Evaluation of Potential Oxidative Stress in Egyptian Patients with Acute Zinc Phosphide Poisoning and the Role of Vitamin C

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Abstract

Objective: To evaluate potential oxidative stress in patients with acute phosphide poisoning and the effect of vitamin C.

Methods: Participants were females and divided into three groups; group I: healthy volunteers group II: healthy volunteers received vitamin C, group III: patients with acute phosphide poisoning received the supportive and symptomatic treatment and group IV: patients with acute phosphide poisoning received the supportive and symptomatic treatment in addition to vitamin C. All the participants were subjected to thorough history, clinical examination, ECG and laboratory investigations were carried on collected blood and gastric lavage samples on admission. Blood samples were divided into two parts, one for measurement of routine investigations and the second part was used for evaluation of malondialdehyde and total thiol levels before and after receiving the treatment regimen.

Results: Most of the cases in this study were among the age group of 15-25 years, females, single, secondary school education, from rural areas and suicidal. All vital signs were within normal range and the most common complaint was vomiting and abdominal pain. All cases in this study showed normal routine investigations. The mean MDA levels after receiving treatment decreased significantly in groups II and IV. The mean total thiol levels increased significantly after receiving treatment in groups II and IV.

Conclusion: It can be concluded that vitamin C has a potential benefit due to its antioxidant property on zinc phosphide induced-oxidative stress in acute zinc phosphide poisoned patients.

Keywords: Oxidative Stress, Zinc Phosphide, Vitamin C

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Introduction

Phosphide compounds (zinc, aluminium, magnesium or calcium phosphides) have been used as pesticides for many years to protect grains in stores and during its transportation. Phosphides are normally found as powders or pellets. The wide spread use of phosphides is attributed to being highly effective, inexpensive, easily available and not leaving toxic residues in the environment. [1] Due to its low price and easy availability, phosphide is emerging as a common self-poisoning agent in Egypt.[2] Reported morbidity and mortality rate due to phosphide poisoning is too high, especially in developing countries and in Egypt. [2, 3]

Acute poisoning with zinc phosphides may occur either through direct ingestion of the salts themselves or indirect inhalation of the phosphine gas generated on exposure to moisture. [1]

When zinc phosphide comes in contact with water, moisture in the air, or hydrochloric acid in the stomach, a chemical reaction starts releasing phosphine gas which is rapidly absorbed throughout the gastrointestinal tract and the lungs. Phosphine acts by blocking cytochrome C oxidase resulting in inhibition of mitochondrial oxidative phosphorylation leading to histotoxic hypoxia. [4] The first symptoms on presentation are often profuse vomiting and abdominal pain. [5] Severer manifestations have also been associated with zinc phosphide ingestion including raised liver transaminases, hepatomegaly, hepatic failure, severe metabolic acidosis with acute distal renal tubular acidosis. [6] Circulatory failure is a common and frequent cause of death after ingestion of zinc phosphides. [7]

Zinc phosphides induces oxidative stress. [8] It inhibits the activity of catalase (CAT) and peroxidase and stimulates superoxide dismutase (SOD). [9] It elevates malondialdehyde (MDA) levels in cardiac tissues. [10]

Vitamin C (ascorbic acid) is a water-soluble vitamin that is necessary for normal growth and development. [11] When ascorbic acid interacts with a reactive and possibly harmful free radical, it donates two electrons, the reactive free radical is reduced and an ascorbyl free radical is formed in its place. As compared to other free radicals, ascorbyl radical is relatively stable with a half-life of 5-10 seconds and is fairly unreactive. This property explains why ascorbate may be a preferred antioxidant. [12]

Since there is no specific antidote, the management of phosphide poisoning is mostly supportive till phosphine is excreted from the body through lungs and the kidneys**.** Research still continues for an effective remedy to phosphide poisoning. [13] Therefore, the aim of this study was to evaluate potential oxidative stress by measuring serum malondialdehyde and total thiol in patients with acute phosphide poisoning and to evaluate the potential therapeutic effect of vitamin C in these patients.

