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Profile of patients with acute aluminium phosphide poisoning in a tertiary care institute of Haryana, India

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Abstract

The Pesticides have proved to be a boon to the farming community to save the grains from pests and rodents etc. Aluminium phosphide (AIP), a solid fumigant pesticide is widely used as a grain preservative in Northern India. The mode of poisoning is usually intentional, occasionally accidental and rarely homicidal. The agriculture community is more at risk due to illiteracy and easy availability of the pesticide in the house hold. Forty five patients of aluminium phosphide poisoning irrespective of age, sex, dose and duration, admitted in Medical College, Rohtak were studied. The patients were grouped in I,II, IIa, IIb. Data was collected on a semi structured schedule and consent was taken. Appropriate statistical tests were applied. It was found that the poisoning was more common in younger age group. 22 (73%) patients out of 30 in group-I and 14 (93%) patients out of 15 in group-II fell in age group between 14-30 years. The poisoning was more common in males than in females. Male to female ratio was 2:1. The earliest symptoms and signs pertain to gastrointestinal tract followed by cardiovascular system.

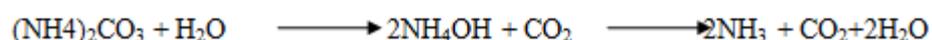
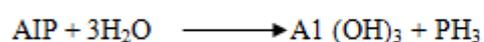
Keywords: Patients, Aluminium phosphide, poisoning

Introduction

The Pesticides have proved to be a boon to the farming community to save the grains from pests and rodents etc. Aluminium phosphide (AIP), a solid fumigant pesticide is widely used as a grain preservative in Northern India. It is available in India in tablet form with the brand names of Celphos and Quickphos in air tight container. Each tablet contains 56% aluminium phosphide as an active ingredient and 44% ammonium carbonate and is 3g by weight, has capacity to liberate 1g of phosphine gas on contact with moisture or humidity present in grains or in atmospheric air. Phosphine being gaseous in nature, diffuses uniformly throughout the stored grains. It does not affect the food value of grains. After fumigation, the non-toxic residues left in the grains are phosphite and hypophosphite of aluminium. Aluminium phosphide was declared as ideal fumigant pesticide in 1973 because of its efficacy, being cheap and easy to use.^{1,2}

The mode of poisoning is usually intentional, occasionally accidental and rarely homicidal. The agriculture community is more at risk due to illiteracy and easy availability of the pesticide in the house hold. The peak season of this poisoning occurs commonly in post-harvesting period i.e. May to September every year. This poisoning has now become a major challenge to medical profession; a problem which was almost non-existent a decade ago, has now acquired epidemic proportion³.

Aluminium phosphide is a systemic poison and its toxic effect is due to liberation of toxic phosphine gas in the stomach. The reaction is as follows⁴:



The diagnosis of acute aluminium poisoning is based upon (i) history of ingestion of fresh and active compound in the form of tablet which is compact, full of texture, lusture, foul smelling and about 50 paisa coin size. (ii) (ii) Decaying fish or garlic like odour imparted to breath with presence of shock of hypotension. (iii) Positivity of silver nitrate paper test with gastric fluid or with breath or both. The positivity⁵ test of the silver nitrate paper only confirms the diagnosis of this poisoning but it cannot determine severity of the poisoning. Hence, to know the severity of poisoning the estimation of blood phosphine level is mandatory.

The clinical symptomatology is more or less the same irrespective of the mode of toxicity, except that the initial symptom pertain to the route of entry. The signs and symptoms depend on the dose and severity of poisoning. Mild inhalation exposure produces mucous membrane irritation and acute respiratory distress. Other symptoms include dizziness, easy fatiquability, nausea, vomiting, diarrhea and headache etc. More severe toxicity produces ataxia, numbness, paraesthesia, tremors, diplopia and jaundice. Very severe toxicity is accompanied by development of adult respiratory distress syndrome (ARDS), cardiac arrhythmias, convulsions and coma. Acute hepato toxicity, acuter renal damage and leucopenia occur late. At a concentration of 290-600 PPM, it is lethal within half an hour, at 100 PPM it is rapidly fatal.⁶ With this background in mind the present study to study demographic and clinical profile of patients with Acute Aluminium Phosphide Poisoning was planned.

Material and Methods

Fourty five patients of aluminium phosphide poisoning irrespective of age, sex, dose and duration, admitted in Medical College and Hospital, Rohtak constituted the subject material. The patients were grouped as under depending on active v/s exposed compound.

