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Original Article

Aluminium Phosphide Poisoning- A prospective clinico therapeutic study in tertiary care Hospital of Eastern U.P. (Gorakhpur)

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Abstract

Aluminium Phosphide (AlP) poisoning is extremely lethal poisoning. Ingestion is usually suicidal in intention Phosphine, which is liberated when AlP comes in contact with moisture, is injurious and effect the cellular respiration there by it became lethal. The present study was under taken to study the salient features and ways to combat the deleterious effect of poisoning.

One hundred and twelve cases of proved AlP poisoning by $AgNO_3$ 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.

The overall incidence of all type of poisoning was 0.29% i.e. 200 cases of poisoning out of total admission 67189 in medical wards during Oct. 2012 to March 2014. Amongst these 200 cases there was 112 cases (56%) of AlP poisoning. They were mainly from 3rd decade of life (mean age ± SD = 22.45 ± 5.3 years) with male to female ratio of 2.7: 1. 73.2% 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.

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.

1. 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. Aluminium phosphide (AlP) an extremely lethal poisonous compound, is a solid fumigant, which has been in extensive use since the 1940 [2,3]. 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 [2-4]. Acute poisoning is on the rise in many countries and similar trend has been observed in India. AlP is now a day's more common because of its easy availability and widely used as grain preservatives at homes and in ware houses. It becomes poisonous by liberating phosphine (PH3) gas after coming in contact with moisture PH3 affects all the body systems and proves fatal.[5]

The poisoning is generally suicidal rarely homicidal and occasionally accidental (Children) and is common in younger age group. Poisoning is on rise in many countries and is often a neglected health problem compounded with the problem of difficulty in the diagnosis. Poisoning is largely preventable and majority of them dies within one to six hour of poison exposed or 6-8 hours of ingestion. AlP poisoning is the most common cause of suicidal death in India.[5]

Keeping in mind of increasing incidence of poisoning specially AlP amongst younger population the present study was undertaken for their clinical presentation with salient features and effective treatment so as to prevent the morbidity and mortality and evaluate Prognostic variables.

2. Materials & Methods

All cases of poisoning suspected or confirmed admitted in the Nehru Chikistisalya B.R.D. Medical College, Gorakhpur during the period of October 2012 to March 2014 constituted the clinical material of this study. 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 diarrhoea, Efforts will be 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 ($KMNO_4$, 1: 10000) two to three times was performed and gastric aspirate was preserved. After gastric wash 100 gms 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 Dobatamine). 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 then 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 ($AgNO_3$) test in gastric aspirate from breathing air and urine which turns the $AgNo_3$ black if AlP (Celphos) is taken.

They 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.

3. Observations

200 cases of poisoning out of 67189 total admissions (0.29% or 2/1000 admissions) in Medical Ward of Nehru Chikitsalya, B.R.D. Medical College, Gorakhpur during the period of October 2012 to March 2014 were included in this study. Of these 200 cases, 112 cases (56 %) were of Aluminium Phosphide (AlP) poisoning whose diagnosis was confirmed by $AgNO_3$ test. The incidence of AlP poisoning amongst total admissions was 0.16%, There was one hundred and twelve cases (56%) of Aluminium Phosphide poisoning followed by 40 cases (20%) of organo phosphorus poisoning and rest of them were of different poisoning/over dosage of material etc. (Table 1)

Table -1: Incidence of poisoning in hospitalized cases (n=200)

Type of poisoning	No of cases	%
Aluminium Phosphide	112	56
Organo phosphorus	40	20
Rodenticides	14	7
Anti-psychotic	2	1.0
Dhatura	2	1.0
Naphthalene	10	5
Diazepam	8	4
Bhang	2	1.0
Acid	4	2.0
Gamaxene	2	1.04
Unknown	4	2.0
TOTAL	200	100%

The age ranged from 12 to 35 years (mean ± SD= 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 accidental in 4 cases only. Eighty two of them (73.28 %) were from rural area and students (46.4 %). (Table-2)

Table 2: Socio –Economic status in Aluminium Phosphide cases (n=112)

Occupation	No. of Cases	%
Students	52	46.4
House wife	18	16.1
Farmers	2	1.8
Labourer	6	5.3
Salesman	4	3.6
Employed persons	14	12.5
Unemployed persons	16	14.2
Marital status:		
Married	44	39.3
Unmarried	68	60.7

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 %) (Table 3 and 4).