Patients and Methods

The study was carried out following approval of the medical research ethical committee of Tanta Faculty of Medicine on admitted patients with acute phosphide poisoning to Toxicology Unit, Emergency hospital, Tanta University in the period from March 2013 to September 2013. A written informed consent was taken from every participant or guardian before starting the study.

The study was carried out on 40 subjects who were divided into four equal groups as followings: Group I (control group): healthy normal volunteers. Group II (control group): healthy normal volunteers received vitamin C (2400mg daily as an intravenous infusion for 3 days) [11] and served as a control group. Group III (phosphide group): patients with acute phosphide poisoning received the standard treatment without administration of vitamin C. Group IV (phosphide and vitamin C group): patients with acute phosphide poisoning received the standard treatment in addition to vitamin C2400 mg daily as an intravenous infusion for 3 days). [14]

All Patients with acute phosphide poisoning from March 2013 to September 2013 were included in group II and III. Patients with medical disease or medication which may induce oxidative stress, such as liver, renal, cardiac and respiratory diseases, autoimmune diseases, cancer or active infections were excluded for all the studied groups. Patients with mixed poisoning were excluded in group II and III. All groups were subjected to the following:

History: Detail history was obtained including personal history (age, sex, occupation, education, residence and marital state), toxicological history (the form and amount of the metal phosphide ingested, manner of poisoning and duration between exposure and arrival to the hospital), history of medical diseases which induce oxidative stress (liver, renal, cardiac and respiratory diseases, cancer or active infections) and history of medication causing alteration in oxidative status such as other anti-oxidant drugs.

Clinical Examination: It included vital signs (respiratory rate, heart rate, blood pressure and temperature), level of consciousness, respiratory, cardiovascular and abdominal examination.

Investigation:

Electrocardiogram (ECG): 12 lead ECG was performed for all participants.

Laboratory investigations: Sample collection: Laboratory investigations were carried on blood and gastric lavage sample, 2 samples of blood were drawn from all participants on admission. The non-heparinized blood sample (4 ml) was used for measurement of liver enzymes (serum aspartate transaminase (AST) and alanine transaminase (ALT), blood urea and serum creatinine, sodium and potassium. The heparinized blood sample (8 ml) was used for evaluation of Arterial blood gases (ABG) and oxidative stress markers (Malondialdehyde and total thiol).

Biochemical analysis: Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities were measured colourimetrically according to the method described by Reitman and Frankel [15] and Tietz. [16] Serum urea levels were measured by the enzymatic colourimetric method as described by Tabacco et al. [17] Serum creatinine levels were measured by the colourimetric kinetic method as described by Fabiny and Eringhausen. [18] The previous biochemical parameters were assayed using commercial diagnostic kits supplied by Stanbio-Laboratory, USA. Serum sodium and potassium were estimated by the colorimetric method of Berry et al. [19] and Sunderman and Sunderman. [20] Serum malondialdehyde (MDA) was determined according to Uchiyama and Mihars (1978).Briefly, 1 vol of serum was added to 6 vol of phosphoric acid [1.00% (w/v)] in 0.1 N HCl, 2 vol of TBA [0.60% (w/v)] in 0.1 N HCl. The samples were heated in a boiling water bath for 45 min. After cooling, the chromogen was extracted in 8 vol of n-butanol. The absorbance of the organic phase was measured at a wavelength of 532 nm. Total thiol was determined according to Ellman (1959). Briefly, reaction mixture containing 0.2 M Tris-HCl and 0.02 M EDTA buffer (pH 8.2), plasma and 0.01 M DTNB (in methanol) was incubated for 15 minutes at room temperature then was centrifuged at 1,200 x g for 5 minutes. The supernatant was collected and the absorbance was read at 412 nm. Results were expressed as nmoles of T-SH/mg protein using molar extension coefficient of DTNB (13,600 cm^{-1} M^{-1}).

