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Cardiotoxicity in Acute Zinc Phosphide Intoxicated Patients (A Prospective Study)

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Abstract Phosphides are common pesticides widely used as a grain preservative. Zinc phosphide toxicity is a major health problem with a high mortality rate especially in developing countries. Cardiotoxicity is the main cause of death in phosphide poisonings. **Aim of study:** This prospective study aimed to evaluate the cardiovascular affection among zinc phosphide intoxicated patients admitted to the Poison Control Center, Ain Shams University Hospitals (PCC- ASUH) during the period from 1st June 2013 to 1st June 2014 and to determine factors predicting the outcome of patients with zinc phosphide cardiotoxicity with special reference to determination of serum troponin I.

Methods: Clinical characteristics (systolic blood pressure and central venous pressure), laboratory parameters (liver enzymes, renal function, serum electrolytes: (sodium and potassium), random blood sugar, serum bicarbonate and qualitative assessment of cardiac troponin I, electrocardiographic (ECG) findings and treatment characteristics were all recorded.

Results: Cardiotoxicity was evident in 18 patients out of 144 (12.5%) with zinc phosphide intoxication, twelve of them died. Systolic blood pressure was significantly lower among non survivors (60 ± 7 mmHg) compared to survivors (75 ± 5 mmHg). Central venous pressure was elevated in 6 patients, all of them died. Serum cardiac troponin I was positive in 67% (12 patients) and it did not predict mortality. Eight patients out of the 18 patients had dysrhythmias which found to be a predictor of mortality.

Conclusion: The statistically significant factors useful in predicting mortality in our study were shock (which required vasoactive drugs), increased central venous pressure and ventricular arrhythmias.

Introduction

Phosphides are common pesticides widely used as a grain preservative. It is a major health problem with a high mortality rate especially in developing countries because of its low cost and easy accessibility (Mehrpour et al., 2008 and Louriz et al., 2009). Zinc phosphide is emerging as a common self-poisoning agent in Egypt (El Naggar and El Mahdy, 2011).

In 2012, zinc phosphide represented about 0.01% (196 of 19744 cases) of the cases presented to the Poison Control Center, Ain Shams University Hospitals; (PCC-ASUH) however, it represented about 16% (5 of 30 cases) of the mortality cases (Halawa et al., 2013)

Cardiotoxicity is a significant cause of death in phosphide poisonings (Jadhav et al., 2012). It manifests as refractory hypotension, congestive heart failure and / or electrocardiographic (ECG) abnormalities and elevation of cardiac markers, such as creatine phosphokinase- MB (CPK-MB) fraction (Nakakita et al., 2009).

The aim of the study

Is to evaluate the magnitude of cardiovascular affection among zinc phosphide intoxicated patients presented to the PCC-ASUH during the period from 1st June 2013 to 1st June 2014 and to assess the role of some investigational parameters as predictors for the outcome in these patients with special reference to serum troponin I.

Subjects and Methods

Subjects

Eighteen patients were selected from all patients with acute zinc phosphide intoxicated patients presented to PCC-ASUH during the period from 1st June 2013 to 1st June 2014 and were prospectively investigated for evidence of cardiovascular affection by the presence of hypotension and /or electrocardiographic (ECG) changes. The diagnosis of phosphide intoxication was based preliminary on history of consumption of the

fine black powder rodenticide (Bumbrah et al., 2012) obtained from the patient or his relatives, and manifestations on admission.

Exclusion criteria

Patients presented with unclear diagnosis of poisoning, consumption of more than one substance or those with previous history of cardiovascular disorders as well as patients with risk factors such as hypertension, diabetes or smoking were all excluded from the study.

Patients were divided into 2 groups, according to their mortality outcome:

The first group: (Survivors) and the second group: (Non Survivors)

Ethical Considerations

Approval of the Ethical Committee and the Head of the PCC-ASUH were obtained. Informed consents were also obtained from the patients or their relatives. They were assured that any information they provided would be kept strictly confidential and anonymous.

