

Cardiotoxicity of Aluminium Phosphide Poisoning Mimicking Acute Coronary Syndrome: A Rare Case Report and a Brief Review of Literature

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ABSTRACT :

Intense Aluminum Phosphide (ALP) or Celphos harming is very deadly and perpetually self-destructive in nature. Lamentably it's high poisonousness and nonattendance of a particular cure brings about high mortality. Aluminum phosphide harming is regular in India. It is one of the most deadly toxic substances. The clinical range of harming differs relying on the measurements and span of utilization. The fundamental impact of the toxic substance is because of the arrival of phosphine which hinders cytochrome oxidase and in this manner hampers cell oxygen usage. Practically any organ can be influenced by aluminum phosphide poisoning. We report a situation where the heart was the dominantly influenced organ. The result is poor, generally because of deferral in proper administration and incredulity among clinicians in regards to the result.

INTRODUCTION :

Aluminium phosphide (ALP) poisoning, has emerged as a common cause of accidental poisoning in children with a mortality ranging from 37-100%-[1] Since ALP is commonly used as a fungicide and rodenticide in India, many reports of accidental poisoning with severe consequences have been noted both in adults and children. However, only a few cases from outside India have been reported. The spectrum of symptoms and signs and their severity depends upon the time tag between ALP ingestion and hospitalisation. The most common presentation is shock with cold and clammy skin, a weak thready pulse and severe hypotension often refractory to vasopressors. Arrhythmias are common in ALP poisoning and are attributed to various causes including, neurological, gastrointestinal and renal involvement is also common and documented in many case reports.

Aluminium phosphide(ALP) poisoning is common occurrence in accidental and suicidal cases, predominantly in rural India, which is mainly attributable to poor regulation regarding the accessibility of this gravely toxic rodenticide[2]. Aluminium Phosphide is the most common farm pesticide in India, with humans commonly exposed to it accidentally or with suicidal tendencies. It causes cellular level damage and end organ damage; cardiac toxicity being the major trauma inflicted. There are only supportive therapies postulated but no specific antidote. We present a case Cardiotoxicity of Aluminium Phosphide Poisoning Mimicking Acute Coronary Syndrome.

CASE REPORT :

METHOD:

A 37-year-old female patient came to the hospital with a history of ingestion of ALP with a suicidal intent (two tablet of Celphos 6 gm). The time between consumption and hospital arrival was approximately 12 hours.

On arrival the patient complained of vomiting 8-10 episodes, nausea, epigastric pain, giddiness and dyspnoea on exertion since 6 hours. She had tried to vomit three to four times in an attempt to remove the tablets. She was conscious but irritable and confused. Her pulse rate was 120 beats/minute and her blood pressure was 60 mmHg systolic, feeble peripheral pulses, cold clammy skin, and respiratory rate was 26/min & Oxygen saturation was 90% with pulse oximetry at atmospheric temperature and air.

Examination of the respiratory system was unremarkable. Cardiovascularly, she was in shock. Patient was immediately shifted to intensive care unit where lavage was performed with normal saline, and intravenous fluids along with vasopressor were started. Vasopressor norepinephrine was administered as per the standard dosage and 1liter of normal saline was infused within one hour.

Laboratory investigations showed a HB of 11 gm%, total white cell count of 7500/Cumm and Arterial blood gas showed severe metabolic acidosis (pH-7.08, HCO₃-7.3), Serum total calcium was 9.2 mg/dl, serum magnesium was 2.33 mg/dl, serum sodium 143 meq/L, serum potassium 5.3 meq/L, Blood urea, serum creatinine and LFTs were normal. After two hours of gastric lavage we gave coconut oil through Ryle's tube. ECG recorded on arrival was normal. 2 D echocardiography was performed which revealed no regional wall motion abnormality.

Over the next two hours the patient's blood pressure continued to fall despite being on maximum doses of norepinephrine infusion, we therefore started the patient on a dopamine infusion as per standard recommended doses. Even after intensive use vasopressors (nor-adrenaline and dopamine) the shock did not respond. At this time colloid infusion at rate of 100ml per hour was started.

Over the next six hours of aggressive supportive measures, the patient continued to deteriorate. After six hours, She was drowsy, arousable, respond to painful stimuli, blood pressure was non recordable and could not maintain her saturation (80% saturation with oxygen) and therefore, the patient was intubated and ventilatory support was initiated. Blood pressure after six hours was non recordable and did not improve despite the maximum dose of dopamine and norepinephrine. Repeat ECG was suggestive of ST elevation in anterior leads which showed acute M.I. with severe myocardial injury. Inj. Heparin 5000 IU I.V. Was given and inotropic supports were continued.

After 2 hours, patient had VT (ventricular tachycardia), 200 Jules shock was given. Patient continued to have ST-T changes along with ectopics on monitor. During these events, patient had bradycardia followed by sudden cardiac arrest. Cardio-pulmonary resuscitation was done for 40 mins, but despite of all these cardio-pulmonary resuscitation, patient could not be reviewed and died. We conducted a post mortem examination.

