



Study of ECG Manifestations in Patients of Aluminum Phosphide Poisoning

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Abstract

Alphos or poisoning (Celphos), a solid fumigant pesticide is widely used as a grain preservative grain preservative. It is one of the dreaded poisons, although found all over the world but more common in developing countries like India. Several ECG changes ranging from ST segment elevation/depression, PR and QRS interval prolongation, complete heart block to ectopics and fibrillation have been observed. Reversible myocardial injury has also been reported. Controversy exists regarding the various ECG changes seen in association with aluminum phosphide poisoning. Some authors have indicated in their studies that ECG abnormalities were poor prognostic markers other have observed that presence of ECG abnormalities did not predict mortality. Persistence of ECG changes after symptomatic recovery is a dilemma for the attending physician. There is paucity of literature and no specific guidelines are available for their management. A clinical profile of 45 patients, who got admitted in Intensive Care Unit (ICU) of sree Mookambika institute with alleged intake of celphos pellets, was studied. In all the 45 patients with alleged celphos poisoning, extensive gastric lavage was done with a mixture of coconut oil, KMNO₄ and sodium bicarbonate solution. And patients were given symptomatic treatments along with supportive hemodynamic care with help of invasive and non-invasive monitoring. ECG changes and other blood parameters were recorded and studied at admission and during the course of hospital admission. Out of 45 patients, 23 patients (51.11%) succumbed and 22 patients (48.89%) were successfully discharged, reflecting the graveness of this poisoning. Cardiotoxicity represents the primary cause of death in acute aluminium phosphide poisoning. ECG monitoring was done for all the patients. Most common physiological ECG manifestation was sinus tachycardia and most common pathological ECG manifestation was atrial fibrillation. On 1st day of admission, mortality rate for normal sinus rhythm was 16.67%, for patients with sinus tachycardia it was 33.3% and sinus bradycardia it was 25%. Among rhythm disturbances viz. Supra ventricular tachycardia, ventricular tachycardia and atrial fibrillation and ST-T changes 100% mortality was seen. On day 3rd of admission among patients with sinus rhythm 12.50% patients died, With sinus bradycardia none died while with sinus tachycardia there was 50% mortality. Again 100% mortality was observed in patients with atrial fibrillation but 83.33% patient with ventricular tachycardia. 100% mortality was observed with T wave inversion on day 3. On day 5th, T wave inversion was found most commonly (13.33%) besides sinus bradycardia (20%) followed by ventricular tachycardia and supra ventricular tachycardia. No mortality was observed in patients having T wave changes but 100% mortality in patients with supra ventricular tachycardia and ventricular tachycardia. Majority of our study population consist of younger male adults. Most of the patients consumed 1 tablet of Aluminium phosphide and significant correlation between amount of celphos ingested and mortality was found. Out of 45 patients, 23 patients (51.11%) succumbed and 22 patients (48.89%) were successfully discharged, reflecting the graveness of this poisoning. Hypotension, low SPO₂ at the time of admission is associated with bad prognosis and high case fatality. Biochemically high CPK-MB, high creatinine, high SGPT is associated with grave prognosis. Patients who had persistent ECG changes from day 1 to day 5 and beyond have grave prognosis than those whose ECG changes only appeared later on in the course of their admission. Development of persistent tachy arrhythmia since day 1 is poor prognostic sign. The ECG changes appearing late after 5 days may be non-fatal. Death in first 24 hours appears to be cardiogenic as evidenced by shock and fatal arrhythmia.

INTRODUCTION

Aluminum phosphide poisoning (Celphos), a solid fumigant pesticide is widely used as a grain preservative grain preservative. It is one of the dreaded poisons, although found all over the world but more common in developing countries like India^[1]. Celphos poisoning has always been a significant problem and threat for intensivists around the world, perhaps as a result of the lack of an antidote (unlike organ phosphates) and high case fatality. Multiple ECG abnormalities, including ectopics and fibrillation, ST segment elevation/depression, PR and QRS interval prolongation, complete heart block, have been noted. One has to be vigilant to look after these ECG changes, as it can precipitate into dangerous ventricular fibrillation and ultimately death. The ECG changes in Aluminum Phosphide poisoning are transient and reversible in most cases within few days as the patient becomes asymptomatic. Controversy exists regarding the various ECG changes seen in association with aluminum phosphide poisoning. Some authors have indicated in their studies that ECG abnormalities were poor prognostic markers other have observed that presence of ECG abnormalities did not predict mortality. Persistence of ECG changes after symptomatic recovery is a dilemma for the attending physician. There is paucity of literature and no specific guidelines are available for their management.