Table 3: Educational Status

Education	No. of cases	Percentage
Uneducated	10	8.9
Class VIII or less	14	12.5
High School	22	19.5
Inter mediate	26	23.2
Graduate	36	32.1
Post graduate	4	3.6
Total	112	100

Table 4: Profile of various precipitating Factor

Precipitating Factors	No. of cases	%
Family conflicts	38	33.9
Failure in love	30	26.8
Financial crisis	20	17.8
Failure in examination	16	14.3
Accidental	4	3.6
Unknown	4	3.6
TOTAL	112	100

As depicted in Table 5 all of them had nausea and vomiting with increased thirst (71.4 %) and pain in abdomen (42.8 %) more over epigastrium and adjacent area with epigastric tenderness Respiratory system revealed increased respiratory rate, creptiations 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%).

Table 5: Clinical Manifestation

Symptoms	No. of Cases	%
Vomiting	112	100
Thrust	80	71.40
Abdominal pain	48	42.85
Headache	12	10.71
Loose motion	4	3.57
Malaena	2	1.78
Haematemesis	2	1.78
Respiratory Distress	26	23.21
Oliguria	6	5.35
Palpitation	6	5.35
1. Shock (Systolic BP)		
< 90 mm hg	84	75.0
> 90 mm hg	28	25.0
2. Pulse		
Normal Pulse	28	25
Tachycardia	60	53.5
Bradycardia	4	3.57
Not recordable	20	17.8
3. Level of Conciousness	38	33.92
Delirium	14	12.5
Stupor	10	8.8
Coma	18	16.1
4. Pallor	12	10.70
5. Icterus	10	8.92
6.CVS		
S1`. Muffled	12	10.70
S3 Present	16	19.20
7. B/L Basal crepts	36	32.14
8. Epigastric tenderness	10	8.92
9. Paralytic ileus	2	1.78

Laboratory evaluation (Table 6) was mainly to assess the general condition and prognosis. 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 ± 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.

Table 6: Findings of the Investigations

Investigations	No. of Cases	%
Hemoglobin – 8 to 10 gm. % 10 to 12 gm % range, mean ± SD	8 12	7.1 10.7 8.4-12 gm, 10.2 ±4.6 gm. %
Leucopenia TLC < 5000/ cm. Range mean ± SD	5	4.4 3100- 4500/cmm 3102. 5 ± 2805/cmm
Blood Urea> 40mg % Range , mean ± SD	36	32.1% 45.6- 108.6 mg % 92.8 ± 12.92 mg %
S-creatinine > 1.5 mg % Range, mean ± SD	35	32.1 % 1.6-4.6 mg % , 3.2 ± 1.92mg %
SGOT (AST) > 40 w Range, mean ± SD	10	8.8 % 45- 92 IU ,, 68.9 ± 10.6 IU
SGPT (ALT) > 40 w Range, mean ± SD	25	22.3 % 56- 172 IU, 126.8 ± 30.9 IU
S.bilirubin > 1.5 mg % Range, means ± SD	6	5.3 % 1.5-5.2 mg % , 3.8 ± 2.4 mg %
S. magnesium < 1.5 mg % Range, means ± SD	18	16.1 % 0.98-1.5 mg%, 1.24±0.36 mg%
S. Magnesium > 2.5 mg Range, means ± SD	6	5.3 % 2.8-4.2 mg % , 3.2 ± 0.96 mg %
Serum potassium > 5 meq/l Range, means ± SD	10	8.8 % 5.2-7.8 meq/l, 6.26± 1.8 meq/l

Similarly raised level of transiminases 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 ± in 6 cases 5.3 %. 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.

Electrocardiogarms (Table 7) revealed various changes indicating towards the cardiac injury as evidenced by sinus /ventricular tachycardia (60 cases, 53.5%) ST-T changes in the form of ST elevation with concavity upwards, or depression (28.8%) while 2 of them had .wide QRS and bundle branch block (L.B.B.B – 3.5% R.B.B.B 1.7 %) and ventricular eptopics observed in 6 and 4 cases respectively.

Table 7: Electro Cardiogram Findings

E.C.G. Changes	No. of cases	%
Sinus Tachycardia	60	53.5
Sinus Bradycardia	4	3.5
ST-T Changes	30	26.8
ST-T Changes with wide QRS	2	1.7
Ventricular ectopics	4	3.5
Atrial Fibrillation	2	1.7
R.B.B.B.	2	1.7
L.B.B.B.	4	3.5

All patient were managed by gastric levage with Potassium Permanganate (1: 10,000) two to three times depending on amount consumed then placed activated charcoal 100 gm and liquid paraffin in stomach just to absorb the toxin and to accelerate excretion by diarrhea, Acceleration of excretion through urine (renal route) achieved by maintaining the adequate hydration and renal perfusion with IV fluids. The systolic BP was maintained around 90mm Hg by Dopamine and Dobutamine which will combat the shock. Dialysis was done in 6 cases and 4 of them expired due to acute renal failure Oxygen was administered through polymask or by endotracheal intubation (18 cases)

