International Journal of Medical Research Professionals
P-ISSN: 2454-6356; E-ISSN: 2454-6364
DOI: 10.21276/ijmrp

IJMRP

Original Research Article.

Clinical Characteristics and Outcomes in Poisoning with Aluminium Phosphide: A Single-Centre Experience

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ABSTRACT

Background: Aluminium phosphide (AlP) poisoning is common form of poisoning which is associated with adverse outcomes.

Objective: To understand clinical characteristic and outcomes in patients with AlP poisoning.

Methods: In a cross-sectional study, we enrolled all patients with AlP visiting a tertiary care hospital for one-year period. Demographic, clinical and laboratory data was recorded. Prevalence of mortality was assessed. Data was analysed with descriptive statistics.

Results: In total 101 patients enrolled, mean age was 32.45 ± 13.08 years and 67.33% were males. All cases were with ingestion of AlP and 85.06% patients had ingested up to two tablets. Lag time from ingestion to presentation was more than two hours in 77.41% cases. Shock defined by systolic blood pressure < 90 mmHg was found in 45.54% cases. 31.74% patients had magnesium levels <2 mg/dL. Troponin I was positive in 37.35% cases and pH <7.1 was seen in 16.13% cases. Myocarditis was seen in 76.24%. Mortality occurred in 33.66% cases.

Conclusion: AlP is common poisoning usually seen young ages and males. Early diagnosis and treatment is necessary to improve adverse outcomes.

Keywords: Aluminium Phosphide; Poisoning; Mortality; Treatment.

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Article History:

Received: 30-11-2017, **Revised:** 24-12-2017, **Accepted:** 27-01-2018

Access this article online	
Website: www.ijmrp.com	Quick Response code
DOI: 10.21276/ijmrp.2018.4.1.116	

INTRODUCTION

Pesticide poisoning is a global public health. Self-poisoning accounts for one-third of the world's suicide rate.[1] Aluminium Phosphide (AlP) is a solid fumigant pesticide, cheap, easily available, orally effective and widely marketed in India.[2] In few areas of developing countries, pesticide poisoning causes more deaths than infection.[3] Human toxicity with AlP is usually acute, occurring either due to AlP ingestion or PH_3 gas inhalation.[2] Cellular toxicity occurs by non-competitive inhibition of cytochrome oxidase, causing coagulation necrosis.[4]

Clinical symptomatology occurs according to route of poisoning, i.e. the gastrointestinal tract in case of ingestion, and the respiratory tract in case of inhalation, causing nausea, vomiting, diarrhoea, transaminitis, and cough, bronchospasm and even acute respiratory distress syndrome (ARDS) respectively.[2,5] More severe toxicity causes tachycardia, hypotension, cyanosis, myocarditis and pulmonary edema. A wide variety of rhythm and rate disturbances in ECG is seen.[6] Treatment of AlP toxicity, in absence of a specific antidote, is mainly supportive. It includes rapid removal of maximum possible toxin before the formation of phosphine gas; maintaining hemodynamic stability with i/v fluids inotropes, anti-arrhythmics, sodium bicarbonate steroids and magnesium (by stabilizing myocardial membrane); and providing respiratory support wherever required.[2,7] In recent years, there have not been studies looking in to aspects of AlP poisoning. Hence, this study was planned clinical presentation, response to treatment and outcome in patients, with Aluminium Phosphide poisoning.

MATERIALS AND METHODS

In a cross-sectional study spanned over one-year duration, we studied patients of AlP poisoning admitted in the intensive care unit of a tertiary care hospital. Study began after ethical approval

from institutional ethics committee. Informed consent was obtained from all conscious patients whereas legally acceptable representative signed the informed consent in cases with unconsciousness condition. The diagnosis was based on history with or without a corroborative evidence to the effect. We included all patients with history of exposure to AlP, regardless of age and sex. We excluded patients pre-existing cardiac, respiratory, hepatic or renal disorder, concomitant exposure to another toxin, prisoners and orphans were not included being dependent population. As per protocol, each patient was personally evaluated at the time of admission. A standard proforma was followed in each case, recording demographic details, AlP exposure details, clinical features and laboratory parameters. The treatment followed as per protocol in ICU was recorded in each case. Definitions used for various complications at the time of presentation to hospital were as shown in table 1.

Statistical Analysis: The data was entered in Microsoft excel. Data was presented as frequency and percentage for categorical parameters and as mean and standard deviation for continuous parameters. Data was analysed with descriptive statistics.