Methods

I) Data collection

1. Demographic data
   - Demographic data: age, sex, occupation and residence.
2. Intoxication data
   - Route of poisoning.
   - Manner of poisoning.
   - Delay time between intoxication and arrival to PCC-ASUH.
3. Clinical data for cardiac affection: (systolic blood pressure and central venous pressure). Normal central venous pressure: (8-20 cm H_2O) (Marino, 2010).
4. Treatment

Gastric lavage (according to patient's hemodynamic status) was performed to patients who presented within the first two hours of intoxication. Then they received oral sodium bicarbonate (Proudfoot, 2009).

Intravenous fluids were administered for resuscitation of patients. Inotropic drugs such as dopamine (20 µg/kg/min) or dobutamine (10 µg/kg/min) (Marino, 2010) and antiarrythmics drugs were given to all of them. Mechanical ventilation was instituted if indicated. All patients were admitted into intensive care unit (ICU).

II) Studied parameters

1) Laboratory parameters

- Liver enzymes: aspartate aminotransferase (AST) (Normal range: up to 37 U/L) and alanine aminotransferase (ALT) (Normal range: up to 42 U/L) (Nyblom et al., 2006).
- Renal function: serum creatinine (Normal range: up to 1.4 mg/dl) (Bazari, 2007) and urea (Normal range: 10- 50 mg/dl) (Sue and Vintch, 2005)
- Serum electrolytes: serum sodium (Normal range: 135- 150 mEq/L) and serum potassium (Normal range: 3.5 -5.5 mEq/L) (Faix, 2000), random blood sugar (Normal range: 72- 144 mg/dl) (Lehman and Krumholz, 2009) and sodium bicarbonate (Normal: 22-26 mEq/L) (Cox, 2001).
- Qualitative assessment of cardiac troponin I (CAL-Tech Diagnostics, Inc Chino, California, USA).

2) ECG: An initial was recorded and repeated when required.

III) Statistical Analysis

The SPSS (Statistical Package for the Social Science) version 13, computer program was used. $P < 0.05$ was considered significant. $P < 0.001$ was considered highly significant.

Results

Cardiotoxicity was evident in 18 patients presented with zinc phosphide intoxication during the study period. Twelve cases of them died, death occurred within mean 16±12 hours from the time of zinc phosphide ingestion.

The mean age of patients was 31±7 years. Table (1) represents that females were predominant than males, 50% of cases were unemployed, about 28% were laborers while 4 cases were employed. Eighty three percent of cases were from urban areas while about 17% were from rural areas. Oral ingestion was the main route and inhalation poisoning occurred during pesticide application on grains in one case only. As regards the manner of poisoning; suicide was the main manner of poisoning in all cases except the case in which accidental exposure occurred during pesticide application on grains. Most of the patients were presented with delay time between 6-8 hours.

Clinical manifestations at presentation:

Nausea and vomiting were the initial manifestations in 13 patients (72%), abdominal colic was found in 11 patients (61%), while agitation was the first manifestation in 10 patients (55%). All of them were oriented and fully conscious.

Mean systolic blood pressure was low in all cases (65±10) mmHg, but it was significantly lower in non survivors (60 ± 7) mmHg compared to those who survived (75 ± 5) mmHg, (Table 2). Diastolic blood pressure was barely auscultated.

Central venous pressure was elevated in 6 patients (33%), all of them died. There was significant difference between both groups as regards central venous pressure (Table 3).

Laboratory findings

Increased serum levels of AST (165.5±45.7 U/L for the survivors and 127.25±83.35 U/L for the non survivors) and ALT (104±32 U/L for the survivors and 105.92±70.58 U/L for the non survivors) were observed in all patients. Serum urea was statistically indifferent between both groups (44±9.96 mg/dL for the survivors and 40.67±4.05 mg/dL for the non survivors). Also, there was insignificant difference between serum creatinine in both groups (1.2±0.37 mg/dL for the survivors and 1.05±0.24 mg/dL for the non survivors) (Table 4).

Although mean serum bicarbonate was lower in non survivors group 11.75 ± 3.25 mEq/L compared to the survivors (13±1.1 mEq/L), yet this was not statistically significant (Table 4).

