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Original Article

Arterial Blood Gas as a Prognostic Tool in Organophosphorus Poisoning Patients – A Prospective Observational Study

S Subikshavarthni[1], R Selvan[2]

[1]Consultant, Department of Emergency Medicine, Meenakshi Memorial Hospital, Rajapalayam, Tamil Nadu, India, [2]Professor, Department of Neurosurgery, Kanyakumari Government Medical College, Kanyakumari, Tamil Nadu, India

Abstract

Introduction: Organophosphorus (OP) compounds constitute a heterogeneous category of chemicals specifically designed for the control of pests, weeds, or plant diseases. Intentional ingestion of OP pesticides has been common and now the preferred form of poisoning due to its easy access in central and Southern parts of India. This predominantly occurs in rural communities. As a result of widespread use, OP poisoning is a major cause of morbidity and mortality worldwide.

Aim: This study aims to access the arterial blood gas (ABG) analysis as a prognostic prediction tool in OP poisoning patients.

Materials and Methods: This is a prospective study from February 2017 to March 2018, out of 114 patients who presented to the emergency department, 14 patients were excluded from the study and the remaining 100 patients were included in the study.

Results: Males were the most predominant group in OP ingestions in this study. In 29% of patients, the ABG interpretation was normal and metabolic acidosis in the initial ABG had a mean intensive care unit (ICU) stay value of 12.92; respiratory acidosis had a mean ICU stay value of 12.36; mixed acidosis had a mean ICU stay value of 9.33; respiratory alkalosis had a mean ICU stay value of 5.37; metabolic alkalosis had a mean ICU stay value of 4.66. *P*-value was calculated and found to be statistically significant. Moreover, it was also found that the patients who presented with extreme acidosis (≤7.1) had increased mean ICU stay value (17.5).

Conclusion: This study concludes that ABG analysis at the initial presentation could help in assessing the prognosis of OP poisoning patients much earlier, which could help in intensifying the management.

Key words: Arterial blood gas analysis, Organophosphorus, Poisoning

INTRODUCTION

Pesticides include a wide variety of compounds which include insecticides, herbicides, fungicides, and others. Thus, far more than 1000 active substances have been determined in approximately 35,000 preparations of pesticides used in agriculture.[1] Organophosphorus (OP) pesticide poisoning is a serious hazard. OP compounds (OPC) poisoning is an important clinical problem in the rural regions of the developing countries.[2] The adult mortality rate due to OP poisoning in rural South India is 0.97/1000 persons/year.[3] Around 200,000 people around the world die each year from OPC poisoning, especially in developing countries.[4] OP groups of poison exist since the 19th century.[2] The first OPC was developed as an insecticide for agriculture and now different varieties of OP pesticides are available in the market each having different toxicity levels.[2] Acute OP pesticide poisoning is common in developing countries due to easy availability and less awareness among poorly educated farmers. Hence, farmers are at high risk for accidental exposure to OPC poisoning. However, poisoning with suicidal intent is more common than accidental exposure.[5] Intentional ingestion of OP pesticides has been common for the past 40 years. A fatal outcome is often related to delay in diagnosis or improper management. This predominantly occurs in rural

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Corresponding Author: Dr. R Selvan, Department of Neurosurgery, Kanyakumari Government Medical College, Kanyakumari, Tamil Nadu, India.

communities and is often an impulsive act comparable to self-poisoning with medication in the west; the critical difference is 10–20% of case fatality rate (compared to 0.3% in Britain).[4]

OPCs are available as dust, granules, or liquids. OPCs are very well absorbed from the lungs, gastrointestinal tract, skin, mucous membranes, and conjunctiva following inhalation, ingestion, or topical contact. Most OPCs are lipophilic. Peak levels of most OP insecticides measure around 6 h after oral ingestion in man.[6,7]

