OUTCOMES OF ACUTE ALUMINIUM PHOSPHIDE POISONING AND ROLE OF MAGNESIUM SULFATE AS AN ANTIDOTE IN MINIA POISONING CONTROL CENTER, MINIA GOVERNORATE, EGYPT.

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ABSTRACT

Background: Aluminium phosphide (AlP) is an affordable and efficient solid pesticide used for grain storage. **Objectives:** The present study was designed to determine the outcomes of acute aluminium phosphide toxicity and the role of magnesium sulfate as an antidote. **Methodology:** The present study was conducted on 53 patients admitted to the Minia Poison Control Center with a history of acute aluminium phosphide toxicity to assess the outcomes and role of magnesium sulfate as an antidote. Only 32 patients manifested AlP toxicity, and they were randomly divided into two groups, A and B, each with 16 patients. Group (A) received magnesium sulfate infusion using the bolus protocol of administering 4 g intravenously, 2 g after one hour, and then 1 g/4 h, in addition to other supportive therapies, while group (B) received only supportive therapy. **Results:** Mortality occurred in 23 cases: 9 cases (56.25%) from group A and 14 cases (87.5%) from group B. In survivors (no. = 9), serum magnesium levels and ABG parameters were significantly higher than those of non-survivors (no. = 23). There was no statistically significant difference between survivors and non-survivors regarding the treatment with Mg sulfate. According to the simple logistic regression test, the riskiest variables were the ingested amount followed by the PO2. Blood PH, PCO2, and serum Mg level had the highest sensitivity (91.3%) in the prediction of mortality, while PO2 was the most specific predictor (specificity = 88.89). **Conclusion:** Magnesium sulfate therapy did not improve patient outcomes as the mortality rate reached 72% in the current study, so until now, prevention has been the most valuable management strategy. Future studies are required to identify a suitable antidote for AlP toxicity.

Keywords: Aluminium phosphide; poisoning, outcome; magnesium sulfate.

INTRODUCTION

Aluminium phosphide (AlP) is an affordable and efficient solid pesticide used for grain storage. It is available in dark grey tablets called Rice Tablets (Gurjar et al. 2011). During the last few decades, AlP has been used more frequently for both agricultural and non-agricultural sectors, increasing in AlP-poisoned cases (Karimani et al., 2018). These tablets release phosphine gas when they meet gastric acidity or absorb environmental moisture. Therefore, AlP pellets or tablets lose their potency when left in the air as they gradually

absorb atmospheric moisture and release phosphine gas, leaving a non-toxic residue of Aluminium hydroxide (Sedaghattalab, 2022).

Suicide is the most often reported method of intake, followed by unintentional ingestion or workplace exposure. In developed countries, accidental cases have been reported where AlP is used as a fumigant and rodenticide (Ghazi, 2013). The number of AlP poisoning cases has increased dramatically since 1990 (Mahajan and Pargal, 2012).

Aluminium phosphide inhibits cytochrome oxidase and produces phosphine gas (PH 3), resulting in the production of highly reactive hydroxyl radicals (Bumbrah et al., 2012). A 3-gram tablet can produce 1 g of phosphine gas, which is an odorless and colorless toxic gas (Shadnia et al., 2005).

For an adult human, the lethal dose of AlP ranges from 0.15 to 0.5 grams (Bogale et al., 2021). Poisoning with AlP has an extremely high mortality rate, even with admission to the ICU. The mortality rate ranges from 30 to 100%, even in specialized hospitals with pioneering life-support systems. Actually, it ranks among the major global reasons for fatal poisoning (Farzaneh et al., 2018).

Since there is currently no known particular antidote, vigorous supportive care is essential for ALP-poisoned patients. In experimental and clinical research, several drugs have been proposed and evaluated (Jha et al., 2022).

Magnesium sulfate is a well-known anti-arrhythmic and cardio-protective drug that can stabilize membranes (Shadnia et al., 2005). Numerous trials carried out over the world have demonstrated its capacity to lower mortality in wheat pill poisoning, as long as it is administered within 4 hours of ingestion. It is well-documented that magnesium can scavenge free radicals. Magnesium sulfate is administered intravenously to eliminate free radicals from the body's circulation and serves as a safeguard against fatal arrhythmias (Hassan et al., 2013).