Table 4 also shows that both groups of patients under the study had normal mean serum levels of sodium (Na^+) (136.50±1.64 mEq/L for the survivors and 136.75±2.01mEq/L for the non survivors), mean serum potassium (K^+) (3.95±0.16 mEq/L for the survivors and 3.90±0.20 mEq/L for the non survivors), and mean random blood sugar (RBS) was (105.33±11.33gm/dL for the survivors and 128.50±52.53 gm/dL for the non survivors).

Table 5 shows that serum cardiac troponin I was positive in 12 patients (66.7%), which did not predict mortality as no significant difference was found between both groups as regards the presence of cardiac troponin I.

Electrocardiographic findings

Eight patients out of the 18 patients included in the current study had dysrhythmias, in the form of wide complex ventricular tachycardia, atrial fibrillation, diffuse ST segment elevation or abnormal repolarization in the form of (inverted T wave) as presented on ECG (Table 6 and figures 1, 2, 3 and 4). The presence of electrocardiographic abnormalities predicted mortality (Table7).

Treatment and outcome

Gastric lavage was performed in 7 patients. All of them received sodium bicarbonate. All of the 18 patients were hypotensive despite adequate fluid resuscitation and required vasoactive support, predominantly dopamine and dobutamine (Table 8).

Mechanical ventilation was required in 6 patients (33%) whom all died (Table 9).

For prediction of zinc phosphide induced cardiotoxicity, the analysis showed that, increased central venous pressure (CVP) had the highest sensitivity (100%) and specificity (100%) and followed by shock represented by decreased systolic blood pressure with sensitivity (75%) and specificity (100%). ECG found to have sensitivity (66.7%) and with (100%) specificity. Cardiac troponin I shows non significance value as a predictor of cardiac involvement (Table 10).

Table (1): Percentage of distribution of demographic and intoxication data in patients admitted to PCC-ASUH with acute zinc phosphide induced cardiotoxicity.

Description	Number (N)	Percentage (%)
Sex		
Female	13	72.2
Male	5	27.8
Total	18	100
Occupation		
Unemployed	9	50
Laborers	5	27.8
Employed	4	22.2
Total	18	100
Residence		
Urban areas	15	83.3
Rural areas	3	16.7
Total	18	100
Route of poisoning		
Oral route	17	94.4
Inhalation	1	5.6
Total	18	100
Manner of poisoning		
Suicidal	17	94.4
Accidental	1	5.6
Total	18	100

Table (2): Student "t" test comparing mean systolic blood pressure in patients admitted to PCC-ASUH with acute zinc phosphide induced cardiotoxicity (Survivors versus Non Survivors).

Patient groups	Systolic blood pressure (mmHg) Mean ± SD	Range (mmHg)	t-test	p-value
Survivors (N=6)	75 ± 5	70-80	4.382	<0.001*
Non Survivors (N=12)	60 ± 7	50-70		

**P < 0.001 is considered highly significant, N: Number*

Table (3): Fisher's exact test comparing central venous pressure in patients admitted to PCC-ASUH with acute zinc phosphide induced cardiotoxicity (Survivors versus Non Survivors).

	Survivors		Non Survivors		Total		P-value
	N	%	N	%	N	%	
Decreased	0	0%	6	50%	6	33.3%	<0.001*
Normal	6	100%	0	0%	6	33.3%	
Increased	0	0%	6	50%	6	33.3%	
Total	6	100%	12	100%	18	100.0%	

**P < 0.001 is considered highly significant, N: number*

Table (4): Student "t" test comparing laboratory parameters in patients admitted to PCC-ASUH with acute zinc phosphide induced cardiotoxicity (Survivors versus Non Survivors).