Group-I : This group included 30 patients of acute aluminium phosphide poisoning with following characteristic features :

- i) History of ingestion of fresh aluminium phosphide compound in the form of tablet which was compact, full of texture, lusture, foul smelling and about 50 paisa coin size.
- ii) Decaying fish or garlic like odour imparted to breath.
- iii) Presence of shock and other clinical signs and symptoms.
- iv) Confirmation of diagnosis by positive silver nitrate paper test with gastric fluid or in breath.

The diagnosis of shock in these patients were based on the presence of auscultatory systolic BP less than 90mm of Hg alongwith presence of atleast two of the features listed below.⁷

- i) Cold, moist, peripheral extremities.
- ii) Impaired state of consciousness, agitations, sommolence, confusion and coma.
- iii) Urine output less than 30ml/hr.
- iv) Metabolic acidosis.

Group-II : This group included 15 patients of acute aluminium phosphide poisoning and further sub-grouped into A and B depending upon clinical parameters and characteristics of the tablet.

Group-II A : This group included 10 patients of acute aluminium phosphide poisoning with following characteristic features :

- i) History of ingestion of old preserved tablet which was friable, textureless, lustureless and slightly foul smelling.
- ii) Presence of clinical symptoms like nausea, vomiting, pain epigastric.
- iii) Decaying fish or garlic like odour in breath may or may not be present.
- iv) Presence of mild hypotension (BP 70-90mm of Hg) only. There was no clinical evidence of shock.

v) Confirmation of diagnosis by positive silver nitrate paper test with gastric fluid.

Group II B : This group included 5 patients of acute aluminium phosphide poisoning with following characteristics :

- i) History of ingestion of old preserved compound in the form of powder.
- ii) Mild symptoms in the form of nausea, vomiting and pain epigastrium.
- iii) No decaying fish or garlic like odour in breath.
- iv) No hypotension.
- v) Silver nitrate paper test was negative both with gastric fluid as well as with breath.

Patients clinical profile were charted out on a proforma. The following investigations were carried out in each patient :

1. Routine Investigation : Haemoglobin, total and differential leucocyte count, complete urine examination.
2. Blood urea was estimated by diacetyl monoxime.⁸
3. Serum sodium and potassium were estimated by flame photometry.
4. Blood sugar was done by orthotoludine method.⁹
5. Serum creatinine by picric acid method.¹⁰
6. Liver function test :
 - a) Serum bilirubin both direct and indirect was measured by diazo method.¹¹
 - b) SGOT/SGPT were measured by Retimane and Frankel method.¹²
 - c) Serum alkaline phosphatase was done by King and Amstrong Method.¹³
7. Frequent ECG monitoring.
8. Blood Gas Analysis.
9. Portable X-ray Chest PA view

Data collection and Analysis

A pre-tested, semi-structured schedule was used for interviewing the study subjects. Written and informed consent was taken from all the subjects before initiating the interview. The confidentiality of the information was assured. Collected data were entered in the Excel spreadsheet and analysis was carried out using appropriate statistical tests. Normally distributed data were presented as means and standard deviation, or 95% confidence intervals (CI) . All tests were performed at a 5% level of significance, thus an association was significant if the p value was < 0.05.

Results / Observations

Fourty five patients of aluminium phosphide poisoning irrespective of age, sex, dose and duration, admitted in Medical College, Rohtak were studied. The patients were grouped as under:

Group – I (Severe Toxicity) : it included 30 patients of aluminium phosphide poisoning, who ingested fresh compound and received in a state of severe shock (BP < 70mm of Hg).

Group-II: It included 15 patients of aluminium phosphide poisoning and further sub-divided into group-II A and II B.

Group – II A (Mild Clinical Toxicity) : It included 10 patients, who had taken old preserved compound. They had clinical signs and symptoms. Mild hypotension (BP between 70-90 mm of Hg) was present.

Group – II B (No Toxicity) : It included 5 patients, who had taken old preserved compound in the form of powder. They had mild clinical symptoms without any hypotension.

Age and sex distribution

The patients varied in their age from 15 to 50 years with an average of 28.6 years. In Group-I, maximum number of patients were in between 21-30 years constituting 46% followed by 27% patients below the age of 20 years. Sex ratio of male : female was 2:1.

In Group-II, maximum number of patients were in between 21-30 years constituting 53% followed by 40% patients below the age of 20 years. Sex ration of male to female was 2:1.

