CASE SERIES

Management of Aluminum Phosphide Poisoning in a ICU setup

Dr. Rajesh Mahajan

Associate Professor, Department of Anaesthesia & Critical Care, Mahaveer Institute of Medical Sciences & Research, Bhopal, Madhya Pradesh, India

Corresponding Author
Dr. Rajesh Mahajan
Associate Professor, Department of Anaesthesia & Critical Care, Mahaveer Institute of Medical Sciences & Research, Bhopal, Madhya Pradesh, India
Email: rajeshmahajan71@yahoo.com

Received: 12 April, 2024 Accepted: 10 May, 2024

ABSTRACT
Aluminum phosphate is a commonly used pesticide in developing countries like India. It is available as fumigant in solid or powder form. It is most common & deadly cause of suicidalpoison in rural areas. Its lethal dose is 150-500mg for an adult. 3gm single tablet can cause mortality.In spite of a poor predicted outcome the patient survived due to prompt aggressive resuscitative measures. In our case series mortality rate was 40% and 60% survived from aluminum phosphide poisoning.
Key words: Aluminum phosphide, resuscitation, poisoning.

INTRODUCTION

Aluminum Phosphide Poisoning is a solid type of fumigant which is available in tablet & powder form.
In our study, it was found to be the most common cause of acute poisoning in rural India(1).
As the lethal dose of aluminum phosphide is 150-500mg for an adult, even a single tablet of 3gm can cause mortality(2).
It is colorless and odorless, however on exposure to air, it gives a foul odour (garlicky or decaying fish) due to the presence of substituted phosphine and diphosphines(3).
This odour also helps in the diagnosis of the poisoning.
Aluminum Phosphide Poisoning after exposure to moisture or acid in the stomach releases the complicating phoshphine (PH3) gas(4).
Following oral ingestion, Aluminum Phosphide Poisoning reacts with water and stomach acid to produce phosphine gas, which may account in large parts for its observed toxicity. Phosphine generated in the gastro intestinal tract is readily absorbed into blood strain, phosphine may denature and induces oxyhemoglobin in addition to enzymes important for respiration and metabolism and may also affect cellular metabolism(5).
The mortality rate of Aluminum Phosphide Poisoning is 77%-100%(6,7). The dominant clinical feature is severe and refractory hypotension. Complications include fatal diaphoresis, congestivecardiac failure, gastrointestinal hemorrhage & acute respiratory distress(8).
This kind of pesticide ingestion accounted for over 60% of suicides in rural areas of countries like South East Asia and China(9).

Case 1: 34 years female came with history of consumption of Celphos on admission patient GCS was full heart rate was 102/min BP was no recordable SPO2 was 93% ABG shows Ph 6.898, pco2-25.4, P02-63.4, HCO3 4.9, Lactate out of range Urea-24mg %, Creatinine-1.41mg%, Sodium-141mmol/l, Potassium-4.74 mmol/l, Hb-12.3 gm%, TLC-38200, Platelet 5,43.000, PT-18.7 INR-1.49. Patient was resuscitated but had cardiac arrest after 5 hours and could not be revived.

Case 2:17 years female came with history of consumption of Celphos on admission patient GCS was full heart rate 90/min BP 80/60mmhg SPO2 96% ABG shows PH-7.37, PCO2-28.9, P02-84.3, HCO3 – 16.3, Lactate – 1.02, Urea – 27mg %, Creatnine – 0.6 mg%, Sodium – 140 mmol/I, Potassium – 3.88 mmol/l – Hb-12.0 gm% TLC -10500, Platelet 1,39000, PT-16, INR-1.29 patient was resuscitated to which she responded and was discharged after 3 day.

Case 3: 34 year male came with history of consumption of Celphos on admission patient GCS was full heart rate 90/min BP 130/90mmhg SPO2 98% ABG shows PH-7.334, PCO2-41.3, P0-75.7, HCO3 -21.3, Lactate -2.41, Urea-30 mg %, Creatinine – 1.16 mtg% , Sodium -143 mmol/l, Potassium- 3.76,

Hb-13.6 mg%, TLC-6900, Platelet 2,45.000, PT-16, INR-1.29 patient was resuscitated to which he responded and was discharged after 2 days.

