



A prospective study of the salient features of Aluminium phosphide poisoning from northern part of India

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Abstract

Background: Aluminium Phosphide (AIP) poisoning is extremely lethal poisoning. Ingestion is usually suicidal in intention. Phosphine, which is liberated when AIP comes in contact with moisture, is injurious and effect the cellular respiration there by it became lethal. Absence of specific antidote results in high mortality.

Aims and objectives: To study the salient features and ways to combat the deleterious effect of poisoning.

Material and Methods: One hundred and twelve cases of proved AIP poisoning by AgNO₃ Test constituted the clinical material. Each and every patient and /or relatives, friends were interrogated regarding amount of exposed or unexposed poison taken, when taken and the time when symptoms started appearing after ingestion of poison. They were submitted to relevant investigations to assess the severity of poisoning, hepato - renal and cardio respiratory status.

Results: The overall incidence of all type of poisoning was 0.29% i.e. 200 cases of poisoning out of total admission 67189. Amongst these 200 cases there was 112 cases (56%) of AIP poisoning. They were mainly from 3rd decade of life (mean age was 22.45± 5.3 years) with male to female ratio of 2.7: 1. 73.2%. Maximum were from rural area and mostly were educated from high school to Graduate level (85 cases or 75.6%). Main causes were set-back in life, unemployment, home conflicts etc. Presenting symptoms reflect the irritation of the mucous membrane of GIT, presented with nausea, vomiting (100%) increased thirst (71.1%), pain in abdomen (42.8%) with altered bowel habits, respiratory distress (23.2%) altered consciousness, dizziness (33.9%) and Cardio Vascular abnormalities (53.5%) in the form of tachycardia profound shock. Laboratory findings reflect the severity and prognosis of poisoning. Leucopenia was reported in 4.4% cases, two to three fold increase in serum transaminases (22.3%), hyperbilirubinemia (5.3%) and raised level of blood urea and serum creatinine in 32.1 of cases, hypomagnesemia was present in 16.18% of cases. Mortality in present study was 42.8%.

Conclusion: Good prognosis depends on the earliest the hospitalization with effective measures taken to combat the shock and promote the rapid excretion of poison through urine and gastro intestinal tract.

Keywords: aluminium phosphide, phosphine shocks, renal perfusion, renal and cardiac status

Introduction

The past 3 to 4 decades have seen a remarkable change in the incidence and type of poisoning. A number of new compounds have been added to the list of potentially poisonous materials. Poisoning is the fourth most common cause of mortality in rural India [1-4].

Aluminium phosphide (AIP) an extremely lethal poisonous compound is a solid fumigant, which has been in extensive use since the 1940 [5-7]. It has rapidly become one of the most commonly used grain fumigants because of its properties which are considered to be near ideal.

It is toxic to all stages of insects highly potent and does not affect seed bioavailability as well as free from toxic residues. [7-12]. Acute poisoning is on the rise in many countries and similar trend has been observed in India [12-14].

AIP is now a day's more common because of its easy availability and widely used as grain preservatives at homes and in ware houses [14-18]. It becomes poisonous by liberating phosphine (PH₃) gas after coming in contact with moisture PH₃ affects all the body systems and proves fatal [19-23].

Hence in present study we tried to study the salient features

and ways to combat the deleterious effect of poisoning.

Materials and methods

Present prospective study was performed on cases of poisoning suspected or confirmed admitted in the Nehru Chikistisalya B.R.D. Medical College, Gorakhpur during the period of March 2017 to February 2018.

All of them and /or their relatives and accompanying person were interrogated for the detailed history regarding, poison ingested, its amount, time, whether sealed or exposed and any poison material seen on the body. Any symptom appeared when and severity in the form of nausea and vomiting, pain in abdomen and diarrhea and efforts were made to know the cause of poisoning. Thorough clinical examination was performed specially for vital signs, level of consciousness pupils and systemic examination to reveal any involvement of the system.

