ALUMINIUM PHOSPHIDE POISONING CASES IN A TERTIARY CARE HOSPITAL

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Abstract

Background: *Aluminium phosphide is used to control rodents and pests in grain storage facilities. It produces phosphine gas, which is a mitochondrial poison. Unfortunately, there is no known antidote for aluminium phosphide intoxication and the mortality rate is very high. This type of poisoning (known as Kerry or Rice tablet poisoning) is common in Comilla region in Bangladesh.*

Methods: *The study was an observational study and was conducted in the Department of Medicine and in Emergency Room of Comilla Medical College Hospital, Comilla, between February and August, 2013. Twenty five cases of Kerry tab. Poisoning were studied about their causes, clinical features, treatment and outcome.*

Results: *Most of the patients (92%) were female and age range was 15-45 yrs. Familial disharmony was the main reason of their suicidal attempts. This poisoning was common in Chandina, Sadar, Debidar, Burichong, B.para, Muradnagar and Daudkandi upazillas of Comilla District. Vomiting and features of shock were the major clinical menifestations. Routine ECG was not done to these patients. Most of the patients (80%) were referred to higher centres in Dhaka after the primary management. Only 20% patients were survived due to early stomach wash and supporting medical management. One patient who survived also mentioned of taking date expired Kerry tablet.*

Conclusion: *Strict implementation of nationwide pesticide regulation, including restricting the availability of poison, being aware of its toxicity and providing improved medical management and improved intensive care facilities can further reduce the mortality due to AlP toxicity as there is no antidote available presently.*

Key words: *Kerry poisoning, Aluminium Phosphide, pesticides.*

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Introduction:

Acute aluminium phosphide poisoning is a large, though under-reported, problem in the Indian subcontinent. Aluminium phosphide, which is readily available as a fumigant for stored cereal grains, sold under various brand names such as *Quickphos* and *Celphos*, is highly toxic, especially when consumed from a freshly opened container.[1,2] Death results from profound shock, myocarditis and multi-organ failure.[3] Aluminium phosphide has a fatal dose of between 0.15 and 0.5 grams (0.0053 and 0.018 oz).[4] It has been reported to be the most common cause of suicidal death in North India.[5] This type of poisoning (known as Kerry or Rice tablet poisoning in our country) is also common in Comilla region in our country, Bangladesh. However, no such study was done before in our country about this poisoning. For this reason, this study was done to find out the present situation of this poisoning. To find out the poisoning prevalent areas, cause and nature of poisoning (suicidal, accidental or homicidal), clinical presentation, current treatment facilities and outcome of the poisoning were the main targets of the present

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study. The mortality rates from acute aluminium phosphide poisoning vary from 40–80%.[1] The actual numbers of cases may be much larger, as less than 5% of those with acute aluminium phosphide poisoning eventually reach a tertiary care center.[3] Since 1992, when aluminium phosphide became freely available in the market, it had, reportedly, overtaken all other forms of deliberate poisoning, such as organophosphorus and barbiturate poisoning in North India.[6] In a 25 year long study on 5,933 unnatural deaths in north-west India, aluminium phosphide poisoning was found to be the major cause of death among all cases of poisonings.[7] The toxicity of aluminium phosphide is attributed to the liberation of phosphine gas, a cytotoxic compound that causes free radical mediated injury, inhibits vital cellular enzymes and is directly corrosive to tissues. The following reaction releases phosphine when aluminium phosphide reacts in contact with moisture:

$AlP + 3\,H_2O$ '! $Al(OH)_3 + PH_3$ (in air), and $AlP + 3$ HCl '! $AlCl_3 + PH_3$ (in air and stomach)

After ingestion, toxic features usually develop within a few minutes. The major lethal consequence of aluminium phosphide ingestion is profound circulatory collapse, is reportedly secondary to these toxins generated, which lead due to direct effects on cardiomyocytes, fluid loss, and adrenal gland damage.[8] The signs and symptoms are non-specific, dose dependent and evolve with time passing. The dominant clinical feature is severe hypotension refractory to dopamine therapy.[7] Other features may include dizziness, fatigue, tightness in the chest, headache, nausea, vomiting, diarrhoea, ataxia, numbness, paraesthesia, tremor, muscle weakness, diplopia and jaundice.[9-11] If severe inhalation occurs, the patient may develop acute respiratory distress syndrome (ARDS), heart failure, arrhythmias, convulsion and coma. Late manifestation includes liver and kidney toxicities.[9-11] The diagnosis of aluminium phosphide poisoning usually depends on the clinical suspicion or history (self-report or by attendants). At some places, tablets of aluminium phosphide are also referred to as "Rice Tablets" and, if there is a history of rice tablet ingestion, then it should be treated differently than other types of rice tablets that are made up of herbal products.[14] For a Silver nitrate test on gastric aspirate, diluted gastric content can be positive. The management of acute aluminium phosphide poisoning remains purely supportive because no specific antidote is exists. Mortality rates approach 60%. The role of magnesium sulfate as a potential therapy in acute aluminium phosphide poisoning may decrease the likelihood of a fatal outcome, and has been described in many studies.[3,11] After ingestion, removal of unabsorbed poison from the gut ("gut decontamination"), especially if administered within 1–2 hours, can be effective. Potassium permanganate (1:10,000) gastric lavage can decompose the toxin. All patients of severe aluminium phosphide poisoning require continuous invasive hemodynamic monitoring and early resuscitation with fluid and vasoactive agents.