On autopsy, the heart appeared to be normal on gross examination. The heart was removed for histopathological examination. Histopathological findings showed that both the left and right ventricles as well as the interventricular septum were involved. Sections

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from both the apex and base of the heart showed changes. The right ventricle showed minimal changes and the interventricular septum was the worst affected. The changes comprised of areas of mild to severe myocyte vacuolation and areas of myocytolysis and degeneration. There were areas of increased waviness of myocardial fibers.

DISCUSSION :

Pesticide harming is the absolute most significant methods for self destruction internationally. It is a significant general medical issue in rustic Asia, where it has prompted an exceptionally high-case casualty proportions than in the created world. Self destruction is a significant reason for untimely mortality representing an expected 849,000 passings consistently [WHO][3]. The first historically speaking instance of ALP harming was accounted for in 1981 in India, from that point forward, such frequencies have been persistently expanding, especially in country area of northwest and focal India to a great extent because of absence of mindfulness and poor guideline in regards to the openness of this gravely poisonous compound[4].

Ongoing investigations have demonstrated that the quantity of passings so far have surpassed the quantity of fatalities in the Bhopal gas tragedy.[5] In an examination directed by Siwach and Gupta, aluminum phosphide harming was seen as the most widely recognized reason for intense harming in India[6]. It was likewise seen as the most well-known reason for self-destructive passing in north India.[7] Poisoning shows an unmistakable male prevalence in the lower financial layers and in rustic zones, presumably because of the overwhelming social pressure trouble in this group.[8]

Chugh et al. has demonstrated that ingestion prompts high superoxide dismutase movement and low catalase levels that bring about expanded development of free radicals and quickened lipid peroxidation[9]. Lipid peroxidation thusly brings about harm to cell layer, disturbance of ionic obstruction, nucleic corrosive harm and cell demise. Central myocardial rot and changes in layer activity potential because of modified penetrability to sodium, magnesium and calcium show as different types of ECG anomalies and cardiovascular arrhythmias.

Phosphine causes broad organ harm. It ties with and squares Cytochrome oxidase, bringing about cell hypoxia. It likewise causes central myocardial putrefaction that presumably results in transmembranal trade of particles (Na⁺, K⁺, Mg⁺, Ca⁺) causing arrhythmias and fast passing. Introduction of ALP has multisystem indications as it influences numerous frameworks, including GI, respiratory, CNS, CVS, musculoskeletal, and urinary System[10],[11]. Aluminum phosphide on contact with dampness structures PHOSPHINE (PH₃) gas which Leads to harming on inward breath, ingestion and dermal contact[12].

The introducing indications rely upon the course of organization. Harming by inward breath produces disturbance of the mucous film, dazedness, simple fatigability, queasiness, regurgitating, cerebral pain and looseness of the bowels in mellow introduction. Ataxia, deadness, paraesthesia, muscle shortcoming, loss of motion, diplopia and jaundice result from moderate level of presentation. In extreme instances of inhalational harming, the patient presents with intense respiratory misery condition (ARDS), congestive heart disappointment, seizure and unconsciousness [13].

Sickness, retching and stomach torment are the most punctual indications that show up after ingestion. Gastrointestinal side effects which present in moderate to extreme harming are exorbitant thirst, stomach torment and epigastric delicacy while cardiovascular variations from the norm seen are significant hypotension, dry pericarditis, myocarditis, intense congestive cardiovascular breakdown and arrhythmias. Inclusion of the respiratory framework may prompt dyspnoea, which may advance to

Type I or II respiratory disappointment. Sensory system appearances incorporate migraine, tipsiness, changed mental status, seizure, intense hypoxic encephalopathy and trance state. Renal and hepatic disappointments are different appearances. Some uncommon finding in aluminum phosphide harming are solid shortcoming, squandering, delicacy in proximal lower appendage muscles, draining diathesis because of hairlike harm, intense adrenocortical deficiency and the pseudoshock condition because of impeded liquid dissemination which brings about small scale circulatory disappointment [14].

ECG changes found in ALP harming cases included range of atrial fibrillation, SVT (supra ventricular tachycardia) untimely ventricular withdrawals and ST-T changes. These progressions were credited to central myocardial rot and changes in real life layer potential because of the shift in the porousness of Na⁺ Mg⁺⁺&Ca⁺⁺ particles. Metabolic acidosis came about, most likely because of lactic acidosis which was brought about by the obstructing of oxidation phosphorylation [15]

One of the demonstrative signs of cardiovascular affront in this harming, is the worldwide LV function[16,17]. It has been accounted for before that heart work begins on improving, by the multi day with a range enduring up to 10-14 days[18]. Forceful cardiovascular help during this stage has been proposed to forestall end organ harm because of poor perfusion. It has been for the most part done as liquid revival and vasopressor support[19]. A positive history of ingestion is the premise of conclusion by and large. The nearness of run of the mill clinical highlights, garlicky smell from the mouth and profoundly factor arrhythmias in a youthful patient with stun and no past history of cardiovascular sickness focuses towards aluminum phosphide poisoning[20].

