www.jmscr.igmpublication.org
Impact Factor 5.84
Index Copernicus Value: 83.27
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v5i7.256

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Aluminium Phophide Poisonoing: Can Acidosis Predict Mortality? A Study in North Bihar

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Abstract
Aluminium phosphide poisoning has emerged as a major problem in rural India. No specific antidote is available. This study was conducted to evaluate relation between metabolic acidosis and outcome in terms of mortality.
Materials and Methods: *An observational study was done on patients admitted with ALP (Aluminium phosphide) poisoning in DMCH, Bihar. Diagnosis was done clinically and biochemically. Clinical variables and outcomes were recorded and statistically analysed using appropriate test.*
Results: *Metabolic acidosis was significantly associated with mortality and higher duration of hospital stay.*

Introduction

Agrochemical poisoning is a major public health problem in our country due ro low education and poor regulatory frameworks. Aluminium phosphide is a solid fumigant pesticide, marketed in India as tablets of celphos and quickphos. Aluminium phosphide has aroused interest with increasing number of cases in the past three decades due to increased use in agricultural and non-agricultural purpose. In India, this poisoning was not known before 1980. The first case in India was reported in 1981 from M.G.M. Medical College, Indore. The incidence of the poisoning has been increasing steadily. Although the percentage of poisoning with AlP is low, but the mortality rate of this kind of intoxication is very high. Overall mortality in cases of aluminium phosphide poisoning varies between 37–100%. The wide variability in survival rate and inability to find out effective antidote aroused interest in search of predictors of outcome.

Background of study: Aluminium phosphide is an efficacious and easy to use and freely available rodenticide in India in form of chalky white or brown 3 gm. tablets containing 56% of ALP and 44% of ammonium carbonate. The tablets are taken out of sealed container and placed on stored grains and storage container is closed for few days to combat moles and vermines in granaries. ALP has relatively high vapour pressure that allows it to penetrate porous material effectively. On coming into contact with water or moisture or OH radical of air or hydrochloric acid in stomach,3 gm. tablet of ALP liberates I gm. of phosphine or phosphorus hydrogen, i.e., ALP + H20 = ALP + 3H20 = AL(OH)3 + PH3,ALP + 3HCL = ALCL3

+ PH3 Phosphine is a colourless gas with fishy or garlic odour. Irrespective of routes of exposure, the inhalational, ingestional or ocular, the toxic effects of PH3, are same .Some of ALP is directly absorbed from stomach to reach liver to liberate PH, slowly to prolong the toxic effects of poisoning. It is rapidly absorbed from stomach or lungs by simple diffusion, oxidised slowly and is excreted in urine as hypophosphite and also excreted unchanged through lungs significantly. PH3 inhibits the electron transport resulting from preferrential inhibition of cytochrome oxidase leading to respiratory chain inhibition which leads to cellular hypoxia and small vessel injury which is further potentiated by cardiotoxicity due to anoxic myocardial damage and shock. Direct toxic effect of ALP leads to arrhythmias. Hypotension and shock ensue within 3-6 hours of ingestion of ALP. In survivors, the cardiotoxicity and hypoxia disappear within 5-7 days due to excretion of PH3, and restoration of normal cellular metabolism. The toxic chemical myocarditis leads to varied fatal ECG changes. The non-fatal ECG changes appear within 12 to 24 hours in survivors and disappear within 56 to 80 hours. Death in first 24 hours appears to be cardiogenic as evidenced by shock and ECG abnormality. Since the survivors show complete normal ECG recovery, it denotes that the effect of poisoning is due to some reversible factor leading to disturbance in the permeability of sodium, potassium, calcium and magnesium ions leading to change is transmembrane action potential due to focal myocardial involvement and subsequent myocardial necrosis. The peripheral circulatory failure (PCF) due to wide spread small vessel injury leads to peripheral vasodilatation leading to shock. Direct toxic effects of PH3, on adrenal cortex accompanied by decreased cortisol levels, leads to shock and high mortality. Injury to alveolar capillary membrane by PH3, while being inhaled, leads to ARDS (Adult Respiratory Distress Syndrome) which may also occur rarely in ingestion. Wide spread capiIlary damage leads to bleeding diathesis, disseminated intravascular coagulation (DIC) and acute tubular necrosis (ATN). Shock and DIC lead to terminal renal failure. The clinical features are more or less the same irrespective of the mode of toxicity, except the initial symptoms pertaining to the route of entry. Ingestional Toxicity manifests asi) mild: Nausea, vomiting, headache, abdominal pain and discomfort. These patients usually recover. (ii) Moderate and Severe Systemic Manifestations: gastrointestinal System: Nausea, vomiting, diarrhoea, pain epigastrium, retrosternal pain and epigastric burning sensation, hepatobiliary System: Acute hepatic failure ,jaundice, hepatitis and soft tender hepatomegaly. cardiovascular System (60-100'%) : Increased JVP, feeble heart sounds, S3 gallop ,hypotension, shock, arrhythmias, myocarditis and pericarditis ,respiratory System (within 2 to 3 hours) : Cough, dyspnoea, cyanosis, bilateral basal rales and rhonchi, respiratory failure and ARDS, (24) rrenal System: Acute (oliguric or nonoliguric) renal failure, central Nervous System : Headache, dizziness, diplopia, paraesthesias, ataxia, altered sensorium, restlessness, intention tremors, convulsion, hypoxic encephlopathy, coma and delayed haemorrhagic stroke muscular System: Muscle pain, severe muscle weakness, myopathy with muscle wasting and proximal muscle weaknes, haemopoietie System: Bleeding diathesis, DIC and jaundice endoerinal system: hypoglycemia and hyperglycemia,. Bad Prognostic Signs include intractable shock, anemia, chest infection, metabolic acidosis, severe hypoxia, electrolyte disturbances, arrhythmias, oliguria, aspiration pneumonia, haemolysis, coma and DIC.

