Section: General Medicine

Original Research Article

ACUTE ALUMINIUM PHOSPHIDE POISONING AND HYPERGLYCEMIA: A POSSIBLE WARNING SIGN FOR PROGNOSIS

Surekha Kadamati[1], Purnima B Potharlanka[2], Rajesh Kumar Palaparthi[3], Lalitha Palaparthi[4]

[1]*Assistant Professor, Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India*
[2]*Assistant Professor, Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India*
[3]*Assistant Professor, Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India*
[4]*Assistant Professor, Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India*.

Received *: 28/02/2024*
Received in revised form *: 04/05/2024*
Accepted *: 21/05/2024*

Corresponding Author:
Dr Lalitha Palaparthi
Assistant Professor, Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India.
Email: lalitha5589411@gmail.com

DOI: 10.5530/ijmedph.2024.2.74

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2024; 14 (2); 379-382

ABSTRACT

Background: Aluminum phosphide is a pesticide that is often used to keep grain holding areas clean. It is possible to buy AlP in the form of 3 g pills. There are 56% AlP and 44% ammonium carbonate in each pill.

Materials and Methods: We did a prospective case-control study in the Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India. People who were taken to the hospital because they were poisoned by AlP from a single drug were part of this study. The study began on February 2023 to January 2024. 45 people with a normal body mass index who had not been diagnosed with diabetes mellitus before began the study. The patient's or their family's information about the exposure agent was used to make the determination.

Result: Phosphorus overdose happens quickly after AlP is eaten, and most people die within the first 12 to 24 hours, mostly from heart problems. The death rate from AlP overdose is said to be between 60% and 80%, according to reports. The numbers we have for this case series match these numbers. When AlP comes into contact with water, a very deadly gas called phosphine is released. One way that phosphine is harmful is by making free radicals and stopping biochemical enzymes like cytochrome-C oxidase from working. In a rabbit model of AlP poisoning, organs were looked at and the liver, heart, and kidneys all showed clear signs of degeneration. When people are exposed to AlP, it can cause problems in many organs.

Conclusion: There needs to be more research done to find out if hyperglycemia-lowering medicine could help treat AIP poisoning.

Keywords: Hyperglycemia, aluminium phosphide poisoning, potential prognostic factor.

INTRODUCTION

Aluminum phosphide (AlP), which is a common pesticide, is used to kill germs in grain storage sites. It is possible to buy AlP in the form of 3 g pills. There are 56% AlP and 44% ammonium carbonate in each pill. When someone eats AlP, it reacts with water and hydrochloric acid in the gut, which releases phosphine gas.[1-3] Phosphine gas doesn't have any color and smells like garlic or fish that has gone bad. It can catch fire and is very dangerous.

As a mitochondrial toxin, phosphorus stops the production of proteins and enzymes in ways that aren't fully known. Studies in real life show that messing with mitochondrial oxidative phosphorylation could make many organs not work right. Thus, treatment methods that keep enzymes working properly might be helpful for helping people who have been poisoned by AlP. Also, there is proof that the membranes of erythrocytes and blood vessels may be directly damaged.[2-4]

It is cheap, easy to get, and very dangerous to work with aluminum phosphide. The reason why poisoning is mostly blamed on it in poorer countries

is now clear. People who are thinking about committing suicide take drugs.[3-5] When phosphine gas is mixed with liquids in the gut, it is quickly released and quickly taken in by the lungs. This stops oxidative phosphorylation, harming the whole body, and low oxygen levels in cells. PH3 can increase the production of free radicals and stop catalase from working, which leads to oxidative stress after being poisoned by aluminum phosphide. AAlPP is very dangerous because it has a systemic poison that is very deadly and for which there is currently no known cure.[4-6]

AlP is thought to be one of the most common chemical poisonings that happens quickly in Asia. Accidental exposure to AlP in humans is very rare. On the other hand, people are often intentionally exposed to it by intentionally ingesting it with the goal to kill themselves. It is very dangerous, cheap, and easy to get in countries that aren't very well developed. Adult respiratory distress syndrome, abrupt metabolic acidosis, rapid start of shock, and cardiac arrhythmias are some of the signs that someone has been poisoned with ALP.[5-7] Phosphine has been shown to change the amount of glucose in the blood of animals. One theory says that AlP poisoning might either stop the production of insulin or cause glucagon, epinephrine, and cortisol to be released. Previous research has mentioned that hyperglycemia can happen in people who have been poisoned by AlP, but this information has not been used to figure out the risk.[6–8]

