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Aluminium phosphide : A fatal poisoning

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Abstract

Aluminum phosphide (ALP) is a low-cost solid fumigant and a extremely toxic pesticide which is generally used for grain protection. Consumption is usually suicidal in Committed, unusually accidental and rarely homicidal. Death by Aluminium phosphide (ALP) poisoning are the commonest forms of poisoning in India. There is no effective antidote or treatment for its intoxication. The effects of ALP poisoning Vomiting, abdominal pain, loose motions, and restlessness are seen. Cardiovascular complications include thread pulse, tachycardia, tachypnea, acidosis, marked hypotension.

Key-words:- *ALP, Poisoning, Effect, etc.*

Introduction

Aluminium phosphide (ALP) is a solid fumigant which has been in extensive use since the 1940s. It has rapidly become one of the most commonly used grain fumigants because of its properties which are considered to be near ideal; it is toxic to all stages of insects, highly potent, does not affect seed viability, is free from toxic residues and leaves little residue on food grains [1]. Agrochemical poisoning is a major public health problem in developing countries particularly in setting of low education and poor regulatory frameworks. Aluminium phosphide is a solid fumigant pesticide. It is marketed in India as tablets of celphos and quickphos. Aluminium phosphide has currently aroused interest with increasing number of cases in the past three decades due to increased use in agricultural and non-agricultural purpose; hence easy availability has increased its misuse to commit suicide [2]. Unfortunately, its widespread use has been associated with a galloping rise in the incidence of alphos poisoning. Recent studies have indicated

that the number of deaths so far have exceeded the number of fatalities in the Bhopal gas tragedy [3]. In a study conducted by Siwach and Gupta, aluminium phosphide poisoning was found to be the most common cause of acute poisoning in India [4]. It was also found to be the most common cause of suicidal death in north India [5]. Poisoning shows a distinct male preponderance in the lower socio-economic strata and in rural areas, probably due to the heavy social stress burden in this group [6]. .If the former view is accepted the smell emanating from phosphide poisoned patients is probably due to contaminants in the pesticide formulations and not phosphine itself. It has been suggested that these volatile contaminants may be alkylphosphines [7].

Epidemiology

Annually about 300 000 deaths are reported by pesticides poisoning worldwide [8].The most reports of acute pesticide poisoning only based on hospital records admission and as a result absolutely reflect a small part of the real incidence. In Asian region about 25 million agricultural workers suffer from an episode of poisoning each year [9]. In "phosphine" poisonings reported from Germany, 28% were planned and mostly by eating, whereas the majority of the 65% accidental exposures were by inhalation [10]. A report has also been publishedfrom the United Kingdom where the majority of 93 aluminum phosphide exposures were accidental and concerned inhalation of phosphine in agricultural locations [11].

Physical and chemical properties of ALP

Aluminium phosphide is available in the form of 3 gm tablets or 0.6 gm pellets. Tablets are dark brown or grayish in colour and contain two compounds: aluminium phosphide and aluminium carbonate in a ratio of 56:44. Aluminium phosphide is the active component of the mixture whereas aluminium carbonate is added to prevent self-ignition of phosphi (PH3) which is liberated when aluminium phosphide comes in contact with moisture:

AlP + 3H20 □ Al (OH)3+ PH3 (in air)

AlP + 3HCl □ AlCl3 + PH3 (in air and stomach)

Each 3 gm tablet releases 1 gm and each 0.6 gm pellet 0.2 gm of phosphine gas on exposure to moisture and leaves behind a non-toxic grayish residue of alumunium hydroxide. Phosphine gas is colourless and odourless. However on exposure to air it gives a foul odour (garlicky or decaying

fish) due to the presence of substituted phosphines and diphosphines [12].

Mechanism of action

The exact mechanism of action of aluminum phosphide poisoning is still unknown, however an initial survey on different animals showed non-competitive cytochrome oxidase binding by phosphine, changes valences of haeme component of haemoglobin.Other than later articles, distinguished significant inhibition of catalase goes to hydrogen peroxide agglomeration [13], On the other hand, other studies declared there is no erythrocyte cholinesterase activity reduction in humans after accidental phosphine inhalation [15, 16]. After ingestion of aluminium phosphide, phosphine gas is released in the stomach which after absorption into the circulation results in early signs and symptoms. However, some of the aluminium phosphide is absorbed and metabolised in the liver with slow release of phosphine causing delayed onset of toxicity [17]. Phosphine is excreted through the breath and urine. AlP causes widespread organ damage due to cellular hypoxia as a consequence of non-competitive inhibition of the enzyme cytochrome oxidase of the mitochondria [18, 19]. Chugh et al. has shown that ingestion leads to high superoxide dismutase activity and low catalase levels that result in increased formation of free radicals and accelerated lipid peroxidation [20]. Lipid peroxidation in turn results in damage to cellular membrane, disruption of ionic barrier, nucleic acid damage and cell death. Focal myocardial necrosis and changes in membrane action potential as a result of altered permeability to sodium, magnesium and calcium manifest as various forms of ECG abnormalities and cardiac arrhythmias. The various exposure limits for phosphine gas have been defined as [12]:

• Permissible exposure limit (PET): 0.3 ppm over an 8 hr shift

• Short term exposure limit (STEL): 1 ppm

• Immediately dangerous to life and health: 200 ppm

• Lethal in 30 min: 400-600 ppm

Fatal dose

The specified fatal dose is 0.15-0.5 gm. However, most of the patients present with ingestion of three or more tablets which invariably results in death [12].

