ORIGINAL RESEARCH PAPER

General Medicine

ECG CHANGES IN ALUMINIUM PHOSPHIDE POISONING

KEY WORDS: Aluminium phosphide, Cardiac toxicity, ECG changes .

Dr. Shailendra Kumar Bilonia Post Graduate student, Department of medicine, Jhalawar medical college, Jhalawar, Rajasthan, Pin Code- 326001

Dr. Nikhil Kumar Nehra* Post Graduate student, Department of medicine, Jhalawar medical college, Jhalawar, Rajasthan, Pin Code- 326001*Corresponding Author

Dr. Karan Singh Charan Post Graduate student, Department of medicine, Jhalawar medical college, Jhalawar, Rajasthan, Pin Code- 326001

Dr. Deepak Gupta senior professor, Department of medicine, Jhalawar medical college, Jhalawar, Rajasthan, Pin Code- 326001

ABSTRACT

Background: Aluminium phosphide is a solid fumigant pesticide. Aluminium phosphide (AlP) is a common suicidal poison with a high mortality rate due to its cardiovascular impact and lack of antidote. The clinical spectrum of poisoning varies depending upon the dosage and duration of consumption. The main effect of the poison is due to the release of phosphine which inhibits cytochrome oxidase and thereby hampers cellular oxygen utilization. Death is due to its direct toxic effect over the heart leading to peripheral circulatory failure. The aim of the study was to describe the electrocardiographic changes in Aluminium phosphide poisoning. **Methods:** 36 patients, who were admitted in MICU, following consumption of aluminium phosphide tablets were included in the study. Patient s clinical evaluation was done. ECG of the patients was done to study the cardiac abnormalities. **Results:** Out of 36 cases, ECG was normal in 16 cases and abnormal in 20 cases. Atrial fibrillation was most common finding-6 patients (30%), supraventricular tachycardia-2 (10%), ventricular tachycardia-2 (10%),ST-T changes-6 { ST elevation-4 (20%) and ST depression-2 (10%) },bundle branch block-1 (5%),prolonged QTc-3 (15%). **Conclusions:** ECG was done bedside, it is a cost effective tool and should be done in all cases. Whenever it is available, it must be done in cases of aluminium phosphide poisoning. Thus, proper clinical work out along with relevant investigations and management as per standard protocol may save many more lives.

INTRODUCTION

Agrochemical poisoning is a major public health problem in developing countries particularly in India in the setting of low education and poor regulatory frameworks. Aluminium phosphide has currently aroused interest with increasing number of cases in the past two to three decades due to increased use for agricultural and non-agricultural purpose; hence easy availability has increased its misuse to commit suicide. Unfortunately the absence of a specific antidote results in very high mortality.(1)

Aluminium phosphide is a solid fumigant pesticide and it currently aroused interest with an increasing number of poisoning cases in the past three decades. Due to its increased use for agricultural and non-agricultural purpose, easy availability has increased its misuse to commit suicide. Initially the poisoning was accidental but gradually it became an effective and widely used medium for suicide and homicide. A suicidal, accidental and homicidal poisoning may occur at all times. The choice depends upon its availability, cost and effectiveness. The green revolution in India on one hand opened the door for prosperity but had something hidden in it to ruin human life. Among the large number of poisoning related deaths in India most of them have been due to aluminium phosphide poisoning (ALP).

Aluminium phosphide is reported to be highly toxic when consumed from a freshly opened container and the fatal dose for an average sized individual is 150 to 500 mg. Death is reported to result from profound shock, myocarditis and multiorgan failure.(2)

The most important factor for cardiac involvement appears to be systemic toxic effect of phosphine gas as it is known that it causes non-competitive inhibition of cytochrome oxidase in the mitochondria in experimental animals. (3)

The mechanism of death from ALP appears to be myocarditis, as evidenced by cardiogenic shock and ECG abnormalities in all those who ultimately died. ECG abnormalities appeared before a substantial fall in blood pressure in a number of cases, even in those cases where conduction blocks or bradyarrhythmia appeared. G.S. Wander, Arora S et al found acute pericarditis in ALP poisoning.(4)

The various ECG changes observed were ST-T wave changes (elevation/depression), supraventricular tachycardia with conduction defects, atrial fibrillation, AV dissociation and LBBB.(5)

This study was conducted in MICU ward, department of medicine, JHALAWAR MEDICAL COLLEGE, Jhalawar, Rajasthan, India from July 2021 to July 2022. The aim of the study was to register 36 cases of aluminium phosphide poisoning admitted in MICU ward. Ecg was done and treatment started according to standard treatment guidelines.

METHODS

Observational study was conducted in the department of medicine, Jhalawar medical college, Jhalawar, Rajasthan, India from July 2021 to July 2022 on 36 patients who were admitted in MICU, following consumption of aluminium phosphide tablets. 36 patients, who fulfilled the inclusion criteria and having none of the exclusion criteria and who/their relatives gave consent to participate in this study were included in the study.

Inclusion criteria

Patients with a history of exposure to aluminium phosphide poisoning.