Vitamins C and E, melatonin, N-acetylcysteine, coconut oil, sodium selenite, triiodothyronine, vasopressin, liothyronine, milrinone, 6-aminonicotinamide, boric acid, acetyl-L-carnitine and Laurus nobilis L. are additional substances that could be used as AlP poisoning antidotes (Moghadamnia, 2012).

The present study was designed to determine the outcomes of acute aluminium phosphide toxicity and the role of magnesium sulfate as an antidote in patients admitted to the Minia Poison Control Center (MPCC) from December 15, 2020, to December 15, 2021.

PATIENTS AND METHODS

The current study is a clinical perspective, one that was conducted on 53 patients admitted to the Minia Poison Control Center (MPCC) with acute aluminium phosphide poisoning between December 15, 2020, and December 15, 2021. The Minia University Faculty of Medicine's Research Ethics Committee approved this research (Approval no. 751:12/2020). Patients or their guardians gave their consent to take part in this study. All personal data were kept secret.

The patients included in this study were of any age or sex and presented with acute aluminium phosphide toxicity. Patients who co-ingested other toxic substances, those with any chronic illness such as diabetes, cardiovascular, respiratory, renal, or hepatic diseases, or those who were pregnant were excluded from this study.

History was obtained from selected patients or those accompanying them, including personal history (name, age, sex, occupation, and residence), amount of poison ingested, manner of exposure, route of exposure, delay time, and symptoms. Clinical examination was performed, including vital signs, skin examination, and systemic examination (cardiovascular, respiratory, GIT, and neurological examinations).

Baseline electrocardiograms were recorded at admission. Arterial blood samples were drawn for arterial blood gases (ABG) and venous samples for serum Mg levels from all patients.

Initial resuscitation was then performed. Gastric lavage was carried out with paraffin oil and sodium bicarbonate. Infusion of vasoactive agents and inotropes was employed if needed, and mechanical ventilation was used when indicated.

Among the 53 patients, 21 were asymptomatic, while the remaining 32 patients who manifested AlP poisoning were randomly

divided into two groups, A and B, each with 16 patients.

Group A (16 patients) received magnesium sulfate infusion using the bolus protocol of administering 4 g intravenously, 2 g after one hour, and then 1 g/4 h (Garg, 2020), in addition to other supportive therapies. Group B (16 patients) received only supportive therapy, and no magnesium sulfate was administered.

The patients were monitored for acidosis, hypotension, respiratory failure, arrhythmias, and ECG changes. All patients were followed until discharge from the hospital (survivors) or death (non-survivors). The follow-up data were recorded.

Statistical analysis:

Statistical Package for Social Sciences (SPSS) version 25 was used to code, tabulate, and statistically analyze the collected data. For parametric quantitative data, descriptive statistics were carried out using mean, standard deviation (SD), and minimum and maximum range. In contrast, non-parametric quantitative data was performed using median and interquartile range (IQR). Frequency and percentage were used to represent the qualitative data. The distribution of the data was determined using the Shapiro-Wilk test.

For comparisons between the study groups using parametric and non-parametric quantitative data, the Independent Samples T-test and Mann-Whitney U test were utilized, respectively. The Chi-square test was used to analyze qualitative data between two groups if up to 20% of cells had an expected count less than 5, while Fisher's exact test was used if more than 20% of cells had an expected count less than 5.

Simple logistic regression was used to evaluate the risk variables predicting poor prognosis. In contrast, the Receiver Operating Characteristics (ROC) curve was used for calculating the optimal cut-off, AUC, sensitivity, specificity, PPV, NPV, and the accuracy of risk factors predicting poor prognosis.

RESULTS

Cases ranged in age from 1.5 to 45 years (23.1 ± 9.3 years), with 58.5% females and 41.5% males, and all of them were from rural areas (100%). Students made up 30.2% of the cases, followed by housewives (28%), employees (17%), farmers (9.4%), non-workers (7.5%), and children (7.5%). For the mode of poisoning, 88.7% of cases were suicide attempts, 7.5% were exposed to AlP accidentally, and 3.8% were homicides. Poisoning was manifested in 60.4% of cases, while 39.6% were non-manifested (Table 1).

Table 1: Demographic data of all patients.