Objectives

To study the predictive ability of metabolic acidosis in AlP poisoning.

Methods

Information of all patient admitted with AIP poisoning to the DMCH from September 2015 to September 2016 were collected and reviewed.

Only patients who were hospitalized were included in the study. Information regarding age, cause of intoxication, amount of AIP consumed, route of exposure, therapeutic intervention and laboratory tests including arterial blood gas (ABG), electrocardiogram (EKG), and outcome were obtained. The factors of positive history of ingestion, symptoms compatible with AlP ingestion and chemical test for phosphine positive in gastric aspirate in combination used for diagnosis. All patients were managed with supportive care .Data were analysed with appropriate statistical analysis.

Results

60 patients with aluminium phosphide poisoning were studied in this study. 24(40%) patients survived with a mortality rate of 60%. (figure 1)

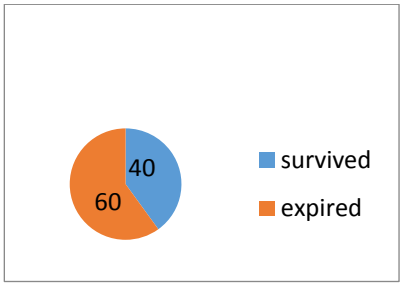

Figure 1

The mean age in the study group was 23.46+/-4.6 years. There was no significant difference in age among survivors and non survivors. (table1)

Table 1:

Mean age (in years)	Survived (mean+/-SD)	Non survived (mean+/-SD)	P value
	23.78+/-2.6	24.64+/-3.1	>0.05 Not significant

The mean intake of tablets were 3.9+/-0.05.There was no significant difference of ingested dose among survivors and non survivors.(table 2)

Table 2:

Mean intake of tablets (in numbers)	Survivor(mean+/-SD)	Non survivor (mean=/-SD)	P value
	3.41+/-1.72	3.55+/-1.24	0.39 (not significant)

In our study, metabolic acidosis was diagnosed in 40 (66.66%) patients.

