>
</tr>
<tr>
<td>Restlessness</td>
<td>4</td>
<td>13.33</td>
</tr>
<tr>
<td>Headache</td>
<td>3</td>
<td>10.00</td>
</tr>
<tr>
<td>Blurred vision</td>
<td>3</td>
<td>10.00</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>2</td>
<td>6.67</td>
</tr>
</tbody>
</table>

---

### Table: Symptoms of Organophosphorus Poisoning

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Present study cases</th>
<th>%</th>
<th>A P N Kumar et al</th>
<th>Goel et al</th>
</tr>
</thead>
<tbody>
<tr>
<td>Offensive odour</td>
<td>30</td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Altered sensorium</td>
<td>23</td>
<td>72%</td>
<td>75%</td>
<td>75%</td>
</tr>
<tr>
<td>Pin point pupil</td>
<td>21</td>
<td>70.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasciculations</td>
<td>15</td>
<td>50</td>
<td>55%</td>
<td></td>
</tr>
<tr>
<td>Oronasal froth</td>
<td>10</td>
<td>33.33</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bradycardia</td>
<td>8</td>
<td>21%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Tachypnoea</td>
<td>6</td>
<td>20%</td>
<td>42.5%</td>
<td></td>
</tr>
<tr>
<td>Bronchospasm</td>
<td>6</td>
<td>20.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Miosis</td>
<td>5</td>
<td>16.7%</td>
<td>95%</td>
<td></td>
</tr>
<tr>
<td>Coma</td>
<td>2</td>
<td>6.67</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

**Sign**

- Offensive odour
- Altered sensorium
- Pin point pupil
- Fasciculations
- Oronasal froth
- Bradycardia
- Tachypnoea
- Bronchospasm
- Miosis
- Coma

**Present study cases**: The percentage of symptoms observed in the study is compared with the studies of A P N Kumar et al and Goel et al.
Tiwari et al observed this in 3.2% of patients leading to dehydration was not seen in any cases it was mild and watery. General weakness was observed in 20% of cases. The increased fatigability and muscular weakness is due to the nicotinic effect of poisoning and was seen mainly in sever poisoning cases. Headache and blurred vision were seen in 10% patient. Convulsions was not observed in any case.

Amongst signs, miosis was the most consistent sign of organophosphorus poisoning was miosis, it was observed in 86.67% of the patient with pin point pupil in 70%. In fact the poison was suspected in many of the patient with kerosene or garlic odor and miosis which may reactive initially and become non reactive as they assume pin point pupil size, with treatment miosis was the last sign to appear and it may take few hours or 1 to 2 days to disappear. Shankar et al observed miosis in 58% cases whereas Panduranga et al 70% of cases. It does not indicate the severity of poisoning and its disappearance is not constant.

Fasciculation were observed in 50% cases. They are because of increased in amplitude of miniature and end plate potentials due to accumulation of acetyl choline and increase in the spontaneous frequency of the end plate potentials. With administration of PAM the fasciculation disappeared within few hours to 1 to 2 day.

Shankar et al observed fasciculation in 20% of case and Balani et al observed them in 30% and Gupta et al observed the same in 32 % of the cases.

Unconsciousness was observed in 6.67% of cases i.e. the two cases who were admitted in grade 5 poisoning one of them expired. Shankar et al observed coma in 14.7 % of patient and Vishwanathan and Srinivasan in 10% of cases.

In our study we did not observed any cranial nerve paralysis or cerebellar sign or delayed neurotoxicity or observed in some of the previous studies done by other worker.

Bronchospasm was observed 12.20% of the patient and are due to bronchospasm. With the treatment bronch disapeared in few hours to 1 to 2 days. Vishwanathan and Srinivasan et al observed bronchospasm in 45% of patients and Gupta, Patel et al found in 30% of cases.

Respiratory distress is multifactorial, the excessive secretions in the oral cavity and bronchial passage are likely to bring about to sign of pulmonary congestion and edema. The bronchospasm from muscarinic effect add to the difficult in breathing leading to shortness of breath and cyanosis. Accumulation of fluid in air passage and bronchospasm leading to obstruction ventilator in sufficient. The CNS effects add to respiratory difficulty by depression of respiratory centre.

Respiratory paralysis is due to the nicotinic effect on respiratory muscle and it was observed in 4 patients in our study (13.33%). they were put on ventilator. 3 of them expired and 1 patient recoverd. Panduranga Rao et al observed the same in 8.6 % of patient.

Sinus bradycardia was observed in 6.67 % of patient. Sinus tachycardia was observed in 10%. Bradycardia can occur due to direct effect of the compound on the sinus node. It is a muscarinic sign but not frequently observed. Gupta et al observed bradycardia in 12% cases. Tachycardia may be due to nicotinic effect of poisoning or due to the anxiety of the patient. Shankar et al observed tachycardia in 19.6% of patient.

Hypotension as a terminal event was observed in 3 patient and none of them responded to dopamine and other supportive measure.

CONCLUSION

Organophosphorous compound are frequently used for suicidal purpose by young population. Most from them are agriculturists and their family members. It is one of the number one killer of young in medical and allied specialties of any hospital.

Common manifestations of poisoning are nausea, vomiting, excessive secretion, miosis, fasciculation and sign of bronchospasm and pulmonary edema. SGO levels through showing an mild increase does not appear to be of any prognosis value. Clinical deterioration was observed in some cases from day1 to 10 days in spite of effective therapy and has to be watched for energetic therapy with atropine, PAM and ventilator support allows survival after poisoning with doses of organophosphorous compound vastly exceeding the usual fatal dose.

The best preventive measure would be improving the socioeconomic standard of the rural poor which will help in improving their educational standards, family atmosphere and the quality of life. Unless these changes come about, the Organophosphorous poisoning will continue to claim its share of young lives plunging many families into chaos.

Bibliography

2. Gupta and Patel: Diazinon Poisoning japi 16; 457,1968
4. Panduranga Rao et al: Clinical profile of 100 cases of poisoning admitted to AMC Unit JAPI16; Vol 40, No12, 839.

*******