ISSN:0975 -3583.0976-2833 VOL 02. ISSUE 01. 2011

Original research article

Arterial blood gas analysis correlation with acute organophosphorus compound poisoning outcome prediction

¹Dr. Kalaivani M, ²Dr. Vijay M. Benglorkar

¹Associate Professor, Department of General Medicine, Raja Rajeshwari Medical College, Kambipura, Karnataka, India

²Professor, Department of General Medicine, Raja Rajeshwari Medical College, Kambipura, Karnataka, India

Corresponding Author:

Dr. Kalaivani M

Abstract

Background and Objective: Organophosphorus (OP) compounds are a diverse group of chemicals that are especially created to manage pests, weeds, or plant diseases. Due of its extensive usage, poisoning from organophosphates (OP) is a significant contributor to illness and death on a global scale. The objective of this study is to evaluate the use of arterial blood gas (ABG) analysis as a prognostic predictor in patients with OP poisoning.

Methods: This is a study that looks ahead and examines potential outcomes from the Department of General Medicine, Raja Rajeshwari Medical College, Kambipura, Karnataka, India. Among the 130 patients who arrived at the emergency department, 20 individuals were not considered for the study, leaving a total of 110 patients who were included in the study.

Results: In this study, males were the most prevalent category in cases of OP ingestions. Among the patients, 29% had a normal ABG interpretation. For those with respiratory alkalosis, the mean ICU stay was 5.37. For those with metabolic alkalosis, the mean ICU stay was 4.66. The P-value was computed and determined to be statistically significant. Furthermore, it was discovered that patients who exhibited severe acidosis (\leq 7.1) experienced a higher average length of stay in the intensive care unit (17.5).

Conclusion: The findings of this study suggest that analyzing arterial blood gases (ABG) at the time of initial presentation can provide valuable information for evaluating the prognosis of patients with OP poisoning. This early assessment can aid in implementing more aggressive care strategies.

Keywords: Arterial blood gas analysis, organophosphorus, poisoning

Introduction

Pesticides encompass a diverse range of compounds, such as insecticides, herbicides, fungicides, and other similar substances. Over 1000 active chemicals have been identified in around 35,000 pesticide formulations currently employed in agriculture. Exposure to organophosphorus (OP) pesticides poses a significant risk of poisoning. OP compounds (OPC) poisoning is a significant medical issue in rural areas of developing nations. The annual adult death rate caused by OP poisoning in rural South India is 0.97 per 1000 individuals. Approximately 200,000 individuals worldwide succumb to OPC poisoning annually, with a particular emphasis on impoverished nations. Groups of poisonous substances have been in existence since the 19th century. The initial OPC was formulated as an agricultural insecticide, and currently, various types of OP pesticides are accessible on the market, each exhibiting distinct levels of toxicity. Acute pesticide poisoning is prevalent in underdeveloped nations as a result of the widespread availability of these pesticides and the lack of awareness among farmers with limited education. Therefore, farmers are most susceptible to unintentional exposure to OPC toxicity. Nevertheless, intentional poisoning with the intention of self-harm is more prevalent than unintentional exposure. The deliberate consumption of organophosphate insecticides has been prevalent for the previous four decades. A catastrophic outcome is frequently associated with a delay in diagnosis or inadequate care. This phenomenon is mainly observed in rural areas and is typically a spontaneous action similar to selfpoisoning with medication in Western countries. However, the crucial distinction lies in the fact that the fatality rate in these cases is 10-20%, whereas in Britain it is only 0.3% [1-4].

ISSN:0975 -3583.0976-2833 VOL 02. ISSUE 01. 2011

OPCs are offered in the forms of dust, granules, or solutions. The majority of OPCs have lipophilic properties. The highest concentrations of most organophosphate pesticides are typically observed approximately 6 hours after they are consumed orally by humans. The OP pesticide hinders the actions of both cholinesterase and pseudocholinesterase, leading to an excess of acetylcholinesterase at synapses. The time it takes for clinical symptoms to appear can differ across individuals and different types of OPCs, but it typically ranges from minutes to hours, depending on the dosage. Certain potent insecticides might result in a delayed onset of acute poisoning, typically occurring within one to two days. Nonetheless, acetylcholinesterase can be inhibited by directly acting OP pesticides without undergoing any structural modifications [5, 6].