OP pesticide inhibits both cholinesterase and pseudocholinesterase activities; as a result, there is an accumulation of acetylcholinesterase at synapses causing overstimulation and disruption of neurotransmission in both central and peripheral nervous systems. Although organophosphates differ structurally from acetylcholine, they can bind to acetylcholinesterase molecule at the active site and phosphorylate the serine moiety. When this occurs, the resultant conjugate is infinitely more stable than the acetylcholine-acetylcholinesterase conjugate although endogenous hydrolysis does occur. The rate of onset of clinical features will vary between people and between OPCs but is mostly within minutes to hours depending on the dose. Some OP insecticides can cause delayed onset of severe poisoning after a day or 2 days. Directly acting OP insecticides can inhibit acetylcholinesterase without being structurally altered. However, many OPCs, such as parathion and malathion, are indirect inhibitors requiring partial metabolism within the body to become active.[8]

Exposure to OP vapors rapidly causes upper airway irritation and bronchospasm followed by systemic symptoms. Acute respiratory failure is the most common cause of death in OPC poisoning due to increased secretions and inadequate ventilation. One of the causes of complications is an acid-base imbalance. Subsequent measures must be carried out by recognizing and correcting the acid-base disturbance at the earliest possible time.[9,10]

Evaluation of acid-base status in the OPC poisoning patients plays a critical role as early recognition of acid-base disturbance can alter the management and prognosis. Moreover, it is also very essential to determine the respiratory failure following which endotracheal intubation and, mechanical ventilation can be done.

Aim

This study aims to access the arterial blood gas (ABG) analysis as a prognostic prediction tool in OP poisoning patients.

MATERIALS AND METHODS

This is a prospective study from February 2017 to March 2018 where a specific set of data was collected from the patients who fulfilled the inclusion criteria. A total of 114 patients were enrolled, of which 14 were excluded from the study and the remaining 100 patients accounted.

Inclusion Criteria

The following criteria were included in the study:

- Age of more than 18
- Clinical features suggestive of OP poisoning
- Written and informed consent (in the local language)
- Consumption of OPC as per.

Patient, relatives, referring doctor, and the pesticide container.

Exclusion Criteria

The following czriteria were excluded from the study:

- Any mixed ingestions
- Age <18
- Time of consumption more than 24 h
- Discharge against medical advice (no follow-up)
- Other forms of OP poisoning other than oral ingestion, for example, inhalation
- Comorbidities such as chronic kidney disease, chronic obstructive pulmonary disease, congestive heart failure, and chronic lung disease.

The acid-base status being the priority, data were collected at the initial presentation of the OP poisoning. Arterial blood collected from the radial artery and femoral artery. The radial artery at the wrist was the most preferred site of ABG specimen collection as it has adequate collaterals. When this was not feasible, femoral artery was chosen to collect the sample. Estimation was done using the ABG analyzer. The results of the ABG were recorded and analyzed. Then, according to the report, acid-base status determined. Data were collected by the study doctor, wherein during the examination, initial resuscitation was carried out. Initial resuscitation was done in emergency department (ED) and further treatment was subjected to the intensive care unit (ICU) physician.

The treating critical care physician was not notified of the study and was blinded so as to avoid any bias in assessing the ICU stay in the selected group of patients.

The acid-base status being the priority, data were collected at the initial presentation of the OP poisoning.

RESULTS

Most numbers of patients who consumed OPCs were found to be between 20 and 40 years which were 69 in number. The next significant group was 40–50 years old which had 15 patients Figure 1. Males were the more predominant group in OP ingestions in this study. Most of the patients consumed monocrotopos and chloropyripos compounds [Figure 2]. Most cases presented within 1 h-4 h of ingestion, which was 53 in number. 25 cases presented within 30 to 60 min of ingestion and 15 cases presented within 30 min of ingestion [Table 1 and Figure 3].