Case 4: 24 year male came with history of consumption of Celphos on admission patient GCS was full heart rate 102/min BP 120/80 mmhg SPO2 96% ABG shows PH-7.39, PCO2 – 38.3, P02-147.70, HCO3 – 20.4, Lactate – 8.41, Urea 17 mg %, Creatinine – 1.18mg%, Sodium -140 mmol/l, Potassium – 3.46 mmol/l, HB-13.1 gm%, TLC-12300, Platelet 2,64,000, PT – 15,. INR – 1.19 patient was resuscitated to which he responded and was discharged after 2 days.

Case 5: 51 year male came with history of consumption of Celphos on admission patient GCS was full heart rate 92/min BP 110/70 mm hg SPO2 96% ABG shows PH-7.451, PCO2-22.1, P02 – 62.4, HCO3 -15, Lactate – 6.76, Urea – 14mg %, Creatinine – 0.70 mg%, Sodium – 136 mmol/l, Potassium – 4.04, Hb14.7 gm%, TLC-12000, Platelet 1,13.000, PT-18.3 INR-1.44 patient was resuscitated to which he responded and was discharged after 4 days.

Case 6: 21 years male came with history of consumption of Celphos on admission patient GCS was full heart rate 104/min BP 160/100mmhg SPO2 98% ABG shows PH – 7.45, PCO2-35.9, P02-113.90, HCO3 – 24.60, Lactate – 2.03, Urea – 13mg%, Creatinine – 1.03mg%, Sodium-145 mmol/Potassium – 3.5 mmol/l, HB-15.2 gm%, TLC-13200, Platelet 2,00,000, PT-16, INR-1.29 patient was resuscitated to which he responded and was discharged after 3 days.

Case 7: 60 year female came with history of consumption of Celphos on admission patient GCS was full heart rate 90/min BP 100/70mmhg SPO2 96% ABG shows PH – 7.46, PCO2 – 36.90, P02-120.70, HCO3-25.4, Lactate – 3.27, Urea 23 mg %, Creatinine – 0.86mg%, Sodium – 140 mmol/l, Potassium – 3.28 mmol/l, Hb-12.3gm %, TLC – 10700, Platelet 1,82.000, PT-18.3, INR-1.44 patient was resuscitated to which she responded and was discharged after 5 days.

Case 8:44 year male came with history of consumption of Celphos on admission patient GCS was full heart rate 68/min, BP non recordable SPO2 non recordable ABG shows PH -7.261, PCO2-13.5, P02-115.5, HCO3-5.9, Lactate -16.07,Urea-45mg% Creatinine-1.26mg%, Sodium-140 mmol/l, Potassium -3.57 mmol/l, Hb-14gm%, TLC-7600, Platelet 1,67.000, PT-22, INR-1.68 patient was resuscitated but had cardiac arrest after 2 hours and couldn't be revived.

Case 9: 20 year male came with history of consumption of Celphos on admission patient GCS was full heart rate 108/min BP non recordable SPO2 88% ABG shows PH-7.215, PCO2-25.3, P02-171, HCO3-10 , Lactate-14.05, Urea-25mg%, Creatinine-1.61mg%, Sodium-137 mmol/l, Potassium-4.36 mmol/l, Hb-14.3 gm%, TLC-16900, Platelet 3,05,000, PT-19.1, INR-1.53. Patient was resuscitated but had cardiac after 5 hours and couldn't be revived.

Case10:50 year male came with history of consumption of Celphoson admission patient GCS was full heart rate 102/min BP non recordable SPO2 88% ABG shows PH-7.336, PCO2-16.1, P02-86.1, HO3-8.4, Lactate-12.99, Urea-15 mg%, Creatinine-1.38mg%, Sodium-144 mmol/l, Potassium-3.91 mmol/l, Hb-19gm%, TLC-17400, Platelet 1,56.000, PT-18.7, INR-1.49 patient was resuscitated but had cardiac arrest 10 hours and couldn't be revived.

Case11:31 year male came with history of consumption of Celphos on admission patient GCS was full heart rate 152/min BP non recordable SPO2 93% ABG shows PH-7.82, PCO2-42.2, P02-17.2, HCO3-12.13, Lactate-15.13, Urea-25mg%, Creatinine-1.06mtg%, Sodium-143 mmol/l, Potassium-3.01mmol/l, Hb-12.9gm%, TLC-14100, Platelet 1,99.000, PT-21.8, INR-1.80. Patient was resuscitated but had cardiac arrest after 3 hours and couldn't be revived.