Laboratory parameter	Survivors (N = 6)		Non Survivors (N = 12)		Independent t-test	
	Mean± SD	Range	Mean± SD	Range	T	p-value
AST (U/L)	165.5±45.7	120 – 211	127.25±83.35	49 – 245	0.532	0.31
ALT (U/L)	104±32	77 – 135	105.92±70.58	36 – 200	-0.679	0.97
Urea (mg/dL)	44±9.96	32 – 57	40.67±4.05	38 – 48	0.907	0.28
Creatinine (mg/dL)	1.2±0.37	0.8 – 1.6	1.05±0.24	0.8 – 1.4	0.89	0.26
Serum bicarbonate (mEq/L)	13.00±1.10	12 – 14	11.75±3.25	7 – 15	0.904	0.379
Serum sodium (mEq/L)	136.50±1.64	135 – 138	136.75±2.01	135 – 140	-0.263	0.796
Serum potassium (mEq/L)	3.95±0.16	3.8 – 4.1	3.90±0.20	3.7 – 4.2	0.537	0.599
RBS gm/dl	105.33±11.33	95 – 116	128.50±52.53	92 – 215	-1.053	0.308

P > 0.05 is considered insignificant, N: number, RBS: Random blood sugar

Table (5): Fisher's exact test comparing cardiac troponin I in patients admitted to PCC-ASUH with acute zinc phosphide induced cardiotoxicity (Survivors versus Non Survivors).

Outcome	Survivors		Non Survivors		Total		P-value
	N	%	N	%	N	%	
Negative	0	0.0%	6	50.0%	6	33.3%	0.054 *
Positive	6	100.0%	6	50.0%	12	66.7%	
Total	6	100.0%	12	100.0%	18	100.0%	

** P > 0.05 is considered insignificant, N: number, %: percentage*

Table (6): ECG abnormalities in patients admitted to PCC-ASUH with acute zinc phosphide induced cardiotoxicity.

ECG abnormalities	N	%
Wide complex ventricular tachycardia	4	50
Atrial fibrillation	1	12.5
Diffuse ST segment elevation	2	25
Abnormal repolarization (inverted T wave)	1	12.5
Total	8	100

N: Number, %: percentage

Table (7): Fisher's exact test of ECG changes in patients admitted to PCC-ASUH with acute zinc phosphide induced cardiotoxicity (Survivors versus Non Survivors).

ECG abnormality	Survivors		Non Survivors		Total		P-value
	N	%	N	%	N	%	
Normal	6	100.0%	4	33.3%	10	55.6%	0.013 *
Abnormal	0	0.0%	8	66.7%	8	44.4%	
Total	6	100.0%	12	100.0%	18	100.0%	

**P < 0.05 is considered significant, N: number,%: percentage*

Table (8): Fisher's exact test of vasoactive drugs administration in patients admitted to PCC-ASUH with acute zinc phosphide induced cardiotoxicity (Survivors versus Non Survivors).

Vasoactive drug	Survivors		Non survivors		Total		P-value
	No.	%	No.	%	No.	%	
Dopamine	6	100.0%	6	50.0%	12	66.7%	0.054*
Dopamine, dobutamine	0	0.0%	6	50.0%	6	33.3%	
Total	6	100.0%	12	100.0%	18	100.0%	

**P > 0.05 is considered insignificant, N: number,%: percentage.*

Table (9): Fisher's exact test of patients admitted to PCC-ASUH with acute zinc phosphide induced cardiotoxicity who required mechanical ventilation (Survivors versus Non Survivors).

	Survivors		Non Survivors		Total		P-value
	N	%	N	%	N	%	
Did not require mechanical ventilation	6	100.0%	6	50.0%	12	66.67%	0.054*
Required mechanical ventilation	0	0.0%	6	50.0%	6	33.33%	
Total	6	100.0%	12	100.0%	18	100.0%	

**P > 0.05 is considered insignificant, N: number,%: percentage.*

Table (10): Sensitivity and specificity of CVP, systolic blood pressure, ECG and cardiac troponin I among patients admitted to PCC-ASUH with acute zinc phosphide induced cardiotoxicity.

