

Upper Gastrointestinal Bleeding in Aluminium Phosphide Poisoning

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ABSTRACT

Aluminium phosphide is one of the most common poisons used for suicide in an agricultural country like India. Death is usually due to myocarditis which occurs within few hours of ingestion. There are many late complications reported in medical literature, however toxic effects on the gastrointestinal system, particularly corrosive action leading to massive haemorrhage is rarely reported. A 30 year old male developed upper gastrointestinal bleeding on the second day after consumption of aluminium phosphide. An exploratory laprotomy was done followed by adhesinolysis, gastrostomy closure with feeding jejunostomy and drainage. He died after eight days of ingestion. The autopsy findings of this rarely reported case along with review of literature on corrosive action of Aluminium Phosphide is discussed.

KEY WORDS

Aluminum phosphide, bleeding, corrosive, mucosal erosion, stomach mucosa

Citation

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INTRODUCTION

Aluminium phosphide (ALP) is a solid fumigant pesticide, insecticide and rodenticide, easily available in India in the form of grayish-white tablets with trade name of Celphos, Alphos, and QuickPhos etc.¹ On exposure to moisture, ALP liberates phosphine gas (PH₃), which is a systemic poison that commonly manifests with acute cardiovascular collapse followed by gastrointestinal, neurological, respiratory, and musculoskeletal manifestations.² Currently, there is no specific antidote for this poisoning. The fatality rate is as high as 90% and the fatal duration is as short as one to four hours. Most of the victims do not survive to manifest the delayed complications of ALP poisoning. However in patients who survive for some time, systemic complications like pleural effusion, cardiac arrhythmias, myocardial injury, subendocardial hemorrhage, features of hepatic and renal failure, pancreatitis, methemoglobinemia, intravascular

hemolysis and multisystemic organ failure have been documented.³⁻¹¹ Local effects of ALP poisoning like esophageal strictures, erosions, adhesions and necrosis of the area of contact with the poison poses a new challenge to the treating doctor and pose a threat to the survival of the victim.^{1,11-16} We hereby report a case of aluminium phosphide poisoning, where the victim manifested with massive gastrointestinal bleeding, due to corrosive effect on the stomach mucosa. The corrosive effects of ALP are discussed in this paper with reviews of literature.

CASE REPORTS

A 30 year old male was found by his wife in a state of altered sensorium on his bed, soiled with vomitus and stools. There was an open aluminium phosphide (ALP) container found beside him. As per his wife, the victim

had consumed one tablet of ALP. She took him to a local hospital, where he was intubated, underwent gastric lavage with potassium permanganate and managed with IV fluids, magnesium sulfate infusion. On the second day of admission, the patient vomited about 500 ml of dark coloured blood (hematemesis). In view of active upper gastrointestinal bleeding, exploratory laprotomy was done followed by adhesiolysis, gastrostomy closure with feeding jejunostomy and drainage. Intraoperative findings revealed diffuse gastric mucosal damage, erosive gastritis and necrosis with sparing of submucosa and serosa. Toxicological analysis of blood and stomach contents (silver nitrate test) established positive evidence for aluminium phosphide ingestion. The patient was a non alcoholic and there was no history of peptic ulcer disease or any coexisting morbidity.

After 2 days at the hospital, the patient left against medical advice, due to financial constraints and got admitted in a tertiary care hospital. At admission, the patient's vitals were stable but urine output was low. Although he was monitored in the Intensive Care Unit along with vasopressors and extensive antibiotic therapy, yet he developed coagulopathy, acute renal failure leading to multiple organ dysfunction syndromes and expired on the 8th day of ALP ingestion.

Autopsy Findings

The body was that of a male adult, medium built. Rigor mortis was all over the body. A midline laprotomy incision with surgical sutures in situ was present. A feeding Jejunostomy opening onto the skin surface was present left to the umbilicus. Multiple injection marks were present over the cubital region. On dissection, brain was congested and edematous. Peritoneal cavity and chest cavity contained about 1 liter and 500ml of straw coloured fluid respectively. Right lung was adherent to chest wall and both lungs were heavy, congested and a cut section showed froth exuding out of the parenchyma. Examination of the esophagus showed deep mucosal congestion. Stomach showed blackish coloured necrotic patches on its anterior surface at the fundus (fig. 1). A surgically stitched gastrostomy wound was present on the wall of the stomach. About 100 ml of blackish coloured blood was present in the stomach. Stomach mucosa was congested, soft, and spongy to touch, with flattening of mucosal ridges, especially in the pyloric region (fig. 2). Intestinal mucosa showed congestion. Feeding Jejunostomy was found in situ sutured on to the abdominal wall by surgical sutures. Liver was enlarged, heavy and a cut section revealed yellowish discolouration. Both adrenal and spleen were congested. Kidneys were congested and cut section revealed flakes of pus on the cortico-medullary junction. Tracheal mucosa was congested.

