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REVIEWARTICLE

GROSS AND ULTRASTRUCTURAL REVIEW OF AFFECT OF CELPHOS ON DIFFERENT HUMAN ORGANS

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ABSTRACT

Aluminium phosphide being economical and its ease for availability, extensively used in developing countries as an effective grain fumigant and rodenticide, poses significant toxicity risks to humans, particularly in cases of intentional ingestion for suicidal attempts.After reviewing the electronic and print media and various textbooks we got to know the effect of AlP poisoning on the vital orangs of human body. It affects the gastrointestinal, cardiovascular, respiratory, and nervous systems, with later manifestations of hepatic and renal failure and disseminated intravascular coagulation.This paper explores the ultrastructural and gross pathological effects of aluminum phosphide (AlP) poisoning on various human vital organs,offering valuable insights for medical professionals and researchers in improving treatment protocols and patient outcomes.The study compiles findings from multiple autopsies and histopathological analyses to detail the impact of AlP on the heart, liver, lungs, kidneys, brain, and spleen. The results reveal extensive cellular and tissue damage caused by AlP poisoning, such as myocardial necrosis and rhexis due to hypercontracted sarcomeres, centrizonal necrosis, alveolar thickening, acute tubular necrosis, and dilated and congested sinusoids., which leads to high mortality rates due tomulti-organ failure.It emphasizes the critical need for enhanced clinical management strategies and the development of specific antidotes for AlP poisoning. Thus,this review thoroughly investigates the toxicological effects of AlP poisoning on various human organs, detailing both microscopic and gross pathological alterations.

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INTRODUCTION

Aluminium phosphide (AlP) is widely utilized in developing countries as an economical and efficient grain fumigant and rodenticide. It's popularity stems from its potent efficacy against a wide range of insect species, non-interference with seed viability, cost-effectiveness, and minimal residue on food grains. However, despite these benefits, it poses severe toxicity risks to humans with no specific antidote available[1–4].Poisoning with AlP is associated with a high mortality rate, with a significant surge in poisoning cases and deaths through suicidal ingestion noted in India during the 1990s. Notably, it stands as one of the primarycause of poisoning incidents in suburban and rural areas of northern India[2].AlP is accessible in three-gram tablets that are either dark brown or gray in color, as well as 0.6-gram pellets. Figure. Represents the chemical structure of Aluminium Phosphide Molecular weight of AlP is 57.9552,*this information is gathered from the Computed by PubChem 2.1 (PubChem release 2021.05.07).*The size and dimensions of the AlP tablets are 20 mm in diameter and 5 mm thick[1].Usually, they are kept in airtight, sealed aluminium canisters.

Tablets composed of AlP are commonly referred to as "tobacco tablets," "rice tablets," or "wheat tablets" in general. This substance is sold under a number of brand names, including Phosphotex, Phosphume, Bidphos, Celphos, Quickphos, and Alphos. Up to one gram of Phosphine gas can be released from each three-gram tablet[3].Ammonium carbamate/carbonate and ureaare the inert constituents in the tablets, whereas pure aluminum phosphide is the active ingredient. The ratio of active to inert components is typically 56:44. The inert ingredient's primary purpose is to release gases to prevent PH_3 from ignitionAlP reacts with water, moisture and acids to form phosphine gas. The reaction as follows:

$$AlP + 3H_2O \rightarrow Al(OH)_3 + PH_3$$
$$AlP + 3H^+ \rightarrow Al^{3+} + PH_3$$

PH_3in its pure form is a colourless and odourless gas, but when released from tablets available in the market there are some impurities(in the form of phosphate moieties), owing to which this gas has a typical garlic odor[5].Phosphine is extremely toxic to organisms that respire aerobically but not to anaerobic or metabolically dormant organisms.

