Clinical Profile of Acute Organophosphorus Poisoning with Special Reference to Its Metabolic Complications

Abdul Barik Ahmed[1], Hussain Ahmed[2], Sangitanjan Dutta[3]

[1]Assistant Professor, Department of General Medicine, Jorhat Medical College and Hospital, Jorhat, Assam.
[2]Associate Professor, Department of General Surgery, Jorhat Medical College and Hospital, Jorhat, Assam.
[3]Professor, Department of General Medicine, Gauhati Medical College and Hospital, Guwahati, Assam.

ABSTRACT

BACKGROUND
Organophosphorus poisoning accounts for significant morbidity and mortality worldwide, especially in developing countries.

METHODS
This prospective observational study was done on 50 cases of acute organophosphorus poisoning admitted in Gauhati Medical College and Hospital, Guwahati, for a period of one year, from August 2015 to July 2016. A detailed history was obtained, and thorough clinical examination and laboratory tests were done in each case.

RESULTS
In our study, most OP compound poisoning cases were observed in 2nd and 3rd decades of life (96%) with a male preponderance (male to female ratio is 3:2). 46 (92%) cases had ingested poison in an attempt to commit suicide. Common clinical manifestations were pupillary constriction (82%), nausea & vomiting (82%), abdominal pain (48%), bradycardia (44%), salivation (44%), breathlessness (32%), headache (30%), and fasciculation (22%). 15 cases had blood sugar more than 140 mg% (6 cases had blood sugar ≥200 mg%) while 4 had less than 60 mg% at admission. 7 cases showed a rise of ALT, 15 cases showed hypokalaemia, 5 cases showed a rise of lipase and 12 cases showed a rise of urea.

CONCLUSIONS
In our study, majority of cases belonged to the younger age group. The common symptoms seen were nausea, vomiting, miosis, abdominal pain, bradycardia, salivation, breathlessness, headache, tachypnoea and fasciculation. Common metabolic complications are hyperglycaemia, hypokalaemia, elevated serum urea and aminotransferases.

KEYWORDS
Metabolic, Miosis, Organophosphorus, Poisoning

Corresponding Author:
Dr. Hussain Ahmed,
Associate Professor,
Department of Surgery,
Jorhat Medical College and Hospital,
Jorhat- 785001, Assam.
E-mail: dr.abahmed@gmail.com

DOI: 10.18410/jebmh/2020/99

Financial or Other Competing Interests: None.

How to Cite This Article:
Abdul Barik A, Hussain A, Dutta S. Clinical profile of acute organophosphorus poisoning with special reference to its metabolic complications. J. Evid. Based Med. Healthc. 2020; 7(10), 460-463. DOI: 10.18410/jebmh/2020/99

Submission 06-02-2020,
Peer Review 10-02-2020,
Acceptance 21-02-2020,
Published 03-03-2020.

BACKGROUND

Organophosphorus (OP) compounds have come as a boon to the farmers to control pests and insects affecting the plants and crops. The widespread use and easy availability of these highly toxic substances have resulted in many instances of accidental as well as suicidal poisoning. Case fatality following deliberate ingestion is high (5-20%)[1] OP compounds inactivate acetylcholinesterase, resulting in the accumulation of acetylcholine in cholinergic synapses.[1,2] OP poisoning causes acute cholinergic phase, which may occasionally be followed by intermediate syndrome or organophosphate induced delayed polyneuropathy.[1] Acute cholinergic syndrome starts within few minutes of exposure and muscarinic or nicotinic features may be present.[1,3] Common muscarinic features are vomiting, diarrhoea, salivation, miosis, sweating, bradycardia, hypotension, bronchoalveolar secretions, bronchoconstriction and nicotinic features are fasciculation, paralysis, tachycardia, reduced ventilation, mydriasis.[1,4] the present study is undertaken to assess the clinical feature and metabolic complications of acute OP poisoning.