Aims and Objectives of the Study:

- Primary Objective Study.
- Secondary Objective.
- Study of clinical, hematological and biochemical profile of patients of celphos.
- CPK-MB as a marker of cardiac involvement in each patient and its co-relation with outcome.

MATERIALS AND METHODS

The present study entitled "ECG manifestations in patients of aluminium phosphide poisoning" was carried out in Department of emergency medicine in Sree Mookambika College of Medical Sciences after taking ethical clearance from Institutional Ethical Committee. In our study we evaluated variable cardiac manifestations in form of electrographic findings (based on isolated 12 lead ECG records) in 45 patients of aluminium phosphide poisoning. ECG changes. ECG was recorded on day 1, day 3 and day 5 of admission. Temporal correlation in ECG changes were noted during hospitalization. The study duration was of one and half years from 1st March 2023 to 31st August 2024. The age, gender, ingested dose, time elapsed between ingestion and hospitalization, hemodynamic parameters i.e., systolic and diastolic blood pressure (DBP), heart rate, SPO₂, score on Glasgow coma scale was recorded and CPKMB (creatinine

phospholipase-myocardial band), hematological, liver and kidney function test and ECG manifestations for each patient was recorded at the time of admission and at serial interval till death or discharge from the hospital. Most of them had features of cardiogenic shock, hypotension and arrhythmias, while few got admitted with respiratory distress and rest of them had multitude of symptoms related to different organ systems. On admission to ICU, the patients were made comfortable on the bed, monitoring gadgets were attached for Heart Rate (HR), Non-Invasive Blood Pressure (NIBP), ECG, Pulse Oximetry (SpO₂) and Ryle's tube was inserted through nasal route. The patients who were grossly unstable hemodynamically or had respiratory distress were sedated with midazolam and intubated. Endotracheal intubation was done with appropriate size cuffed endotracheal tube and patients were put on mechanical ventilation. Gastric lavage was initiated with KMnO₄, 50 ml of coconut oil and 50 ml of sodium bicarbonate solution and continued for the next half an hour, with simultaneous aspiration being done after every 2-3 minutes through Ryle's tube for 1 hour. An intravenous access through internal jugular vein was established for central venous pressure monitoring as well as for guiding the fluid therapy in majority of patients who had presented with cardiovascular instability, respiratory distress and renal failure. In a few patients who had severe cardiogenic shock, an arterial line was also secured through radial/dorsalis pedis artery for observing beat to beat variation of HR and BP. Symptomatic treatment was initiated on a patient to patient basis. Magnesium sulfate, dopamine, dobutamine, amiodarone infusions and other appropriate intravenous drugs were given depending on the patient's clinical presentation and symptomatology, as well as arrhythmia and blood pressure variations. Urine output was monitored through Foley's catheter attached to uro bag. Patients who required mechanical ventilation were kept sedated with injection midazolam. During this period, strict and vigil monitoring of all vital parameters was done and treatment regimens were titrated according to the clinical condition of the patients. Gastric lavage was again performed after 1 hour of admission with the same solution for next half an hour. After admission in the ICU, all the baseline routine and specific investigations were carried out including regular arterial blood gas analysis (ABG). Soda bicarbonate was given empirically to all patients in a dose of 1-1.5 mEq/kg body weight and further adjusted for correction of metabolic acidosis as per ABG reports. At the end of the study period, all the data were arranged systematically and were subjected to statistical analysis using non-parametric tests. Value of P<0.05 was taken as significant value. Statistical analysis was done using the statistical package for social sciences (SPSS). Different statistical methods were used as appropriate. Mean±SD was determined

for quantitative data and frequency for categorical variables. The independent t-test was performed on all continuous variables. The normal distribution data was checked before any t-test. The Chi-Square test was used to analyze group difference for categorical variables. A p-value <0.05 was considered significant.

RESULTS AND DISCUSSIONS

Table 1: Distribution of Age (Years) of Study Subjects

Age(years)	Frequency	Percentage
15-20	9	20.00%
21-30	20	44.44%
31-40	6	13.33%
>40	10	22.22%
Total	45	100%

Majority of our cases lie in the age group 21-30 years accounting for 44.44% followed by those >40 years (22.22%). 9 (20%) were of 15-20 years old and rest 6 (13.33%) were between 31-40 years.