Silver nitrate test: The diagnosis of zinc phosphide poisoning usually depends on clinical suspicion or history, but can be made easily by the simple silver nitrate test on gastric content.

Five ml of gastric aspirate and 15 ml of water is put in a flask and the mouth of the flask is covered by filter paper impregnated with 0.1N silver nitrate (16.987 gm of silver nitrate in 1L distilled water). The flask is heated at 50°C for 15 to 20 minutes; if phosphine is present the filter paper turns black. [21]

Statistical analysis: The results obtained were subjected to statistical analysis using SPSS version 15. Results were expressed as mean±SD. While Student's t test was used to determine the level of significance between each group and control, analysis of variance (ANOVA) is used to test inter-group differences; value of $P \leq 0.05$ is considered as significant.

Results

All patients in this study showed a positive result of the silver nitrate test as the silver nitrate impregnated filter paper turned brownish to black color as compared to the light brown color of the control group. All participants are females who are presented to our center. The socio-demographic data of the studied groups concerning age, gender, marital state, residence, level of education, occupation and past history of medical disease was illustrated in table 1.

There were no significant differences between the studied groups as regards the socio-demographic data.

Table 1. Socio-demographic data of the studied groups

Groups / Socio-demographic data		Group I		Group II		Group III		Group IV		X^2	P
		No (10)	%	No (10)	%	No (10)	%	No (10)	%		
Age (Years)	**15 - 25**	6	60	6	60	5	50	7	70	**4.231**	**0.645**
	25 - 35	4	40	3	30	3	30	3	30		
	35 - 45	0	0	1	10	2	20	0	0		
Sex	**Male**	3	30	4	40	4	40	5	50	**0.833**	**0.842**
	Female	7	70	6	60	6	60	5	50		
Marital Status	**Single**	7	70	6	60	5	50	6	60	**0.833**	**0.841**
	Married	3	30	4	40	5	50	4	40		
	Divorced	0	0	0	0	0	0	0	0		
	Widow	0	0	0	0	0	0	0	0		
Residence	**Rural**	8	80	6	60	5	50	7	70	**2.198**	**0.532**
	Urban	2	40	4	40	5	50	3	30		
Level of Education	**Illiterate**	4	40	3	30	2	20	0	0	**15.41**	**0.080**
	Elementary education	2	20	1	10	2	20	3	30		
	Secondary school	3	30	2	20	4	40	7	70		
	High education	1	10	4	40	2	20	0	0		
Occupation	**Unemployed**	6	60	7	70	5	50	5	50	**1. 125**	**0.771**
	Employed	4	40	3	30	5	50	5	50		
Medical Disease	**-**	-	-	-	-	-	-	-	-	**-**	**-**

As regards the manner of poisoning in group III and group IV, all patients had taken phosphide in a suicidal attempt with no homicidal or accidental Participants.

The mean amount of zinc phosphide was (1.10+0.82) packets (each packet contains 5 grams of zinc phosphide) in group III and was (0.57+0.37) packets in group IV. This difference was statistically insignificant (P = 0.224).

As regards the duration between exposure to zinc phosphide and arrival to hospital, there was insignificant difference in group III (2.45±0.86 hours) compared to group IV (3.15±0.78 hours) (Table 2).

Table 2. Comparison between the studied groups regarding the ingested amount of zinc phosphide and duration before arrival to hospital

	Group I **No.** (10)	Group II **No.** (10)	Group III **No.** (10)	Group IV **No.** (10)	X^2	P
	Mean ±SD	**Mean ±SD**	**Mean ±SD**	**Mean ±SD**		
Amount (Packet)	0	0	1.10±0.82	0.57±0.37	**1.325**	**0.224**
Duration between exposure and arrival to hospital (Hour)	-	-	2.45+0.86	3.15+0.78	**1.220**	**0.325**

The vital signs of the studied groups regarding systolic blood pressure, diastolic blood pressure, pulse, respiratory rate and temperature showed insignificant difference between the studied groups as P value was > 0.05 (Table 3).

