Original Article

Study of acid base imbalance in organophosphorous poisoning patients

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Abstract

Introduction: In Asian countries the most frequently used pesticide in agriculture field and industrial settings is organophosphorous. The morbidity and mortality from acute OP poisoning is attributed to respiratory failure. In such patients acid base imbalance is commonly seen. **Aims and Objectives:** To study acid base imbalance in organophosphorous poisoning patients. **Materials and Method:** Acid base imbalance was determined by Arterial Blood Gas Analyzer reports from three variables PH, PCO2 and HCO3. **Results:** Patients included in this study, belong to age group 25- 45 years. Total 50 patients were studied. Majority of them were having acid base disturbance with marked low levels of cholinesterase. On ABG reports 58% of cases were showing respiratory alkalosis, 22% respiratory acidosis, 8% Metabolic alkalosis, 2% Metabolic acidosis and 10% showed normal acid base balance. **Conclusion:** Acid base imbalance was seen in organophosphrous poisoning patients with low levels of cholinesterase level and its interpretation can help in early recovery by appropriate respiratory ventilation to Organophosphorous poisoning patients.

Keywords: Organophosporous poisoning, Cholinestrase, Arterial Blood Gas, Respiratory Alkalosis, Respiratory Acidosis, Metabolic Acidosis, Metabolic Alkalosis.

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INTRODUCTION

Organophosphorous compound is a part of pest control and have been used worldwide for more than 60 years as insecticide, herbiside, fungicide in agriculture field and household garden. Accidental organophosphorous poisoning is common in farmers and also in suicidal attempts and rarely homicide, as it is readily available in market everywhere and easily accessible with rapid onset of action. Poisoning occurs through oral, inhalational, transcutanious routes which causes adverse effect on tissues, organ function, central nervous system, cardiovascular, urogenital system as well as neuromuscular junction and respiratory system which is the major cause of mortality in organophosphorous poisoning. Over 3 million people die every year due to organophosphorous poisoning in developing countries and fatality rate is rising year by year. The morbidity and mortality from acute OP poisoning is attributed to respiratory failure, by affecting the central and peripheral nervous system, and causing collapse of the respiratory system. Acute OP exposure produces a range of clinical findings related to increased acetylcholine levels centrally and peripherally acting on post-junctional receptors. such as central apnoea are life threatening if not treated immediately. Many of the clinical effects of acute OP poisoning have the potential to cause respiratory dysfunction, and respiratory failure, but the exact mechanism of respiratory failure has not been determined.[1] A number of synergistic mechanisms have the potential to contribute to decreased ventilation post OP exposure. Acutely, peripheral acetylcholine at the neuromuscular junction induces fasciculation and weakness of voluntary muscle groups (including those involved in respiration) and necrotic lesions within skeletal muscle cells. Exposure of the neuromuscular junction to an OP results in dysfunction. Alternatively,

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OP agents could cause a central respiratory depression or central apnoea.[2] In our institute, such O.P. poisoned Patients with respiratory failure are kept under observation and regular Arterial Blood Gas reports are strictly monitored to evaluate acid base disturbances and improve alveolar ventilation. Thus considering the importance of Arterial Blood Gas analysis, we are trying to study acid base imbalance in o. p. poisoning patients with the help of ABG report. Despite several parameters available in the arterial blood gas report, acid base disturbances can be determined by using three variables pH, $PaCO_2$, and HCO_3. These three parameters are sufficient to interpret of acid base imbalance. Arterial Blood Gas analysis represents the ultimate test in clinical practice for the evaluation of severity and causes of Lung gas exchange abnormalities using arterial oxygen tension (PaO_2) and arterial carbon dioxide tension ($PaCO_2$) and also Acid base disturbances using pH, $PaCO_2$ and serum biocarbonate (HCO_3). Arterial blood gas analysis is the sampling of blood from an artery for the determination of oxygen (O_2), carbon dioxide (Co_2) and acid content.[3]