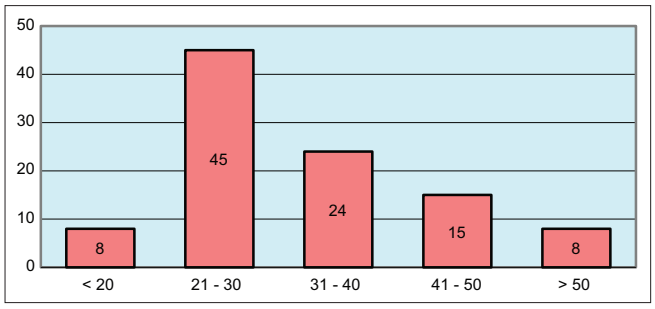

Figure 1: Age distribution

Out of 24 cases who are initial ABG showed metabolic acidosis, 3 deaths were reported. Out of 11 cases, who initially presented with respiratory acidosis, 2 deaths were reported; and out of 3 cases, 1 death was reported. In cases who presented with respiratory and metabolic alkalosis, no deaths were reported. Out of 100 cases, 42 cases required invasive ventilation. Out of 100 patients, 94 patients recovered and death reported in 6 patients. Patients with pH <7.10 had a mean ICU stay value of 17.5; patients with pH ranging from 7.11 to 7.20 had a mean ICU stay value of 12.1; patients with a pH ranging from 7.21 to 7.35 had a mean ICU stay value of 11.23. The mean ICU stay of patients presenting with pH of 7.45–7.49 is 5.6; and patients presenting with pH >7.50 had a mean ICU