Exposure to organophosphate (OP) fumes quickly leads to irritation and constriction of the upper airways, which is then followed by symptoms affecting the entire body. OPC poisoning commonly leads to mortality mostly due to acute respiratory failure, which is caused by excessive secretions and insufficient breathing. An acid-base imbalance is a contributing factor to problems. It is essential to promptly identify and rectify the acid-base imbalance through subsequent actions. The assessment of acid-base status in patients with OPC poisoning is crucial, as timely identification of acid-base imbalances can impact the treatment and outcome. Furthermore, it is crucial to identify respiratory failure before proceeding with endotracheal intubation and mechanical ventilation [6,7].

Material and Methods

This study is a prospective investigation conducted at Department of General Medicine, Raja Rajeshwari Medical College, Kambipura, Karnataka, India from April 2010 to March 2011 where a distinct dataset was gathered exclusively from patients who met the predefined inclusion criteria. A total of 130 patients were initially included in the study, however 20 of them were later eliminated, leaving a final count of 110 patients.

Inclusion Criteria

The study incorporated the following criteria:

- Age exceeding 18 years.
- Presence of clinical characteristics indicating organophosphorus (OP) poisoning.
- Submission of written and informed permission in the local language.
- Confirmed consumption of organophosphorus compound (OPC) as prescribed.

Exclusion Criteria

The study excluded the following criteria:

- Any instances of consuming a combination of substances.
- Age under 18.
- Consumption time exceeding 24 hours.
- Discharge without medical guidance and lack of follow-up.
- Other routes of organophosphate poisoning outside from oral ingestion, such as inhalation.

Results

Table 1: Comparison ABG analysis and ICU stay

Comparison	Mean	Standard deviation	<i>P-</i> value
Metabolic acidosis (24) versus metabolic alkalosis (3)	11.85	4.26	0.002
	5.26	0.89	
Metabolic acidosis (24) versus respiratory acidosis (11)	11.95	4.15	0.565
	11.97	3.56	
Metabolic acidosis (24) versus respiratory alkalosis (30)	13.21	4.08	< 0.002
	659	3.04	
Metabolic acidosis (24) versus mixed acidosis (3)	13.19	4.06	0.135
	10.21	2.56	
Metabolic acidosis (24) versus normal (29)	11.67	4.56	< 0.002
	5.67	2.96	
Respiratory acidosis (11) versus normal (29)	11.93	3.57	< 0.002
	4.37	1.69	
Respiratory alkalosis (30) versus normal (29)	6.34	3.27	0.289
	5.23	2.56	
Mixed acidosis versus normal (29)	10.35	1.96	0.004
	3.95	3.11	
Respiratory acidosis (11) versus respiratory alkalosis (30)	13.49	4.19	< 0.002
	5.12	2.58	
Respiratory acidosis (11) versus metabolic alkalosis (3)	11.65	4.19	0.002
	5.29	0.68	

ISSN:0975 -3583.0976-2833 VOL 02. ISSUE 01. 2011

Metabolic acidosis (24) versus respiratory alkalosis (30)	11.94	2.58	< 0.002
	6.56	3.28	
Respiratory alkalosis (30) versus metabolic alkalosis (3)	7.58	3.18	0.728
	5.18	0.84	

Discussion

Diagnosing a patient with a favorable medical history, a distinct smell on their breath, specific symptoms, and reduced levels of cholinesterase in their red blood cells and plasma is not a challenging task. Regrettably, history is frequently inaccessible. In addition, the clinical manifestations of OP poisoning may go unnoticed if the patient exhibits symptoms such as heart block, gastroenteritis, convulsions, or ketoacidosis. Recognizing this variety of manifestations is the initial stage towards making a precise diagnosis [7,8].