Table 3. Comparison between the studied groups regarding the vital signs

Vital signs / Groups	Group I No. (10)	Group II No. (10)	Group III No. (10)	Group VI No. (10)	ANOVA	
					F	p
	Mean ±SD	Mean ±SD	Mean ±SD	Mean ±SD		
Systolic Blood pressure	119.2±8.3	118.0±10.3	121.0±13.7	121.0±16.6	**0.135**	**0.938**
Diastolic blood pressure	75.0±6.2	70.0±8.1	79.0±8.7	73.0±6.7	**2.538**	**0.072**
Pulse	77.5±6.1	77.8±4.3	93.3±15.5	95.9±14.1	**7.835**	**0.000**
Respiratory rate	20.4±2.5	20.3±1.6	21±2.4	20.5±2.3	**0.195**	**0.899**
Temperature	37.0±0.2	36.9±0.17	37.1±0.14	36.8±0.17	**5.679**	**0.003**

SD**=** Standard deviation.

Table 4 illustrated that, all patients in the three studied groups were conscious with normal chest and cardiovascular system examination. However, in group I and II all participants had no complaint from GIT. While, 10% in group III and IV were represented by abdominal pain while 30% in group III and 20% in group IV were manifested by vomiting. Additionally, both vomiting & abdominal pain were the main GIT complaint in 50% and 60% of group III and IV respectively.

Table 4. Comparison between the studied groups regarding conscious level, chest, cardiovascular and GIT examination

	Group I		Group II		Group II		Group III		X^2	P
	No	%	No	%	No	%	No	%		
Consciousness level										
Conscious	10	100	10	100	10	100	10	100	**1.362**	**0.052**
Drowsy	-	-	-		-	-	-	-		
Comatose	-	-	-		-	-	-	-		
Chest										
Normal chest	10	100	10	100	10	100	10	100	**1.362**	**0.052**
Crepitation	-	-	-		-	-	-	-		
Wheezes	-	-	-		-	-	-	-		
CVS										
Normal CVS	10	100	10	100	10	100	10	100	**1.362**	**0.052**
Abnormal CVS	-	-	-	-	-	-	-	-		
ECG										
ECG Normal	10	100	10	100	10	100	10	100	**1.362**	**0.052**

ECG Abnormal	-	-	-	-	-	-	-	-		
										GIT
No complaint from GIT	10	100	10	100	1	10	1	10		
Vomiting	0	0	0	0	3	30	2	20		
Abdominal pain	0	0	0	0	1	10	1	10	**21.954**	**0.000***
Vomiting & Abdominal 0pain	0	0	0	0	5	50	6	60		
Total	10	100	10	100	10	100	10	100	**-**	**-**

SD: Standard deviation *****Significant at P ≤ 0.05

The routine laboratory investigations of the studied groups regarding serum alanine transaminase (ALT),serum aspartate transaminase (AST)**,** serum creatinine , Blood urea, serum sodium (Na),serum potassium (K), PH , PCO_2,HCO_3 , PO2 and SO_2 showed insignificant difference between the studied groups as P value was > 0.05 (Table 5).