Table-I

AGE AND SEX DISTRIBUTION OF PATIENTS WITH AIP POISONING

AGE GROUPS IN YEARS	GROUP-I PATIENTS				GROUP-II PATIENTS			
	MALE	FEMALE	TOTAL	%	MALE	FEMALE	TOTAL	%
15-20	6	2	8	27	5	1	6	40
21-30	12	2	14	46	5	3	8	53
31-40	3	3	6	20	-	-	-	--
>40	1	1	2	7	-	1	1	7

The amount of AIP ingested varied from one tablet (3.0 gm) to 3 tablets (9.0 gm) or more. The majority of patients (80%) consumed one or two tablets. The number of patients with dose of pesticides consumed in Group-I and II are shown in Table No.II.

Table II
NUMBER OF PATIENTS AND AMOUNT OF PESTICIDE CONSUMED

AMOUNT CONSUMED	GROUP-I		GROUP-II		
	NO OF PATIENTS	%	NO OF PATIENTS	%	
1 tablet	(3.0gm)	7	23.310	10	66.7
2 tablet	(6.0gm)	17	56.73	3	20.0
3 tablet	(9.0gm)	6	20.02	2	13.3
Or more					

Clinical profile

Symptoms of poisoning started within 10 minutes to half an hour of ingestion and patients were brought to hospital within 30 minutes to 5 hours (except one patient who was brought after 8 hours of ingestion). The mean time interval at admission (between ingestion and arrival to hospital) was 2.3 hours.

Patients symptom and signs were recorded on a specially designed proforma. In Group-I patients, nausea, vomiting, retrosternal burning, pain epigastrium, severe shock and garlic like odour in breath were present in all the patients. 25 patients showed restlessness without alteration in consciousness. Metabolic acidosis was present in 21 patients while 20 patients showed EKG Changes (Sinus Tachycardia, Sinus Bradycardia, Ventricular ectopics, V.T., V.F., A.F., block and ST-T changes). Two patients developed jaundice with raised serum transaminases levels.

In group-IIa patients, nausea, vomiting, retrosternal burning, pain epigastrium and mild hypotension (BP 70-90mm of Hg) were present in all the patients. Garlic like odour in breath was present in 4 patients while 2 patients showed EKG changes.

In group-IIB patients, nausea, vomiting, pain epigastrium were present in all the case without any clinical toxicity. The clinical profile of patients is depicted in table no III.

Table -III
CLINICAL PROFILE OF PATIENTS OF AIP POISONING

CLINICAL FEATURES	GROUP-I		GROUP-IIA		GROUP-IIB	
	NO OF CASES	%	NO OF CASES	%	NO OF CASES	%
1. Nausea, vomiting, burning	30	100	10	100	5	100
2. Shock (Group-I BP<70mm of Hg and for group-IIA BP 70-90mm of Hg)	30	100	10	100	NIL	NIL
3. Garlic like odour in breath	30	100	4	40	NIL	NIL
4. Rest lessness without alteration in consciousness	25	83.33	NIL	NIL	NIL	NIL
5. Metabolic acidosis	21	70	NIL	NIL	NIL	NIL
6. EKG changes	20	66.67	2	20	NIL	NIL
7. Jaundice	2	6.67	NIL	NIL	NIL	NIL

Routine investigation (Hb, TLC, DLC, Urine) Were normal . Blood urea, serum electrolytes , serum creatinine, serum transaminases (SGOT/SGPT) and serum bilirubin were also normal in all patients except two patients, which showed increased serum bilirubin and SGOT/SGPT levels. X-ray chest PA view was also normal in all patients. All patients were treated conservatively as per W.H.O recommendation (1988) and modified by Chugh et al (1992).

Discussion

The present study was undertaken on 45 patients of acute aluminium phosphide poisoning irrespective of age, sex and dose, admitted in Medical College and Hospital, Rohtak. The gastric lavage was done and medico legal formalities were completed. The diagnosis of aluminium phosphide was confirmed by silver nitrate paper test, which was positive with gastric fluid or breath or both in Group-I and with gastric fluid in Group-IIA patients while it was negative in Group IID patients. The silver nitrate paper test was found to be simple and valuable for bedside poisoning diagnosis of acute aluminium phosphide.