Table 3: Distribution of patients according to arterial pH.

pH	Number of patients
≥7.35	20
7.1-7.34	24
<7.1	16

Table 4: Mortality in relation to arterial pH

pH	Mortality	
≥7.35	6(0.3%)	P value=<0.001 (significant)
7.1-7.35	16(66.66%)	
<7.1	14(87.5%)	

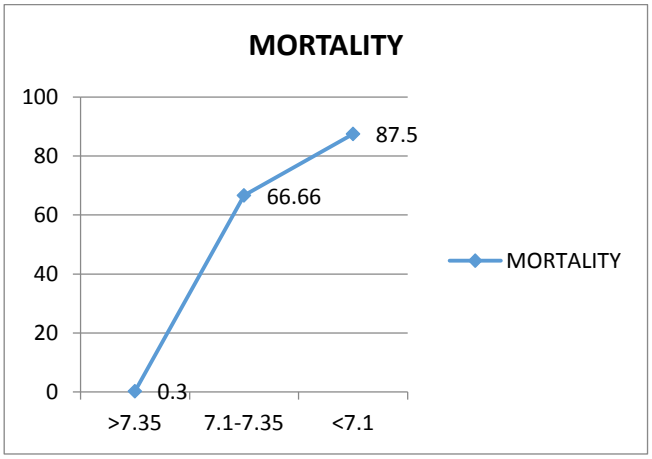

Fig 2: Mortality in relation to Arterial blood pH

Duration of hospital stay was also higher in patients with metabolic acidosis who survived. (Table 4)

pH	Hospital stay in days (Mean±SD)	
≥7.35	1.85+/-0.89	P value= <0.001 (significant)
7.1-7.34	3.57+/-1.90	
<7.1	10.2+/-4.20	

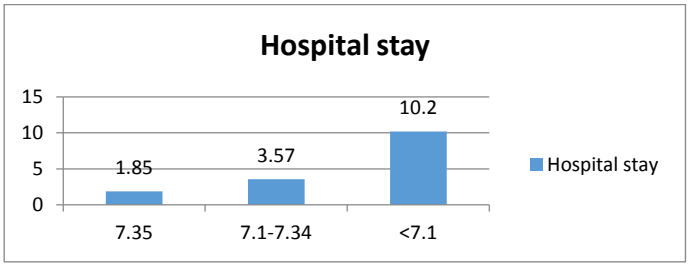

Figure3: Hospital stay in relation to blood pH

The average mean arterial pressure on arrival was 53.73 ± 17.96 mm Hg. It was significantly high among survivors. (table 5)

Table 5:

Mean arterial pressure (mm of Hg)	survivor	Non survivor	P value
	73.91+/-2.77	40.27+/-8.15	<0.0001 (highly significant)

Clinical manifestations at time of admission involved different systemic manifestations. They include nausea and vomiting (83.33%), dyspnoea and palpitations (40%), cyanosis (16.66%) hypotension (80%) and shock (60%). Cardiac arrhythmias were present in (53.33%) cases.

Discussion

The aluminium phosphide is a highly lethal poison with no definite antidote till now. The LD50 dose of ALP is 10 mg/kg of body weight. The specified fatal dose is 0.15-0.5 gm. However, most of the patients present with ingestion of three or more tablets which invariably results in death. For a 70 kg man 0.5 gm. ALP is lethal. The exact mechanism of action of aluminum phosphide poisoning is still unknown. A three-gram tablet can release almost one gram of phosphine gas in contact with water. After ingestion, phosphine will be released due to contact between AlP and water/acid in the gastrointestinal (GI) tract. Some phosphide may be absorbed by the GI tract without hydrolysis and convert into phosphine. The mortality varies in different studies from 37-100%.In some studies no significant correlation was found between the dose of aluminium phosphide ingested and outcome. In our study we also found no significant difference between quantity of tablets consumed. In a retrospective study of seven years in Teheran, Shadnia S et al found significant association between mortality and arterial pH. In their study, 100% patients with blood >7.35 survived, whereas 100% patients with pH<7.1 died. In our study, we also found significant association between acidosis and outcome of poisoning.

Conclusion

Aluminium phosphide poisoning is a great health hazard in developing country like India due to easy and wide availability. The exact mechanism of action of aluminum phosphide and an effective antidote of this poison is still unknown. But we assume that contact with water prior ingestion may predict a better outcome.Further studies in this field is needed.

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