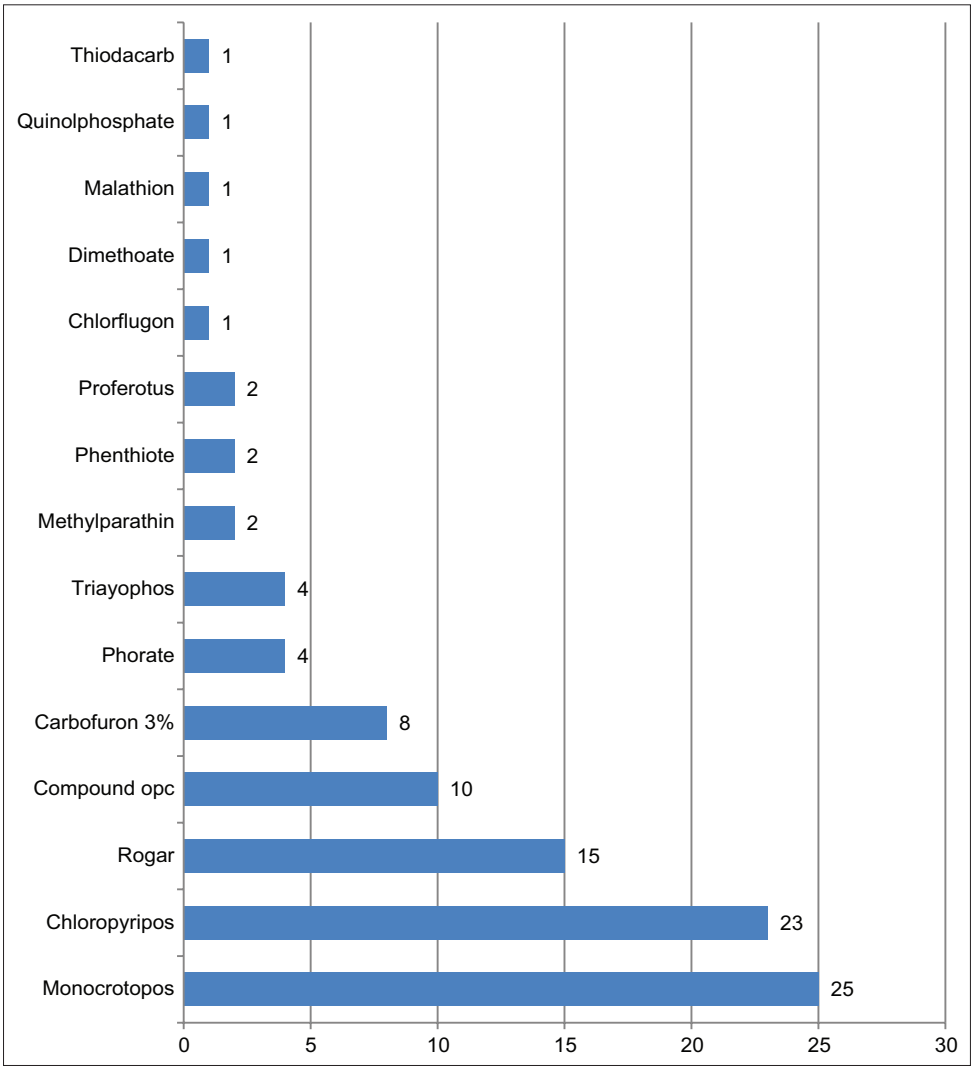

Figure 2: Organophosphorus compounds

Table 1: Comparison ABG analysis and ICU stay

Comparison	Mean	Standard deviation	*P*-value
Metabolic acidosis (24) versus metabolic alkalosis (3)	12.92	3.92	0.001
	4.67	0.58	
Metabolic acidosis (24) versus respiratory acidosis (11)	12.92	3.92	0.684
	12.36	3.11	
Metabolic acidosis (24) versus respiratory alkalosis (30)	12.92	3.92	<0.001
	5.37	2.54	
Metabolic acidosis (24) versus mixed acidosis (3)	12.92	3.92	0.137
	9.33	2.08	
Metabolic acidosis (24) versus normal (29)	12.92	3.92	<0.001
	4.65	2.61	
Respiratory acidosis (11) versus normal (29)	12.36	3.11	<0.001
	4.65	2.61	
Respiratory alkalosis (30) versus normal (29)	5.37	2.54	0.293
	4.65	2.61	
Mixed acidosis versus normal (29)	9.33	2.08	0.005
	4.65	2.61	
Respiratory acidosis (11) versus respiratory alkalosis (30)	12.36	3.11	<0.001
	5.37	2.54	
Respiratory acidosis (11) versus metabolic alkalosis (3)	12.36	3.11	0.001
	4.67	0.58	
Metabolic acidosis (24) versus respiratory alkalosis (30)	12.92	3.92	<0.001
	5.37	2.54	
Respiratory alkalosis (30) versus metabolic alkalosis (3)	5.37	2.54	0.642
	4.67	0.58	

ABG: Arterial blood gas, ICU: Intensive care unit

stay value of 5.2. Thus, alkalosis does not play a role in increasing the ICU stay.

The mean ICU stay of metabolic acidosis and metabolic alkalosis shown a statistically significant difference and it confirms that patients presented with metabolic acidosis had increased ICU stay.

The mean ICU stay of metabolic acidosis and respiratory acidosis showed statistically insignificant. Thus, patients who presented with respiratory and metabolic acidosis did not show the difference in their ICU stay.

The mean ICU stay of metabolic acidosis and respiratory alkalosis was shown a statistically significant difference and confirm that patients who presented with metabolic acidosis had increased ICU stay.

The mean ICU stay of metabolic acidosis and mixed acidosis showed statistically insignificant. Thus, it is proved that patients presenting with metabolic acidosis and mixed acidosis did not show variation in their ICU stay.

The ICU stay of patients presenting with respiratory acidosis and respiratory alkalosis was shown a statistically significant difference and it was confirmed that patients presenting with respiratory acidosis had increased ICU stay.

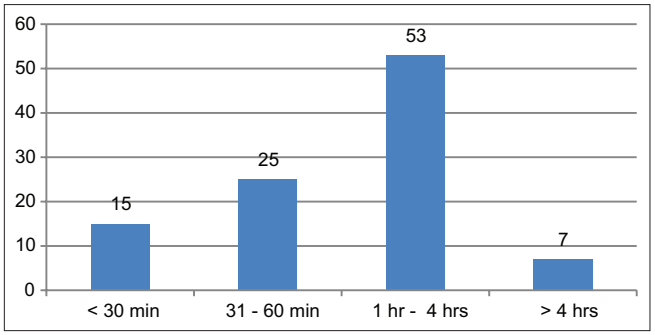

Figure 3: Presentation at hospital after consumption

The ICU stay of patients presenting with metabolic alkalosis and respiratory alkalosis was shown a statistically insignificant. Thus, it is proved that patients presenting with metabolic and respiratory alkalosis did not show the difference in their mean ICU stay.

The mean ICU stay of patients presenting with respiratory acidosis and metabolic alkalosis was shown a statistically significant difference and it confirms that patients presenting with respiratory acidosis had increased ICU stay [Figure 4 and Table 1].

