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Outcome of Patients with Acute Aluminium Phosphide Poisoning In a Tertiary Care Hospital of Haryana

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Abstract

Background: *The only factor that determines the severity and monitors the progress of cases of AIP poisoning is blood phosphine level. The mortality is highly variable depending on the freshness and dose of the compound consumed. The present study has been designed to determine the serial levels of phosphine in blood and to correlate them with outcome of the patients.*

Materials: *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 in I,II, IIa, IIb. Data was collected on a semi structured schedule and consent was taken. Appropriate staistical tests were applied.*

Observations: *The blood phosphine levels at admission, at 12 hours and 24 hours in group-I patients were significantly higher than patients of group-II A & B, making the conclusion that group-I patients had severe intoxication and consumed active and fresh compound. In group-II A patients, blood phosphine levels at admission, at 12 hours and 24 hours were significantly lower than group-I but higher than group-II B paitents. This indicated that compound consumed by group-II A patients was still active but less than group-I. These patients had mild intoxication.*

Introduction

Aluminium phosphide (AIP), a solid fumigant pesticide is widely used as a grain preservative in Northen India. It is available in India in tablet form with the brand names of Celphos and Quickphos in air tight container. Each tablet contain 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. Aluminium phosphide is a systemic poison and its toxic effects are due to liberation of toxic phosphine gas in the stomach.

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 positivity[1] 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 only factor that determines the severity and monitors the progress of cases of AIP poisoning is blood phosphine level. The mortality is highly variable depending on the freshness and dose of the compound consumed. The safe limit of the dose of AIP has been claimed to be less than 500mg of active and fresh compound but the safe limit of phosphine in blood has not been defined.

Keeping in view the above facts, the present study has been designed to determine the serial levels of phosphine in blood and to correlate them with outcome of the patients.

Materials & 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 (Severe Toxicity): 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 along with presence of atleast two of the features listed below.[87]

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 (Mild Clinical Toxicity): 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 (No Toxicity): 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.

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.

Observations

Serial blood phosphine estimation (in mg %)was done in all the patients at the time of admission (at mean ± SEM time interval of 2.3 ± 0.21 hours) and at 12 and 24 hours after the admission.

Table 1: Comparison of serial blood phosphine levels (Mean± SEM) in group-I and group-II A&II B patients

GROUP	NO. OF PATIENTS	SERIAL BLOOD PH_3 LEVELS		
		AT ADMISSION (Mean time interval after ingestion 2.3 ± 0.21 HRS)	TIME INTERVAL AFTER ADMISSION	
			AT 12 hrs	AT 24 hrs
I	30	3.18 ± 0.49	2.7 ± 0.91	1.58 ± 0.75
II A	10	0.097 ± 0.013	0.0270. ± 017	Undetectable
II B	5	Undetectable	Undetectable	Undetectable
	p value	<0.001	<0.01	-

In group-I patients the blood phosphine levels (Mean ±SEM) were 3.18 ± 0.49 mg%, which were significantly higher (p < .001) than in patients of Group-IIA and IIB. In Group-IIA the levels were detectable upto 12 hours then became undetectable level throughout the period of observation.

Table 2: Serial blood phosphine levels (Mean± SEM) and amount of pesticide consumed in group-I patients

AMOUNT OFPESTICIDECONSUMED	SERIAL BLOOD PH_3 LEVELS		
	AT ADMISSION (Mean time interval after ingestion 2.3 ± 0.21 HRS)	TIME INTERVAL AFTER ADMISSION	
		AT 12 hrs	AT 24 hrs
1 tablet (3.00gm)	1.43 ± 0.25	1.07 ± 0.33	0.69 ± 0.31
2 tablet (6.00gm)	2.11± 0.18	1.44 ± 0.24	1.12 ± 0.42
3 tablets (9.00gm)	8.26 ± 0.82	8.04±1.58	7.13± 0
Statistical analysis			
Comparison of one Tablet with two	p< 0.05	n.s.	n.s.
Comparison of two Tablets with three	p< 0.001	< 0.01	< 0.001
Comparison of one Tablet with three	p< 0.001	< 0.01	< 0.001

Patients who consumed 3 tablets or more had mean blood phosphine levels of 8.26± 0.82 mg% which were significantly higher (p <0.001) those who consumed 2 tablets or 1 tablet. The blood phosphine levels showed steady declination as the time progressed (Fig.-1).