PH_3makes it effect for killing insect pests in grain without compromising the grain's viability. Its stable breakdown products are harmless phosphorus oxides that integrate into normal cellular metabolism as phosphate. Due to its ease of application, effectiveness, lack of residues, and low cost, AlP is widely used on nearly all internationally traded grain intended for human consumption[6].Prior to 1980, this poisoning was unknown in India; the first case was documented in 1981 from MGM Medical College in Indore. Phosphide and additional ingredients, such as ammonium carbonate, which releases phosphine gas even more forcefully when it comes into touch with stomach acid or water present in stomach, are found as tablets, pellets, and compacted discs, usually the fatal dose of AlP is 500mg, therefore according to toxicity rating it is (4 i.e, 51 to 500mg/kg), hence, it is a very toxic poison.[33]

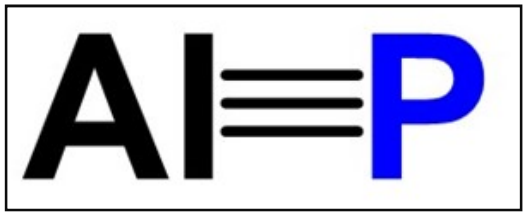

Figure 1. Chemical Structure of Aluminium Phosphide (AlP) [Molecular Weight: 57.955300 g/mol]

MATERIALS AND METHODS

For doing this research work in the form of review study we have reviewed thevast area of print and electronic media. This incorporates various research articles available in the online journals then offline journals. We have also gone through almost all the standard textbooks of forensic medicine and toxicology and toxicology book alone.

RESULTS AND DISCUSSION

Effect of Aluminium Phosphide poisoningon vital organs of human beings: After ingestion of AlP tablets, toxic symptoms typically manifest within a few minutes. In cases of mild poisoning, common clinical features include nausea, recurrent vomiting, diarrhea, headache, abdominal discomfort or pain, and tachycardia, with patients generally experiencing recovery. Conversely, in cases of moderate to severe ingestion poisoning, initial signs and symptoms affect the gastrointestinal, cardiovascular, respiratory, and nervous systems, with later manifestations of hepatic and renal failure and disseminated intravascular coagulation[15].Malik et al. studied a case of a married women 35-year-old, On the external examination cannula in-situ was discovered on the right forearm, close to the elbow. There were erythematous areas all over the body, ranging in size from 2 to 5 cm. Upon internal inspection, it was discovered that the pharynx, esophagus, and mouth mucosa were all clogged. There were erosions along the lesser curvature and congestion of the stomach mucosa. Histopathology showed that the liver had changed to a fatty state, and the heart had mild to moderate atherosclerosis. Congestion was seen in the kidneys, spleen, and lungs. Skin samples from erythematous patches had modest inflammatory infiltrates in the upper dermis and localized regions of dermo-epidermal separation. It was unable to determine the precise etiology of the erythematous spots. The cause of death was identified as poisoning with AlP found in the viscera that were sent for chemical investigation[4].

Effect of Aluminium Phosphide on Heart tissue: Kumar et al. examined a 30-year-old individual who died two hours prior to the autopsy, with no prior medical history. The heart sample was preserved in formaldehyde, refrigerated, and prepared for SEM analysis. During the autopsy, all organs were congested, and visual examination revealed petechial haemorrhages on the heart's atrial surface, a hematoma over the ascending aorta root, myocardial congestion with focal infarction patches, and epicardial haemorrhagic spots on the left atrial surface. The right atrium showed sub-endocardial hemorrhages and myocardial edema. SEM analysis at 6000X magnification showed ruptured and separated striated muscle fibers and intercalated discs, with focal myocardial necrosis and rhexis due to hypercontracted sarcomeres. At 3000X magnification, SEM images revealed swollen myocardial Fibers, separated myofibrils, and intercalated discs, with well-separated myofibrils and entangled striated muscle fibers. Additionally, there were areas of myocytolysis with vacuolated cytoplasm and polymorphonuclear cell infiltration[3,20].