Magnesium sulphate therapy was given in 62 cases that is immediately 1 g, in 100 ml of 5% dextrose within 15 to 20 minutes and later on hourly for 3 hours , later on 6 hourly for 5-6 days. It was observed that there was no significant .effect on reducing the mortality i.e. 28 death out of 62 cases (45.1%) as compared to 20 deaths in 50 patient who have not received Mg SO 4 (P> 0.05)

Fourty eight (42.8 %) deaths were seen in the present series of cases (Table -8). Mortality was not related to amount (tablet) taken even though 16 deaths (50%) were seen in person who had taken to 2 tablets. Maximum deaths occurred within 6 hours (33 deaths out of 90 cases or 35 %) of ingestion of poison. Similarly the unexposed (sealed) tablet resulted in more deaths (39.2 %). Persons having abnormal ECG findings and in shock had also high mortality (39.2 %). There were more deaths in persons having low magnesium level (10 death out of 18 cases or 8.4 %, Table -8) as compared to patient with normal magnesium level(38 deaths out of 88 cases or 33.9 %).Deaths were mostly seems to be due to multi organ failure.

Table 8: Factors responsible for mortality (N= 112)

Factors	No. of Cases	%	Deaths	%
1- Tablets (ALP) - 1 tab.	52	46.4	20	17.8
consumed 2 tab	32	28.5	16	14.2
3 tab	4	3.5	2	1.7
4 tab	2	1.7	2	1.7
Not known	22	19.6	8	7.1
Duration of Ingestion < 6 Hours	90	80.3	33	29.4
>6 Hours up to 24 Hours	22	19.6	15	13.4
- Exposed tablets	14	13.3	4	3.5
-Unexposed tablets	98	87.5	44	39.2
2- Abnormal ECG.	82	73.2	44	39.2
3- No. shock (normal vitals)	28	25	4	3.5
- In shock (syst. BP < 90mm Hg)	84	75	44	39.2
-Deaths within 24 hours			38	32.1
- Deaths after 24 hours			10	8.98
4- Altered Renal function	8	7.1	4	3.5
5- Altered Liver Function	16	14.26	12	10.7
6-Magnesium -<1.5 mg(1.34±0,7)	18	15.96	10	8.4
(meq /l – 1.5 -2.5 (1.92±0.15)	88	78.5	38	33.9
- >2.5 (3.2±0,2)	6	5.3	-	-
7- Deaths	112	100	48	42.8

4. Discussion

Aluminum phosphide (AlP), commonly marketed as Celphos. Quickphos sunfume and Phosphene is easily available and a deadly poison due to Phosphine gas which is liberated when it comes in contact with water and / or hydrochloric acid of stomach. It is rapidly becoming a very commonly used agent for self poisoning in many countries [1,2, 9, 10] and now termed as Delebrate Self Poisoning (DSP) [11].

In the present study the incidence of poisoning was 0.29 % i.e. 200 cases of poisoning out of total admissions of 67189 in medical ward during the period of October 2012 to March 2014. Amongst these 200 cases of different poisoning there were 112 cases of AlP poisoning giving the incidence of 56%. Amongst total admissions the incidence of (AlP) poisoning came to 0.16%. This study confirms the finding of the literature [1,2,9,10] and agreed with the term DSP[11]. Kapoor *et al*[12] in their study at Allahabad reported 2.3 % in total admissions and 11.7 % of AlP poisoning amongst the all poisoning cases. They reported the sex ratio of male to female 2:1 as we reported 2.7: 1 and main age group involved was 16 to 25 years (73.2 %).

As described in literature [1,2,6,10-12] the present cases too manifested with nausea, vomiting, abdominal pain, loose motion and restlessness. Cardiovascular manifestation was in the form of tachycardia, thready pulse, hypotension, palpitation and breathlessness. ECG changes were observed in 26.8 % of cases ranging from ST-T elevation to prolonged PR and QRS with various blocks. These ECG changes and cardiac manifestations were due to toxic injury to myocardium and presented as focal myocadardial necrosis [3]. The changes in action membrane potential due to alteration in permeability of Na^{+}, Mg^{++} and Ca^{++} ions [14] Both hypo and hypermegnesemia were also results in ECG abnormalities [14,15] and seen in present series (16.1 % of cases) which might be due to an oxidative stress which buffer the Mg^{++} leading to increased susceptibility to oxygen , free radical , injury and accelerated liquid peroxidation [16].