Table 1: Definition of complications

Complication	Definition
Shock	Systolic blood pressure < 90 mmHg
Acute renal failure	Serum creatinine ≥ 2 mg/dl
Acute hepatic injury	Serum bilirubin ≥ 2 mg/dl or SGOT ≥ 160 U/L or SGPT ≥ 120 U/L
Respiratory failure	SpO2 < 60 mmHg

Table 2: Baseline characteristics of study population

Parameter	Observation (n=101)
Age	32.45 ± 13.08
Age groups	
≤20	20 (19.80)
21-30	33 (32.67)
31-40	29 (28.71)
>40	19 (11.88)
Sex	
Male	68 (67.33)
Female	33 (32.67)
POISONING DETAILS	
Mode of exposure	101 (100.00)
Number of tablets consumed	
1-2	74 (85.06)
>2	13 (14.94)
Ingestion of fresh tablets	54 (64.29)
Lag time (hours)	
≤ 2	21 (22.58)
> 2	72 (77.41)
Altered sensorium	21 (20.79)
HEMODYNAMIC ALTERATIONS	
Bradycardia (HR < 50)	1 (0.99)
Shock (SBP<90 mmHg)	46 (45.54)
LABORATORY EVALUATIONS	
Serum Magnesium (n=63)	
≤ 2	20 (31.74)
> 2	43 (68.25)
Troponin I (n=83)	31 (37.35)
pH ≤ 7.1 (n=93)	15 (16.13)
ORGAN DYSFUNCTIONS	
Hepatic (n=60)	4 (6.67)
Acute renal failure (n=95)	13 (13.68)
Myocarditis (n=101)	77 (76.24)

Table 3: Treatment administered for AlP

$MgSO_4$ dose (gm/d) (n=98)	Frequency (%)
1	18 (18.36)
2	37 (37.75)
3	22 (22.44)
4	19 (19.38)
≥ 5	2 (2.20)
Hydrocortisone dose (mg/day) (n=98)	
100	23 (23.46)
200	21 (21.42)
300	22 (22.44)
400	25 (25.51)
≥ 500	7 (7.14)

Table 4: Outcome of patients

Outcome	Frequency (%)
Discharged	49 (48.51)
Discharge against medical advice	18 (17.82)
Mortality	34 (33.66)

RESULTS

In 101 patients included in study, mean age of the patients was 32.45 ± 13.08 years with most patients being in age group of 20-40 years. Among them, 2/3rd were males and 1/3rd were females. 14.94% patients had consumed more than two tablets of AlP. 77.41 % patients presented to hospital after lag time of more than 2 hours from ingestion of poison. One-fifth of all patients had altered sensorium. Bradycardia with heart rate below 50 per minute was seen in one patient only. 45.54 % patients had shock on presentation to hospital. 68.25% had serum magnesium above 2 mg/dL and troponin I was positive in 37.35% cases. pH below 7.1 was observed in 16.13 % cases. Myocarditis, acute renal failure and hepatic dysfunction was found in 76.24 %, 13.68 % and 6.67 % cases. Table 2 enlists the baseline characteristics of study population. MgSO4 and steroids were administered to majority of cases. Table 3 shows proportion of patients receiving different doses of both drugs.

The outcome of patients as shown in table 4 suggest that 48.51% were discharged from hospital whereas 17.82% were discharged against medical advice. 33.66% patients expired.

DISCUSSSION

Aluminium Phosphide (AlP) is an epidemiologically significant poison. It is a potent toxin, with rapid deleterious effects, associated with high mortality. We found 32.67% patients being in their third decade and a large majority (81.19%) fell between ages of 11 and 40 years. Males were affected more frequently than females. Similar results have been reported by Siwach et al. where 43.8% patients belonged in their third decade, while 86.8% were between 10 and 39 years of age with 61.4% of all patients as being male.[5] Greater preponderance in males is may be due to easier access of males to the toxin, especially in the agriculture industry. Majority of patients had ingested up to two tablets of AlP in the present study. Comparable results were obtained by Siwach, et al. in their study where 81.6% patients had taken up to 2 tablets of AlP.[5] The reason is high and quick oral effectiveness of AlP causes imminent toxicity.

Analysis of patients admitted with shock revealed that out of all patients with recordable vitals on admission nearly 45.54% had shock (SBP < 90 mmHg). Only 11.67 % patients remained in

shock at the end of 24 hours. Hence, it was inferred that shock is an important predictor of mortality in AlP poisoning. Positive troponin in blood probably indicates of myocardial damage from myocarditis. In our opinion, cardiac enzymes are a useful and consistent marker for myocarditis.