	Sensitivity	Specificity	AUC	Accuracy
CVP	100	100	100	100
Systolic BP	75	100	93.7	87.5
ECG	66.7	100	83.3	83.35
Cardiac Troponin I	50	100	75	75

CVP: Central venous pressure

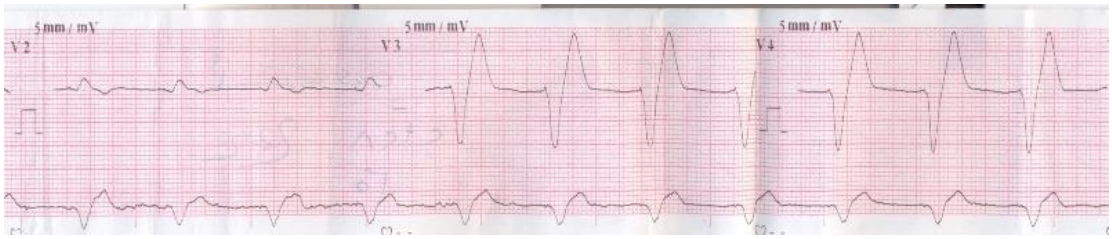

Figure (1): A strip of ECG of a 38- year-old male patient acutely intoxicated with zinc phosphide, his systolic blood pressure 50 mmHg shows wide complex ventricular tachycardia.

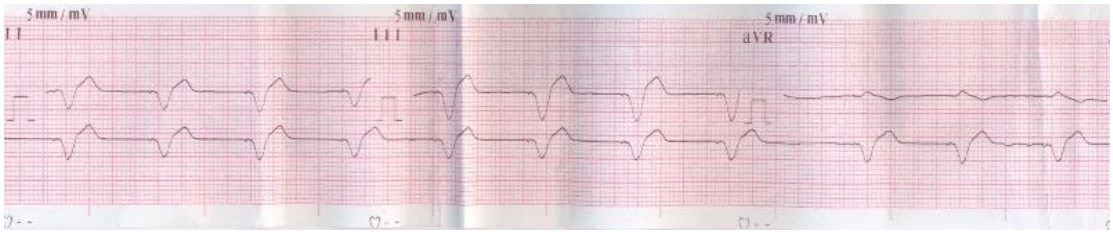

Figure (2): A strip of ECG of a 31 - year-old male patient, acutely intoxicated with zinc phosphide, his systolic blood pressure was 60 mmHg shows wide complex ventricular tachycardia.

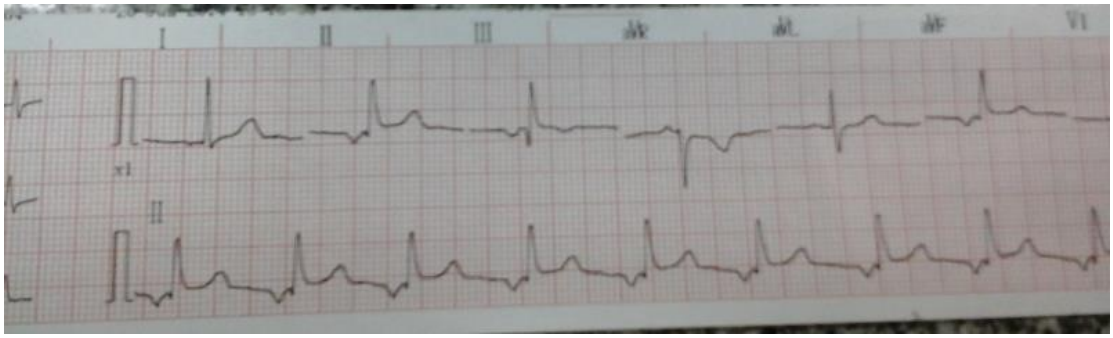

Figure (3): A strip of ECG of 23 - year-old female patient acutely intoxicated with zinc phosphide, her systolic blood pressure was70 mmHg shows diffuse ST segment elevation.

