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Brief communication

Acute exposure to white phosphorus: a topical problem in Ecuador (South America)

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Abstract

There is evidence of a growing number of cases of intoxication due to white phosphorus (WP) in Ecuador. These cases mainly occur after consuming the so-called 'little devils'. This work involved a retrospective-type observational descriptive analysis of all the cases registered in the country in a time interval of 19 years. A total of 590 cases were described in this period. The age of the intoxication cases ranged from 2 to 70 years, with an average of 18.27 years. With respect to genre, women were the most affected (68%). The aetiology observed was intentional or violent in 98.6% of the cases registered, the onset of the problem being the most commonly cited cause. The quantity consumed varied between 1 and 70 tablets (0.3–21 g). WP produces hepatic and renal failure, which leads to secondary cardiac failure, which is the cause of death.

The mortality observed exceeds 10% of the intoxicated people. The main reason for the ingestion of WP is for autolitical purposes, due to a high component of depressive affectation in the majority of patients. Supplementary psychiatric studies will be required in the future to determine the main causes. The posterior hepatic damage, which is considered as an after-effect could not be confirmed, as there are no follow-up protocols, although the hepatotoxity of this substance is obvious. The utility of a specific antidote for this type of intoxication has not been demonstrated. However, the importance of an early gastric wash/aspiration and the administration of large doses of activated carbon prove to be very important. A determinant factor in the quality of survival is early and appropriate health care for the intoxicated people.

Keywords: White phosphorus; Poisoning; Epidemiology; Explosives

1. Introduction

Over the past few years, an increase in the growth of acute intoxications due to white phosphorus (WP), also called yellow phosphorus, has been observed in Ecuador. This substance is forbidden in the majority of the countries of the region and in the world, but it is still used in the traditional and homemade manufacture of explosives and firecrackers, and above all, in the manufacture of the so-called 'little devils'. Little devils, explosive tablets lit by violent friction, weigh approximately 0.3 g, and comprise quantities of WP that vary between 1 and 10% of their weight, and also comprise up to 40% potassium chlorate in addition to a mixture of flour and other non-declared compounds [1].

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Classically, WP intoxication was observed in match factory workers. This substance has been forbidden or restricted in the majority of the developed countries due to its dangerous effects, which is why this problem is no longer mentioned in the majority of the toxicological registers, and therefore, there are no epidemiological data in this regard.

WP is a semi-metal element, which exists in three allotropic forms: white, black and red. The typical pale white colour turns yellow due to the presence of impurities such as arsenic, toluene and carbon [2]. It is smooth in consistency, not sandy, it smells like 'garlic' and is soluble in absolute alcohol, ammoniac, ether, toluene and benzene [3,4]; furthermore, it rusts quickly in the presence of air, turning into phosphoric acid [4,5].

WP is used to manufacture insecticides in paste-form, made up of a mixture of yellow phosphorus crushed with water and flour; it is also used in fume control systems and to analyse gases [2,6,7]. In Ecuador, the clandestine use of WP is aimed at the manufacture of homemade explosives and firecrackers. The toxic dose is 15–100 mg (1 mg per kg weight) in adults, and a small dose of 50 mg can be lethal [8]; although there have been cases of survival after ingesting 1570 mg [9]. The lethality rates vary between 20 and 50% and the prognosis is good if the patients survive for more than 6 days [12]. The prognosis depends on the quantity of phosphorus absorbed in the intestine, which seems to be greater if the absorption vehicle is liquid.

The acute intoxication normally develops in three stages. (1) Contact with the skin produces painful second and third degree burns that are slow in healing. Ingestion causes burns in pharynx, oesophagus and stomach, nausea, vomition, diarrhoea, acute abdominal pain, garlic-smelling breath; luminescent faeces and vomition have been described, which facilitate the diagnosis. Shock may occur, which causes death 24–48 h after ingestion. (2) Relative periods of 7 days free from symptoms, which seem to indicate recovery [10]. (3) Nauseas, copious vomition, diarrhoea, profuse and massive haematemesis, hepatomegalia, jaundice, pruritus, haemorrhages on skin and mucosa due to important coagulation alterations; renal damage with oliguria, haematurai, albuminuria and anuria. In the terminal phase there may be convulsions, delirium and coma. If the patient survives, the brain symptoms may persist for a long time. The WP by-products cause second and third degree burns when in contact with the skin, as well as respiratory symptomatology when inhaled. Death occurs 4–8 days later [11].

The effects of the associated potassium chlorate are the rapid oxidative destruction of the erythrocytes, possibly followed by increased methaemoglobinemia and possible peripheral cyanosis. Later it causes gastritis, toxic nephritis, depression of the central nervous system and transitory amblyopia [10,15,16]. The lethal dose is estimated at 5–30 g of potassium [15].