DISCUSSION

The diagnosis of OP pesticide poisoning is based on the patient's history, clinical presentation, and laboratory tests. In a patient with a positive history, a typical odor

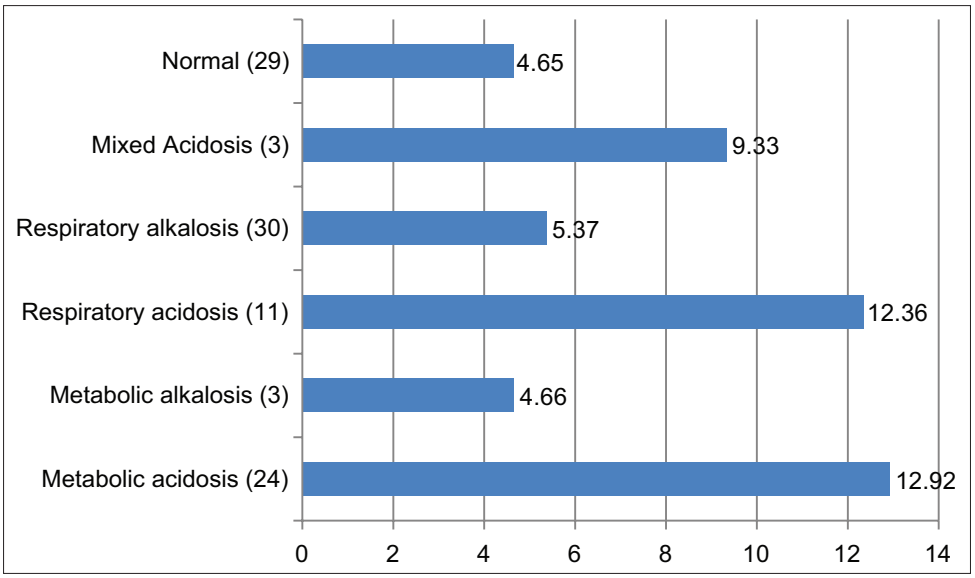

Figure 4: Mean days of intensive care unit stay versus arterial blood gas analysis interpretation

on the breath, characteristic symptoms, and depressed erythrocyte and plasma cholinesterase activities, diagnosis is not difficult to make. Unfortunately, history is often unobtainable. Moreover, the clinical features of OP poisoning may not be recognized as such if the patient presents, for example, with heart block, gastroenteritis, convulsions, or ketoacidosis. An awareness of this diversity of presentation is the first step to an accurate diagnosis.

In OP poisoning, OPC inhibits acetylcholinesterase activity, increases the accumulation of acetylcholine in the synaptic gap, and decreases degradation of acetylcholine, thus leading to excessively increased cholinergic symptoms, which disturbs neurotransmission of the central and peripheral nervous system. This excess synaptic acetylcholine stimulates muscarinic receptors and then depresses or paralyzes the nicotinic receptors. Abnormal neuromuscular transmission mediated through nicotinic receptors may cause carbon dioxide retention and alter the acid-base balance.[8] A retrospective analysis of OP poisoning patients, carried by Liu *et al.*, found a direct correlation between the severity of poisoning and mortality and the presence of pre-treatment of metabolic and respiratory acidosis.[11]

Among the patients who presented with acidosis, extreme acidosis (<7.1) was associated with increased ICU stay when compared with mild-to-moderate acidosis. Patients who presented with alkalosis had decreased ICU stay irrespective of their ranges. Death was reported in 6% of cases. Among them, 3% were cases who presented with metabolic acidosis; 2% with respiratory acidosis; and 1% with mixed acidosis. Thus, the present study showed that among the acid-base disorders in the initial ABG in OP poisoning patients, acidosis was associated with increased morbidity. In patients presenting with acidosis, metabolic acidosis is predominantly associated with increased morbidity and mortality followed by respiratory acidosis subsequently. Mixed acidosis ranks the third in increasing the morbidity and mortality of the patients presenting with OPC poisoning. Metabolic alkalosis and respiratory alkalosis were associated with decreased morbidity and no death was reported in these cases.