		Descriptive statistics N = 53
Age	*Range*	(1.5 - 45)
	Mean ± SD	23.1 ± 9.3
	Median/IQR	22/ (19 - 28)
Sex	*Male*	22 (41.5%)
	Female	31 (58.5%)
Occupation	*House-wife*	15 (28.3%)
	Farmer	5 (9.4%)
	Worker	9 (17%)
	Student	16 (30.2%)
	Not working	4 (7.5%)
	Child	4 (7.5%)
Mode of poisoning	*Suicidal*	47 (88.7%)
	Homicidal	2 (3.8%)
	Accidental	4 (7.5%)
Manifested	*No*	21 (39.6%)
	Yes	32 (60.4%)

No difference between group A and group B was statistically significant for the ABG findings, the serum Mg levels, and the ECG data (Table 2). Survivors who recovered after AlP poisoning were 9 cases; 7 cases (77.7%) from group A and 2 (22.2%) cases from group B, while non-survivors who died after AlP poisoning were 23 cases; 9 cases (56.25%) from group A and 14 cases (87.5%) from group B, with no statistically significant difference in the outcome between the two groups (Table 2).

The difference was statistically significant between non-survivors and survivors regarding the amount ingested (P = 0.010). Survivors received 0.3-1.5 tablets of AlP of a 3 gm tablet (0.7 ± 0.5) while the non-survivors ingested 0.3-3 tablets (1.5 ± 0.8). The delay time before presenting to the hospital was 2 – 6 hr (4.2 ± 1.4)

and 1.5 – 6 hr (3.3 ± 1.5) for the survivors and non-survivors, respectively, with no statistically significant relationship between it and the mortality (Table 3).

Table 2: Comparisons of the ABG data, serum Mg level, ECG data and outcome between group A and B

		Groups (A) Treated with Mg sulfate N=16	Groups (B) Not treated with Mg sulfate N=16	P value
pH	*Range*	(4.8 - 7.3)	(4.2 - 7.3)	0.570
	Mean ± SD	7 ± 0.6	6.9 ± 0.8	
PCO_2	*Range*	(5.4 - 25.6)	(8.6 - 31.3)	0.742
	Mean ± SD	18.1 ± 4.3	18.7 ± 5.5	
HCO_3	*Range*	(3 - 12.5)	(3.9 - 14.5)	0.781
	Mean ± SD	8.7 ± 2.3	8.5 ± 2.7	
PO_2	*Range*	(16.2 - 83.5)	(10.8 - 89)	0.685
	Mean ± SD	61.2 ± 16.1	58.6 ± 20.4	
Serum Mg level	*Range*	(1.5 - 2.3)	(1.5 - 2.6)	0.393
	Mean ± SD	1.8 ± 0.2	1.8 ± 0.3	
*** ECG data**				
ST	*No*	4 (25%)	3 (18.8%)	1
	Yes	12 (75%)	13 (81.3%)	
VT	*No*	9 (56.3%)	7 (43.8%)	0.480
	Yes	7 (43.8%)	9 (56.3%)	
SVT	*No*	12 (75%)	12 (75%)	1
	Yes	4 (25%)	4 (25%)	
AF	*No*	11 (68.8%)	14 (87.5%)	0.394
	Yes	5 (31.3%)	2 (12.5%)	
SB	*No*	16 (100%)	15 (93.8%)	1
	Yes	0 (0%)	1 (6.3%)	
****Outcome**	*Recovery (survivors, N=9)*	7 (77.8%)	2 (22.2%)	0.113
	Death (non-survivors, N=23)	9 (39.1%)	14 (60.9%)	

- Independent Samples T test for parametric quantitative data between the two groups.
- *Fisher's exact test for qualitative data between the two groups.
- **Chi square test and Fisher's exact test for qualitative data between the two groups.
- Significant level at P value < 0.05.
- pH: potential hydrogen, PCO_2: Partial pressure of carbon dioxide, HCO_3: Bicarbonate, PO_2: Partial pressure of oxygen, Mg: Magnesium.
- ST: Sinus tachycardia, VT: Ventricular tachycardia, SVT: Supraventricular tachycardia, AF: Atrial fibrillation, SB: Sinus bradycardia.