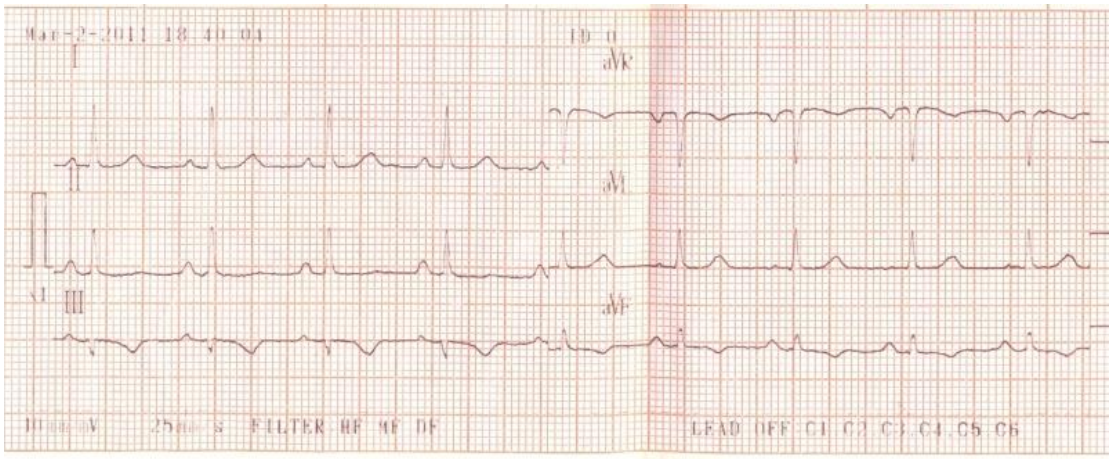

Figure (4): A strip of ECG of a 34 - year-old female patient acutely intoxicated with zinc phosphide, her systolic blood pressure 70 mmHg shows abnormal repolarization in the form of inverted T wave.

Discussion

As regards the mean age of patients in the current study, it was 31±7 years which could be due to the susceptibility of this age group to social and economic problems (Kar, 2010). In this study, the female to male ratio was 2.6:1, similar results of increased female to male ratio was reported by (Abdelwahab et al., (2011) and Moghaddam and Pajoumand (2007)). This may be due to liability of females to attempt suicide more than males. Concerning their occupation, half of the patients were unemployed. Most of our patients were from urban areas which were also in accordance with Abdelwahab et al., (2011) who attributed that to the location of PCC-ASUH in Great Cairo, and that patients from rural areas can be presented only in severe cases.

Oral ingestion was the main route of poisoning except in a single accidental case, where inhalation poisoning occurred. This is explained by the availability of the zinc phosphide in the form of powder that can be easily ingested. As regards the manner of poisoning; suicide was the manner of poisoning in all cases except one case in which accidental exposure occurred during pesticide application on grains. This agrees with Mishra et al., (2006) who reported that the majority of cases with zinc phosphide intoxication were suicidal. This may be attributed to its easy availability and low cost.

The majority of the patients presented with delay time between 6-8 hours. This is due to the fact that zinc phosphide is recognized as the slowest acting of the commonly used rodenticide giving the victim time to seek medical advice (Assem and Takayama, 2007)

Nausea, vomiting, abdominal colic and agitation were the initial manifestations. This comes in agreement with El Naggar and El Mahdy (2011) who found abdominal pain and vomiting in 75.8% of their patients. Nausea and vomiting were common among both survived and non-survived patients in the study done by Hosseinian et al. (2011). This could be attributed to irritation of gastrointestinal tract mucous membrane (Jain et al., 2005).

Systolic blood pressure was low in all cases, but it was significantly lower in patients who died compared to those who survived. Central venous pressure was elevated in 6 patients (all of them died), indicating cardiogenic shock (DeCotiis et al., 2014) Singh et al., (2003)and Akkaoui et al., (2007) reported high CVP and revealed left ventricle hypokinesia by echocardiography in acute aluminum phosphide intoxicated patients. They explained their results to be due to myocardial lesion which might be responsible for hemodynamic failure. Cardiac manifestations in the form of hypotension and arrhythmias were predominant and indicated poor prognosis in the study done by Mathai and Bhanu (2010). However in the current study, shock could be attributed to hypovolemia due to repeated episodes of vomiting or peripheral vasodilatation, which can explain the cause of shock in some of these cases as evidenced by decrease central venous pressure. Although we cannot exclude the cardiac element as in these cases as fluid therapy did not fully correct the shock.

Increased serum levels of (AST) and (ALT) were observed in all patients. However, this was of no significant value. Hepatotoxicity manifested as elevated ALT was found in the studies done by (Karanth and Nayyar (2003), Chomchai (2004), Khurana et al. (2009), and El Naggar and El Mahdy (2011)). Deranged liver function test was associated with poor outcome in the study performed by Hosseinian et al. (2011).

