Cardiac injury in acute ZINC phosphide ingestion

Thesis

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Cardiovascular medicine

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بســـــم الله الـــــرحمن الـــــرحيم

(قَـــالُوا سُـــبْحَانَكَ لا عِلْـــمَ لَنَـــا إِلَّـــا مَـــا
عَلَّمْتَنَـــا إِنَّـــكَ أَنْـــتَ الْعَلِيـــمُ الْحَكِيـــمُ)

صـــــدق الله العظيـــــم
(البقـــرة : 2 3)

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"God bless them all"

Abstract

Cardiac injury in acute zinc phosphide ingestion is a serious, relatively common manifestation in zinc phosphide toxicity especially in severs intoxicated patients The management of zinc phosphide ingestion represents a therapeutic challenge and is still controversial. The main aim of management is to sustain life with appropriate resuscitation measures till PH_3 is excreted from the body. Hence early recognition and institution of therapy are mandatory.

Key words :

Zinc phosphide - Cardic injery - Metabolic acidosis - Echocardiography - Electrocardiography - Troponin T.

DEDICATION

Words will never be able to express my deepest gratitude to my **Mother, father, Wife, Daughter yasmine, and my Son Omar** For their help.

LIST OF CONTENT

`LIST OF ABBREVATION

ABG	Arterial blood gases
AF	Atrial fibrillation
ALT	Alanine transaminase.
AMA	Against medical advice
ANOVA	Analysis of variance
ARDS	acute respiratory distress syndrome
AST	Aspartate transaminase
ATSDR	Agency for Toxic Substance and Disease Registry
CNS	Central nervous system
CVS	Cardiovascular system
CVP	Central venous pressure
ER	Emergency room
ECG	Electrocardiography
EPA	Environmental Protection Agency
GIT	Gastrointestinal tract
GC/MS	Gas chromatography/Mass spectrometry
H2	Histamine 2
HCO_3^-	Bicarbonate ion
ICU	Intensive Care Unit
IV	Intravenous
K	Potassium
Na	Sodium
NECTR	National Egyptian Center of Clinical and Environmental Toxicological Research
PaCO2	Arterial carbon dioxide partial pressure
PaO2	Arterial oxygen partial pressure
PCCA	poison control center of ain shams
PEEP	Positive end expiratory pressure
pH	Blood H ion concentration
PH3	Phosphine gas
Ppm	Part per million
PVC	Premature ventricular contraction
QTc	QT corrected

RTA	Renal tubular acidosis
SD	Standard deviation
SPSS	Statistical Package for Social Science
WHO	World Health Organization

List of tables

List of Figures

INTRODUCTION

Acute poisoning with pesticides is a global public health problem and accounts for as many as 300,000 deaths worldwide every year **(Goal and Aggarwal, 2007).** The incidence of fatalities from self-poisoning (suicidal or accidental) in the developing world is commonly 10–20%, but for some pesticides in particular it may be as high as 50–70% (**Eddleston, 2000)**. The causes of the high case fatality are multifactorial but include the high toxicity of locally available poisons, difficulties in transporting patients across long distances to hospital, the paucity of health care workers compared with the large numbers of patients, and the lack of facilities, antidotes, and training for the management of pesticide-poisoned patients (**Eddleston, 2000 ; Buckley et al., 2004**).

Rodenticides pose a risk of accidental poisonings for several reasons; they are agents specifically designed to kill mammals, often their toxicity is very similar for the target rodents and for humans. Since rodents usually share environments with humans and other mammals, the risk of accidental exposure is an integral part of the placement of baits for the rodents **(Fishel, 2005).**

Zinc phosphide has been widely used as a rodenticide since 1942 and it is acceptable for continued registration **(PACR, 2006).** Although zinc phosphide used as rodenticide has largely been replaced in the United States, by anticoagulants because of its hazard to non-target organisms and its acute oral toxicity **(Extoxnet, 2002*),*** however, it is still widely used in the developing countries because it is cheap and effective **(Perry, 2007).**