There were statistically significant differences between survivors and non-survivors for all the arterial blood gases (ABG) parameters. The mean PH value in survivors was 7.2 ± 0.1 compared to 6.8 ± 0.8 in non-survivors (P = 0.028). PCO2 mean values were 16.7 ± 3.9 and 22.6 ± 4.8 in non-survivors and survivors, respectively (P = 0.001). For HCO3 mean levels, they were 7.8 ± 2.1 and 10.8 ± 2.2 in non-survivors and survivors, respectively (P = 0.001).

At the same time, PO2 mean values were 54.5 ± 17.6 in non-survivors and 73.6 ± 11.1 in survivors (P = 0.005). The difference between survivors and non-survivors was statistically significant with regard to the serum magnesium level, with mean levels of 2 ± 0.3 and 1.7 ± 0.2, respectively (P = 0.021) (Table 4).

Table 3: Comparisons of the outcome of patients with ingested amount and delay time

		Outcome		**P value**
		Recovery *(Survivors)* **N=9**	**Death (***non-survivors***) N=23**	
	Range	(0.3 - 1.5)	(0.3 - 3)	
Amount (tablet)	*Mean ± SD*	0.7 ± 0.5	1.5 ± 0.8	**0.010***
	Median/IQR	1/(0.3 -1)	1/(1 - 2)	
	Range	(2 - 6)	(1.5 - 6)	
Delay time (hr)	*Mean ± SD*	4.2 ± 1.4	3.3 ± 1.5	0.121
	Median/IQR	4/(3-5.5)	3/(2 - 4)	

- Mann Whitney test for non-parametric quantitative data between the two groups
- *: Significant level at P value < 0.0

Table 4: Comparisons between the outcome of patients with ABG data and serum Mg levels

		Outcome		
		Survivors N=9	**Non survivors N=23**	**P value**
pH	*Range*	(7.2-7.3)	(4.2-7.3)	**0.028***
	Mean ± SD	7.2±0.1	6.8±0.8	
PCO_2	*Range*	(17.5-31.3)	(5.4-23.2)	**0.001***
	Mean ± SD	22.6±4.8	16.7±3.9	
HCO_3	*Range*	(7.9-14.5)	(3-12.5)	**0.001***
	Mean ± SD	10.8±2.2	7.8±2.1	
PO_2	*Range*	(60.2-89)	(10.8-82)	**0.005***
	Mean ± SD	73.6±11.1	54.5±17.6	
Serum Mg levels	*Range*	(1.6-2.6)	(1.5-2.1)	**0.021***
	Mean ± SD	2±0.3	1.7±0.2	

- Independent Samples T test for parametric quantitative data between the two groups.
- *: Significant level at P value < 0.05.
- pH: potential hydrogen, PCO2: Partial pressure of carbon dioxide, HCO3: Bicarbonate, PO2: Partial pressure of oxygen, Mg: Magnesium.

A simple logistic regression test showed that different variables were significant (P < 0.05) to predict mortality, including ingested amount, pH, PCO_2, HCO_3, PO_2, and serum Mg level. The riskiest variables were the amount consumed (odds ratio was 9.3), followed by the PO_2 (odds ratio was 0.87) (Table 5).

The receiver operating characteristics (ROC) curve analysis of ABG and serum Mg levels for prediction of mortality is shown in Figure 2. Blood PH, PCO2, and serum Mg level had the highest sensitivity (91.3%) in the prediction of mortality in AlP-intoxicated patients with an optimal cut-off point of ≤ 7.21, ≤ 19.5 and ≤ 1.9, respectively, followed by HCO3 with sensitivity of 82.61% and optimal cut-off point of ≤ 9, then PO2 levels with sensitivity of 69.57% and optimal cut-off point ≤ 62. PO2 was the most specific predictor (specificity = 88.89%), followed by HCO3 (specificity = 77.78%), then PCO2 and PH values (specificity = 66.67%), and finally, the serum Mg level (44.44%) (Table 6, Chart 1).