Methods:

This observational study was conducted in the Department of Medicine and in Emergency Room of Comilla Medical College Hospital, Comilla, between February and August, 2013. Twenty five cases of Kerry tab. Poisoning were studied about their causes, clinical features , treatment and outcome. Patients having the history of Kerry tablet ingestion were included in the study. History was taken from the patient or from the attendance or in some cases from the medical records.

Results:

Most of the patients (92%) were female and 8% were male. Age range was15-45 years. Familial disharmony was the main reason of their suicidal attempts. This poisoning was common in Chandina, Sadar, Debidwar, Burichong, Brahmonpara, Muradnagar and Daudkandi upazillas of Comilla District. Vomiting and features of shock were the major clinical menifestations. Routine ECG was not done to these patients. In our study, it was found that magnesium sulfate, oral coconut oil, charcoal or Potassium permanganate gastric lavage

were not used. Digoxin, Trimetazidine or intra-aortic balloon pump were also not practised. Sometimes, i.v. sodium bicarbonate was used along with fluid and vasopressor. Most of the patients (80%) were referred to higher centres in Dhaka after the primary management. Only 20% patients survived due to early stomach wash and supporting medical management and rest of them (80%) died. One patient who survived also mentioned of taking date expired Kerry tablet.

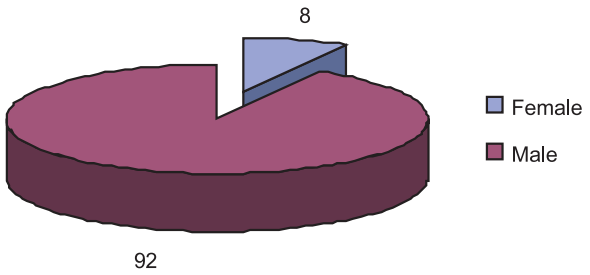

Fig 1: *Male-female ratio of poisoning (1=male 8%; 2=female 92%.)*

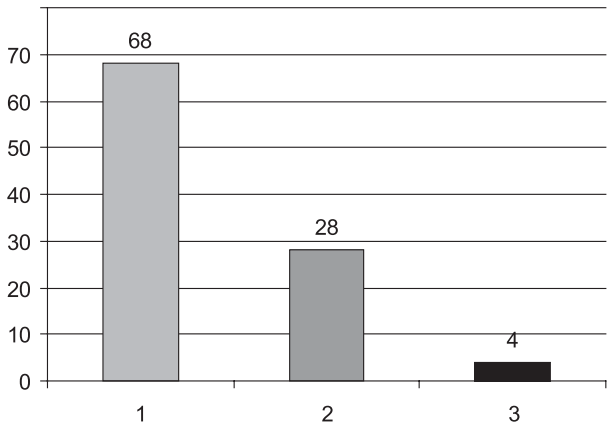

Fig 2: *Percentage of poisoning cases according to age group. Here, column 1 represents age group 15-25 years (68%); 2 represents 26-40 years (28%) and 3 represents >40 years (4%).*

Discussion:

It was found in the study that this poisoning was common in Chandina, Sadar, Debidwar, Burichong, Brahmonpara, Muradnagar and Daudkandi upazillas of Comilla District. These are the areas where agricultural activities are prominent and these areas are near Indian border. Moreover, this poisoning has been reported to be the most common cause of suicidal death in North India.[5] It is also noted that most of the patients were young, age group was 15-25 years (68%) and most of them were female (92%). In a 25 year long study on 5,933 unnatural deaths in north-west India, aluminium phosphide poisoning was found to be the major cause of death among all cases of poisonings.[7] In our study, only 20% patients survived and rest of them (80%) died due to aluminium phosphide poisoning. However, no such large long duration study was done in our country due to some limitations. Every year, about 300,000 people die because of pesticide poisoning worldwide. The most common pesticide agents are organophosphates and phosphides, aluminium phosphide in particular. Aluminium phosphide is known as a suicide poison that can easily be bought and has no effective antidote.[12] In our study, most of the patients (92%) were female and age range was15-45 years. Familial disharmony was the main reason of their suicidal attempts. Diagnosis of this poisoning, is based on clinical suspicion, positive silver nitrate paper test to phosphine, and gastric aspirate and viscera biochemistry. Treatment includes early gastric lavage with potassium permanganate or a combination with coconut oil and sodium bicarbonate, administration of charcoal, and palliative care.[13] In this study, diagnosis was done based on only the history and no other confirmatory test was done. Specific therapy includes intravenous magnesium sulphate and oral coconut oil. Moreover, acidosis can be treated with early intravenous administration of sodium bicarbonate, cardiogenic shock with fluid, vasopresor, and refractory cardiogenic shock with intra-aortic baloon pump or digoxin. Trimetazidine may also have a useful role in the treatment, because it can stop ventricular ectopic beats and bigeminy and preserve oxidative metabolism.[13] However, in our study, it was found that magnesium sulfate and oral coconut oil were not used. Digoxin, trimetazidine or intra-aortic balloon pump were also not practised. Sometimes, i.v. sodium bicarbonate was used along with fluid and vasopressor. Routine ECG was not done in every case. Gastric lavage was given to the

patients who presented early (within 2-3hrs) but no potassium permanganate or a combination with coconut oil and sodium bicarbonate was given. Supportive medical management was given. Critical patients were referred to the higher medical care centres in Dhaka, especially for intensive care facilities.

Conclusion

Kerry poisoning is a serious poisoning as the mortality rate is very high. Hence, prevention is better than cure. Strict implementation of nationwide pesticide regulation, including restricting the availability of poison, being aware of its toxicity and providing improved medical management and improved intensive care facilities can further reduce the mortality.

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