Table 5. Comparison between the studied groups regarding the routine laboratory investigation

Groups / Routine Laboratory investigations	Group I	Group II	Group II	Group III	ANOVA	
	Mean ±SD	Mean ±SD	Mean ±SD	Mean ±SD	F	P
Serum alanine transaminase (ALT)	19.1±2.8	19.4±3.9	17.5±5.4	19.9±5.1	**0.055**	**0.651**
serum aspartate transaminase (AST)	19.0±1.7	18±3.1	20.2±6.64	22.8±9.4	**1.185**	**0.329**
Serum creatinine	0.91±0.02	0.89±0.08	0.77±0.18	0.88±0.21	**1.901**	**0.147**
Blood urea	22.0±0.9	21±1.05	22.7±10.2	26.1±8.2	**1.131**	**0.350**
Serum sodium (Na)	141.7±2.9	139.1±1.4	143.1±5.6	140.6±2.36	**2.426**	**0.081**
Serum potassium (K)	4.0±0.3	4±0.31	3.79±0.20	4.21±0.58	**2.091**	**0.119**
PH	7.40±0.05	7.39±0.02	7.43±0.04	7.40±0.03	**2.222**	**0.102**
PCO_2	40.8±1.6	40.7±1.4	41.4±4.1	37.1±5.8	**2.789**	**0.054**
HCO_3	23.4±2.8	22.9±1.1	24.7±3.1	23.8±4.3	**0.624**	**0.604**
PO2	84.0±3.4	84.5±3.1	81.36±5.2	84.8±9.2	**0.744**	**0.533**
SO_2	95.9±0.8	96.4±1.07	95.1±1.3	96.1±2.2	**1.487**	**0.234**

SD=Standard deviation.

The plasma malondialdehyde (MDA) level was statistically insignificant in the three studied groups before and after treatment with vitamin C. However, statistically significant difference in the MDA level before and after treatment was observed in group II & IV as shown in Table 6.

Table 6. Comparison between the studied groups regarding the plasma malondialdehyde (MDA) levels (nmol/ml).

Groups / Plasma MDA levels	Group I No. (10)	Group II No. (10)	Group III No. (10)	Group IV No. (10)	ANOVA	
	Mean ±SD	Mean ±SD	Mean ±SD	Mean ±SD	F	P
MDA levels before treatment (nmol/mL)	1.21±0.78	1.19±0.83	2.11±0.99	2.35±1.88	**2.500**	**0.075**
MDA levels after treatment (nmol/mL)	-	0.71±0.73	1.47±0.59	1.27±1.51	**1.427**	**0.259**
Wilcoxon Signed Ranks Test						
Groups	**Group I (Before & after treatment)**	**Group II (Before & after treatment)**	**Group III (Before & after treatment)**	**Group IV (Before & after treatment)**		
Z	-	-2.803	-1.886	-2.803		
P-value	-	0.005*****	0.06	0.005*****		

SD: Standard deviation.
No**.:** Number of subjects.
*****Significant at P ≤ 0.05

Table (7) revealed significance difference in the plasma total thiol level between the three studied groups both before and after treatment. The plasma total thiol levels showed statistically significant increase in the group II and group IV after receiving treatment.

Table 7. Comparison between the studied groups regarding the plasma total thiol levels (×10^5 µmol/L).

Groups / Plasma total thiol level	Group I No. (10)	Group II No. (10)	Group III No. (10)	Group IV No. (10)	ANOVA	
	Mean ±SD (×10^5µmol/L)	Mean ±SD (×10^5µmol/L)	Mean ±SD (×10^5 µmol/L)	Mean ±SD (×10^5 µmol/L)	F	P
Thiol levels before treatment	15.97±9.03	14.75±11.4	7.09±5.33	5.28±1.52	**4.765**	**0.007***
Thiol levels after treatment	-	23.1±17.2	6.98±4.28	13.68±7.56	**5.621**	**0.012***
Post hoc test						

Groups	GII&GIII	GII&GIV	GIII&GIV
Thiol levels before treatment	0.001*	0.001*	0.024*
Thiol levels after treatment	0.024*	0.014*	0.008*

Wilcoxon Signed Ranks Test				
Groups	Group I (Before & after treatment)	Group II (Before & after treatment)	Group III (Before & after treatment)	Group IV (Before & after treatment)
Z	-	-2.497	-1.580	-2.803
P-value	-	0.013*	0.114	0.005*