Saleki et al. (2007) stated that phosphine can cause liver dysfunction, especially after the first day of poisoning, and the main histopathological changes found were fine cytoplasmic vacuolization of hepatocytes and sinusoidal congestion. Congestion, edema and centrizonal necrosis of the liver were found on histopathological examination in the study of Musshoff et al. (2008).

Phosphine a nucleophile, acts as a strong reducing agent capable of inhibiting cellular enzymes involved in several metabolic processes. It blocks the enzyme cytochrome C oxidase as a result of which

mitochondrial oxidative phosphorylation is inhibited (Singh et al., 2006). It also disturbs the mitochondrial morphology, inhibits respiration by 70% and causes severe drop in mitochondrial membrane potential, causing in turn, rapid cellular death (Proudfoot, 2009).

All cases in the current study had metabolic acidosis with decreased level of serum bicarbonate. Although serum bicarbonate was lower in the patients who died compared to the patients who survived, yet it was not statistically significant.

Metabolic acidosis was also observed by (Orak et al. (2008), Proudfoot (2009), and El Naggar and El Mahdy (2011)). Hosseinian et al. (2011) considered severe metabolic acidosis as a predictor of mortality. Also, Louriz et al. (2009) considered low serum bicarbonate value (less than 15 mEq/L) as a predictor of mortality.

In the current study both groups of patients had normal serum levels of sodium and potassium. Electrolyte disturbance was not found to be an indicator of mortality in a study performed by Mehrotra et al. (2012).

In this study random blood sugar was within normal range. However, Louriz et al. (2009) reported hyperglycemia which was not related to mortality. Mehrotra et al. (2012) in their study found variable random blood sugar levels in patients of acute phosphide poisoning. The difference in random blood sugar between group of survivors and non survivors group was found to be border line, although it was higher among non survivors (Mehrpour et al., 2008). However, Abder-Rahman (1999) in his study reported that hyperglycemia could be used as a prognostic factor in phosphide intoxicated patients.

Direct myocardial injury was evidenced in our study by the finding of increased serum cardiac troponin I. Myocardial muscles injury was demonstrated by Shah et al., (2009) who found that in non survivors patient showed myocyte vacuolation, areas of myocytolysis and degeneration. This finding could be due to myocardial necrosis resulted from the release of reactive oxygen species (Chugh et al., 1997).

Phosphine inhibits mitochondrial cytochrome C oxidase enzyme which may lead to cardiotoxicity. It is also known to inhibit protein synthesis and enzymatic activity, particularly in the mitochondria of heart cells. This can lead to a blockage of mitochondrial electron transport chain (Aggarwal, 2007).

Our finding that half of deaths due to zinc phosphide cardiotoxicity and with negative troponin I represent a significant population at risk and reason for not relying on troponin alone as a predictive of mortality.

Eight patients out of the 18 patients in our study had dysrhythmias, in the form of wide complex ventricular tachycardia, atrial fibrillation, ST segment elevation and diffuse repolarization abnormalities which were predictive of mortality.

Electrocardiographic changes were also detected in other studies performed by Shadnia et al., (2009). Atrial fibrillation, junction rhythm, ventricular and atrial extra systoles and ventricular tachycardia were reported by Gupta et al. (1995). Siwach et al. (1998) reported the incidence of various arrhythmias in aluminum phosphide intoxicated patients in the form of ventricular tachycardia in 40%, ventricular fibrillation, supraventricular tachycardia in 46.7% and atrial flutter/fibrillation. ST-segment elevations and diffusely abnormal repolarization were found by Akkaoui et al. (2007). Louriz et al. (2009) reported repolarization disorders, such as ST segment depression, ST segment elevation and T wave inversion.

Katira et al. (1990) showed that during the initial 3–6 hours, sinus tachycardia was predominant, in the 6–12 hours period; ST segment-T wave changes and conduction disturbances were reported.