Toxicity

Permissible exposure limit (PEL) is 0.3 ppm over an 8 h shift (for factory stuffs). The

range of short term exposure limit (STEL) is 1 ppm a dimidiate danger to life and health would be 200 ppm. For lethal dose in 30 min, the range of 400 – 600 ppm (10 mg/Kg AlP) has been determined. It has been reported that its LD50 in mice (inhalation of fumes) is 0.68 g/m3 during 65 –75 min of exposure and for rats is 1.47 g/m3 during 35 –50 min of exposure. LD50 for cats is 25 pp m (2 –4 h daily during 3 days) [21].

Clinical features

Aluminium phosphate poisoning affects the most organs and a variety of signs and symptoms appear in patients. Early symptoms include nausea, vomiting, retrosternal and epigastric pain, dyspnea, anxious, agitation and smell of garlic [22, 23, 24]. On the breath. Moreover shock and peripheral circulatory failure are mainly imperative early signs of toxicity. Mortalities in past studies have ranged from 40–77% and in one survey 55% occurred within 12 h of ingestion and 91% within 24 h [25].

Cardiac toxicity

Cardiac toxicity comprises circulatory failure hypotension [26, 27, 28], congestion of the heart, separation of myocardial fibres by edema, fragmentation of fibres, non-specifc vacuolation of myocytes, focal necrosis, neutrophil and eosinophil infiltration were found in autopsy [29-32]. Also, significantly increasing left ventricular dimensions [33], hypokinesia of the left ventricle and septum, akinesia, ejection fractions reduction [34], severe hypotension, raised systemic venous pressure, normal pulmonary artery wedge pressure, inadequate systemic vasoconstriction and ECG abnormalities (ST and T-wave changes) are other signs and symptoms [35].

Respiratory toxicity

Tachypnea, dyspnea, crepitations, and rhonchi were present on examination in 192 out of 418 cases (46%) of phosphide poisoning [31], and have been found by others [36].Pulmonary edema is common but it is not always clear whether it is cardiogenic or non-cardiogenic in etiology. It tends to develop 4–48 h after ingestion and the finding of a reduced arterial pressure of O2 without an increase in pulmonary artery wedge pressure, suggested it was non-cardiogenic [35]. Others have confidently diagnosed adult respiratory distress syndrome [25, 33, 37, 38], and non-specifed pulmonary edema [39,40]. The edema fluid

may be protein-rich and hemorrhagic [40].

Gastrointestinal toxicity

Hematemesis [36], corrosive lesions of the esophagus and stomach [41, 42], vomiting, epigastric pain, severe gastric erosions, duodenal erosions, esophageal strictures tracheo-oesophageal fistulae,dysphagia. Dysphagia may be apparent as soon as 3 or 4 days after ingestion of aluminium phosphide but is more usual about 2 weeks later [41,43].

Hepatic toxicity

Transient elevations of alanine aminotransferase and aspartate aminotransferase activities are not infrequent after ingestion of metal phosphides [44-47], but jaundice secondary to liver damage is much less common [39]. It was present in 12 out of 92 cases (Singh, et al, 1991) and was said to be common in another series of 15 patients [48], but confirmatory laboratory data were not provided. Jaundice was alleged to be present in 16 (52%) members of the crew of a grain freighter who inhaled phosphine after an accidental release [49], but, in the six tested, serum bilirubin concentrations were normal and transaminase activities only minimally disturbed, casting doubt on the clinical observation. Acute hepatic failure and encephalopathy was considered to be the cause of death in one man [50], while a 12-yearold girl died from a combination of acute hepatic failure and encephalopathy with renal failure [27]. Portal edema, congestion of the portal tract and central veins, and vacuolization of hepatocytes are the most frequent findings at autopsy [51].

DISCUSSION

Aluminium Phosphide (ALP) tablets products a fatal Phosphine gas when it comes in contact with Hydrochloric Acid or liquid in the stomach. Phosphine is an extremely toxic colourless gas that discharges a fishy or garlic-like odour. Due to the toxic nature of the compound it is not generally available; though, it is frequently sold in the black markets. In the previous, ALP has been used by Indian villagers as a way of committing suicide. The ingestion or inhalation of ALP has multisystem exhibitions as it affects several systems, including gastrointestinal, respiratory, cardiovascular, musculoskeletal, central nervous system, and urinary system. Intentional or accidental contact to this compound can effect in the Human

nausea, vomiting, abdominal pain, diarrhoea, thirst, arrhythmia, Sino-atrial block, chest tightness, decreased EF on echocardiography, dyspnoea, pulmonary oedema, muscle pain, electrolyte imbalance, burning sternal pain, and renal and liver damage. Also, the example of unidentified poisons must be sent for analysis to initiate effective and appropriate managing of the cases. It is suggested that cases of poisoning must be taken seriously.

Conclusions

Metal phosphide are poisonous to rodents and used to protector the grain in stores and fields. Aluminium phosphide is a commonly used grain fumigant because of its highly powerful characteristic, cost efficiency and easy availability. Once administered in the body, a metal phosphide gets disintegrated by dilute hydrochloric acid in stomach, and liberates highly toxic phosphine gas; the final performances as respiratory and mitochondrial poison. All body vital organs are effected in the aluminum phosphide poisoning. Silver nitrate test & ammonium molybdate this test are used for the detection of ALP in biological & non-biological samples. Instrumentation analysis of samples are mostly used for Gas Chromatography (GC), (GCMS), nitrogen–phosphorous detector or mass selective detector are identified the presences of phosphine gas in biological & non-biological Samples.

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