MATERIALS AND METHODS

This study was conducted at Dr Shankar Rao Chavan Government Medical College, Vishnupuri, Nanded, Maharashtra. This study was approved by our Institutional Ethical Committee. Organophosphorous poisoning patients who were admitted in Intensive care unit and Casualty of Dr S.C.GMC and hospital, were taken for the study. In this study total 50 patients were included, out of them 40 were male while 10 were female, belonging to age group 25-45 years. Pateints history and clinical condition was carefully reviewed. Written informed consent from all the patients was taken. The diagnosis of organophosphorous poisoning was based on clinical manifestation, presentation, and history of exposure. Routine biochemical investigation like random blood sugar, urea, creatinine, liver enzymes, serum electrolyte done in our laboratory was recorded as data. Serum cholinesterase level in patients was estimated by using biolab kit method on Erba chem 7, Semi auto analyser with a normal reference value of 3500-12000 IU/L. Serum sample was collected immediately at the time of admission of patients, before administration of pralidoxime and starting treatment. Acid base balance was studied using "Arterial Blood Gas Analyzer, Cobbas b 221". 2 ml Arterial blood sample was collected prior to therapy by direct arterial puncture, in a 2 ml heparinised syringe from either radial or dorsalis pedis artery, rarely femoral artery was used. Arterial blood gases were analyzed from parameters pH, PCO_2, HCO_3. The results were recorded.

Inclusion Criteria

Patients with organophosphorous poisoning immediately after admission to our hospital and before starting any kind of treatment. Cases were included irrespective of gender. Patients with consent were included in this study.

Exclusion Criteria

Patients with poisoning other than organophosphorus. Patients who administered pralidoxamine and atropine or started treatment before taking sample.

RESULTS

Total 50 organophosphorous poisoning patients admitted to Dr S.C. Government Medical College and Hospital were taken for study. Out of them 40 (80%) were male and 10 (20%) were female.

Table 1: Mean value of cholinesterase level in study group is represented as mean ± S.D

	Organophosphorous poisoning patients
Mean of serum Cholinestrase level (IU/L)	1756.4 ± 915.3

Mean of cholinestrase level is 1756.4 with S.D. 915.3.

Table 2: Data showing variation in acid base imbalance in our study group

	Patients	Average Percentage
Respiratory alkalosis	29	58%
Respirator acidosis	11	22%
Metabolic alkalosis	04	8%
Metabolic acidosis	01	2%
Normal	05	10 %

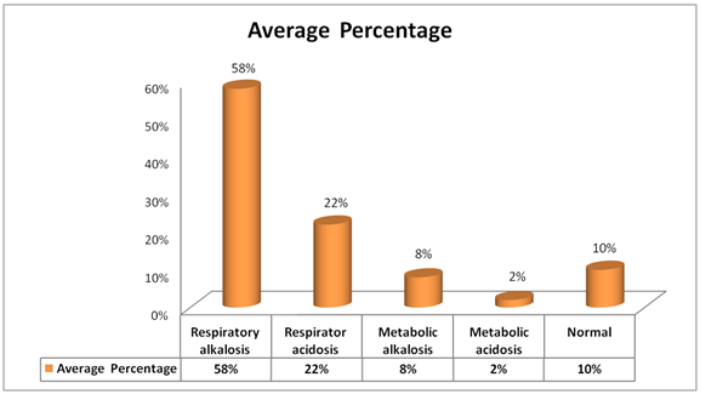

Table 3: PH, PCO_2 And HCO_3 in Primary Acid Base Abnormality are as follows.[4]

ACIDOSIS	ALKALOSIS
PH < 7.35	PH > 7.35
HCO_3 <22mmols/L(metabolic)	HCO_3 > 26 (metabolic)
PCO_2> 45 mmHg (respiratory)	PCO_2< 35mmHg(respiratory)

Serum cholinesterase level was significantly low in all o.p.poisoning patients taken for study. Out of total 50 patients taken for study, 89% patients had acid base disturbances and only 10% patients indicated to have near

normal acid base balance. Respiratory Alkalosis and Acidosis in patients was confirmed as per table no.3. Maximum arterial blood gas reports showed lower partial pressure of oxygen and inadequate ventilation. About 29 patients (58%) confirmed as Respiratory alkalosis, as they showed *pH towards alkaline side*, *decrease in PCO_2 and normal levels of biocarbonate.* 11 patients (22%) showed respiratory acidosis *with pH towards acidic side*, *increase in PCO_2 and slight increase in HCO_3.* 4 patients(8%) showed metabolic alkalosis by indicating *high biocarbonate*, *low pH* while 1 patient (2%) showed metabolic acidosis indicating *low bicarbonate and acidic pH.* Surprisingly, 5 patients (10%) were having *near to normal* acid base balance.