All the 18 patients under the study were hypotensive despite adequate fluid resuscitation and required vasoactive support, predominantly dopamine and dobutamine. This was in agreement with the findings of Akkaoui et al. (2007) and Mathai and Bhanu (2010).

In our study, mechanical ventilation was required in 6 patients (non survivors). Louriz et al. (2009) considered the need for mechanical ventilation as an indicator of poor outcome.

It is concluded that the overall mortality from zinc phosphide poisoning in the PCC-ASUH during the study period was 12 out of 18 patients. The statistically significant factors which were useful in predicting mortality in our study were increased central venous pressure, shock (which required vasoactive drugs) and ventricular arrhythmias

One of the limitations of this study was our inability to measure the blood level of phosphine, which prevented us from drawing firm conclusions with regard to its temporal relation to cardiotoxicity. However, the consistency of our results with previous studies suggests that phosphide induced cardiotoxicity is the main cause of mortality and morbidity in cases of zinc phosphide poisoning. This cardiotoxicity manifested with refractory shock, ECG changes, and increased serum cardiac troponin I level.

Recommendations

More policies are needed to restrict the availability and sale of zinc phosphide to limit the magnitude of the problem. Larger prospective studies in the future are needed to support our results. Early sensitive markers of cardiac affection and echocardiography performance are required to detect zinc phosphide cardiotoxicity. Finally further researches to find specific antidote is highly recommended.

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الملخص العربى

السمية القلبية فى مرضى التسمم الحاد بفوسفيد الزنك (دراسة مستقبلية)

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يعد فوسفيد الزنك من أكثر المبيدات الحشرية انتشارا و يستخدم لحفظ الغلال. و يمثل التسمم بفوسفيد الزنك مشكلة صحية كبيرة و يصاحبه ارتفاع فى معدل الوفيات خصوصا فى الدول النامية. و تعتبر السمية القلبية من أهم أسباب الوفاة فى حالات التسمم بالفوسفيد.

الهدف من الدراسة: تهدف هذه الدراسة المستقبلية الى تقييم حجم التأثير على القلب و الاوعية الدموية لمرضى التسمم بفوسفيد الزنك. وتم حجزهم بمركز علاج التسمم بمستشفيات جامعة عين شمس خلال الفترة بين يونيو ٢٠١٣ الى يونيو ٢٠١٤ و تحديد عوامل التنبؤ بنتائج مرضى التسمم بفوسفيد الزنك مع الاشارة الى أهمية الكشف عن التروبونين I بالدم.

طريقة الدراسة: تم تسجيل الخصائص الاكلينيكية (ضغط الدم الانقباضى، الضغط الوريدى المركزى)، الفحوص المعملية (الاملاح بالدم: الصوديوم و البوتاسيوم)، سكرالدم العشوائى ، انزيمات الكبد و وظائف الكلى، الغازات بالدم، التقييم النوعى للتروبونين I القلبى ونتائج رسم القلب الكهربائى وكذلك الخصائص العلاجية.

النتائج: وجد التسمم القلبى فى ١٨ حالة من اجمالى ١٤٤حالة قدمت بالتسمم بفوسفيد الزنك وتوفى منهم ١٢حالة. وتبين وجود انخفاض فى ضغط الدم الانقباضى بشكل كبير فى حالات الوفاة (٧± ٦٠مم زئبق) بالمقارنة بالناجيين (٥± ٧٥مم زئبق) ولقد ارتفع الضغط الوريدى المركزى فى ستة حالات و توفوا جميعا.و كانت نسبة وجود التروبونين I بالدم ايجابية فى ٦٧% (١٢ حالة) ولم يكن مؤشرا للوفاة. كما وجدت اضطرابات فى الرسم القلب الكهربائى فى ٨ مرضى من اجمالى ١٨ حالة وكانت مؤشرا للوفاة.

الخلاصة: إن العوامل ذات دلالة احصائية مفيدة لتنبؤ الوفاة فى هذه الدراسة هم الهبوط القلبى (و الذى تتطلب الادوية الفعالة على الاوعية) ، ارتفاع الضغط الوريدى المركزى وعدم انتظام ضربات القلب البطينى.

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