Table 2: Distribution of Gender of Study Subjects

Gender	Frequency	Percentage
Female	13	28.89%
Male	32	71.11%
Total	45	100.00%

Out of total 45 patients male patients were more-32 (71.11%) than female-13 (28.11%).

Table 3: Distribution of Amount of Celphos Tablets of Study Subjects

Amount of Celphos tablets	Frequency	Percentage
1	24	53.33%
1.5	1	2.22%
2	16	35.56%
3	3	6.67%
4	1	2.22%
Total	45	100.00%

Majority of patients took 1 tablet around half of the cases-24 (53.33%) followed by 2 tablets-16 cases (35.56%). 3 patients took 3 tablets and only 1 patient took >4 tablets (2.22%).

Table 4: Descriptive Statistics of Time Since Ingestion and Hospitalization of Study Subjects

Time since ingestion and hospitalization	Frequency	Percentage
<4 hours	6	13.33%
4-6 hours	31	68.89%
>6 hours	8	17.78%
Total	45	100.00%

Majority (31-68.89%) patients got hospitalized between 4-6 hours of celphos ingestion, followed by 8 patients after 6 hours and only 6 patients(13.33%) in <4 hours.

Table 5: Distribution of GCS of Study Subjects

GCS	Frequency	Percentage
12-15	25	55.55%
9-11	15	33.33%
6-8	2	4.44%
3-5	3	6.67%
Total	45	100.00%

Majority of the patient presented with a GCS score of 12 or higher around 55.55% patients. 5 (11.11%) patients presented with GCS below 8 and around

15patients (33.33%), presented with GCS in between (9-11).

Table 6: Distribution of Vitals of Study Subjects

Vitals	Frequency	Percentage
Blood pressure at the time of admission		
<90/60 mm Hg	20	44.44%
90/60 -120/80 mm Hg	24	53.33%
Pre HTN (120/80-139/89) mm hg	0	0
Stage 1 HTN (SBP=140-159)(DBP=90-99)	1	2.22%
Stage 2 HTN (SBP>160), (DBP>100)	0	0
SpO2 on room air		
<=90%	14	31.11%
>90%	31	68.89%
Pulse rate (bpm)		
<60/minute	3	6.67%
60-100/minute	32	71.11%
>100/minute	10	22.22%

53.33% patients had blood pressure within normal range. 20 patients (44.44%) presented in shock and only 1 patient presented in stage 1 hypertension. Around 2/3rd of patients had SpO2 >90% (31-68.89%). And 14 patients had Spo2 <90% (31.11%). Majority of patients had pulse rate within normal physiological limit-71% (32 patients). 3 patients (6.67%) had bradycardia or pulse rate below 60 on presentation and 10 patients (22.22%) had pulse rate >100/min.

Table 7: Distribution of Lab Parameters of Study Subjects

Lab parameters	Frequency	Percentage
Hemoglobin(g/dL)		
<11 g/dL	7	15.56%
>=11 g/dL	38	84.44%
Total leucocyte count(cells/cumm)		
<11,000 cells/cumm	39	86.67%
>=11,000 cells/cumm	6	13.33%
Platelet count(lacs/cumm)		
>=1 lacs/cumm	45	100.00%
Random blood sugar(mg/dL)		
<70 mg/dL	10	22.22%
>=70 mg/dL	35	77.78%
Serum creatinine(mg/dL)		
<=1.3 mg/dL	34	75.56%
>1.3 mg/dL	11	24.44%
Urea(mg/dL)		
<=40 mg/dL	41	91.11%
>40 mg/dL	4	8.89%
Bilirubin(mg/dL)		
<=1.2 mg/dL	39	86.67%
>1.2 mg/dL	6	13.33%
SGOT(IU/L)		
<=40 IU/L	27	60.00%
>40 IU/L	18	40.00%
SGPT(IU/L)		
<=40 IU/L	28	62.22%
>40 IU/L	17	37.78%
Sodium(mEq/L)		
<135 mEq/L	3	6.67%
135-155 mEq/L	42	93.33%
Potassium(mEq/L)		
<3.5 mEq/L	3	6.67%
3.5-5.5 mEq/L	41	91.11%
>5.5 mEq/L	1	2.22%
CPKMB(IU/L)		
<25 IU/L	13	28.89%
25-70 IU/L	11	24.44%
>70 IU/L	21	46.67%