This leads to a decrease in the breakdown of acetylcholine and subsequently causes a significant increase in cholinergic symptoms. These symptoms disrupt the transmission of signals in both the central and peripheral nervous systems. The surplus amount of synaptic acetylcholine triggers the activation of muscarinic receptors and subsequently inhibits or immobilizes the nicotinic receptors. Disrupted neuromuscular communication, facilitated by nicotinic receptors, can lead to the accumulation of carbon dioxide and disrupt the balance of acid and base. Liu *et al.* conducted a retrospective review of patients with OP poisoning and discovered a clear link between the severity of poisoning and death, as well as the presence of pre-treatment of metabolic and respiratory acidosis ^[8].

Therefore, this investigation demonstrated that acidosis in patients with OP poisoning was linked to higher morbidity rates among the several acid-base abnormalities observed in the initial ABG. Metabolic acidosis is the primary cause of increased morbidity and mortality in individuals with acidosis, followed by subsequent respiratory acidosis. OPC poisoning is associated with a significant increase in morbidity and mortality, with mixed acidosis being the third most common contributing factor. Metabolic alkalosis and pulmonary alkalosis were linked to reduced morbidity, and no fatalities were documented in these instances [9, 10].

Metabolic acidosis is a common consequence of OP poisoning and has been identified as a significant factor in determining the patient's outcome. Nevertheless, the occurrence of severe metabolic acidosis that does not respond to typical treatments such sodium bicarbonate is infrequently recorded and always suggests a negative prognosis. Four cases of severe metabolic acidosis and hypotension that were unresponsive to catecholamines can be illustrated using examples [11].

The majority of the patients arrived to the Emergency Department (ED) within a time frame of 1 to 4 hours after ingesting the substance. The majority of them had undergone stomach lavage during prehospital care. ABG, serum cholinesterase levels, electrolytes, and other normal biochemical examinations were conducted in all instances. The levels of serum cholinesterase were predominantly reduced in all instances, indicating a likelihood of organophosphate poisoning. A sample of arterial blood was collected for the purpose of analyzing its acid-base composition. Approximately 71% of patients exhibited acid-base imbalances, while only 29% had ABG findings within normal ranges. Moulali *et al.* found that out of the 20 patients studied, 85% had acid-base imbalance while 15% had normal ABG results [12].

Conclusion

The current study emphasizes the significance of arterial blood gas (ABG) analysis as a valuable tool for promptly evaluating the prognosis of patients with organophosphate (OP) poisoning. This study demonstrates that the initial arterial blood gas (ABG) analysis conducted on patients who have been exposed to organophosphate (OP) poisoning can be used to assess the patients' prognosis even prior to hospitalization. Since this study concludes that acidosis (mostly metabolic acidosis) is linked to higher rates of illness and death. Consequently, the treatment can be escalated and executed promptly without any procrastination.

Funding support: None.

Conflict of interest: Nil.

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. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet. 2008;371:597-607.
- 3. Eddleston M, Phillips MR. Self-poisoning with pesticides. BMJ. 2004;328:42-4.
- 4. Banerjee I, Tripathi S, Roy AS. Clinico-epidemiological characteristics of patients presenting with organophosphorus poisoning. N Am J Med. Sci. 2012;4:147-50.

ISSN:0975 -3583.0976-2833 VOL 02. ISSUE 01. 2011

- Keservani, R. K., Kesharwani, R. K., Vyas, N., Jain, S., Raghuvanshi, R., & Sharma, A. K. Nutraceutical and functional food as future food: a review. Der Pharmacia Lettre, 2010a:2(1), 106-116.
- 6. Keservani, R.K., Kesharwani, R.K., Sharma, A.K., Vyas, N., Chadoker, A. Nutritional Supplements: An Overview. International Journal of current pharmaceutical review and research, 2010b: 1 (1), 59-75.
- 7. 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.
- 8. 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.
- 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.