Table 5: Simple logistic regression analysis of variables for prediction of mortality

	OR	95% CI	P value
Amount	9.3	1.2-70	0.031*
pH	0	0-0.012	0.020*
PCO_2	0.6	0.38-0.93	0.024*
HCO_3	0.46	0.26-0.82	0.009*
PO_2	0.87	0.78-0.97	0.012*
Serum Mg level	0.02	0-0.96	0.048*

- Simple logistic regression test
- OR: Odds Ratio
- CI: Confidence Interval
- *: Significant level at P value < 0.05
- pH: potential hydrogen, PCO_2: Partial pressure of carbon dioxide, HCO_3: Bicarbonate, PO_2: Partial pressure of oxygen, Mg: magnesium.

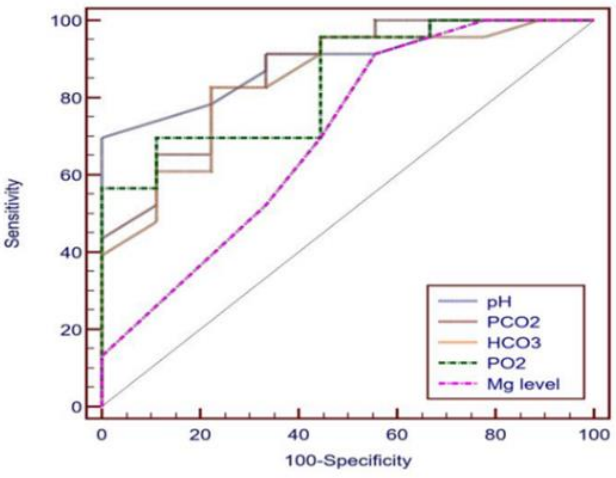

Chart (1): ROC curve for ABG parameters and serum Mg level.

Table 6: ROC curve analysis of ABG and serum Mg level for prediction of mortality

	pH	PCO_2	HCO_3	PO_2	Serum Mg level
Optimal cutoff	≤ 7.21	≤ 19.5	≤ 9	≤ 62	≤ 1.9
AUC	0.896	0.870	0.843	0.841	0.700
95% CI	0.737-0.976	0.703-0.962	0.671-0.947	0.668-0.945	0.513
P value	**<0.001***	**<0.001***	**<0.001***	**<0.001***	0.076
Sensitivity	91.3	91.3	82.61	69.57	91.3
Specificity	66.67	66.67	77.78	88.89	44.44
PPV	87.5	87.5	90.5	94.1	80.8
NPV	75	75	63.6	53.3	66.7
Accuracy	84.4	84.4	81.2	75	78.1

- ROC curve test
- AUC: Area Under Curve
- CI: Confidence Interval
- PPV: Positive Predictive Value
- NPV: Negative Predictive Value
- *: Significant level at P value < 0.05

DISCUSSION

In the current study, among the 53 patients, 21 were asymptomatic, primarily due to exposure to expired forms of AlP, receiving a minimal non-toxic amount, or being deceitful that they did not receive AlP and only tried to gain sympathy.

Regarding the sociodemographic variables in this study, the patients were children and young adults, which is in agreement with several studies which explained that the exposure of young adults to the stress of modern life, educational and emotional complexity, family disharmony, in addition to financial instability and unemployment (Mathai and Bhanu, 2010; Kalawat et al., 2016; Abd Elghany et al., 2018; Darwish et al., 2020; Mwaheb and Hassan, 2021; Deraz et al., 2022; Dadpour et al., 2016). However, Gupta et al. (2011) observed that AlP poisoning in children was prevalent in India through accidental ingestion due to illiteracy and the availability of AlP in the agricultural community there.

Comparable to our research, Qureshi et al., 2018, reported a higher frequency for women (59.1%) than for men (49.9%); Soltaninejad et al., (20120b) revealed 54.71% women and 45.29%

men, Alnaseer et al., **(**2018) showed 56% women versus 44% men, and Louriz et al., (2009) reported 63.27% women versus 36.73% men. Regarding occupation, students were more liable to toxicity (30.2%), in contrast to the findings of Badawi et al. (2018), who stated that civil personnel constituted the majority of cases. Also, Deraz et al. (2022) revealed that unemployed cases were the more represented (30.4%). It may be due to failure or low grades in exams or as a result of their teachers' reprimands.

One hundred percent of the patients studied were from rural areas, which is in agreement with Moghadamnia (2012) and Mehrpour et al. (2012). This may be due to the easy availability of phosphides in rural areas, failure in education, and financial problems.