Majority of subjects had normal hemogram parameters including total leucocyte count, hemoglobin and platelet count. 10 patients presented with hypoglycemia. 24.44% (approx. 1/4th patients)

had deranged serum creatinine of >1.3 but only 4 patients that is 8.89% have raised urea levels. CPK-MB a very important prognostic parameter was found to be within normal limit in only 13(28.89%) cases and between 25 to 70 IU/L in 24.44% cases and above 70 IU/L in around half of the cases-21 (46.67%). Serum electrolytes were almost within normal range in all patients only 3 (6.67%) patients had serum sodium below 135 meq/dl and only 4 patients had deranged potassium values. Derangement in LFT (Liver function test) could be seen. Around 13.33% patients had bilirubin >1.2 mg/dl and approximately 40% patients had raised SGOT and SGPT values above 40 IU/L.

Table 8: Distribution of ECG Changes on Day 1 of Study Subjects

ECG changes on day 1	Frequency	Percentage
Rate		
Sinus tachycardia	9	20.00%
Sinus bradycardia	8	17.78%
Rhythm		
Sinus rhythm	12	26.67%
Supra ventricular tachycardia	1	2.22%
Ventricular tachycardia	4	8.89%
Atrial fibrillation	5	11.11%
Atrial flutter	0	0.00%
ST changes		
ST segment elevation	2	4.44%
ST segment depression	1	2.22%
T changes		
Peaked T waves	1	2.22%
T wave inversion	3	6.67%

Cardiotoxicity represents the primary cause of death in acute aluminium phosphide poisoning. ECG monitoring was done for all the patients. Most common physiological ECG manifestation was sinus tachycardia and most common pathological ECG manifestation was atrial fibrillation. On day 1st of admission 12 patients (26.67%) had sinus rhythm. Rate disturbances were found in 17 patients, 9 (20%) had sinus tachycardia and 8 (17.78%) had sinus bradycardia. 10 patients had rhythm disturbances, out of which most common was atrial fibrillation (11.11%) followed by ventricular tachycardia (8.89%) then supra ventricular tachycardia 1 (2.22%). ST changes were found in 3 patients out of which 2 (4.44%) had ST segment elevation and 1 had ST segment depression (2.22%). Peaked T wave could be seen in 1 patient (2.22%) and T Wave Inversion in 3 (6.67%) patients. Overall 33 patients had one or other ECG changes, with rate disturbances seen on 17 patients and rest 21 patients had other rhythm or ST changes.

Table 9: Distribution of ECG Changes on Day 3 of Study Subjects

ECG changes on day 3	Frequency	Percentage
Rate		
Sinus tachycardia	2	6.06%
Sinus bradycardia	5	15.15%
Rhythm		
Sinus rhythm	16	48.48%
Supra ventricular tachycardia	0	0.00%
Ventricular tachycardia	6	18.18%
Atrial fibrillation	2	6.06%
Atrial flutter	0	0.00%
ST changes		
ST segment elevation	0	0.00%
ST segment depression	0	0.00%
T changes		
Peaked T waves	1	2.22%
T wave inversion	1	2.22%
TOTAL	33	100%

On day 3rd out of 33 patients remaining 16 (48.48%) had sinus rhythm, 6.06% (2) had sinus tachycardia and 15.15% (5) had sinus bradycardia. Ventricular tachycardia could be seen as major ecg manifestations on day 3rd in 18.18% patients followed by atrial fibrillation (6.06%). Hyper acute T waves and T wave inversion together accounts for 4.44%.

Table 10: Distribution of ECG Changes on Day 5 of Study Subjects

ECG changes on day 5	Frequency	Percentage
Rate		
Sinus tachycardia	1	6.67%
Sinus bradycardia	3	20.00%
Rhythm		
Sinus rhythm	7	46.67%
Supra ventricular tachycardia	1	6.67%
Ventricular tachycardia	1	6.67%
Atrial fibrillation	0	0.00%
ST changes		
ST segment elevation	0	0.00%
ST segment depression	0	0.00%
T changes		
Peaked T waves	1	6.67%
T wave inversion	2	13.33%

On 5th day out of 16 patients, 7 (46.67%) patients were having normal ECG. 6.67%^[1] patient had sinus tachycardia and 3 patients (20%) had sinus bradycardia. Among rhythm disturbances 6.67%^[1] patient had supra ventricular tachycardia and 6.67%^[1] patient presented with ventricular tachycardia. 3 patients had T wave changes (20%).