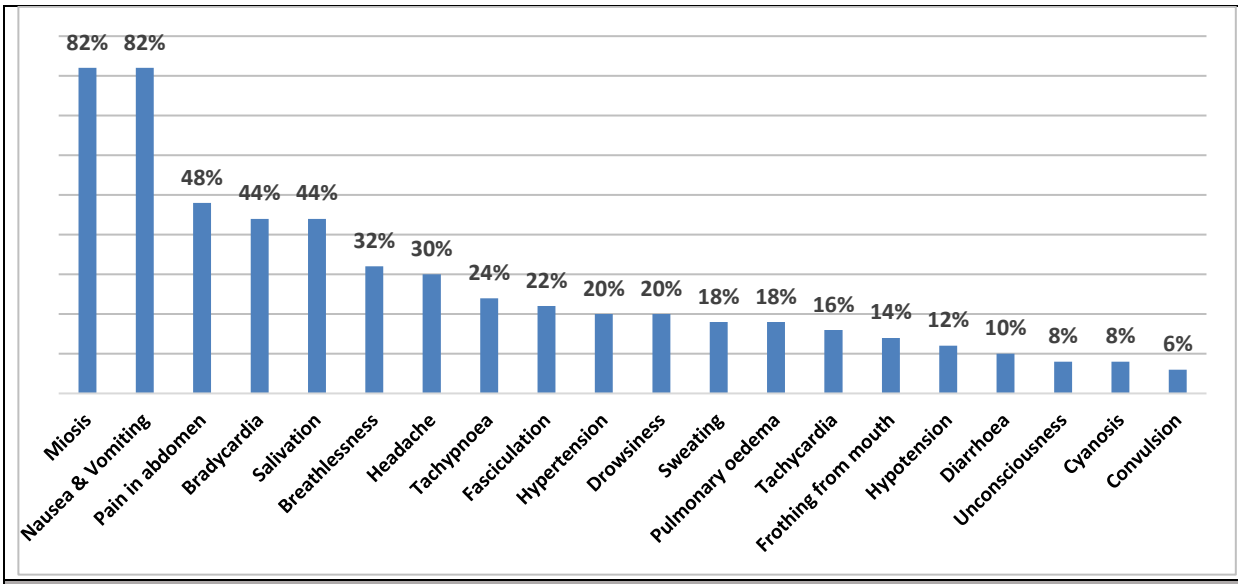
We wanted to study the clinical profile of acute organophosphorus poisoning with special reference to its metabolic complications.

METHODS

This prospective observational study was done on 50 cases of acute organophosphorus poisoning admitted in Gauhati Medical College and Hospital, Guwahati for a period of one year, from August 2015 to July 2016. Cases were selected based on history of exposure to OP compounds, characteristic clinical signs and symptoms of OP poisoning and improvement of the signs and symptoms of poisoning after administration of atropine and oximes.[4] Patients with co-morbidities like diabetes, renal disease, liver disease, cardiovascular disease were excluded. A detailed history regarding age, occupation, residence, socio-economic status, approximate time of ingestion, circumstances leading to poisoning were recorded. A thorough clinical examination was carried out at the time of admission in each case with particular attention to the signs and symptoms attributed to OP compound poisoning. Special attention was paid to the level consciousness, lung, cardiovascular system, and pupils. Each case was examined immediately on admission and was observed every 15 minutes interval for the first two hours. Subsequently they were monitored at two hourly intervals or oftener if required till the patient were atropinised. Atropinisation was confirmed by dry secretions, dilated pupils and tachycardia.[4] Laboratory investigations viz. Blood R/E, Urine for R/E, Liver function tests, Renal function tests, blood sugar, ECG were done in each case. Investigations like USG of abdomen, Echocardiography and Chest X-Ray were done if indicated. In all cases, standard treatment was started. In our study, pralidoxime and atropine were used as antidote to OP compound poisoning.[5] Patient was said to be atropinised with the following signs- dilatation of pupils, dry secretions and tachycardia.[4] all data were documented and analysed as per laid down protocol.

RESULTS

50 cases of OP compound poisoning were included in this study. Highest number of cases (52%) were found in the age group of 11-20 years. 30 (60%) cases were male and remaining 20 (40%) were female. Male to female is 3:2. Out of 50 cases, 33 (66%) cases were unmarried, 33 (66%) cases were from rural background and 46 (92%) had ingested poison in an attempt to commit suicide.