This study agreed with Qureshi et al. (2018), finding that 79.6% of their cases received AlP with suicidal intent and referred to bodily or psychological illness. Soltaninejad et al. (2012a) also found that the majority of AlP-studied cases ingested it, while inhalation exposure was only identified in individuals who handled grains during storage or transport.

These present results are nearly similar to those of El-Sarnagawy (2017), who found that the ingested amount was between 0.25 and 2 tablets with a significant prediction of mortality. Additionally, Wahdan and Khalifa (2020) reported a significant relationship between mortality and the amount of AlP. Teimoory et al. (2013) reported that the lethal dose of AlP was 150-500 mg (0.05-0.2 tablet), and people who survived after AlP exposure mainly used expired forms.

The current study is in accordance with Wahdan and Khalifa (2020), who found that the difference in the delay time between non-survivor and survivor cases was not statistically significant, ranging between 0.5-10 h.

Regarding the ABG changes in the present study, all manifested cases developed metabolic acidosis, but this was more severe in non-survivors, and it was directly proportional to the severity of the clinical manifestations. This is consistent with the findings of Meena et al. (2015) along with Abdel Wahab et al. (2020). Bashardoust et al. (2017) explained that lactic acidosis was secondary to hypoperfusion and inhibition of oxidative phosphorylation.

The survivors had a statistically higher serum magnesium level than the non-survivors, and none of the patients developed hypermagnesaemia. Its optimal cut-off point was ≤ 1.9, which was related to mortality, with a specificity of 44.44% and a sensitivity of 91.3%. In accordance with these results, Bajwa et al. (2010) found that AlP intoxication induced hypomagnesemia in the studied cases, leading to the development of arrhythmias. The study done by Chugh et al. (1991) observed significant hypomagnesemia in patients with severe AlP toxicity. Similarly, Chugh et al. (1994) found that the high mortality in AlP poisoning patients may be attributed to hypomagnesemia.

Regarding the ROC curve analysis in the present study, PH and PCO2 had the highest sensitivity in predicting mortality in AlP-intoxicated patients, followed by HCO3 and PO2 levels. However, PO2 was the most specific predictor, followed by HCO3, PCO2, and PH values.

For the blood PH cut-off, The present results are quite similar to those of Pannu et al. (2020), who found that the cut-off point of PH was <7.2, with a specificity of 71% and a sensitivity of 91%. Moreover, Wahdan and Khalifa (2020) recommended blood pH for early prediction of mortality in cases of ALP poisoning. For the present HCO_3 cut-off (≤ 9), Mathai and Bhanu (2010) agreed with the present results, as they revealed that low serum bicarbonate value (< 10) in patients with acute metal phosphide toxicity indicated poor prognosis and mortality.

The optimal cut-off point of PO_2 was ≤ 62, associated with mortality, with a specificity of 88.89 and a sensitivity of 69.57, while the optimal cut-off point of PCO2 was ≤ 19.5, with a specificity of 66.67% and a sensitivity of 91.3%. There have been no previous studies concerning the ROC curve analysis for PO2 and PCO2 in AlP acute poisoned cases.

According to the simple logistic regression analysis in the present study, the amount consumed (odds ratio = 9.3) followed by PO_2 (odds ratio = 0.87) was linked to an elevated risk of mortality. This result was contrary to that of

El-Sarnagawy (2017), who found that high risk of mortality was related to the development of abnormal ECG (odds ratio = 48.488), then metabolic acidosis and hypotension (odds ratio = 10.251 and 0.964, respectively).

Unfortunately, the absence of a specific antidote for AlP toxicity resulted in high mortality, reaching 72% in the current study. This is consistent with several studies that found high mortality in AlP patients that exceed 70% and can reach 100% (Mehrpour et al., 2019). However, Mathia and Bhanu (2010) stated that death after AlP exposure occurred only in 40-80% of the studied patients. This may be secondary to mild toxicity in most cases.

Not enough research has been done on MgSO4's potential benefits in AP poisoning. Chugh et al. (1994) examined 105 patients. They found that while magnesium levels increased right away following parenteral MgSO4 administration and continued to be consistently above normal in the group treated with MgSO4, hypomagnesemia was consistently reported in cases of AlP poisoning that were not treated with MgSO4. In a different trial with 155 patients, the same author noted that MgSO4 helped to lower mortality.