Research center assessment is principally done to survey the forecast. Leukopenia demonstrates extreme poisonousness. Expanded SGOT or SGPT and metabolic acidosis demonstrate moderate to extreme ingestional harming. Electrolyte examination shows diminished magnesium while potassium might be expanded or diminished. Estimation of plasma renin is critical as its level in blood conveys an immediate relationship with mortality and is raised direct extent to the portion of pesticide. The serum level of cortisol is generally seen as diminished in serious harming.

Chest X beam may uncover hilar or perihilar blockage if ARDS creates. ECG shows different signs of heart injury (ST despondency or group branch square, ventricular tachycardia, ventricular fibrillation [20]. Divider movement variations from the norm, summed up hypokinesia of left ventricle, diminished discharge division and pericardial radiation can be seen in echocardiography.[21]

The mortality (as much as 70% upto 100%. in certain investigations) identified with Aluminum phosphide ingestion is expected absence of explicit remedy and the system of activity of phosphine gas, that is acting at the mitochondrial level causing cell hypoxia heart being the ideal objective. The normal time span among ingestion and demise is accounted for to be 3 hours (extend 1-48hrs) with 95% mortalities happening inside 24 hours cause cardiovascular dysrhythmias [22]. Central myocardial rot and film activity potential changes happen because of modified penetrability to sodium, calcium and magnesium prompting different ECG changes and arrhythmias[23].

Aluminum phosphide has no particular counteractant thus ideal result corresponded best with the seriousness of retching and the immediacy of the commencement of treatment after poisonousness. Negative result was firmly corresponded to the level of hypotension and acidosis[24]. The most significant factor for progress is revival of stun and foundation of steady measures at the earliest opportunity.

Intravenous access ought to be built up and 2-3 liters of ordinary saline are directed inside the initial 8-12 hr guided by focal venous

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weight (CVP) and pneumonic slim wedge pressure (PCWP). The point is to keep the CVP at around 12-14 cm of water[6]. Low portion dopamine (4-6 ug/kg/min) is given to keep systolic circulatory strain >90 mm Hg. Hydrocortisone 200-400 mg each 4-6 hr is offered intravenously to battle stun, lessen the portion of dopamine, check slim spillage in lungs (ARDS) and to potentiate the responsiveness of the body to endogenous and exogenous catecholamines[13,14].

Oxygen is given for hypoxia. ARDS require serious consideration checking and mechanical ventilation. To lessen the assimilation of phosphine, gastric lavage with potassium permanganate (1:10,000) is finished. Permanganate is utilized as it oxidizes PH₃ to shape non-poisonous phosphate. This is trailed by a slurry of initiated charcoal (around 100 gm) given through a nasogastric tube. A purifying (fluid paraffin) is given to quicken the discharge of aluminum phosphide and phosphine.

Acid neutralizers and proton siphon blockers are included for indicative relief[25]. Phosphine discharge can be expanded by keeping up satisfactory hydration and renal perfusion with intravenous liquids and low portion (4-6 ug/kg/min) dopamine. Diuretics like frusemide can be given if systolic circulatory strain is >90 mm Hg to upgrade discharge as the primary course of end of phosphine is renal[26]. Bicarbonate level under 15 mEq/L requires sodabcarb) in a portion of 50-100 mEq intravenously every 8 hour till the bicarbonate level ascents to 18-20 mEq/L. Patients may require up to 300-500 ml of sodium bicarbonate[7].

Dialysis might be required for extreme acidosis and intense renal disappointment. The result relates best with the quantity of vomiting the patient gets after ingestion and the seriousness of hypotension the patient creates. It doesn't connect with the ingested dose[7]. The normal time stretch between admission of toxic substance and passing is three hours with a scope of 1-48 hours, 95% of the patients pass on inside 24 hours and the commonest reason for death in this gathering is arrhythmia.

Demise following 24 hours is because of stun, acidosis, ARDS and arrhythmia. The death rate is profoundly factor, extending from 37-100% and can arrive at over 60% even in experienced and well prepared focuses.

CONCLUSION:

Presentation to phosphine gas discharged from ALP fumigants builds danger of significant grimness and mortality. The most significant factor that may assist with improving endurance is giving primer clinical guide inside 0.5-1 h of ALP admission at grassroots levels. ALP is extremely deadly toxic substance with no particular antitoxin. Early identification, disposal of poison and concentrated steady consideration are the backbone of treatment. Coconut oil, magnesium sulfate and potassium permanganate are a portion of the other treatment modalities in ALP harming. The great result of Celphos harming connects best with the brief expulsion of toxic substance from the body and great steady treatment.

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