AlP poisoning is linked to a number of clinical and test signs, but there isn't a lot of information from the past about how to tell if someone will get better. The point of this study was to look into what happens to blood sugar levels when someone is poisoned by AlP and whether this could be used as a sign of how someone who has been exposed to AIP will do in the future.[7-9]

MATERIAL AND METHODS

We did a prospective case-control study in the Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India. People who were taken to the hospital because they were poisoned by AlP from a single drug were part of this study. The study began on February 2023 to January 2024. 45 people with a normal body mass index who had not been diagnosed with diabetes mellitus before began the study. The patient's or their family's information about the exposure agent was used to make the determination.

As a way to measure internal control, we chose patients who stayed awake while they were drunk. As a test, we looked at their blood sugar levels and matched them to those of people who had died from AlP poisoning. The Student t-test, Pearson association coefficient, and logistic regression were all used by SPSS software to look at the data. It was thought that p-values below 0.05 were statistically significant.

RESULTS

The study looked at 40 people who had been identified with AlP poisoning. There were 20 men and 20 women in the group. In general, they were 26.8 years old. Table 1 and figure shows that most of the cases were between the ages of two and three years. Thirteen of the patients were able to stay alive, while 32 died. All of the patients who got AlP exposure did so on purpose. This table (Table 2) shows that 44.4% of patients were hurt by AlP pill.

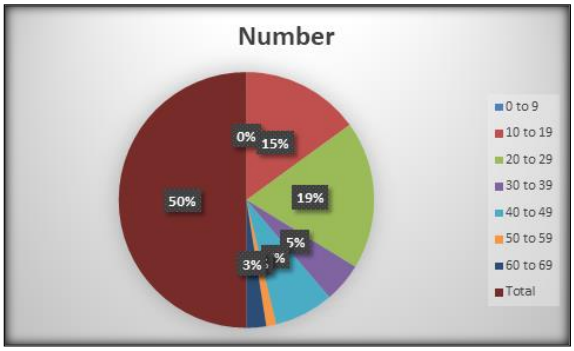

Figure 1: Age wise distribution

The groups that lived and the groups that did not had no major differences in age, gender, or the amount of time before treatment. Endotracheal intubation and mechanical breathing were needed for every patient. The survivors stayed in the, while the non-survivors stayed. The blood glucose level wasn't significantly linked to age, gender, and time since treatment, or amount eaten. In addition, there were no significant links found between the blood glucose level and the pH or HCO3 levels.

The average blood sugar levels of patients who lived and those who died were 142.5 mg/dL and 223.1 mg/dL, respectively, as shown in Table 3. The odds ratio for hyperglycemia as a death risk was found to be 5.7, taking into account things like age, gender, amount taken, pH, and HCO3 concentration. [Table 2]

Table 1: Age distribution of aluminum phosphide poisoning patients

Sr. No.	Age	Number
1	0 to 9	0
2	10 to 19	12
3	20 to 29	15
4	30 to 39	4
5	40 to 49	6
6	50 to 59	1

7	60 to 69	2
	Total	**40**

Table 2: Patients' AIP poisoning dose distribution

Sr. No.	No. tablets taken	AlP ingested (mg)
1	0.26	421
2	0.34	553
3	0.50	841
4	1.0	1681
5	1.6	2521
6	2.0	3361
7	3.0	5041

Table 3: Age, gender, ingestion interval distribution

Sr. No.	Patient group	Age (yrs.)	Gender	Length of stay in ICU	Blood glucose	Elapsed time
1	Survived	24.5	5 female, 6 male	174.2	142.5	2.9
2	Non-survived	27.9	17 female, 15 male	16.5	223.1	4.3

DISCUSSION

When someone takes AlP, phosphorous overdose quickly sets in, and most of the deaths happen in the first 12 to 24 hours, mostly because of heart problems. The death rate from AlP overdose is said to be between 60% and 80%, according to reports. The numbers we have for this case series match these numbers. When AlP comes into contact with water, a very deadly gas called phosphine is released. One way that phosphine is harmful is by making free radicals and stopping biochemical enzymes like cytochrome-C oxidase from working.[9-11]