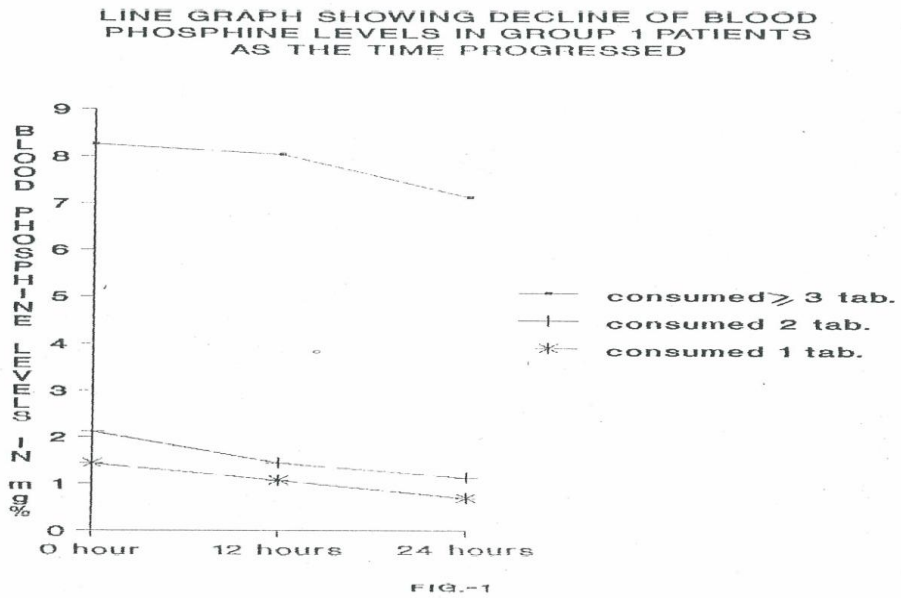

FIG.-1

Table 3: Amount of pesticide consumed and outcome of the patients

AMOUNT OF PESTICIDECONSUMED	GROUP-I			GROUP-II		
	Tota l patients	No. of died	% Patientsdied	Total patients	No. of died	% Patientsdied
1 tablet (3.00gm)	7	3	42.9	10	0	0
2 tablet (6.00gm)	17	15	88.2	3	0	0
3 tablets (9.00gm)	6	6	100.0	2	0	0

In Group-I, the morality was 100% in patients who consumed 3 or more than 3 tablets followed by 88.2% who consumed 2 tablets. The patients who consumed one tablet showed 43% mortality. In Group-II none of the patient died.

Table 4: Comparison of serial blood phosphine levels (Mean ± SEM) withpatients outcome:

GROUP	NATURE OF COMPOUND	OUTCOME	NO.	SERIAL BLOOD PHOSPHINE LEVELS		
				(Mean time interval after ingestion 2.3± 0.32 hrs)	Time Interval after Admission	
					AT 12 hrs	AT 24 hrs
I	Fresh	Died	24	3.73 ± 0.59	4.57 ± 1.37	3.20± 1.31
		Survived	6	1.067 ± 0.16	0.523 ±0.127	0.28 ±0.013
IIA	Semi-exposed	Survived	10	0.097 ±0.013	0.027 ±0.017	Undetectable
IIB	Exposed	Survived	5	Undetectable	Undetectable	Undetectable

Statistical Analysis

Comparison of Group-I died v/s survived	p< 0.001	<0.005	< 0.05
Comparison of Group-I survived v/s Group-II Survived (semi-exposed)	p< 0.001	< 0.01	-

The blood phosphine levels (mean ±SEM) were 3.73 ± 0.59 mg% in patients who died, which were significantly higher ($p < 0.001$) than patients who survived.

Discussion

The dose of fresh pesticides consumed has been correlated with the severity of poisoning in most of the studies.[2,3,4,5] These studies lacked serial blood phosphine estimation. Serial blood phosphine levels at admission, at 12 hours and at 24 hours in patients of group-I who consumed 3 tablets were 8.26 ± 0.86 mg%, 8.04 ±1.58 mg% and 7.13 ± 0 mg% respectively. The levels were significantly higher (P<.001) than in patients who consumed 2 tablets (6.0 gm) and one tablets (3.0 gm.) This finding suggested that higher the dose, higher were the serial blood phosphine levels provided compound consumed was active and fresh. Thus there was direct relationship between dose of active pesticide and blood phosphine level.

The mortality in this poisoning has been found to be highly variable in the reported studies.[6,7,8,9] In the absence of blood phosphine level, the grading of poisoning into mild, moderate and severe was not actually possible but physicians had made impression earlier on the basis of clinical experience they gained as well as dose of fresh pesticide consumed. The blood phosphine level in patients who died in Group-I (n-24) had 3.73 ±0.59mg% at admission, 4.57 ± 1.37mg% at 12 hours and 3.20 ±1.31mg% at 24 hours respectively. These levels were significantly higher than patients (n-6) who survived in Group-I. This finding indicated that survival of patient was possible upto blood phosphine level ≤ 1.067 ± 0.16mg%. Above this level blood phosphine was found to be lethal in this study (Table-4).

In patients who had mild intoxication due to exposed tablets (Group-IIA) had mean blood levels of phosphine 0.097 ± 0.013mg% at admission and then became lower at 12 hours (0.017ng%) and became undetectable at 24 hours. All the patients (n-10) in this group survived. This finding also supported above observations of safe limit of phosphine who had no clinical toxicity (Group-IIB) did not have detectable blood phosphine level and patient exposed compound had detectable level within safe limits. Such observations had not been made in the literature.

From the above discussion it has become amply clear that serial blood phosphine levels not only indicate the severity of poisoning due to fresh and active compound but had been found useful to grade the poisoning into mild and severs cases. It was also possible to define the safe limit of blood phosphine level on the basis of mortality analysis. Last but not least, this blood phosphine level differentiated very well between patients who consumed active compound (Group-I) and patient who consumed exposed compound (Group-IIA & B).

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