The care of the intoxicated person must be carried out in four phases: basic emergency support, elimination of the poison, control of the symptoms and administration of specific antidotes [9]. The basic vital support includes the permeabilisation of the airways, administration of liquids and shock control. The poison is eliminated with gastric aspiration, as the use of a wash is controversial. The third phase includes the control of the convulsions, cardiac arrhythmias and electrolytic misbalances [9]. The administration of substances such as activated carbon in large doses (30 g carbon with 240 ml water) [12–14], and copper sulphate 0.1 M [15] has been tested with some success. Other substances such as potassium permanganate, oxygenated water, antioxidants, *N*-acetylcystein, plasmapheresis or exchange transfusion have not proved to be very efficient.

2. Material and methods

In this article we make a retrospective-type descriptive study of all the cases registered in the country in a time interval of 19 years, between 1980 and 1999. The hospitals where the data have been collected from belong to the Public Health Ministry.

All the patients intoxicated with 'little devils', who were treated in the different health-care centres mentioned, were included in this study. An information search was carried out in Medline and in the Cochrane Library to compare the results with those described in similar studies.

3. Results and discussion

Information from a total of 590 cases was collected

Table 1
Distribution of the number of cases depending on genre, age and health-care centre [17–22]

Code	Location (town)	Health centre	Year of study	Authors (reference no.)	No. of cases	Age range	Average age in years	Women	Men
A	Quito	Eugenio Espejo Hospital	1980–1982	Bustamante, Higalgo, Arroba and Ayala [1]	40	2–59	15	30	10
B	Quito	Pablo Arturo Suarez Hospital	1990–1995	Mosquera et al. [17]	39	Not reported	20.3	20	19
C	Machala	Teófilo Dávila Hospital	1991	González, Bastidas and Jaramillo [18]	2	18–24	21	2	0
D	Quito	Enrique Garcés Hospital	1992–1996	Villacís, Tirado, Orbea, Serrano [19]	38	13–30	20	22	16
E	Quito	Eugenio Espejo Hospital	1992–1996	Brito, Paredes, Salgado, Rivadeneira [20]	273	3–70	21.55	184	89
F	Latacunga	Province Hospital	1993	Chingo et al. [21]	3	15–27	19.3	3	0
G	Quito	Vozandes Hospital	1996	Maldonado, Herrera, Aguinaga and Garcés [22]	1	20	20	1	0
H	Quito	Quito Red Cross	1996–1999	Archives	194	2–59	19	140	54
Total					590	–	–	402 (68%)	188 (32%)

Table 2
Distribution of the quantity of poison ingested and the cause

Code	Quantity of poison ingested (g)	Average quantity ingested (g)	Accidental cause (no. of cases)	Intentional or violent cause
A	Not mentioned	Not mentioned	5	35
B	0.6–10.2	3.3	0	39
C	Not mentioned	Not mentioned	0	2
D	0.3–3	1.5	1	37
E	0.3–21	3.21	5	272
F	Not mentioned	Not mentioned	0	3
G	Not mentioned	Not mentioned	0	1
H	Not mentioned	Not mentioned	20	174
Total	–	–	31 (4.4%)	559 (96.6%)

[17–22], in a variable period of 19 years. The ages registered vary from 2 to 70 years, with an average of 18.27 years; the highest percentage corresponding to women (68%) (Table 1). The most frequently observed cause of intoxication was related to the affective state of the victims and depressive states, which led to intentional or violent suicide attempts in 98.6% of the cases recorded (Table 2). The victims took between 1 and 70 tablets, that is, between 0.3 and 21 g of the poison. The mortality reported varies from 10.5 to 25.5% (Table 3). The most important system affected in this study was the hepatic system (Table 4). Table 5 summarizes the therapeutic measures tested and the results obtained.

In the Cochrane Library, there was no database on meta-analysis on WP intoxication. On the other hand, 657 entries on this topic were found in Medline from 1966 to the present, and these include articles on experimental studies, most of which are on animals.

In 1997, the National Institute of Statistics and Census of Ecuador presented a report which indicated the occurrence of 155 deaths (rate of 2.6/100,000 inhabitants) due to accidental poisoning or exposure to noxious substances [21–23]. This information does not include a specific detail of each one of the causes of the intoxications. As it can be seen, there is an important infraregister of cases, and no work has been published that documents this problem in more detail. A mean of 31 cases on a yearly basis is recorded, although an important increase has been observed over the past few years.

However, it must be underlined that it is a serious public health problem on a national level. In spite of the legal prohibition for the use and manufacture of explosives with WP, illegal use is very common. On the other hand, the by-products, such as the 'little devils' are easy to access and represent a low cost for the whole community. The social situation is conjoined to this, which forces many people in precarious employment conditions to market these products.