Metabolic acidosis is one of the frequent complications of OP poisoning and has been documented as an important determinant of the outcome of the patient.[11] However, severe metabolic acidosis, refractory to standard treatment measures such as sodium bicarbonate, is a rarely reported complication and invariably indicates a poor prognosis. Examples can be cited in four cases with severe metabolic acidosis and hypotension that did not respond to catecholamines.[12]

Most of the patients presented to the ED within 1–4 h of ingestion. Most of them had received gastric lavage in the pre-hospital care setup. In all the cases, ABG, serum cholinesterase levels, electrolytes, and other routine biochemical investigations were carried out. Serum cholinesterase was mostly lowered in all cases suggestive of OP poisoning. Arterial blood was drawn for acid-base analysis. Around 71% of cases had acid-base disturbances, whereas only 29% had normal ABG reports. According to Moulali *et al.*, the study which involved 20 cases 85% had acid-base disturbance and 15% had normal ABG reports.[13]

CONCLUSION

The present study highlights the importance of ABG as an effective tool in the quick assessment of prognosis in

OP poisoning patients. From this study, it is evident that the initial ABG performed on patients presenting with OP poisoning could help in determining the prognosis of the patients even before hospitalization. Special consideration should be given to patients presenting with acidosis (metabolic, respiratory, and mixed) as it is concluded from this study that acidosis (predominantly metabolic acidosis) is associated with increased morbidity and mortality. As a result, the treatment can be intensified and implemented quickly without any delay.

REFERENCES

1. Thunga G, Ganna Sam K, Khera K. Evaluation of incidence, clinical characteristics and management in organophosphorus poisoning patients in a tertiary care hospital. J Toxicol Environ Health Sci 2010;2:73-6.
2. Nurulain SM. Different approaches to acute organophosphorus poison treatment. J Pak Med Assoc 2012;62:712-7.
3. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet 2008;371:597-607.
4. Eddleston M, Phillips MR. Self poisoning with pesticides. BMJ 2004;328:42-4.
5. Banerjee I, Tripathi S, Roy AS. Clinico-epidemiological characteristics of patients presenting with organophosphorus poisoning. N Am J Med Sci 2012;4:147-50.
6. Balali-Mood M, Shahab-Ahmadi A, Salimifar M, Shariate M, editors. Effects of Sodium Bicarbonate in Organophosphate Pesticide Poisoning. Spiez, Switzerland: 3rd Proceedings of Chemical and Biological Medical Treatment Symposium, CBMTS III; 2000. p. 7.
7. Antonijvic B, Stefanovic D, Milovanovic ZA, Stojiljkovic MP, Bokonjic D, Dukic M, editors. Standard Antidotes Along with Sodium Bicarbonate in Organophosphate Poisoning. Spiez, Switzerland: 4th Proceedings of Chemical and Biological Medical Treatment Symposium, CBMTS; 2002.
8. Colović MB, Krstić DZ, Lazarević-Pašti TD, Bondžić AM, Vasić VM. Acetylcholinesterase inhibitors: Pharmacology and toxicology. Curr Neuropharmacol 2013;11:315-35.
9. World Health Organization. Organophosphorus Insecticides a General Introduction. Effects of Organophosphorus Insecticides on the Nervous System. Geneva: World Health Organization; 1986. p. 58-69.
10. World Health Organization. Organophosphorus Insecticides a General Introduction. Metabolism and Mode of Action. Geneva: World Health Organization; 1986. p. 39-48.
11. Liu JH, Chou CY, Liu YL, Liao PY, Lin PW, Lin HH, *et al*. Acid-base interpretation can be the predictor of outcome among patients with acute organophosphate poisoning before hospitalization. Am J Emerg Med 2008;26:24-30.
12. Asari Y, Kamijyo Y, Soma K. Changes in the hemodynamic state of patients with acute lethal organophosphate poisoning. Vet Hum Toxicol 2004;46:5-9.
13. Moulali D, Sarma DV, Jagannath N, Veeresh T, Reddy KE. Spontaneous coronary artery dissection in a case of chronic cocaine abuse. Sci Technol 2014;12:102-4.

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