SD: Standard deviation.
*****Significant at P ≤ 0.05

Discussion

Every year, about 300,000 people die because of pesticide poisoning worldwide. The most common pesticidal agents are organophosphates and phosphides**.** Phosphide is known as a suicidal poison that can easily be bought and has no effective antidote. [22]

In this study, most of patients were at the age group of 15-25 years, unmarried females, at the stage of secondary school education and came from rural areas. The high incidence of acute intoxication with zinc phosphide in patients with such characteristics could be explained by the higher stress and psychic burden that amounts in individuals with such traits due to the higher susceptibility of youth to excitability and depression, scolding from other family members to females, and stresses of failure in education or lack of employment opportunities. [23, 24] In addition, overcrowding and the easy access to toxic pesticides play an important role in rural areas. [5]

All the Participants in this study were suicidal. The higher incidence of suicidal attempts in patients with acute phosphide poisoning than other modes of poisoning could be attributed to the increasing stresses today facing people particularly the youth including the stresses of unemployment, economic instability and failure of education, love or work. [25]

The amount of zinc phosphide ingested by the studied groups were **(**1.10±0.82) packets in group III and (0.57±0.37) packet in group IV. This is in accordance with the finding of El Nagar & El Mahdy (2011) (5) who stated that the amount of zinc phosphide varied from ½ to two sachets (a sachet is 2–3 g).The duration between exposure and arrival to the hospital in the studied groups of zinc phosphide was 2.45 ± 0.86 and3.15 ± 0.78 hours in group III and group IV respectively. The small amount of zinc phosphide ingested (0.25-2 sachets) and the relatively short duration between ingestion of phosphides and admission to the hospital could be explained as most of patients were females who fear death, only want to draw attention, gain sympathy and get help from their relatives.

All patients in this study were conscious at time of admission to the poisoning centre, their vital signs were within normal range and the most common complaint was vomiting and abdominal pain followed by vomiting alone and the least complaint was abdominal pain alone. These findings partially coincide with the results obtained by Lohani et al. (2002) [26] who studied zinc phosphide poisoning Participants in Nepal poisoning center for 5 years and they found that 178 Participants were associated with zinc phosphide, out of them 79 % showed no symptoms at the time of going to hospital and they did not find a relation between entrance time and intensity of symptoms, and the common symptoms were abdominal pain (62%), vomiting (23%), dizziness (19%) and headache (13%). [26]

El Nagar and El Mahdy (2011) [5] reported that 24.2% of patients with zinc phosphide poisoning were asymptomatic and 36.4% manifested with minor GIT symptoms (abdominal pain, nausea and vomiting) and

29.1% presented with respiratory manifestations (dyspnea and excessive secretions). They attributed the severe manifestations in some zinc phosphide poisoned patients to delay in seeking medical help or ingestion of large amount of zinc phosphide. Moreover, Teimoory et al. (2013) [27] revealed that 90% of patients poisoned by zinc phosphide had an abdominal pain and 78% had nausea and vomiting.

In the present study the routine investigations of the studied groups were within normal ranges. In contrast, El Nagar and El Mahdy (2011) [5] in their study reported an elevation of ALT and AST levels (28.5% and 42.8% of patients respectively) without history of previous liver affection. Kidney function tests were nearly within normal range. They reported also some electrolyte disturbances in the form of hyponatremia and hypokalemia (50% and 25% of their patients respectively), which may be due to repeated vomiting.

As regards the oxidative stress markers of the studied groups, the mean MDA levels after treatment decreased significantly in groups II and IV who received vitamin C while group III showed a non-significant decrease compared with MDA levels at admission.