Histopathology sections revealed marked mucosal ulceration with sub mucosal edema, congestion and mixed (acute and chronic) inflammatory cell infiltrate in



Figure 1. Blackish necrotic patch seen on the posterior aspect of the stomach



Figure 2. Corrosive changes in the stomach mucosa

the upper gastrointestinal tract (fig. 3a). Lung parenchyma showed marked alveolar edema and congestion of the septal capillaries (fig. 3b and 3c). Myocardium and brain parenchyma showed areas of congestion. The liver showed centrilobular necrosis with sinusoidal dilatation, hemorrhage along with the presence of histiocytes and polymorphs. Areas of periportal mixed inflammatory infiltrate and focal macrovesicular steatosis was also noted (fig. 3d and 3e). Sections from both the kidney showed acute tubular necrosis (fig. 3f).

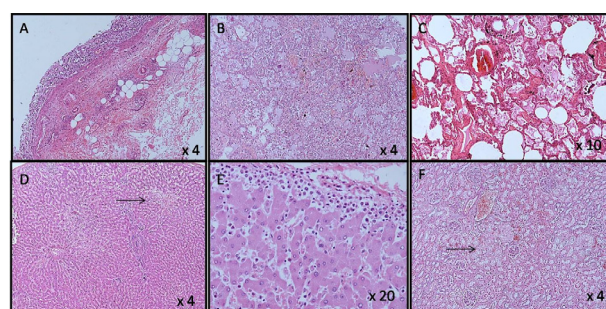


Figure 3. (A) Stomach: ulceration of the mucosa. (B and C) Lungs: Pulmonary edema. (D and E) Liver: Centrilobular necrosis (Arrow) with sinusoidal dilatation, presence of polymorphs and histiocytes (F) Kidney: Acute tubular necrosis (Arrow).

DISCUSSION

Toxic effects of the ALP and the survival of the victim depends on factors like the dose, exposure of the tablet to moisture before ingestion, presence of vomiting,

resuscitating efforts.² Absorption of phosphine gas from ingested tablet in gastrointestinal tract occurs by simple diffusion that causes damage to the internal organs. Apart from that, ALP does have local effects on the gastrointestinal system. Rastogi et al. reported a case of esophago-bronchial fistula in a survivor of ALP poisoning who suffered from dysphagia after five to six days of treatment. They postulated that the mechanism of such injury might be due to severe inflammation and corrosion of esophageal and tracheobronchial walls due to the local release of phosphine gas secondary to local trapping or impaction of the tablet in the esophageal mucosa.² Mishra et al. studied fifteen consecutive patients of ALP poisoning presenting with dysphagia to elucidate the natural history of ALP induced esophageal stricture.¹² They reported that all patients had a single stricture and the median time lag between consumption of ALP and occurrence of dysphagia was 3 weeks. Darbari et al. described 11 cases of esophageal injuries due to ALP ingestion, among which one patient had trachea-esophageal fistula with stricture and ten others had oesophageal stricture only.¹³ Madan et al. described corrosive-like strictures in three cases of ALP ingestion. Nijhawan et al. reported aluminium phosphide induced esophageal strictures.¹⁷ They explained that the large size of the tablet which was ingested might have got stuck in oesophageal lumen and caused local ulceration. They also raised the possibility that some preservative or adulterant in the tablet was responsible for ulcerations. They had observed that in both exposed and unexposed tablets produces strictures which might suggest that some ingredient in the tablet other than phosphine gas may

be responsible for the injury. They suggested that early vomiting and the sticking of the tablet to esophageal lumen might have protected the victims from systemic effects and the brunt of the injury was limited locally.¹⁶

In our case, the victim had developed upper gastrointestinal bleeding on the second day of ingestion of the poison. He was taken up for exploratory laprotomy and intraoperative findings suggested corrosive changes in the stomach mucosa as a source of bleeding. The feeding jejunostomy tube was placed and the patient was monitored closely in ICU set up. However, the patient succumbed to death on 8th day of ingestion. At autopsy, the findings were consistent for diffuse mucosal damage with corrosive changes in the stomach. The damage was more pronounced in the pyloric region which could be due to its reflex spasm, causing prolonged exposure to ALP. Histopathology findings were also conclusive for the corrosive effects of the poison on the stomach. In our case, the victim escaped the fulminant systemic effect of ALP, which could be due to its localization in the stomach mucosa. This observation is similar to that made in oesophageal mucosa by Madan et al.¹⁶ Although Aluminium phosphide related strictures in the esophagus are reported, yet corrosive effects on the stomach leading to fatal complication is not found in available literature. We thus report this case in the view of the rarity of findings that suggests the corrosive nature of Aluminium phosphide in the victim who survives the acute period of poisoning. We also recommend a larger study on corrosive aspects of aluminium phosphide, which might help in planning the management of such delayed complications in the victims.

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