Graph 1. Clinical Features

Blood Sugar	Number of Cases	Percentage
<60 mg/dl	4	8
60-139 mg/dl	31	62
140-199 mg/dl	9	18
≥200 mg/dl	6	12

Table 1. Blood Sugar at the Time of Admission

Serum Level	Number of Cases	Percentage
ALT		
>40 U/L	7	14
<40 U/L	43	86
AST		
>40 U/L	8	16
<40 U/L	42	84

Table 2. Serum ALT, AST

Serum Urea	Number of Cases	Percentage
<15 mg/dl	0	0
15-40 mg/dl	38	76
>40 mg/dl	12	24

Table 3. Serum Urea

Serum K^+ Level	Number of Cases	Percentage
<3.5 mmol/L	15	30
3.5-5.5 mmol/L	35	70
>5.5 mmol/L	0	0

Table 4. Serum K^+

Pupillary constriction (miosis) and "nausea & vomiting" were the commonest manifestations (82% each) followed by, abdominal pain (48%), bradycardia (44%), salivation (44%), Breathlessness (32%) and others as depicted in the Graph 1. Out of 50 cases, 16 showed leucocytosis. 4 cases had hypoglycaemia, 6 had blood sugar in diabetic range and 9 had impaired blood sugar. 6 cases showed a rise of serum amylase and 5 showed a rise of serum lipase. In our study of 50 cases, 7 showed a rise of ALT and 8 showed a rise of AST. Among all cases, 12 (24%) showed a rise of urea more than 40 mg%. Present study showed hypokalemia in 15 (30%) cases. 2 cases showed hyponatremia.

DISCUSSION

Because of their widespread availability and use as household and agricultural products, the OP compounds are common sources of poisoning. In our study, most OP compound poisoning cases were observed in 2nd and 3rd decades of life (52% and 42% respectively). 94% cases were below the age of 31 years. The mean age of cases was 22.14 years with a male preponderance (male to female ratio was 3:2). 17 patients (10 males and 7 females) were married. These findings correlate with the findings of other studies.[6,7,8,9,10] Common occurrence of poisoning in the younger age is remarkable and factors like failure in examination, emotional conflicts, unemployment, failure in love-affairs are likely responsible.

In 46 cases (92%), the intention of poisoning was suicide. This finding is comparable to the studies of Shakuntala, Yogesh G,[6] Subhash Chandra Joshi, Chandra Prakash et al.[7] Viswanathan KG, Gupta A et al.[10] In the rest, the causes were accidental (3 cases) and occupational (1 case). In our study, majority of suicidal cases were due to circumstances like domestic problems (scolding, disagreement with family members), love affairs, failure in examinations, unemployment, marital disharmony and financial crisis.[7, 10] Gastrointestinal system bore the initial effects as the poison was taken orally. The pungent smell and bitter taste provoke the nausea and vomiting. These features persist because of muscarinic effects of OP poisoning. In our study, 41 (82%) cases had nausea and vomiting. Ojha U.K. et al. found nausea and vomiting in 98% of cases in their study.[9] Excessive salivation was noted in 48% cases. Only 4 cases had diarrhoea in the present study.

In the respiratory system, we observed breathlessness in 32%, pulmonary oedema in 16%, bronchospasm 16%, tachypnoea 24% and cyanosis in 8% of cases. Basavaraj GM, Saraf N et al. found breathlessness in 38% of cases in their study[9]. Flooding of lungs by bronchoalveolar secretions is the probable cause of pulmonary oedema. In the cardiovascular system, we observed bradycardia in 44%, tachycardia in 16% and normal pulse rate in 42% of cases. Ojha U.K. et al. found bradycardia and tachycardia in 18% and 12% of cases respectively in their study.[9] Blood pressure was normal in 60%, high in 20% and low in 14% cases. Bradycardia and hypotension are due to potentiation of post-ganglionic parasympathetic activity. Hypotension may also be the effect of vomiting and/or diarrhoea. Miosis was observed in 41 (82%) cases, muscular twitching in 20% cases and generalised muscular weakness in 24% cases. These findings correlate with the findings of other workers.[9]

Oro-nasal froth was seen in 7 (14%) cases in the present study. These cases also had pulmonary oedema. These findings were due to salivation and broncho-alveolar secretions. Sweating was seen in 18% cases, lacrimation in 10% cases. Basavaraj GM, Saraf N et al. found sweating, lacrimation and salivation in 44%, 38% and 44% of cases respectively in their study.[2] In the present study, haematological profile showed leucocytosis in 32% of cases. Kumar S, Agarwal S et al. found leucocytosis in 32.5% of cases in their study.[11] Hyperglycaemia and hypoglycaemia were found in 30% and 8% cases respectively in the present study. Rachita Nanda, Rathod PK et al. and Hui KS also found hyperglycaemia in their study.[12,13] R Raghapriya et al. found hyperglycaemia in 37% and hypoglycaemia in 11% of cases in their study.[14]