Table 11: Distribution of Outcome of Study Subjects

Outcome	Frequency	Percentage
Death	23	51.11%
Discharge	22	48.89%
Total	45	100.00%

The mortality rate in celphos poisoning is very high. In our study also we found death of 52.11% (23 patients) and 48.89% patients (22) got discharged.

Table 12: Association of Age (Years) with Outcome

Age(years)	Death(n=23)	Discharge(n=22)	Total	P value
15-20	4 (44.44%)	5 (55.56%)	9 (100%)	0.946*
21-30	10 (50%)	10 (50%)	20 (100%)	
31-40	3 (50%)	3 (50%)	6 (100%)	
>40	6 (60%)	4 (40%)	10(100%)	
Mean±SD	32.17±12.97	28.77±10.03	30.51±11.62	0.487§
Median(25th-75th percentile)	27(24-40)	26(22.25-35.25)	27(23-36)	
Range	18-65	15-49	15-65	

§ Mann Whitney test, * Fisher's exact test

Age based mortality variation was not found in our study. Mortality rate is comparable among all age groups 50% mortality in age group 21-30 years and 60% in patients above 60 years.

Table 13: Association of Gender with Outcome

Gender	Death(n=23)	Discharge(n=22)	Total	P value
Female	8 (61.54%)	5 (38.46%)	13 (100%)	0.372†
Male	15 (46.88%)	17 (53.13%)	32 (100%)	
Total	23(51.11%)	22 (48.89%)	45 (100%)	

† Chi square test

The mortality rate among both the groups was comparable **61.66%** in females and **46.68%** in males. Thus gender does not have effect on mortality (p value =0.327).

Table 14: Association of Amount of Celphos Tablets with Outcome

Amount of Celphos tablets	Death (n=23)	Discharge (n=22)	Total	P value
1	4 (16.67%)	20 (83.33%)	24 (100%)	<.0001*
1.5	0 (0%)	1 (100%)	1 (100%)	
2	15 (6.25%)	16 (100%)	31 (93.75%)	
3	3 (100%)	0 (0%)	3 (100%)	
4	1 (100%)	0 (0%)	1 (100%)	
Total	23 (51.11%)	22 (48.89%)	45 (100%)	

* Fisher's exact test

Mortality rate in patients with 1 tablet ingestion was only 16.67% but as the number of tablets increases mortality rate also increased with **93.75% mortality** in patients with consumption of 2 tablets to **100% mortality** in patients who have taken 3 or 4 tablets. There is significant association of mortality with number of tablets (**p value=0.0001**).

Table 15: Association of Time Since Ingestion and Hospitalization with Outcome

Time since ingestion and hospitalization	Death(n=23)	Discharge(n=22)	Total	P value
<4 hours	1 (16.67%)	5 (83.33%)	6 (100%)	0.207*
4-6 hours	17 (54.84%)	14 (45.16%)	31 (100%)	
>6 hours	5 (62.50%)	3 (37.50%)	8 (100%)	
Total	23 (51.11%)	22 (48.89%)	45 (100%)	

* Fisher's exact test

Mortality rate in patients who arrive before 4 hours was 16.67% but not much difference in mortality rate could.

Table 16: Association of GCS with Outcome

GCS	Death(n=23)	Discharge(n=22)	Total	P value
12-15	5(20%)	20(80%)	25(100%)	0.0001*
9-12	13(86.67%)	2(13.33%)	15 (100%)	
7-9	2(100%)	0(0%)	2(100%)	
3-6	3 (100%)	0(0%)	3 (100%)	
Total	23(51.11%)	22 (48.89%)	45 (100%)	

* Fisher's exact test

On 1st day of admission, mortality rate for normal sinus rhythm was 16.67%, for patients with sinus tachycardia it was 33.3% and sinus bradycardia it was 25%. Among rhythm disturbances viz. Supra ventricular tachycardia, ventricular tachycardia and atrial fibrillation and ST-T changes 100% mortality was seen. On day 3rd of admission among patients with sinus rhythm 12.50% patients died, With sinus bradycardia none died while with sinus tachycardia there was 50% mortality. Again 100% mortality was observed in patients with atrial fibrillation but 83.33% patient with ventricular tachycardia. 100% mortality was observed with T wave inversion on day 3. On day 5th, T wave inversion was found most commonly(13.33%) besides sinus bradycardia (20%) followed by ventricular tachycardia and supra ventricular tachycardia. No mortality was observed in patients having T wave changes but 100% mortality in Patients with supra ventricular tachycardia and ventricular tachycardia. The age, gender, ingested dose, time elapsed between ingestion and hospitalization, hemodynamic parameters i.e., systolic and diastolic blood pressure (DBP), heart rate, SPO₂, score on glassgow coma scale was recorded and CPKMB (creatinine phosphokinase-myocardial band), hematological, liver and kidney function test and ECG