These 45 patients were seen over a period of eleven months. The poisoning has been found common in younger age group, 22(73%) in Group-I and 14(93%) in Group-II cases were below the age of 30 years in the present study. In both the groups about 2/3rd patients were male. These findings are consistent with the observation made by^{1,14} us as well as by other workers.^{3,15,16} The cause of higher incidence of this poisoning in younger age group may be frustration and dissatisfaction with life due to social, economic and political reason both in males and females. The incidence was higher in rural as compared to urban population. In this study, 17 (60%) patients belonged to rural area of Haryana. The higher incidence of this poisoning in areas could be due to easy availability and accessibility of this pesticide as the latter is kept freely in every household in the village. This finding has already been highlighted by us.^{1,14}

The amount of AIP ingested varied from one tablet (3.0g) to three tablets (9.0g) or more 17(56.5%) patients consumed two tablets, 7(23.%) patients consumed one tablet and rest consumed three tablets or more This observation is consistent with our own previous observations and different with others.^{3,15,16} The cause of varied intake of pesticides was not known. The symptoms of poisoning were immediate and occurred within half an

hour of ingestion and patients were brought to hospital within 30 minutes to 5 hours (except one patient who was brought after 8 hours of ingestion). The mean time interval at admission (between ingestion and arrival to hospital) was 2.3 hours. This was due to the fact that all patients came from nearby places. This poisoning emerged as the commonest suicidal poisoning in this state during the last decade¹ and in the present study the mode of poisoning was suicidal in all the cases. This finding has been reported by most of the previous workers.^{3,15,16}

The early symptoms in this poisoning pertain to G.I tract i.e. nausea, vomiting, pain epigastrium, diarrhoea etc.; followed by cardiovascular manifestations i.e. hypotension or shock, arrhythmias, conduction disturbances, myocarditis, pericarditis etc. The symptoms of liver and renal toxicity and leucopenia are not so common and occur late.¹² In the present study all patients of Group-I (100%) had nausea, vomiting, burning, pain epigastrium and garlic like odour in breath. All patients (100%) were received in a state of severe shock. 25(83.33%) patients remained conscious with clear mentation. Similar findings have been observed by us^{1,17} and other workers also.^{3,15,16} Ten patients of Group-IIA who consumed partial exposed tablets showed nausea, vomiting, burning, pain: epigastrium with mild hypotension in all the cases (100%). 4(40%) cases had garlic like odour imparted to breath. All patients in Group-IIB who consumed fully exposed compound in the form of powder had nausea, vomiting, burning and pain epigastrium without any hypotension. This finding suggest that severity of poisoning is related to the freshness and activity of the compound ingested. This finding had been recently observed by us. There is no comparable study on this finding.

Routine investigations and blood bio-chemistry, blood urea, serum electrolytes (K^+ , Na^+) serum creatinine and X-ray chest PA view were normal in all cases. Serum bilirubin and transaminases levels were normal except two patients (6.67%) who developed raised transaminases and serum bilirubin levels which came down to normal later on. Hepatic manifestations are known to occur late and disappear completely if patient survives.¹⁸

Electrocardiographic manifestations is a cardinal feature of this poisoning. This is due to development of acute cardiotoxicity induced by phosphine either due to direct effect or due to denaturation of intracardiac cellular proteins¹⁹ and peroxidation of lipids.²⁰ In the present series 20(66.67%) patients in Group-I had electrocardiographic manifestations (sinus tachycardia, sinus bradycardia, ventricular ectopics, VT, VF, AF, Block and ST-T changes). This findings is constant with previous reports.^{1,3}

Shock, a cardinal manifestation of acute AIP poisoning produces metabolic acidosis. In the present study 21(70%) patients in Group-I developed variable degree of metabolic acidosis and bicarbonate levels were found to be low in all patients. PaO_2 was found to be low in all patients but was maintained more than 75% by continuous oxygen therapy. Variable degree of metabolic acidosis due to shock have already been observed by us other workers.^{1,3,16}

Summary and Conclusions

1. The poisoning was more common in younger age group. 22 (73%) patients out of 30 in group-I and 14 (93%) patients out of 15 in group-II fell in age group between 14-30 years.
2. The poisoning was more common in males than in females. Male to female ratio was 2:1.
3. The incidence was higher in rural population than in urban population.
4. The symptoms of poisoning were immediate and occurred within half an hour. The mode of poisoning was suicidal in all the cases.
5. The dose of intoxicant was variable. Majority of patients (80%) consumed either one or two tablets.

6. The earliest symptoms and signs pertain to gastrointestinal tract followed by cardiovascular system. Other signs and symptoms included restlessness without alteration in consciousness, dyspnoea and tachypnoea. Various arrhythmias and jaundice was also seen.

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