There have been many studies regarding magnesium levels and the advantages of magnesium sulfate in cases of AlP poisoning. Furthermore, not enough research has been done on MgSO4's potential benefits in this poisoning. Chugh et al. (1994) examined 105 patients. They found that while magnesium levels increased right away following parenteral MgSO4 administration and continued to be consistently above normal in the group treated with MgSO4, hypomagnesemia was consistently reported in cases of AlP poisoning that were not treated with MgSO4. In a different trial with 155 patients, the same author noted that MgSO4 helped lower mortality.

The efficacy of MgSO4 has been attributed to the rapid correction of magnesium levels, and it was suggested that hypomagnesemia might be responsible for the high mortality of patients with AlP poisoning (Chugh et al., 1994).

Magnesium helps in scavenging free radicals and hence acts as an antioxidant, and it is a cell membrane stabilizer and anti-arrhythmic agent. Magnesium sulfate therapy has been consistently used for AlP poisoning in many studies and has been claimed to reduce mortality by up to 50% (Mehrpour et al., 2021 & Navabi et al., 2017). On the contrary, Siwach et al. (1994), in a study involving 50 patients, found no significant difference in AlP-related mortality in patients treated with and without magnesium sulfate and found no evidence of hypomagnesemia in these patients.

The current study revealed that only 9 cases (28%) survived, with 77.8% of them receiving magnesium sulfate, while 23 cases (72%) died, and only 39.1% of them received Mg sulfate. However, the difference between the two groups was not statistically significant, and the small size of the sample may explain that. Siwach et al. (1994) agreed with the current results; they found that magnesium sulfate therapy did not improve survival, but it can be improved to some extent with the use of appropriate anti-arrhythmic agents and continuous cardiac monitoring.

El-Sarnagawy (2017) disagreed with our study, as he demonstrated that administration of MgSO4 caused significant improvement in the patient's outcomes. His study was conducted on 105 patients; 95.7% of non-survivors did not receive MgSO4, whereas 34.5% of survivors received MgSO4. They explained that the anti-oxidation and membrane-stabilizing effects of Mg sulfate resulted in a decrease in the incidence of cardiac arrhythmias. Additionally, Shakeri and Mehrpour (2014) found that the administration of magnesium to AlP-intoxicated rats resulted in a marked improvement in heart failure. In contrast to the current study, Bajwa et al. (2010) found that magnesium sulfate therapy improved patients' conditions due to its anti-arrhythmic properties.

Sodium selenite, melatonin, coconut oil, N-acetylcysteine (NAC), Vitamin E, vasopressin, triiodothyronine, Acetyl-L-carnitine (ALCAR), 6-aminonicotinamide, and boric acid studied with both experimental and clinical researches for the possibility of using them as AlP poisoning antidotes. Like mg sulfate, sodium selenite did not appear to have an impact on mortality latency, although it did appear to be able to lessen hepatic and pulmonary problems (Moghadamnia et al., 2000). ADP/ATP ratio, mitochondrial complex

activities, and intriguing antioxidant properties were all demonstrated by melatonin (Asghari et al., 2017). Still, the impact of melatonin on the death rate has not been discussed.

Coconut oil acts as a powerful barrier against AlP toxicity by covering the stomach and reducing the pace of AlP absorption. Numerous case studies have shown that coconut oil could enhance the outcome of AlP poisoning [Bajwa et al., 2014; Shadnia et al., 2005). NAC may be used as an adjuvant therapy to treat AlP-induced cardiotoxicity, avoid liver necrosis, and improve biochemical and hemodynamic parameters. NAC was well tolerated and did not have any negative effects, even at large doses.

When vitamin E and NAC were given together, the death rate was reduced (Halvaei et al., 2017), and the effects were more pronounced (Oghabian et al., 2015). It has been proposed that AlP-related changes in apoptosis, ATP levels, and cardiovascular function can be improved by vasopressin (Abdolghaffari et al., 2015), T3 (Abdolghaffari et al., 2015), and ALCAR (Baghaei et al., 2016). 6-Aminonicotinamide exhibited hepatocyte-protective effects based on in vitro investigations (Salimi et al., 2017). Finally, boric acid appears to be a possible counteragent by trapping PH3; however, it has not been thoroughly studied (Soltani et al., 2013; Soltani et al., 2016).