manifestations for each patient was recorded at the time of admission and at serial interval till death or discharge from the hospital. Total sample size was 45, out of which 23 patients (51.11%) died and 22 patients (48.89%) were successfully discharged. similar findings were observed in study (6) in which 59.3% mortality was observed. In our study majority of patients were of age group 21-30 years old indicating prevalence of suicide by celphos ingestion more among younger population of 2nd-3rd decade. There was male preponderance in our study, 71.11% (32 patients) of our population were male. Similar findings were observed in other studies^[1] in which 60% of patients were male and mean age was 27±8.7 years. Most of the patient had consumed 1 celphos tablet (53.33%). A very significant correlation in mortality could be observed with the number of tablets ingested with 16.67% mortality in patients who have consumed 1 tablet to 93.75% mortality in patients with 2 tablets ingestion and 100% in patients with consumption of 4 tablets. (With a significant p value=<0.0001). This is in agreement with other studies^[1]. The time interval between ingestion and arrival at the hospital was found to be 4-6 hours in maximum patients (68.89% patients=31 subjects). No significant correlation in survivor and non survivor percentage was found in those arrived in 4-6 hours (54.84% mortality) and after 6 hours (62.50% mortality) (insignificant p value =0.207). This is in agreement with the findings of Mathai and Bhanu, Christian Medical college^[4] who also found no significant association between the time delay in presentation to the hospital with mortality. Significant association between GCS score and mortality could be established with 20% death in patients with GCS 12-15 to 100% mortality in Similar correlation of mortality was observed with Spo₂, of 14 patients presenting with saturation <90%, 92.86% (13) patients died whereas 67.74% (21 patients) gets discharged out of 31 presenting with initial saturation >90%. (P value <0.0002). No significant relation could be established with pulse rate in our study with 66.67% in patients with pulse rate <60/min, 40.63% in patients with pulse rate between 60-100/min and 80% in patients with pulse rate <100/min (insignificant p value =0.072) All the basic routine blood investigations and CPK-MB was done for all patients on day of admission and thereafter till death or discharge of the patient. Significant association with CPK-MB level with mortality is found in our study (P value=0.0001) with 7.21% mortality in patients with CPK-MB <25 to 95.24% with CPK-MB >70 IU/L. These findings correlate well with the studies already published^[3]. On day 1, sinus rhythm was found in approximately one-fourth of population (26.67%) followed by sinus bradycardia (17.78%) and sinus tachycardia (20%). Apart from rate disturbances atrial fibrillation (11.11%) was found as most common ECG, on day 1, followed by ventricular tachycardia (8.89% patients) Raman^[2] found in their study Supra ventricular tachycardia and atrial flutter/

Table 17: Association of Vitals with Outcome

Vitals	Death(n=23)	Discharge(n=22)	Total	P value
Blood pressure at the time of admission				
<90/60 mm Hg	18 (90%)	2 (10%)	20 (100%)	<.0001*
90/60-120/80 mm Hg	5 (20.83%)	19 (79.17%)	24 (100%)	
Stage 1 HTN (SBP=140-15) (DBP=90-9)	0 (0%)	1 (100%)	1 (100%)	
Spo2 on room air				
<=90%	13 (92.86%)	1 (7.14%)	14 (100%)	0.0002*
>90%	10 (32.26%)	21 (67.74%)	31 (100%)	
Pulse rate (bpm)				
<60/minute	2 (66.67%)	1 (33.33%)	3 (100%)	

