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A Case Report of Aluminium Phosphide Poisoning

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Abstract
Aluminium phosphide (ALP) is highly toxic pesticide that rapidly became one of the most commonly used grain fumigants and commonly known as 'rice tablet'. Aluminium phosphide (ALP) is a common cause of suicidal poisoning in India and poisoning can result in high mortality rates. A 23 year male patient who had alleged history of consumption of 1 tablet of Celphos presented with burning sensation of epigastrium. He developed hypotension and respiratory distress with right side pleural effusion which resolve spontaneously. He was discharged one week after his symptom resolved.

Introduction

Aluminium phosphide (ALP) is a widely available fumigant and pesticide. Over the last three decades, aluminum phosphide has emerged as common cause of suicidal poisoning in india When ingested, it is highly toxic and can result in refractory hypotension, Acute Respiratory Distress Syndrome (ARDS) and fatal arrhythmias. High mortality is due to severe mitochondrial dysfunction leading to disruption of cellular respiration leading to tissue hypoxia and organ dysfunction**.** There is no specific antidote for phosphide poisoning and treatment involves supportive care.

Case Report

A 23Yrs male from Bilaspur (H.P) presented with alleged history of consumption of 1 tablet of Celphos followed by burning sensation of epigastrium and not associated with nausea/vomiting/. His father presented a packet of tablet. His medical history was not significant. According to treatment history he was taken to RH Bilaspur within 45 minutes of Celphos consumption. Where gastric lavage was done and injectables were given and referred to IGMC. upon arrival at IGMC emergency department, patient was Conscious, Cooperative and well Oriented to time, place and person. His vital sign were pulse of 118 /min, blood pressure of 70/50 mmHg and saturation of 95 %. Initially his respiratory system, cardiovascular system and central nervous system was normal. Provisional Diagnosis of Aluminium Phosphide Poisoning with haemodynamically compromised was made. The patient was managed with gastric lavage with 3 litre of NS. Further managed included intravenous fluid, inj Mgso4 ,inj dopamine 2 amp

in 50 ml of NS at the rate 0.9 ml /hr . Patient remained hypotensive and finally inotropic support was withdrawn after 1 week. Patient developed respiratory distress on day 6th of admission. His chest X-ray was suggestive of right pleural effusion. Pleural aspirations done. Diagnostic thoracentesis revealed transudative picture with 160 cells 40 % lymphocytes 60 5 neutrophils. Gram stain and AFB stain were negative with no growth after 48 hours of culture for pyogenic organisms. No other cause of effusion which appeared and resolved with the recovery of the patient could be thought. Few case reports are available in the literature with patients of ALP poisoning developing pleural effusions and resolved spontaneuously. Similar to the findings by Kranti Garg et al at GMCH *our patient* also had spontaneous development and resolution of effusion. There was no history of fever during the stay in the hospital. His symptoms resolved spontaneously on day 10th and chest X Ray was normal. He was discharged after about a week of stay in the ward. During follow-up he was healthy with a normal chest X-ray and investigations were normal.

Discussion

Aluminium phosphides have been highly effective insecticides and rodenticides with the major merits of being inexpensive and not leaving toxic residues ALP is being marketed in India mainly as granulated powder in plastic pouches [10 gm] as Celphos [Excel industries]. ALP is a solid pesticide that rapidly became one of the most commonly used grain fumigants. This highly toxic chemical is cheap and usually formulated in tablets or pellets. Each 3 gm tablet releases 1 gm and each 0.6 gm pellet 0.2 gm of phosphine gas on exposure to moisture. *Toxicity is mediated by phosphine which inhibits cytochrome c oxidase.* ALP toxicity is due to the release of lethal phosphine gas following expose to atmospheric moisture and hydrochloric acid in the stomach. This gas causes cell hypoxia due to inhibition of mitochondrial oxidative phosphorylation. The clinical manifestations of ALP poisoning include cardiovascular system such as circulatory failure, congestive heart failure and fatal arrhythmias. Hypotension can develop rapidly and may persist due to continuing absorption of phosphine. ECG abnormalities most commonly comprised ST and T-wave changes, every type of arrhythmia, conduction abnormalities. It also effect gastrointenstinal system which include abdominal pain, loose stool and vomiting. Pulmonary symptom include pulmonary edema and ARDS. Electrolyte and metabolic abnormalities include hypokalemia, metabolic acidosis and hypoglycemia. The confirmation of ALP include by silver nitrate test on gastric lavage content . A positive history of ingestion is the basis of diagnosis in most cases. The presence of typical clinical features, garlicky odour from the mouth and highly variable arrhythmias in a young patient with shock and no previous history of cardiac disease points towards aluminium phosphide poisoning. Currently, there is no antidote for ALP poisoning, therefore supportive care remains the mainstay of treatment. Gastric lavage should be considered if the procedure can be performed by experienced staff within 1 h of ingestion. Phosphine gas can be absorbed cutaneuosly, thus the patient's skin and eyes should be decontaminated with plain water. The initial approach is to stabilise the patient by ensuring adequate oxygenation, ventilation and circulation. A high inspired oxygen concentration together with tracheal intubation may be needed. Intravenous access must be established, and crystalloids administered to restore circulatory volume. Vasopressors will be required. Noradrenaline or phenylephrine is preferable as dopamine and dobutamine have a higher propensity for developing arrhythmias. The role of magnesium sulfate as a potential therapy in AlP poisoning to decrease the likelihood of a fatal outcome has been described in many studies It corrects cardiac arrhythmias by modulating sympathetic, parasympathetic and slow channel kinetics and acts as a cell membrane stabilization

factor, but the exact mechanism is still unclear as this is a weak anti arrhythmic. 1 g intravenously then, 1 h later, 1 g by constant infusion hourly for 3 h followed by 1 g 6-hourly till death or recovery or a maximum of 5 days. Supportive care include correction of hypoglycaemia, hypokalemia /hyperkalemia and metabolic acidosis. There is experimental evidence to suggest that pretreatment with N-acetylcysteine (NAC) and melatonin helps in improving outcome in animal.

Conclusion

Following aluminum phosphide poisoning, apart from the usual complications such as ARDS, arrhythmia and aspiration syndrome, the possibility of polyserositis should be kept in mind especially if patients develop respiratory distress during the course of the illness**.**

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