Effect of Aluminium PhosphidePoisoning on Liver: Kundal et althe current study is based on a thorough examination of the microscopic and gross liver autopsies from 100 poisoning patients that displayed a variety of lesions. Out of 100 instances, the male to female ratio was 3.7:1, with 79 males and 21 females. A maximum of 34% of the liver specimens collected were from individuals between the ages of 21 and 30. The majority of cases (39%) included poisoning with Alp. which examined 50 medico-legal autopsies of AlP poisoning, liver congestion was observed in 44 cases, mild fatty changes in 19 cases, and haemorrhagic necrosis in only 10% of the cases.Congestion (66.6%), degeneration (46%), mononuclear cell infiltration (51%) fatty change (46%), necrosis (13%), sinusoidal dilatation (10%), and bleeding (20.5%) were the histopathological findings in the liver of AlP poisoning victims. This indicates that liver congestion is the most common findings, followed by fatty changes and then haemorrhagic necrosis, although other studies have reported varying results[9]. Another study conducted by Jain et al. the study examined 50 cases of AlP poisoning, with an equal number of males and females aged 13-60. The majority of the individuals were housewives (32%), followed by students (24%), skilled and semi-skilled workers (16%), laborers (6%), and a small percentage were unemployed (2%). The ingestion of the poison mostly occurred between 6 am-11 am (51%), 12 noon-6 pm (27%), and 8 pm-midnight (22%). The amount of AlP consumed ranged from half a tablet to six tablets. Hospitalized cases (28) had an average survival time of 12.8 hours, while non-hospitalized cases (22) had an average survival time of 2.6 hours. Overall, survival times varied from 1 to 47 hours, with post-mortem intervals ranging from 1 to 48 hours This study also shows the similar results of congestion seen in 88% cases, mild fatty infiltration was present in 38% cases and Centrizonal necrosiswas present in 20% cases[16].

According to Scanning Electron Microscopy of Human Liver Sinusoids, the non-fenestrated regions of endothelial cells extend outward from the nuclear swelling, dividing the fenestrated areas. Small fenestrations are usually evenly spaced, forming sieve plates. Adjacent endothelial cells partially overlap.

Table 1. Phosphorus and Aluminium Contents in examined biological vital organs [10]

S.N.	Biological samples	Phosphorusfound	Usual published value	Aluminium found	Usual published value
1.	Brain	4.3 mg/g	2.36 + 0.5mg/g	36 µg/g	2 ± 1 µg/g
2.	Heart	1.37 mg/g	1.27 + 0.30 mg/g	4.6 µg/g	< 1 µg/g
3.	Liver	8.22 mg/g	200 + 0.05 mg/g	75 µg/g	3 ± 1 µg/g
4.	Kidney	2.05 mg/g	1.75 _+ 0.05 mg/g	3 µg/g	3± 1 µg/g

Additionally, parts of the sinusoidal wall exhibit larger fenestrations, through which granular or fibrous materials can be seen in the space of Disse[17].Manoj Kumar et al. have observed fatty changes in the liver in cases of Celphos poisoning. According to the Atlas of Histopathology by Ivan Damjanov, steatosis, also known as fatty liver changes, is characterized by the replacement of hepatocyte cytoplasm with fat droplets. Both micro-steatosis and macro-steatosis can be present to varying degrees. This condition is accompanied by chronic inflammation and ballooning degeneration of hepatocytes. Additionally, "chicken wire" fibrosis is sometimes prominent, and there can be distinct pericellular and perisinusoidal collagen deposition[1,18].The sinusoidal wall shows visible Kupffer cells, characterized by numerous cytoplasmic processes. According to the study "Survey of Histopathological Findings in Autopsy of Poisoned Patients with AlP, clusters of polymorphonuclear leukocytes have been observed in cases of AlP poisoning. Additionally, fine cytoplasmic vacuoles are also visible[1,19].