The gastric lavage with potassium permanganate (KMNO₄, 1: 10000) two to three times was performed and gastric aspirate was preserved. After gastric wash 100 grams of activated charcoal was administered along with medicated liquid

paraffin just to absorb PH₃ and enhance excretion through GIT by purgation. Adequate hydration and renal perfusion was maintained with IV fluids and vasopressors (Dopamine and /or Dobutamine). Efforts were made to combat shock and maintain systolic BP around 90 mm Hg.

Oxygen was given to combat hypoxia. Magnesium sulphate 1 gm in 100 ml of 5% dextrose hourly for 3 to 6 hours than 6 hourly for 5 to 6 days in 62 cases and their result of efficacy was compared with 50 cases who had not given Magnesium.

The diagnosis was established with Silver Nitrate (AgNO₃) test in gastric aspirate from breathing air and urine which turns the Ag No 3 black if AIP (Celphos) is taken.

Subjects were subjected to routine examination of blood for hemoglobin, total and differential leucocytes count, general blood picture. Urine examination in detail especially for the presence of RBC, pus cells etc. Screening and / or X-ray chest and 12 leads standard E.C.G. Serum was subjected to liver and renal function tests along with blood sugar, serum electrolytes and serum magnesium level.

All the data analysis was performed using IBM SPSS ver. 20 software. Quantitative data was expressed as mean \pm standard deviation (SD) whereas categorical data was expressed as percentage. Cross tabulation and frequency distribution was used to prepare the table and Microsoft excel 2010 was used to prepare the required graph. Level of significance was assessed at 5% level.

Results

Out of 67189 total admissions at study place, 200 (2.9%) were of poisoning. Of these 200 cases, 112 cases (56%) were of aluminium phosphide (AIP) poisoning whose diagnosis was confirmed by AgNO₃ test. The incidence of AIP poisoning amongst total admissions was 0.16%.

There was 112 (56%) of Aluminium Phosphide poisoning followed by 40 cases (20%) of organophosphorus poisoning and rest of them were of different poisoning/over dosage of

material etc. There age ranged from 12 to 35 years (mean age of 22.45 ± 5.3 years) with male to female ratio of 2.7:1.

Out of 200 cases, 102 cases (91%) were of suicidal and 4 were accidental cases. Eighty two of them (73.28%) were from rural area and students (46.4%).

Sixty eight (60.7%) were unmarried of whom 54 were males (48.20%). Many of them were educated from high school to graduate level. (84 cases, 75%) and some of them had family conflict (33.9%), failure in examination and unemployment, (26.88%).

Maximum patients' nausea and vomiting with increased thirst (71.4%) and pain in abdomen (42.8%). Respiratory system revealed increased respiratory rate, crepitating and rhonchi mainly at bases. This was progressing to adult respiratory distress syndromes (23.2%). Twenty cases (21.4%) were drowsy and 18 of them (16.9%) were unconscious. Eighty four cases (75%) were in shock with systolic BP < 90 mm Hg and pulse was not recordable in 20 cases (17.8%).

Anemia was mild (10- 12 gm%, 12 cases) to moderate (8 to 10 gm%, 8 cases) degree. Leucopenia indicating towards the severity of toxicity was noted in 5 cases (TLC= $3100 \pm 280.5/c$ mm). Altered renal function in the form of raised blood urea (>40 mg%, 92.6 ± 20.7 mg %) and serum creatinine (> 1.5 mg%, 2.8 ± 1.6 mg %) was observed in 36 (32.1%) and 35 cases (32.1%) respectively.

