PROGNOSTIC FACTORS IN ALUMINIUM PHOSPHIDE POISONING

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ABSTRACT

A prospective study was done on 12 consecutive cases of aluminium phosphide poisoning admitted to a zonal hospital in Punjab. Patients were in 18-36 years age group and mortality was more in females. The dose of aluminium phosphide was more in non-survivors and fresh tablets were more likely to cause fatality. Early vomiting and prompt initiation of treatment resulted in better outcome. Survivors responded to treatment early, but duration of shock had no predictive value. Shock and cardiac arrhythmias were present in all cases and extracardiac complications occurred more commonly in fatal group. With vigorous treatment of shock and other complications 33 per cent of the cases could be saved.

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KEYWORDS: Aluminium phosphide poisoning; Complications; Mortality; Prognostic factors.

Introduction

Aluminium phosphide (ALP) is a potent grain preservative. The brands available locally are Celphose and Quickphos tablets of strength of 0.5 g. In the last decade several reports have come from Central and North India establishing the potentially lethal outcome of ingestion (mortality varying from 20 to 100%) [1-5].

Phosphine, a toxic product of ALP, binds with cytochrome oxidase at cellular level which results in cellular hypoxia and widespread organ damage [6]. Of the various presentations, shock is universal with or without manifestations suggesting variable involvement of respiratory, hepatobiliary, renal and central nervous system [7].

The aim of the study was to analyze mortality and survival rates in relation to dose of ALP consumed and identify prognostic factors predictive of poor outcome.

Material and Methods

Twelve consecutive cases of ALP poisoning in the period 1992-95 admitted to a zonal hospital

formed the material for the present study. The diagnosis of ALP poisoning was based on a definite and reliable history, the clinical profile and confirmation by examination of left over tablets/containers.

All the patients were in a state of shock with systolic blood pressure less than 90 mmHg. History was taken with emphasis on number of tablets consumed, their relative freshness, whether vomiting was induced and time interval from consumption to definitive management.

All patients were attended promptly. Blood samples were taken for estimation of urea, sugar, serum bilirubin, SGPT, haemoglobin, total and differential counts, and serum electrolytes. An intravenous line was established through the same venous puncture. A 12-lead ECG was recorded and a chest radiogram was taken and repeated later in selected cases. Vital parameters, level of consciousness, cardiac rhythm and intake-output was closely monitored.

Treatment line followed was similar for each case. Stomach wash with 1:10,000 potassium permanganate solution, intravenous (IV) fluids,

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dopamine infusion, steroids, oral antacids, forced diuresis, antibiotics, H₂ receptor blockers and antiarrhythmic drugs were used in selected cases as indicated.

Effort was made to correlate mortality with number of ALP tablets consumed, their freshness and whether immediate emesis was induced. Tablets were considered fresh if they were ingested directly from a container and old if taken after being placed in the grain.

Results

The 12 cases studied, (5 males and 7 females) were in the age group of 18 to 36 years. Clinical profile of cases are shown in Table 1. Mean age of the patients who survived was 22.25 years and of the non-survivors it was 25.7 years. Mortality was higher in females. Six out of the 7 female cases died. The dose of ALP consumed ranged from 1 to 4 tablets of aluminium phosphide of brand Celphose or Quickphos. The mean dose among the survivors was 1.75 tablets and 2.87 in non-survivors. Eight out of the 12 patients had consumed tablets directly from the container and all except one died (87.5%) whereas among survivors only 1 out of 4 (25%) had taken fresh tablet.

TABLE 1
Clinical profile of cases

Characteristics	Survivors (n=4)	Non survivors (n=8)
Age (years)	22.25 ± 8.82	25.7 ± 7.12
Sex		
Male	3	2
Female	1	6
No of Tablets ingested	1.75 ± 0.8	2.87 ± 0.07
Time interval in reporting (hours)	1 ± 0	1.87 ± 0.78
Duration of shock (hours)	6.75 ± 6.53	7.12 ± 7.3
Status of ALP tablets		
Fresh	1 (25%)	7 (87.5%)
Old	3 (75%)	1 (12.5%)
No of patient with immediate emesis after taking ALP	4 (100%)	2 (25%)

Two out of 8 non-survivors had vomited immediately after consuming the tablets (25%) while among survivors all had immediate emesis. Time interval from intake of ALP to start of treatment varied from 1 to 3 hours. All survivors had reported to the hospital within one hour of consum-

ing ALP tablets while mean period of reporting for treatment among fatal cases was 1.87 hours. In fatal cases all patients remained in shock from admission to death. Duration of shock was found to be variable ranging from 1 to 24 hours. Survivors responded to treatment in 2 to 4 hours. However one patient was in shock for 18 hours and survived. Duration of shock was of no prognostic value.