Case 12: 27 year male came with history of consumption of Celphos on admission patient GCS was full heart rate 130/min BP non recordable SPO2 92% ABG shows PH-7.2271, PCO2-30.2, P02-39.9, HCO3-13.6, Lactate-9.39, Urea-27mg%, Creatinine-1.0mg%, Sodium-137mmol/l, Potassium-3.90, Hb-13.7gm%, TLC-15600, Platelet 1,73.000, PT-21.8, INR-1.80 patient was resuscitated but had cardiac arrest after 8 hours and couldn't be revided.

Case 13:32 year male came with history of consumption of Celphos on admission patient GCS was full heart rate 96 / min, BP 90/60 SPO2 96% ABG shows PH-7.383, PCO2-21.6, P02-89.6, HOC3-12.6, Lactate – 7.56, Urea – 82mg%, Creatinine – 1.15mg%, Sodium – 139 mmol/l, Potassium – 3.84 mmol/l, Hb-13.8 gm%, TLC – 11600, Platelet 75.000, PT – 14.0 INR-1.19 patient was resuscitated to which he responded and discharged after 3 days.

Case14: 22 year male came with history of consumption of Celphos on admission patient GCS was full heart rate 140/min, BP 70/40 mm/hg SPO2 93% ABG shows PH-7.42, PCO2-42.5, P02-78.5, HCO3-26.9, Lactate – 1.39, Urea-30mg%, Creatinie-0.84mg%, Sodium-139 mmol/l, Potassium – 4.51 mmol/l, HB-14.0 gm%, TLC-7000, Platelet 1,65.000, PT-16.0, INR – 1.19 patient was resuscitated to which he responded and discharged after 3 days.

Case 15: 22 year male came with history of consumption of Celphos on admission patient GCS was full heart rate 78/min, BP 120/80 mm/hg SPO2 98% ABG shows PH-7.40, PCO2-36.3, P02-75.2, HCO3-22.1, Lactate – 0.6, Urea-22mg% Creatinine – 1.05mg%, Sodium -142 mmol/l, Potassium-397mmol/l, Hb-12.9m%, TLC -13400, Platelet 1,08.000, PT – 18.0, INR-1.39 patient was resuscitated to which he responded and discharged after 2 days.

INVESTIGATIONS

	1	2	3	4	5	6	7	8
PH	6.898	7.37	7.334	7.39	7.45	7.45	7.46	7.261
PCO2	25.4	28.9	41.3	38.3	22.1	35.9	36.90	13.5
PO2	63.4	84.3	75.6	47.7	62.4	113.90	120.70	115.5
Lactate	Out of range	1.02	2.41	8.41	6.76	0.03	3.27	16.07
HCO3	4.9	16.3	21.3	20.4	15.0	24.60	25.4	5.9
Na+	141	140	143	140	136	145	140	140
K+	4.74	3.88	3.76	3.46	4.04	3.5	3.28	3.57
Urea	24	27	30	17	14	13	23	45
Creatinine	1.41	0.66	1.16	1.18	0.70	1.03	0.86	1.26
Hb	12.3	12.0	13.6	13.1	14.7	15.2	12.3	14
TLC	38,200	10,500	6,900	12,300	12,000	13,200	10,700	76,00
Platelet count	5,43,000	1,39,000	2,45,000	2,62,000	1,13,000	2,00,0000	1,82,000	167000
PT	18.7	16	16	15	18.3	16	18.3	22
INR	1.49	1.29	1.29	1.19	1.44	1.29	1.44	1.68

	9	10	11	12	13	14	15
PH	7.215	7.336	7.082	7.271	7.383	7.42	7.40
PCO2	25.3	16.1	42.2	30.2	21.6	42.5	36.3
PO2	171	86.1	17.2	39.9	89.6	78.5	75.2
Lactate	14.05	12.99	15.13	9.39	7.56	1.39	0.86
HCO3	10.0	8.4	12.13	13.6	12.6	26.9	22.1
Na+	137	144	143	137	139	139	142
K+	4.37	3.91	3.01	3.90	3.84	4.51	3.97
Urea	25	15	25	27	82	30	22
Creatinine	1.61	1.38	1.06	1.0	1.15	0.84	1.05
Hb	14.3	19.0	12.9	13.7	13.8	14	12.9
TLC	1,69,00	1,74,00	1,41,00	15,600	11,600	7000	13400
Platelet count	305000	156000	199000	173000	75000	165000	108000
PT	19.1	18.7	21.8	21.8	14.0	16.0	18.0
INR	1.53	1.49	1.80	1.80	1.19	1.19	1.39