DISCUSSION

In our study, we tried to asses acid base imbalance in organophosphorous poisoning patients. In the study group, serum cholinesterase level was evaluated immediately on admission and low levels of serum cholinesterase was recorded. Out of 50 patients, most of the patient who were having cholinestrase level between 1501-1900 IU/L, recovered by appropriate treatment with pralidoxamine and atropine and proper adequate mechanical ventilation. While patients who were having very low cholinesterase level between 801 and 1501 IU/L needed more attention towards mechanical ventilation and correction of more and more acid base disturbances. Organophosphorous compounds that bind to acetyl cholinesterase block the conversion of acetylcholine to its degradation products, namely, acetic acid and choline. This leads to a build-up of excessive acetylcholine at synapses, which is the primary cause of most of the toxic effects of Organophosphorous compounds.[5] In such type of poisoning, death is often due to increased pulmonary secretions and inadequate ventilation and respiratory failure.[5] Time of death after a single acute exposure may range from less than 5 minutes to 24 hours depending on the dose, route of exposure and other factors. The cause of death primarily is respiratory failure usually accompanied by secondary cardiovascular complication.[6] Eddleston *et al*, proposed that, mechanism of early respiratory failure during the acute cholinergic crisis in humans is unclear but is likely to involve three components Depression of central respiratory drive from respiratory failure in ventrolateral medulla, respiratory muscle weakness and pulmonary effects (bronchospam and bronchorrhoea)[7] According to Ganendra *et al*, Clinical management of organophosphorous poisoning primarily depends on treatment of bronchial secretions, which can overwhelm the respiratory function of the patients and lead to a death which is often characterised as 'drowning on dry land'[8] From these theories we can say that, o.p.poisoning produces cholinergic crisis by inhibition of acetylcholinesterse in central and peripheral nervous system, with a wide range of clinical effects, inducing central apnoea, pulmonary bronchoconstriction and secreations, seizures and muscle weakness. All these clinical effect, results in flaccid and paralysed muscles, which ultimately leads to respiratory failure and acid base balance disturbance. In O.P.poisoning, Organophosphorous compound inhibits acetylcholinesterase activity, increases the accumulation of acetylcholine in the synaptic gap, decreases degradation of acetylcholine, thus leading to excessively increased cholinergic symptoms. Which disturbs neurotransmission of central and peripheral nervous system. This excess synaptic acetyl choline 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.[9,10,11] A Retrospective analysis of Organophosphorous poisoning patients, carried by Liuon JH *et al*, found a direct correlation between the severity of poisoning and mortality and the presence of pre-treatment of metabolic and respiratory acidosis[12]. This study indicates that management in organophosphorous poisoning case, focuses mainly on acid base disturbances and its complications. Respiratory Acidosis is frequently encountered complication of o.p.poisoning and it was previously suggested that both the degree and type of acidosis can be a predictor of outcome in O.P.poisoning[13] The exact cause of alteration in PH, PCO_2, and biocarbonate values in O.P. poisoning patients is yet unclear. More Reasearch is needed to understand the correlation between o.p.poisoning and acid base alteration leading to respiratory failure.

CONCLUSION

Thus our study concludes that in organophosphorous poisoning patients, with low cholinesterase level, a huge variation is seen in acid base balance. Most commonly respiratory alkalosis and acidosis is seen in these patients, however with proper and adequate ventilation they managed to survive. While Metabolic alkalosis and acidosis proved serious and fatal condition that required rapid diagnosis and treatment, along with clinical correlation in few O.P. poisoning patients. Thus maintenance of acid base imbalance is one of the prime requisites of life and any variation in it may seriously disturb the vital processes and may lead to death in O.P. poisoned patients. Hence more studies are required to explain this variation in detail in o.p. poisoning. Early recognition of this complication, it's careful monitoring and providing appropriate respiratory compensatory

management may help to minimise mortality in o.p.poisoing patients due to respiratory failure.

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