All the 12 patients presented in shock and showed cardiac arrhythmias (Table 2). Survivors had relatively benign arrhythmias such as ventricular premature beats grade 1-3 and junctional ectopics.

TABLE 2
Cardiac arrhythmias*

ECG changes	Survivors (n=4)	Non survivors (n=8)
Sinus arrest	3 (75%)	4 (33%)
Heart block	3 (75%)	6 (75%)
Junctional ectopics	4 (100%)	6 (75%)
Atrial fibrillation	—	2 (25%)
Ventricular ectopics	2 (50%)	6 (75%)
Ventricular tachycardia	—	4 (50%)

*one patient showed more than one type of ECG abnormality

Other complications are shown in Table 3.

TABLE 3
Other complications

Complication	Survivors (n=4)	Non survivors (n=8)
Shock	4 (100%)	8 (100%)
ARDS*	1 (25%)	4 (50%)
Pneumonia	1 (25%)	3 (37.5%)
Confusion, disorientation	2 (50%)	3 (37.5%)
Coma	—	1 (12.5%)
Uraemia	—	2 (25%)
Vomiting, epigastric pain	4 (100%)	8 (100%)
Hepatitis	2 (50%)	2 (25%)

*Adult respiratory distress syndrome

Discussion

No specific antidote is available for treatment of ALP poisoning. Initial enthusiasm for magne-

sium sulphate died down because of subsequent reports questioning its usefulness in reducing mortality. Meta analysis of all published studies have shown that the dose-related mortality was not significantly different among those treated with and without magnesium sulphate [8]. Therefore in our study main stress was on combating shock and preventing complications.

Shock with clear mentation is the cardinal presentation of ALP poisoning. It is an indicator of systemic toxicity by a potentially lethal dose of the poison. The dose ingested varied from 1 to 4 tablets of ALP, but all cases on admission were in shock. The shock was managed with vigorous IV fluid therapy, dopamine infusion support and intermittent forced diuresis with IV Lasix to maintain urine output. The results were favourable in 4 cases (33%). In fact one patient, who was in shock for a long duration (18 hours) without recordable pulse and blood pressure, survived without any sequelae. The same outcome could not be achieved in all cases. This highlights other contributory factors influencing the outcome.

The dose of ALP tablets was more among the non survivors. The freshness of tablets and early emesis assessed by history did show important correlation. When the chemical had prior exposure to atmospheric moisture or was vomited out after ingestion, the chance of survival was better.

Early institution of therapy has two-fold benefit. It removes the residual poison by the gastric wash and permits correction of shock before it can affect the organs irreversibly. This established fact could be verified in our study where survivors received treatment earlier than those who succumbed.

Besides shock, the associated complications have also a bearing on mortality. In earlier series cardiac arrhythmias and adult respiratory distress syndrome have been projected to be the main culprits [7]. Acute cardiotoxicity is due to tissue hypoxia secondary to binding of cytochrome oxidase at the cellular level. It is also due to subcellular trans-membrane exchange of ions brought on by focal myocardial necrosis produced by phosphine

[1]. In our study we encountered cardiac arrhythmias ranging from junctional and ventricular premature beats to ventricular tachycardia and conduction defects. All our patients showed some form of ECG abnormality. However, those who survived had relatively benign arrhythmias like junctional and ventricular beats (grade 1-3) as compared to those who had fatal outcome.

Among other complications adult respiratory distress syndrome was seen more in non survivors. Other complications like uraemia and coma were seen during terminal stage in fatal group. Mild symptoms of altered sensorium and hepatitis had no bearing on the outcome. Complications are a result of inadequate oxygen perfusion due to shock as well as direct toxic effect of the poison. Little can be done to prevent binding of toxin to the tissues but vigorous treatment of shock and complications improves survival. Thirty three per cent of our cases survived after treatment based on this principle.

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