In a rabbit model of AlP poisoning, organs were looked at and the liver, heart, and kidneys all showed clear signs of degeneration. When people are exposed to AlP, many of their functions can stop working properly. Animal studies that looked into how AlP affected blood glucose levels found that the levels of glucose changed over time.[12-14] Blood glucose levels have been shown to change in the past, including going up, down, or staying the same. However, no work has been done to find a link between these findings and clinical toxicity or outcomes. Some studies have shown that the average amounts of magnesium and phosphate in the blood and tissues are higher. There is also more renin activity and plasma cortisol.[15-17]

Out of the 50 cases, 40% had plasma cortisol levels rise significantly. Ten people who had died had mild to moderate changes in their adrenal cortex, such as cellular infiltration, swelling, and congestion. It makes sense that the adrenal axis would be involved in the problem with glucose control that was seen before. This is what the current study is based on. Additionally, there is evidence linking AlP overdose to both acute pancreatitis and high blood sugar in some cases.[16-18] This suggests that the way AlP affects the pancreas may play a part in the development of high blood sugar in this condition. In addition, experiments where AlP was put straight into the stomachs of rats showed that adenosine triphosphate levels dropped significantly. This means that the actions listed above have a big effect on the production of metabolic energy right away.[17-19]

Based on the results of our study, people who go to the emergency room with glucose levels above 140 mg/dL have a higher chance of dying. It is thought that the rise in blood sugar is due to problems with oxidative phosphorylation and glucose consumption, as well as the pancreatic and adrenal axis being involved.[20-22] Some people believe that treating diabetes directly with insulin can help cells take in glucose better, speed up cellular metabolism, and make more ATP. We are currently doing a follow-up study to look at the possible benefits of this direct action in stopping this very dangerous poisoning.[23-25]

New drugs like trimetazidine might help by changing the metabolism of myocytes, specifically by switching them from using fatty acids to glucose. Because of this change in metabolism, there is less need for air. It was impossible to get a good idea of how food from a recent meal affected our patients because they were already very sick from their harmful situations and their family histories were hard to predict because of stress.[24-26] Because of this, our study didn't look at how a previous meal affected blood glucose levels at the start. One problem with our study is that patients were not stopped from joining because they did not have this information. Most patients, though, had to wait at least three to four hours after taking AlP before they could go to the hospital. Because the illness was so bad, no food was eaten during this time.[26-28]

CONCLUSION

In our study, people who did not survive AIP poisoning had a statistically significant higher risk of hyperglycemia than people who did survive. According to these results, diabetes may be a useful way to predict how this poisoning will affect the person and how to treat it. There needs to be more research done to find out if hyperglycemia-lowering medicine could help treat AIP poisoning.

Funding: None
Conflict of interest: None.