Table 18: Association of Lab Parameters with Outcome

Lab parameter s	Death(n=23)	Discharge(n=22)	Total	P value
Hemoglobin(g/dL)				
<11 g/dL	4 (57.14%)	3 (42.86%)	7 (100%)	1*
>=11 g/dL	19 (50%)	19 (50%)	38 (100%)	
Total leucocyte count(cells/cumm)				
<11,000 cells/cumm	21(53.85%)	18 (46.15%)	39 (100%)	0.414*
>=11,000 cells/cumm	2 (33.33%)	4 (66.67%)	6 (100%)	
Platelet count(lacs/cumm)				
>=1 lacs/cumm	23(51.11%)	22 (48.89%)	45 (100%)	NA
Random blood sugar(mg/dL)				
<70 mg/dL	7 (70%)	3 (30%)	10 (100%)	0.284*
>=70 mg/dL (45.71%)	16 (54.29%)	35 (100%)		
Serum creatinine(mg/dL)				
<=1.3 mg/dL	13(38.24%)	21 (61.76%)	34 (100%)	0.004*
.1.3 mg/dL	10(90.91%)	1 (9.09%)	11 (100%)	
Urea(mg/dL)				
<=40 mg/dL	21(51.22%)	20 (48.78%)	41 (100%)	1*
>40 mg/dL	2 (50%)	2 (50%)	4 (100%)	
Bilirubin(mg/dL)				
<=1.2 mg/dL	19 (48.72%)	20 (51.28%)	39 (100%)	0.665*
>1.2 mg/dL	4 (66.67%)	2 (33.33%)	6 (100%)	
SGOT(IU/L)				
<=40 IU/L	11(40.74%)	16 (59.26%)	27 (100%)	0.088†
>40 IU/L	12(66.67%)	6 (33.33%)	18 (100%)	
SGPT(IU/L)				
<=40 IU/L	10(35.71%)	18 (64.29%)	28 (100%)	0.013*
>40 IU/L	13(76.47%)	4 (23.53%)	17 (100%)	
Sodium(mEq/L)				
<135 mEq/L	2 (66.67%)	1 (33.33%)	3 (100%)	1*
135-155 mEq/L	21 (50%)	21 (50%)	42 (100%)	
Potassium(mEq/L)				
<3.5 mEq/L	2 (66.67%)	1 (33.33%)	3 (100%)	1*
3.5-5.5 mEq/L	20(48.78%)	21 (51.22%)	41 (100%)	
>5.5 mEq/L	1 (100%)	0 (0%)	1 (100%)	
CPKMB(IU/L)				
<25 IU/L	1 (7.69%)	12 (92.31%)	13 (100%)	<.0001†
25-70 IU/L	2 (18.18%)	9 (81.82%)	11 (100%)	
>70 IU/L	20(95.24%)	1 (4.76%)	21 (100%)	

* Fisher's exact test, † Chi square test

Table 19: Association of ECG Changes on Day 1 with Outcome

changes on day 1	Death(n=23)	Discharge(n=22)	Total
RATE			
Sinus tachycardia	3 (33.33%)	6 (66.67%)	9 (100%)
Sinus bradycardia	2 (25%)	6 (75%)	8 (100%)
RHYTHM			
Sinus rhythm	2 (16.67%)	10 (83.33%)	12 (100%)
Supra ventricular rhythm	1 (100%)	0 (0%)	1 (100%)
Ventricular tachycardia	4 (100%)	0 (0%)	4 (100%)
Atrial fibrillation	5 (100%)	0 (0%)	5 (100%)
ST CHANGES			
ST segment elevation	2 (100%)	0 (0%)	2 (100%)
ST segment depression	1 (100%)	0 (0%)	1 (100%)
T WAVE CHANGES			
Peaked T waves	1 (100%)	0 (0%)	1 (100%)
T wave inversion	3 (100%)	0 (0%)	3 (100%)

fibrillation in 46.7% and 20% patients respectively. Ventricular tachycardia was recorded In 40% cases and ventricular fibrillation in 23.3% cases. 100% mortality was found in patients who had supra-ventricular rhythm, ventricular tachycardia, atrial fibrillation, ST segment elevation and depression, peaked T waves and T wave inversion on day 1. 25% case fatality rate was found in patients with sinus bardycardia. This was

in accordance with study done by Kambiz Soltaninejad and Mohammed^[5] who found significant correlation between ECG findings and mortality in celphos poisoning. Another study also found significant association of ECG changes with mortality.^[5,6] On day 3rd among ECG manifestations ventricular tachycardia was most common (18.18%) after rate disturbances (sinus tachycardia and sinus bradycardia) followed by

atrial fibrillation (6.06%). Mortality was again 100% in patients with atrial fibrillation and T wave inversion on day 3, whereas 83.33% mortality in patients with ventricular tachycardia. This was in coherence with the study done by M Louriz^[9] found all the 4 types of electric abnormalities (conduction disorders, cardiac dysrhythmias such as atrial fibrillation., depolarization disorders like ST segment elevation and T wave inversion) in variable frequency and also found them to be associated with mortality on uni variate analysis. (p value <0.001). On day 5th, T wave inversion was found most commonly(13.33%) besides sinus bradycardia (20%) followed by ventricular tachycardia and supra ventricular tachycardia (6.67% each) but no mortality was observed in patients having T wave changes but 100% mortality in patients with supra ventricular tachycardia and ventricular tachycardia. Thus ECG Changes on 3rd day or during the course of hospitalization in hospital have no significant association with mortality. These finding are similar to findings of previous studies^[7-9].