Phosphine is an inhibitor to cytochrome c oxidase activity. [26] Cytochrome c oxidase enzyme is responsible for the reduction of oxygen into water as the terminal member of the eukaryotic mitochondrial electron-transport chain. When cytochrome c oxidase is inhibited, electrons were directed toward reacting with oxygen to form superoxide anions and reactive oxygen species. [27, 28]

To the best of our knowledge, no studies have been carried out to investigate the oxidative stress in Participants of acute zinc phosphide poisoning. However, many studies evaluated oxidative stress markers in acute aluminium phosphide poisoning.

In rats, Hsu et al. (2002) [29] examined the production of reactive oxygen species, lipid peroxidation and the effectiveness of endogenous glutathione (GSH) as a protective agent against phosphine-induced oxidative damage. They found that phosphine significantly decreased GSH, GSH peroxidase and catalase, while significantly increased lipid peroxidation, DNA oxidation (as 8-hydroxydeoxyguaonsoine) and superoxide dismutase (SOD) levels in kidney and heart. These changes were significantly alleviated by melatonin with the exception of SOD activity in heart tissue.

Morever, Kariman et al. (2012) [30] found that plasma total thiol concentration was lower in patients with acute aluminium phosphide toxicity versus control subjects, while the level of thiobarbituric reactive substance values showed a significant increase in patients compared with healthy controls.

In addition, Tehrani et al. (2012) [31] studied the oxidative stress in Participants of aluminium phosphide and the potential role of N-acetyl cystiene (NAC) as a treatment. They reported that plasma MDA level was significantly reduced in NAC treatment group during the first 24 hours, while in the group without NAC treatment the MDA level significantly increased. They also found that plasma total antioxidant capacity value significantly increased in NAC treatment group.

In this study, the mean plasma total thiol levels increased significantly after treatment in groups II and IV who received vitamin C. This indicates an improvement of the antioxidant defense status on the fact that vitamin C is a useful and effective antioxidant in humans, also, vitamin C can be used as a free radical scavenger in acute stage of lipid peroxidation by any toxin that produces oxidative stress. [32]

On the other hand, group III that received the supportive and symptomatic treatment only showed a non-significant decrease of mean plasma total thiol levels. This could be explained as when the generation of reactive free radicals overwhelms the antioxidant defense, there will be exhaustion of the endogenous antioxidant defenses without receiving enough replenishment.

All participants of zinc phosphide poisoning in this study survived and were discharged from the hospital after achieving clinical improvement. This may be explained by the fact that the ingested amount of zinc phosphide by patients was relatively smaller than the lethal dose that ranges between 4-5 gm**.** Furthermore, survival was mentioned in some case reports after ingestion of 25 gm and 50 gm of zinc phosphide. [33] Additionally, most patients in this study suffered from repeated vomiting after ingestion of zinc phosphide and this could lead to decrease in the absorbed amount of phosphide.

There is a difference between zinc phosphide poisoned patients and aluminum

phosphide poisoned patients as regards the severity of clinical manifestations and the clinical course. This could be attributed to two factors; firstly zinc phosphide is slower in onset since it releases phosphine gas more slowly than aluminum phosphide. [34] Secondly, the lethal dose of aluminum phosphide is small ranging between 0.15 and 0.5 g and the aluminum phosphide tablet contains about 1.68 g of phosphide (21, 35), so ingestion of less than half a tablet (provided it has been freshly opened and has not been exposed to the atmosphere) is lethal while in zinc phosphide the lethal dose reported is 5 g. [36]

Zinc phosphide is a favorite chemical for suicides in Egypt. [36] In 2006, at Ain Shams University, zinc phosphide intoxicated patients represented 10% of all admitted poisoned patients (2224 out of a total of 21,805 Participants). [37] From May 2010 to April 2011, 50 patients were presented to National Egyptian Center of Clinical and Environmental Toxicological Research (NECTR) with zinc phosphide poisoning. Only 2% of patients died after suicidal ingestion of zinc phosphide. [38]

Conclusion

It can be concluded that vitamin C has a potential benefit due to its antioxidant property on zinc phosphide induced-oxidative stress in acute zinc phosphide poisoned patients.

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