Throughout the present research, death took place during the initial 24 hours of AlP exposure due to the development of cardiotoxicity and severe metabolic acidosis. Similarly, according to Neki et al. (2017), acute phosphide exposure resulted in death within the first 24 hours, and it was linked to arrhythmia, shock, cardiotoxicity, ARDS, and the need for mechanical ventilation. In contrast, Abder-Rahman (2011) and Lehoux et al. (2018) reported sudden fatalities after AlP exposure due to sudden cardiac arrest with no preceding clinical symptoms or signs of toxicity. On the other hand, Yogendranathan et al. (2017) reported that death occurred after 24 hours due to liver cell failure, renal failure, or chemical pneumonitis.

CONCLUSION

Aluminium phosphide represents a fatal chemical with a high mortality rate that reached 72% in the current study, and there is no specific antidote for AlP toxicity. Magnesium sulfate therapy did not improve patient outcomes, so prevention is the most valuable management strategy.

RECOMMENDATIONS

1- The misuse of aluminium phosphide should be restricted.

2- Other non-toxic alternatives to replace AlP must be suggested.

3- Future studies are required to identify a suitable antidote for AlP toxicity.

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

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الملخص العربي

نتائج التسمم الحاد بفوسفيد الألومنيوم ودور كبريتات المغنيسيوم كمضاد في مركزالمنيا لمكافحة التسمم بمحافظة المنيا ، مصر.

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المقدمة: فوسفيد الألومنيوم هو مبيد صلب وفعال للآفات وأيضا متاح بأسعار معقولة حيث أنه يستخدم لتخزين الحبوب. **الأهداف:** صممت هذه الدراسة لتحديد نتائج التسمم الحاد بفوسفيد الألومنيوم ودور كبريتات المغنيسيوم كترياق. **طريقة الدراسة:** أجريت الدراسة الحالية على 53 مريضاً تم إدخالهم إلى مركز علاج التسمم بالمنيا ولديهم تاريخ للتسمم الحاد بفوسفيد الألومنيوم؛ لتقييم نتائج التسمم الحاد بفوسفيد الألومنيوم ودور كبريتات المغنيسيوم كترياق. أظهر فقط 32 مريضا أعراضا للتسمم الحاد بفوسفيد الألومنيوم، وقد تقسيمهم لمجموعتين(أ) و(ب). تلقت المجموعة (أ) تسريب كبريتات المغنيسيوم باستخدام بروتوكول الجرعة المركزة لإعطاء 4 جم عن طريق الوريد، و 2 جم بعد ساعة واحدة ، ثم 1 جم / 4 ساعة ، بالإضافة إلى العلاجات الداعمة الأخرى ، بينما تلقت المجموعة (ب) العلاج الداعم فقط. **النتائج:** كانت مستويات المغنيسيوم في الدم وكذلك معاملات غازات الدم الشرياني في الناجين (عدد الحالات = 9) أعلى بكثير من تلك في غير الناجين (عدد الحالات = 23). لم يكن هناك فرق معتد به إحصائيا بين الناجين وغير الناجين فيما يتعلق بالمعالجة بسلفات الماغنسيوم. وفقًا لاختبار الانحدار اللوجستي البسيط ، كانت المتغيرات الأكثر خطورة هي الكمية المأخوذة، يليها ضغط الأكسجين الجزئي. كان مستوى الأس الهيدروجيني و ضَغط ثاني أكسيد الكَربون الجزئي والماغنسيوم في الدم أعلى حساسية في التنبؤ بالوفيات (91.3٪)، بينما كان ضغط الأكسجين الجزئي هو المؤشر الأكثر تحديدًا (النوعية = 88.89). **الخلاصة:** العلاج بكبريتات المغنيسيوم لم يحسن نتائج المرضى ، لذلك حتى الآن ، الوقاية هي الاستراتيجية الأجدى بالنفع في التعامل مع التسمم بفوسفيد الألومنيوم، لذلك فإن المزيد من الدراسات المستقبلية مطلوبة لتحديد ترياق مناسب للسمية بفوسفيد الألومنيوم.