Age and gender of patients age 18 years to 80 years, both male and female patients .

Patients who were willing to undergo this study their written and informed consent was taken.

Exclusion criteria

Pre-existing cardiac, respiratory, hepatic, metabolic or renal disorder, multisystem disease, Concomitant exposure to another poison.

Non-willing/uncooperative patients were not included.

Mean, standard deviation, probability and coefficient of correlation was done by SPSS20.0 statistical software.

Methodology

All the patients and/or his/her legally acceptable representative were provided complete information regarding the aims and objectives, procedure of the study.

A written Informed consent was taken from each patient or the relative. Patient's clinical evaluation was done as per standard proforma wherein details like exposure to aluminium phosphide tablets, symptoms, physical examination and relevant investigation-ECG (12 lead standard ECG).

RESULTS

The study included 36 patients, out of which 22 were male and 14 were females. The mean age of the study population was 28.4 years with a range of 18 years to 80 years. Age wise distribution of patients as per table no. 1

Table no. 1

Age group	No of cases	Percentage of cases
18-30 years	19	52.77%
31-45 years	11	30.55%
46-60 years	5	13.88%
61-80 years	1	2.77%

ECG was abnormal in 20 cases out of 36; Atrial fibrillation was most common finding 6 patients (30%), supraventricular tachycardia 2 (10%), ventricular tachycardia 2 (10%),ST-T changes 6 { ST elevation-4 (20%) and STdepression -2 (10%) },bundle branch block 1 (5%),prolonged QTc 3 (15%). (as described in table no. 2)

Table no. 2

ECG changes	No of cases(out of 20)	Percentage of cases
Atrial fibrillation	6	30%
Supraventricular tachycardia	2	10%
Ventricular tachycardia	2	10%
St elevation	4	20%
St depression	2	10%
Bundle branch block	1	5%
Prolonged QTc	3	15%

DISCUSSION

Aluminium phosphide poisoning, although rare, is an important cause of morbidity and mortality in India. The reason for human poisoning is usually suicide, occasionally accidental and rarely homicidal. The clinical features depend upon the number and freshness of tablets ingested. In human beings, phosphine causes toxicity by inhalation and ingestion. The exact mechanism of action of phosphine is not clearly understood but appears to be hypoxic as supported by post-mortem findings. Like cyanide, phosphine produces hypoxia by non-competitive inhibition of cytochrome oxidase at mitochondrial level. Acute toxicity is due to direct effect of phosphine inhibiting cytochrome oxidase, potentiated by free radical stress due to inhibition of catalase and induction of superoxide dismutase (SOD) and release of hydrogen peroxide, the net result is interaction of free radicals with cell membrane leading to protein denaturation and cellular dysfunction.(6)

The ECG changes in Aluminium Phosphide poisoning are transient and reversible in most cases within a few days as the patient becomes asymptomatic. Controversy exists regarding the various ECG changes seen in association with aluminium phosphide poisoning. Some authors have indicated in their studies that ECG abnormalities were poor prognostic markers; others have observed that presence of ECG abnormalities did not predict mortality. (7)

According to Nagar KS et al, it would be logical to presume these changes were due to deleterious effects of poisoning on myocardium. ST-T changes do not fit into the distribution area of any specific coronary artery, hence appear to be due to focal myocardial damage as a result of this poison. The reversibility of ECG changes denote that although the effect is focal yet it is due to some reversible factors such a change in trans-membrane action potential as a result of ionic disturbances brought on by focal myocardial involvement. (8)

Our study shows ecg changes in 20 patients out of 36 patients of Aluminium Phosphide poisoning. In our study QTc prolongation in 3(15%).

Nithyapriya TK et al. study shows ECG changes were seen in 42% of the cases, the most common finding was prolonged QTc interval (26%).(9)

Sahoo D et al. study shows ECG changes related to celphos poisoning have been examined in various studies and include atrial fibrillation, supraventricular and ventricular tachycardia, ST-T changes, bundle branch blocks, and atrioventricular conduction disturbances.(10)

In our study, Atrial fibrillation was most common finding -6 patients (30%), supraventricular tachycardia -2 (10%), ventricular tachycardia -2 (10%),ST-T changes- 6 { ST elevation-4 (20%) and ST depression- 2 (10%) },bundle branch block -1 (5%), prolonged QTc- 3 (15%).

Kambiz soltaninejad et al. study shows Elevation of the ST segment was seen in nine cases (45%). Seven patients (35%) had prolonged QTc intervals. Bundle branch block (BBB) was observed in four (20%) patients.(11)

CONCLUSION

Though times have changed, presentation and mortality depends on the number of tablets consumed, whether the tablet is fresh or exposed to air, vomited or not and delay in getting hospitalized at some center with facilities for proper treatment may determine the outcome. Mode of presentation has not changed significantly with passage of time.

ECG should be done in all the cases to find out exact cardiac abnormality, so that proper treatment can be instituted in time. Whenever and wherever it is available, it must be done in cases of aluminium phosphide poisoning.

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