REFERENCES

1. Singh, S, Bhalla, A, Verma, SK, Kaur, A, Gill, K. Cytochrome-C oxidase inhibition in 26 aluminum phosphide poisoned patients. Clin Toxicol (Phila) 2006; 44: 155–158.
2. Lall, SB, Peshin, SS, Mitra, S. Methemoglobinemia in aluminium phosphide poisoning in rats. Indian J Exp Biol 2000; 38: 95–97.
3. Abdollahi, M, Jalali, N, Sabzevari, O, Hoseini, R, Ghanea, T. A retrospective study of poisoning in Teh- ran. J Toxicol Clin Toxicol 1997; 35: 387–393.
4. Siwach, SB, Dua, A, Sharma, R, Sharma, D, Mehla, RK. Tissue magnesium content and histopathological changes in non-survivors of aluminium phosphide poisoning. J Assoc Physicians India 1995; 43: 676–678.
5. Bates SM, Grand'Maison A, Johnston M, et al. A latex D-dimer reliably excludes venous thromboembolism. Arch Intern Med 2001; 161:447-53.
6. Shadnia, S, Mehrpour, O, Abdollahi, M. Unintentional poisoning by phosphine released from aluminum phosphide. Hum Exp Toxicol 2008; 27: 87–89.
7. El-Ebiary A, Elgazzar F, Soliman MA, Shouip O. Predictors of prognosis in acute aluminum phosphide poisoning. Mansoura Journal of Forensic Medicine and Clinical Toxicology. 2015 Jul 1;23(2):13-27.
8. Abder-Rahman, H. Effect of aluminum phosphide on blood glucose level. Vet Hum Toxicol 1999; 41: 31–32.
9. Hajouji Idrissi, M, Oualili, L, Abidi, K, Abouqal, R, Kerkeb, O, Zeggwagh, AA. Severity factors of alumi- num phosphide poisoning (Phostoxin). Ann Fr Anesth Reanim 2006; 25: 382–385.
10. Burgess, JL, Morrissey, B, Keifer, MC, Robertson, WO. Fumigant-related illnesses: Washington State's five- year experience. J Toxicol Clin Toxicol 2000; 38: 7–14.
11. Gupta, S, Ahlawat, SK. Aluminum phosphide poison- ing – a review. J Toxicol Clin Toxicol 1995; 33: 19–24.
12. Soltaninejad, K, Faryadi, M, Sardari, F. Acute pesti- cide poisoning related deaths in Tehran during the period 2003–2004. J Forensic Leg Med 2007; 14: 352–354.
13. Gurjar M, Baronia AK, Azim A, Sharma K. Managing aluminum phosphide poisonings. Journal of emergencies, trauma, and shock. 2011 Jul 1;4(3):378-84.
14. Brautbar, N, Howard, J. Phosphine toxicity: report of two cases and review of literature. Toxicol Ind Health 2002; 18: 71–75.
15. Okolie, NP, Aligbe, JU, Osakue, EE. Phostoxin-induced biochemical and pathomorphologiacal changes in rab- bits. Indian J Exp Biol 2004; 42: 1096–1099.
16. Sheta AA, El-Banna AS, Elmeguid RA, Mohamed HE, Gad NH. A study of the predictive factors of mortality in acute poisoning with aluminum phosphide with special reference to echocardiography and SOFA score. Environmental Science and Pollution Research. 2019 Nov; 26:33135-45.
17. Siwach, SB, Singh, P, Ahlawat, S, Dua, A, Sharma, D. Serum and tissue magnesium content in patients of aluminum phosphide poisoning and critical evalua- tion of high dose magnesium sulphate therapy in reducing mortality. J Assoc Physicians India 1994; 42: 107–110.
18. Singh, RB, Shahria, RB, Sharma, VK. Can aluminum phosphide be poisoning cause hypermagnesemia? A study of 121 patients. Magnes Trace Elem 1990; 9:212–218.
19. Chin, KL, Mai, X, Meaklim, J, Scollary, CR, Leaver, DD. The interaction of phosphine with hemoglobin and erythrocytes. Xenobiotica 1992; 22: 599–607.
20. Chugh, SN, Ram, S, Mehta, LK, Arora, BB, Saini, AS, Malhtra, KC. Adrenocortical involvement in alumi- num phosphide poisoning. Indian J Med Res 1989; 90: 289–294.
21. Singh, RB, Rastogi, SS, Singh, DS. Cardiovascular manifestation of aluminum phosphide intoxication. Assoc Physicans India 1989; 37: 590–592.
22. Chugh, SH, Singhal, HR, Mehta, L, Chugh, K, Shankar, V, Malhotra, KC. Plasma rennin activity in shock due to aluminum phosphide poisoning. J Assoc Physicians India 1990; 38: 398–399.
23. Louriz M, Dendane T, Abidi K, Madani N, Abouqal R, Zeggwagh AA. Prognostic factors of acute aluminum phosphide poisoning. Indian journal of medical sciences. 2009 Jun 1;63(6).
24. Sharma A, Balasubramanian P, Gill KD, Bhalla A. Prognostic significance of blood glucose levels and alterations among patients with aluminium phosphide poisoning. Sultan Qaboos University Medical Journal. 2018 Aug;18(3): e299.
25. Pannu AK, Bhalla A, Gantala J, Sharma N, Kumar S, Dhibar DP. Glucose-insulin-potassium infusion for the treatment of acute aluminum phosphide poisoning: an open-label pilot study. Clinical Toxicology. 2020 Oct 2;58(10):1004-9.
26. El-Sarnagawy G. Predictive factors of mortality in acute aluminum phosphide poisoning: 5 years retrospective study in Tanta Poison Control Unit. Ain Shams Journal of Forensic Medicine and Clinical Toxicology. 2017 Jun 1;29(2):70-9.
27. Ahmed N, El-Mehallawi I, Abo Elnoor M, Hodeib A. Potential clinical and laboratory prognostic factors for prediction of need for ICU admission in acute aluminum phosphide poisoning. Ain Shams Journal of Forensic Medicine and Clinical Toxicology. 2021 Jul 1;37(2):98-106.
28. Ghonem MM, El Sharkawy SI, Lashin HI. Predictive variables of acute aluminum phosphide poisoning outcome: a new proposed model. The Egyptian Journal of Forensic Sciences and Applied Toxicology. 2020 Jun 1;20(2):45-60.