63 patients from our study group underwent serum magnesium measurement on admission before commencement of therapy. It was found that 31.74% of the above had S. Mg levels < 2 mg/dl. This is similar to the finding of Chugh et al. who found hypomagnesemia in patients with shock in AlP toxicity, thereby advocating the use of intravenous magnesium sulphate in AlP poisoning with shock.[8] Contrast to this, Siwach et al. reported no significant difference in the tissue Mg content of their test (those with AlP poisoning) and control patients (randomly selected out of patients who had died in road side accidents), thus rejecting the therapeutic use of intravenous $MgSO_4$ in AlP poisoning.[9] Arterial blood gas values done in 93 patients in our study revealed that 16.13% had a blood pH value of 7.1 or less. Siwach et al reported much higher percentage (43.33%) of patients with pH of up to 7.1. However, their total number of patients was 30 only. [7]

We found that 6.67% developed biochemical evidence of hepatitis and nearly 13.68% developed ARF. Siwach et al. in two different studies reported microscopic organ affection in much higher fractions of patients in their studies based on post-mortem biopsies. The changes in liver and renal abnormalities were found in 83% and 76% patients respectively. Myocarditis was a finding in nearly 75% of patients who had been electrocardiographed.[5,10] The exact mechanism of development of myocarditis is not clear, but it has been attributed to cellular ischaemia caused by the PH_3 gas [11,12] acidosis or hypomagnesemia.[8]

Use of magnesium is advised in ALP poisoning. Chugh et al. studied the therapeutic effect and mortality benefit of intravenous $MgSO_4$ through 2 different schedules of administration – a 1g 6 - hourly schedule, and another regime where an initial loading was done with 4g $MgSO_4$ over 3 hours, then followed by 1g-6 hourly schedule. They recommended the latter schedule as having a significant survival benefit (p value < 0.001) over the former.[13] Use of intravenous corticosteroids in combating shock in AlP poisoning, prevention of ARDS and direct (toxic) tissue insult by AlP. Hence, the use of intravenous Hydrocortisone in doses varying from 100 mg in 24 hours to 500 mg per day. We observed mortality in in 33.36% cases. Siwach, et al in another study reported mortality of 67.6% in patients consuming AlP.[10] If the patients who were discharged against medical excluded, mortality rate in our study becomes 40.9% which is similar to other studies. Siwach et al. in a study of 114 patients reported a fatality rate of 70% in shock with AlP poisoning. [5] This suggests strong association of shock with poor prognosis in AlP toxicity.

CONCLUSION

Aluminium phosphide is important poison leading to high mortality. Magnesium and steroids can be considered in all cases. Development of complications like hepato-renal dysfunction and myocarditis can lead to adverse outcomes. Early identification and institution of rapid treatment remains the cornerstone for improving outcome. Interventions to improve suicidal behavior can be helpful.

REFERENCES

1. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: Systemic review. BMC Public Health. 2007; 7:357–71.
2. Chugh SN. AlP poisoning: Present status and management. JAPI 1992; 40:226-7.
3. Eddleston M, Karalliedde L, Buckley N, Fernando R, Hutchinson G, Isbister G, et al. Pesticide poisoning in the developing world-a minimum pesticides list. Lancet. 2002; 360:1163–7.
4. Hsu CH, Chi BC, Liu MY, Li JH, Chen CJ, Chen RY. Phosphine- induced oxidative damage in rats: Role of glutathione. Toxicology. 2002; 179:1–8.
5. Siwach SB, Yadav Dr, Arora B, Dalal S, Jagdish. Acute AlP poisoning- An epidemiological, clinical, histopathological study. JAPI. 1988; 10: 594-6
6. Gupta MS, Malik A, Sharma VK. Cardiovascular manifestations in AlP with special reference to Echocardiographic changes. JAPI. 1995; 43:773-4,
7. Siwach SB, Jagdish Katyal VK, Dhall A, Bhardwaj. Prognostic indices in AlP poisoning-observations on acidosis and central venous pressure. JAPI 1997; 45: 693-95.
8. Chugh SN, Malhotra S, Kumar P, Malhotra KC. Reversion of ventricular and supraventricular tachycardia by magnesium sulphate therapy in aluminium phosphide poisoning. Report of two cases. JAPI 1991; 39:642-3.
9. Siwach SB, Singh P, Ahlawat S. Magnesium in aluminium phosphide poisoning – where have we erred. JAPI – Editorial 1994; 42:193-4.
10. Siwach SB, A Gupta. The profile of acute poisoning in Haryana- Rohtak study. JAPI 1995; 43:756-9.
11. Siwach SB, H Singh, Jagdish, Katyal VK, Bhardwaj G. Cardiac arrhythmias in AlP poisoning studied on continuous Holter and cardioscopic monitoring. JAPI 1998; 46: 598-601.
12. Dashora UK, Swaroop D. The dreadful Celphos poisoning. JAPI 1986; 34: 227.
13. Chugh SN, Kumar P, Aggarwal HK, Sharma A, Mahajan SK, Malhotra KC. Efficacy of magnesium phsphate in AlP poisoning. JAPI 1994; 42:32.

Source of Support: Nil.

Conflict of Interest: None Declared.

Cite this article as: Ashish Kumar Jain, Parul Jain, Sompal Singh. Clinical Characteristics and Outcomes in Poisoning with Aluminium Phosphide: A Single-Centre Experience. Int J Med Res Prof. 2018 Jan; 4(1):546-48. DOI:10.21276/ijmrp.2018.4.1.116