Age Group	**Male**	**Female**
16-20	1	1
21-25	4	-
26-30	1	-
31-35	4	-
36-40	-	-
36-40	-	-
41-45	1	-
46-50	1	-
51-55	1	-
56-60	-	1

HEMODYNAMIC READINGS

	1	2	3	4	5	6	7	8
HR on admission	102	90	90	102	92	104	90	68
BP on admission	NR	80/60	130/90	120/80	110/70	160/100	100/70	NR
SPO2 (%)	93	96	98	96	96	98	96	NR

	9	10	11	12	13	14	15
HR on admission	108	102	152	130	96	140	78
BP on admission	NR	NR	NR	NR	90/60	70/40	120/80
SPO2 (%)	NR	88	93	92	96	93	98

DISCUSSION OF MANAGEMENT

Patient came to us with clinical manifestations like gastrointestinal symptoms (Nausea, Vomiting, epigastric discomfort), metabolic acidosis & hemodynamic instability, ECG changes/elevated cardiac enzymes, myocardial toxicity and alteration of sensorium(8)

Gastric lavage with mixture of 50ml sodabicarbonate & 50ml coconut oil was done immediately then injection sodabicarbonate 50ml intravenous stat and later as per ABG findings either as infusion or 25ml six hourly given. Injection magnesium sulphide 2gm IV stat followed by 1gm every six hourly was given(1).

Apart from this injection hydrocortisone 100mg IV stat followed by 100mg every 8 hourly, injection Avil 1ampoule IV stat followed by 1 ampoule every 8 hourly, injection pantoprazole40mg IV stat followed by 40mg every 12 hourly, injection ondansetron 4mg IV stat followed by 4mg every 12 hourly. Also, we started 20% intralipid infusion @ 10ml per hour. All baseline investigations, ECG, Chest X-ray done. All patients were catheterized. The gastric lavage sample was preserved for toxicology. If the patient was hemodynamically unstable than invasive blood pressure monitoring along with inotrope support started. Oxygen administered by mask @ 2-3 litres per minute and relatives were explained regarding the poor prognosis and outcome.

CONCLUSION

From our case series of 15 patients. We found that mortality was more in patient having metabolic acidosis & hemodynamic instability.

In our case series. Mortality case was 40% and 60% survived from AluminiumPhosphide poisoning.

REFERENCES

1. Mukesh Kumar Samota et al. Survival of a case of Celphos poisoning with damaged liver function and acute renal failure. J. Evid Based Med. Healthc 2017; 91(4): 5521-22.
2. Siwach SB, Gupta A. The profile of acute poisoning in Haryana-Rohtak study. J Asoc Physicians India 1995; 43(11): 756-59.
3. Goel A, Aggarwal P. Pesticide poisoning. Natt Med J India 2007; 20(4): 182-191.
4. Chugh SN. Aluminum Phosphide Poisoning: Present status and management. J Assoc Physicians India 1992; 40(6): 401-05.
5. MC Meena, S. Mittal, Y Rani. Fatal AluminumPhosphidePoisoning2015; 8(2): 65-67.
6. Gurjar M, Baronia AK, Azim A, Sharma K. Managing AluminumPhosphide Poisoning. J Emerg Trauma Shock. 2011;4: 378-84.
7. Chugh SN, et al. Incidence and outcome of Aluminum Phosphide Poisoning in a hospital study. Indian J Med Res 1991; 94:232-35.
8. Bansal P, Giri S, Bansal R, Tomar LR. Survival in a case of Aluminum Phosphide Poisoning with severe myocardial toxicity. Indian Journal of Health Sciences and Biomedical research. 2017; 3(10): 343-46.
9. Sumit Bhatnagar, Vishnu Pal, Rare survival after Aluminum Phosphide Poisoning after myocarditis with L-Carnitine and Steroid. International Journal of Pharmacy and Pharmaceutical Sciences. 2015; 9(7): 522-23.6