Similarly raised level of transaminase i.e. SGOT > 40 IU (68.9 ± 10.6 I.U.) and SGPT > 40 IU (112.6 ± 30.81 IU), was present in 10 (8.5%) and 25 (22.3%) cases with hyper bilirubinamia (S. bilirubin > 1.5 mg%, $3.6 \pm$ in 6 cases 5.3%) respectively. Serum Magnesium was above 2.5 mg% in 6 cases (5.3%, 3.2 ± 0.98 mg %) and less than 1.5 mg% off in 18 cases (16.1%, 0.98 ± 1.5 mg. %) Sodium and Potassium levels were within normal range except in 10 cases (8.8%) in whom serum potassium was raised above 5 meq / l (6.26 ± 1.8 meq / l). X-ray chest revealed hilar congestion in 26 cases (23.2%) who later on developed respiratory distress.

Table 1: Factors responsible for mortality (N= 112)

Factors		N (%)	Death; n (%)
Tablet (ALP) consumed	1	52 (46.4)	20 (17.8)
	2	32 (28.5)	16 (14.2)
	3	4 (3.5)	(2 (1.7)
	4	2 (1.7)	(2 (1.7)
	Not known	22 (19.6)	8 (7.1)
Duration of ingestion	<6 hours	90 (83.3)	33 (29.4)
	>6 hours up to 24 hours	22 (19.6)	15 (13.4)
Exposed tablet		14 (13.3)	4 (3.5)
Unexposed tablet		98 (87.5)	44 (39.2)
Abnormal ECG		82 (73.2)	44 (39.2)
No Shock (normal vitals)		28 (25)	4 (3.5)
In Shock	Systolic, <90 mmHg	84 (75)	44 (39.2)
	Deaths within 24 hours		38 (32.1)
	Deaths after 24 hours		10 (8.98)
Altered renal function		8 (7.1)	4 (3.5)
Altered liver function		16 (14.26)	12 (10.7)
Magnesium (meq /l)	<1.5 mg (1.34 \pm 0.7)	8 (15.96)	10 (8.4)
	1.5 -2.5 (1.92 \pm 0.15)	88 (78.5)	38 (33.9)
	>2.5 (3.2 \pm 0.2)	6 (5.3)	0 (0)

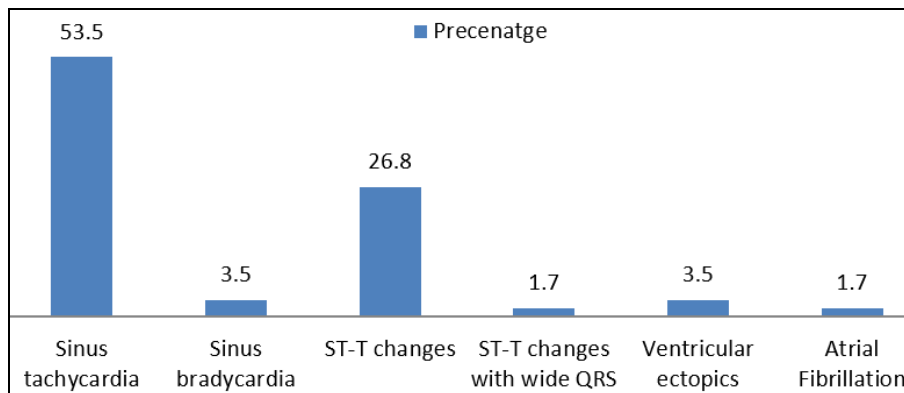


Fig 1: Electro Cardiogram Findings

Discussion and conclusion

Deaths by the AIP and Organ phosphorus poisoning are the commonest form of poisoning in India and in this part of U.P. (East U.P.) It produces phosphine gas which result in mitochondrial poisoning leading to organ damage^[18-23]. There is no known antidote for AIP poisoning which liberate toxic Phosphine when it comes in contact with water and hydrochloric acid of stomach. It cannot be detoxified but with all suitable supportive measures its absorption can be prevented and promote the excretion through kidney, Lung and GIT^[6, 9, 12-16]. It can be managed conservatively to provide symptomatic relief and supportive aid to help to promote excretion of phosphine through Lungs, Kidney and GIT. With the steps for management described by Bajpai SR^[20] can reduce the mortality and morbidity.

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