Cardiovascular toxicology is at an exponential phase of growth. Looking to the future, the demand for a comprehensive understanding of cardiovascular toxicology is very high. The adaptation of more advanced approaches and techniques to formulate more integrated assessment of cardiovascular toxicity will greatly help the creation of new knowledge of cardiovascular toxicology**. (Verloes et al, 1996).**

AIM OF THE WORK

1- Evaluating the cardio- toxic effect of zinc phosphide in patients admitted to (NECTR) on a clinical and laboratory basis.

2- Performing a thorough analysis of the poisoned patients in order to improve the prognosis and the outcome.

3- Suggesting recommendations for prevention in order to reduce the incidence of toxicity with zinc phosphide.

Rodenticides intoxication

Rodenticide is any product commercially marketed to kill rodents, mice, squirrels gophers and other small animals. Rodenticides are a heterogeneous group of chemicals bearing little or no relationship to one another, apart from historic use rodenticids. A perfect rodenticide is one that kills rodents but not toxic to human or non rodent pets. Instead, a wide Varity of less than perfect rodenticide are commercially available differing from one another in chemical composition, mechanism for killing rodents, and toxicity to humans **(Aggarwal and Kaur, 2010).**

Rodenitcide pests cause major economic losses to crops and inflict health risk to people and livestock in villages (**Leung et al, 2007).** They cause yearly losses of 20%to 30% in grains and other food storage facilities. These pests' harbors as well diseases in the form of fleas that carry bacteria and other organism (**Hodgson, 2004**).They are responsible for animal extinctions more than any other cause ***(Burns, 2009)***.

There are three types of rodenticides (Anticoagulants, calcium releaser and acute toxins).

A-Anti coagulants

Before 1940 the use of rodenticide depends on heavy metals such as arsenic, thallium, phosphorus and strychnine. This was changed in 1940s as investigators discovered warfarin (**Ware, 2000).** Dicumarol (4-hydroxy coumarine) and other natural anticoagulants are found in sweet clover. Coumarin derivatives are used both therapeutically and as rodenticides. Warfarin adrevative of dicoumarone is used widely as a therapeutic anticoagulant but is no longer popular as a rodenticide because rats and mice have become resistant. The most common anticoagulant rodenticides available today contain long-acting **"superwarfarins"** such as brodifacoum, diphacinone, bromadiolon, chlorophacinone, difenacoum,

pindone, and valone, which have profound and prolonged anticoagulant effects ***(Ilene, 2004).***

Litovitz and his colleges (2001), reported in the annual report of the American Association of Poison Control Centers**,** 15,855 human exposures to anticoagulant compounds. Standard warfarin accounted for (< 3%) of the total number, leaving greater than (97%) of exposures in the super warfarin group. Thus, the emergence of these compounds as a source of exogenous coagulopathy in humans has become more evident.

Blood in urine , bloody stools, bruising, bleeding, vomiting of blood, low blood pressure and shock can be detected after acute poisoning with anticoagulant rodenticides ***(CRRU, 2009).***

Diagnosis is based on clinical picture and prolonged prothrombin time (PT)

Treatment consists of high doses of vitamin K ***(Poovalingam et al, 2002)***.

B-Calcium releaser

Cholecalciferol :

Calciferous (vitamin D), cholecalciferol (Vitamin D_3) and ergocalciferol (Vitamin D_2) are used as rodenticides. Being toxic to rodents for the same reason they are important to humans: they affect calcium and phosphate homeostasis in the body. Vitamins D are essential in minute quantities (only a fraction of a milligram per kilogram body weight daily) and like most fat soluble vitamins, they are toxic in larger doses, causing hypervitaminosis. If the poisoning is severe enough it leads to death. In rodents that consume the rodenticidal bait, it causes hypercalcemia, ***(Whisson, 1996).***