CONCLUSION

This study included 45 patients of Aluminum phosphide poisoning also known as celphos poisoning admitted in critical care unit of our tertiary care centre. Majority of our study population consist of younger male adults. Most of the patients consumed 1 tablet of Celphos and significant correlation between amount of celphos ingested and mortality was found showing better prognosis than few previous studies. Out of 45 patients, 23 patients (51.11%) succumbed and 22 patients (48.89%) were successfully discharged, reflecting the graveness of this poisoning. Hypotension, low SPO2 at the time of admission is associated with bad prognosis and high case fatality. Biochemically high CPK-MB, high creatinine, high SGPT is associated with grave prognosis. Patients who had persistent ECG changes from day 1-day 5 and beyond have grave prognosis than those whose ECG changes only appeared later on in the course of their admission. In survivors, the cardiotoxicity and hypoxia disappear within 5-7 days due to excretion of phosphine and restoration of normal cellular metabolism. The toxic chemical myocarditis leads to varied fatal and non-fatal ECG changes, 6-24 hours after ingestion in non-survivors, in the form of sinus tachycardia, sinus bradycardia, atrial fibrillation, ST-T changes, atrial fibrillation, ventricular ectopic beats, ventricular fibrillation, aberrant conduction and idioventricular rhythm terminally leading to asystole. Death in first 24 hours appears to be cardiogenic in origin. The serum electrolytes are within normal limits and not correlated with ECG changes. Since the survivors show complete normal ECG recovery, it denotes that the effect of poisoning is due to some reversible factor leading to disturbance in the permeability of sodium (Na⁺), potassium (K⁺), calcium (Ca²⁺) and magnesium (Mg²⁺) ions leading to change

in its transmembrane action potential due to focal myocardial involvement and subsequent myocardial necrosis, resulting in release of reactive oxygen species.

REFERENCES

1. Biswas M.D.G., 2021. Review of Forensic Medicine and Toxicology: Including Clinical and Pathological Aspects: As Per the Competency Based Medical Education Guidelines of Nmc. 5th Ed., Edn., Jaypee Brothers Medical Pub, ISBN-14: 978-9390281428.
2. Raman R. and M. Dubey., 1985. The electro cardiographic changes in quick phos poisoning., Indian heart journal., 3: 193-195.
3. Soltaninejad K., S. Shadnia and O. Mehrpour., 2010. A simplified acute physiology score in the prediction of acute aluminum phosphide poisoning outcome. Indian J. Med. Sci., Vol. 64: 10.4103/0019-5359.75928.
4. Mathai A. and M. Bhanu., 2010. Acute aluminium phosphide poisoning: Can we predict mortality. Indian J. Anaesth., 54: 10.4103/0019-5049.68372.
5. Soltaninejad K., M.R. Beyranvand, S.A. Momenzadeh and S. Shadnia., 2012. Electro cardiographic findings and cardiac manifestations in acute aluminum phosphide poisoning. J. Forensic Legal Med., Vol. 19: 10.1016/j.jflm.2012.02.005.
6. Pannu A.K. *et al.*, 2020. PGI Score": A Simplified Three-point Prognostic Score for Acute Aluminum Phosphide Poisoning. Indian journal of critical care medicine: Peer-reviewed, official publication of Indian Society of Critical Care Medicine., Vol. 24: 10.5005/jp-journals-10071-23555.
7. Bogle, R.G., *et al.*, 2006. Aluminium phosphide poisoning. Emergency Med. J., Vol. 23. 10.1136/emj.2004.015941.
8. Siwach S.B. *et al.*, 1998. Cardiac arrhythmias in aluminium phosphide poisoning studied by on continuous holter and cardioscopic monitoring. The Journal of the Association of Physicians of India., 46: 598-601.
9. Zeggwagh A., M. Louriz, T. Dendane, K. Abidi, N. Madani and R. Abouqal., 2009. Prognostic factors of acute aluminum phosphide poisoning. Indian J. Med. Sci., Vol. 63: 10.4103/0019-5359.53386.