It is the Phosphine (PH_3). Gas which is released in the stomach gets absorbed and circulates which manifest as various early signs and symptoms. The delayed onset to toxicity is because of some AlP is absorbed and metabolaise in liver will result in slow release of PH3 gas thus causing the delayed onset of toxicity signs and symptoms [8-13]. The ingested AlP leads to high superoxide dismulase activity and low catalyses levels that result in increased formation of free radicals and accelerated lipid peroxidation [13,15,17]. This in turn will result in cellular membrane damage with disruption of ionic barrier, nucleic acid damage and cell death. This will give rise to various ECG changes and cardiac arrhythmias. Phosphine is excreted through breath and urine Cellular hypoxia as a consequence of non competitive inhibition of cytochrome oxidase enzyme of mitochondria will lead to wide spread damage to other organs.

Laboratory investigations revealed leucopenia (TLC< 5000/cmm) in 4.4% cases, raised serum creatinine (>1.5 mg %) in 32.1% cases, 2-3 fold increase in serum transaminases in 22. 3% of cases and altered serum electrolytes with hypomagnesimea (< 1.5 mg%) in 16.1 % of cases. All these findings indicate towards the severity of toxicity , metabolic acidosis and poor prognosis ,which emphasized that laboratory evaluation was mainly to assess the severity of the disease and prognosis[1,2,15,17]. The altered level of Magnesium, Sodium and Potassium will lead to various ECG changes in the form of ST-T changes to various blocks even ventricular tachycardia and ventricular fibrillation. We too observed various types of ECG changes as depicted in Table 6 and 7. Which on echo cardiography reveal a generalized left ventricular hypokinesea, decreased ejection fraction etc. [17-20]. Chug *et al*[18] stressed that serum cortisol level was usually decreased in severe AlP poisoning cases which often proved fatal and indicate the severity of toxicity to myocardium . The fatal dose usually varied from 015 to 0.5 gm. However most of the patient has ingested [2-3] or more tablets which invariable results fatal as we observed too [1,2,4].

All patients were managed conservatively as no specific antidote to the poison is known. All the steps in management as described by Bajpai [21]. Were taken to provide symptomatic and

supportive aid to the patients till, Phosphine is excreted through GIT, Lungs and Kidneys. Magnesium Sulphate was given to 62 cases and revealed no significant effect on reducing morbidity and mortality on comparing with controls. But anyhow as described in literature [1,2,21,22], the MgSO4 was proved useful in reducing the various cardiac conduction defects and ventricular rate. MgSO4 stabilizes the cardiac cell membrane and thereby act as a weak anti arrhythmic agent and control the supra ventricular arrhythmias [1,2,21,22]. Similarly Siwacha *et al*[23] also observed no significant effect of $MgSO_4$ in reducing the mortality.

The stress has been laid in the quick and efficient management to prevent toxicity effect and to reduce mortality and morbidity. Measures will be taken to prevent absorption of AlP from GI tract and promote Phosphine excretion from Lung and Kidney by means of maintaining the renal perfusion with intravenous fluids with low dosage (4-6 mg / kg /min.) Dopamine, Dobutamine to maintain the BP around 90 mm Hg [1,2,21,23] Diuretics can be given when systolic BP was above 90mm. Hg. to enhance the excretion of PH3 through urine Acidosis will be managed with Sodium Bicarbonate 50-100 meq/l intravenously till carbonate level raised to 18-20 meq/l^2. The role of cortisone is controversial even some clinicians recommend to give hydrocortisone in the dosage of 200 - 400 mg 4-6 hourly intravenously. This is to combat shock and to reduce the dosage of dopamine [17,18] .

The fatal dosage of AlP is 0.15 – 0.5 gm.4 however most of the person taken 2-3 tablets or even more (38 cases) which invariably results to death (20 deaths 52.6%) The average time interval between the ingestion of AlP and death varies from 3- 48 hours and 95% of death usually occurs within 24 hours [10]. In the present series of cases the mortality was 42.8% and 33 deaths (68.7%) occurred within 6 hours and these persons consumed unexposed AlP which is more injurious and fatal[1,2]. Mortality depends on dosage and time of ingestion of poison and when symptoms start appearing. The shorter the time of appearing clinical manifestation the fatality will be more. It is the severity of poisoning, duration of shock , failure to revert, shock to normal life, resuscitative measures and severity of electrolyte imbalance and acidosis will affect the prognosis . Deaths after 24 hours (10 deaths or 8.9 %) was usually due to shock acidosis ARDS, cardiac dysarrtythmia [10]. Non survivors have more severe hypotension, metabolic acidosis etc. as compared to survivors.

The mortality is highly variable ranging from 37-100% most commonly reach up to 60% even in the experience hands and well equipped hospital 4.11. In the present series of 112 cases of AlP poisoning there were 48 deaths giving the incidence of 42.8%.

5. Conclusion

Deaths by the AlP and Organophosphorus 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. There is no known antidote for AlP 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 absoption can be prevented and promate the excretion through kidney, Lung and GIT. 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 [20] can reduce the mortality and morbidity.

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