One relevant fact is that the composition of the little devils is very variable, which reduces the 'quality' of the poison. The seriousness of the clinical pattern in patients who ingested little devils manufactured in neighbouring countries, particularly Colombia, is greater. It is known in the medical world that this type of intoxication also occurs quite frequently in neighbouring countries such as Colombia and Peru,

Table 3
Relation between primary health-care time, mean hospitalisation days and mortality

Code	Primary health-care (in h/mean)	Mean hospitalisation days	Mortality (%)
A	Less than 24	Not mentioned	25.5
B	15.74	11.10	15.36 ($n = 6$)
C	Not mentioned	10	Not mentioned
D	4–8	Not mentioned	10.5 ($n = 4$)
E	4	8.6	19.5 ($n = 52$)
F	Not mentioned	22.6	Not mentioned

Table 4
Systems affected that are reported in each study

Code	Hepatic	Neurological	Cardiological	Haematological	Digestive	Renal
A	Yes	Yes	No	No	No	No
B	Yes	Yes	Yes	No	Yes	Yes
D	Yes	No	No	Yes	No	No
E	Yes	Yes	Yes	No	Yes	Yes
F	Yes	No	No	No	Yes	No

where it is also manufactured. However, the access to health data and medical information in this regard is limited.

One aspect, which stands out, is the common denominator of self-aggression, suicide attempts or causing harm to third parties by ingesting WP. The lethal effects of this substance and the almost unavoidable progression to death are well known. Unfortunately, the initial collection of information and the complete access to this information is not easy because the intoxication victims and their relations do not usually collaborate. On the other hand, in spite of the seriousness and high incidence of this intoxication there are no standardised protocols either, in this regard, which can facilitate the initial diagnosis, treatment and later follow-up.

Although the mortality is high, there is no detailed study on the variability in survival, and the multiple therapeutic alternatives used have not shown satisfactory results in the majority of the patients. Perhaps, the variability in survival is due basically to individual response factors, and to the basic health-care support. Only activated carbon has been shown to be an important factor in reducing the absorption of the poison in the initial stages. There is no study either that categorically demonstrates the relationship between toxic ingestion doses, mortality and prognosis. However, the fact that the quicker the attention received in a treatment centre, the fewer the hospitalisation days when the patient manages to survive, is relevant. Later studies will have to be made to confirm this statement. Another factor to be highlighted is that we are possibly facing a mixed intoxication by WP and potassium chlorate, as, although the effects of WP are predominant, the added action of the second poison is not known. The methaemoglobinemia caused by the potassium has not been assessed in any patient.

Histopathological observations were made only in one study. These observations basically describe alterations of the hepatocyte characterised by pericellular oedema, cytoplasmatic condensation, intracellular cholestasis, steatosis and finally hepatocellular necrosis. This is translated into blood chemistry by way of leucopoenia, total hyperbilirubinemia, an increase in the hepatic aminotransferase and an increase in the prothrombin time. All these findings agree with the information found in literature. Therefore, it would initially be a hepatic problem, which causes multisystemic failure. The patients die due to shock or cardiac arrhythmias.

Later studies must be aimed at searching for a specific antidote, which can be administered at an early stage, as well as at standardising health-care protocols in all the intoxication stages. It will also

Table 5
Distribution of the therapeutic measures tested and results obtained[a]

Therapeutics administered	A	B	D	E	F
Aspiration/gastric wash	4	4	4	3	4
Potassium permanganate	1	5	5	1	5
Hypertonic glucose solutions	3	6	6	3	5
Lactulose	3	6	6	3	5
Vitamin K (phytomenadion)	3	6	6	3	5
Blood plasma	2	6	6	0	5
L-Dopa	3	6	6	0	5
Antiacids and blockers H2	3	3	3	3	3
Antibiotics	3	1	3	3	3
Plasmapheresis/exchange transfusion	1	1	1	2	1
Antioxidants	1	1	1	2	1
N-acetylcysteine	1	1	1	5	1

[a] 1, Not administered; 2, administered but no therapeutic action; 3, symptomatic measure, patient's condition improves; 4, favourable measure; 5, recommended but not administered; 6, ignored if administered.

be important to establish social action lines that include a specific legislative development aimed at prohibiting the use, handling and marketing of these explosives. This is the first study published, which globally includes data about this important problem.

We obtained the following conclusions:

1. WP produces hepatic and renal failure, which leads to secondary cardiac failure, which is the cause of death. The mortality observed exceeds 10% of the intoxicated people.
2. The main reason for ingestion of WP is for autolitical purposes, due to a high component of depressive affectation in the majority of patients. Supplementary psychiatric studies will be required in the future to determine the main cases.
3. The posterior hepatic damage, which is considered as an after-effect could not be confirmed, as there are no follow-up protocols, although the hepatotoxity of this substance is obvious.
4. The utility of a specific antidote for this type of intoxication has not been demonstrated. However, the importance of an early gastric wash/aspiration and the administration of large doses of activated carbon prove to be very important. A determinant factor in the quality of survival is early and appropriate health-care of the intoxicated people.

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