An elevated urea (>40 mg %) was the commonest renal abnormality, seen in 24% of the patients followed by glycosuria (20%), haematuria (20%) and proteinuria (14%) of patients. All these abnormalities were reversible within few days. Hypokalaemia was observed in 30% of cases in the present study. Basavaraj GM, Saraf N et al. found hypokalaemia in 14% of patients in their study.[2] Hyponatremia was found in 4% cases. These electrolytes abnormality may be related to the increased gastrointestinal secretions, vomiting, diarrhoea and nasogastric suctions. Elevated serum amylase and lipase were observed in 12% and 10% of cases respectively. Rise of serum alanine aminotransferase and aspartate aminotransferase were found in 14% and 16% of cases respectively. Singh S, Bharadwaj U, Verma SK, et al. found that 46.95% of cases had a rise of serum amylase and 16% of cases had a rise of serum transaminases in their study.[15] All cases were treated

according to the standard protocol. In our study, 5 cases died. Those who died had pulmonary oedema.

CONCLUSIONS

In our study, majority of the cases belonged to the younger age group. The common clinical manifestations were nausea, vomiting, miosis, abdominal pain, bradycardia, salivation, breathlessness, headache, tachypnoea and fasciculation. Common metabolic complications were hyperglycemia, hypokalaemia, elevated serum urea and aminotransferases.

REFERENCES

[1] Thomas SH. Poisoning. In: Ralston SH, Penman ID, Strachan M, et al, eds. Davidson's principles and practice of medicine. 23rd edn. Elsevier 2018:145-146.

[2] Bsavaraj GM, Saraf N, Sharma N. Study of clinical profile of organophosphate poisoning with special reference to electrocardiographic changes and electrolyte derangements. J of Evolution of Med and Dent Sci 2014; 67 (3):14514-14523.

[3] Peter JV, Sudarsan TI, Moran JL. Clinical features of organophosphate poisoning: a review of different classification system and approaches. Indian J Crit Care Med 2014; 18 (11):735-745.

[4] Tripathy SK, Rout PK, Debta N, et al. Study of clinical profile of organophosphorus poisoning with special reference to electrocardiographic changes and electrolyte derangement. Int J Adv Med 2018; 5 (1):50-56.

[5] Cherian MA, Roshini C, Visalakshi J, et al. Biochemical and clinical profile after organophosphorus poisoning-a placebo-controlled trial using pralidoxime. J Assoc Physicians India 2005; 53:427-431.

[6] Shakuntala, Yogesh G. Analysis of organophosphorus poisoning, at tertiary are hospital: a report. J Evidence Based Med and Healthcare 2015; 2 (4):421-430.

[7] Joshi Kumar S, Agarwal S, Khan S, et al. Leukocyte count: a reliable marker for the severity of organophosphate intoxication? J Lab Physicians 2018; 10 (2):185-188.

[8] SC, Prakash C, Joshi A, et al. Profile of organophosphorus poisoning at tertiary care hospital in Uttarakhand. J Indian Acad Forensic Med 2013; 35 (4):346-348.

[9] Khajuria V, Shah S, Tandon VR, et al. Organophosphorus poisoning in a tertiary hospital of North India- a preliminary report. Bulletin of Pharmaceutical and Medical Sciences (BOPAMS) 2013;1 (1):41-43.

[10] Ojha UK et al. Profile of organophosphorus poisoning at a tertiary medical centre in Jharkhand, India. Int J Adv Med 2018; 5 (6):1510-1514.

[11] Viswanathan KG, Gupta A, Santosh RC, et al. Profile of fatal organophosphorus pesticide poisoning cases near Davangere. J Punjab Acad Forensic Med Toxicol 2014; 14 (2):96-100.

[12] Nanda R, Rathod PK, Mishra PK, et al. Glycemicstatus in organophophorus poisoning. J Nepal Health Res Counc 2015; 13 (31):214-219.

[13] Hui KS. Metabolic disturbances in organophosphate insecticide poisoning. Arch Pathol Lab Med 1983; 107 (3):154.

[14] Raghapriya R, Dosi RV, Parmar A. Glycemicstatus at the time of presentation in acute organophosphorus poisoning and its correlation with severity and clinical outcome. J Assoc Physicians India 2018; 16 (8):18-22.

[15] Singh S, Bharadwaj U, Verma SK, et al. Hyperamylasemia and acute pancreatitis following anticholinesterase poisoning. Hum Exp Toxicol 2007; 26 (6):467-471.