Effect of Aluminium Phosphideon Lungs: Pathak et al. investigated the histopathological effects of AlP poisoning on lung tissue. A lung sample was taken from a person who had committed suicide by ingesting Celphos. The sample was preserved in formaldehyde, refrigerated, and prepared for Scanning Electron Microscope (SEM) analysis. During autopsy, all organs appeared congested. Lung micrographs revealed general edema, resembling a partially collapsed lung where air fills the space between the chest cavity wall and the lung. Micrographs showed alveolar thickening, type I Pneumocytes cells,dilated capillaries, and increased alveolar macrophages, indicating chronic irritation. The epithelial lining of the alveoli displayed necrosis, cell death, and edema. The study noted that macrophages released toxic factors that led to fibroblast recruitment, collagen deposition, and fibrosis, thus thickening the alveolar walls and reducing gas exchange efficiency, Pneumocyte type II cells are also evident[8].

Effect of Aluminium Phosphide on Kidney: Gross examination revealed generalized congestion of the vital organs, including the kidneys. The kidneys exhibited degenerative and necrotic changes, along with infiltration by inflammatory cells. Histopathological analysis of the kidneys showed a spectrum of changes:

(1) Early Degenerative Changes: Notably, cloudy swelling was observed. (2) Medullary Congestion: There was significant congestion within the renal medulla. (3) Acute Tubular Necrosis (ATN): The tubular epithelial cells displayed acute necrosis. (4) Eosinophilic Hyaline Casts:These casts were present within the renal tubules. (5) Tubular Lumen Findings: Congestion and the presence of hemolyzed red blood cells were noted within the tubular lumens. It is important to highlight that the glomeruli remained unaffected throughout these pathological processes. This indicates that while the tubular and interstitial compartments of the kidney were severely compromised, the glomerular structures were preserved[23].

Effect of Aluminium Phosphide on Brain (cerebellum): Macroscopic examination of the meninges and brain in cases of Celphos poisoning revealed congestion, edema, and petechial haemorrhagic spots on the surface. Histopathological analysis of the cerebral cortex showed a scarcity of neurons, disorganization, and the presence of necrotic patches[24].Histopathological examination revealed several significant abnormalities in the cerebral cortex. There was notable disorganization across its various layers, and neurons appeared round with convex borders. Additionally, there was degeneration of Nissl granules within the cytoplasm and an eccentric, degenerated nucleus.In the cerebellar cortex, the findings included degenerative changes in the neurons. There was also infiltration of round cells into the molecular layer, and a marked loss of the processes of Purkinje cells was observed, indicating severe disruption of cerebellar architecture and function[21].

Effect of Aluminium Phosphide poisoning on Spleen: Study performed by Kumar et al. a man in his 40s after being discovered unconscious, he was taken to the nearby hospital and revealed that he had taken three Celphos pills. There were no visible injuries found during the autopsy. The whole body had rigor mortis, and the back had formed and fixed post-mortem staining, with the exception of pressure points. Both nostrils gushed blood-coloured froth, and the lips, nose tip, and nails on the fingers and toes were all cyanosed. The stomach's mucosa was swollen with haemorrhagic patches and contained 10 milliliters of pasty, greyish-brownish material with an overpowering smell similar to decomposing fish or garlic. In 56% of instances, the stomach cavity will have a pasty substance or grey to greyish-brown fluid from phosphine poisoning.This happens as a result of hemorrhage that causes the stomach's contents to be exposed to gastric mucosa. The 140 g, edematous spleen was severely congested and had minor haemorrhages and necrosis along with localized regions of exudation on histology. The splenic mucosa had haemorrhagic infarction followed by necrosis, and electron microscopy of a portion of the spleen sliced revealed dilated and congested sinusoids. Gross examination of the spleen revealed haemorrhagic infarction, a condition often associated with Celphos poisoning due to its potential to cause extensive organ damage, including congestion, petechial haemorrhages, and organ edema. Consequently, significant congestion of the spleen, along with focal areas of exudation and small haemorrhages, were observed during the post-mortem examination [7,21].The spleen was found to be congested and edematous, with a weight of 140 grams. Histopathological examination revealed significant congestion within the spleen, along with focal areas of exudation, small haemorrhages, and necrosis.

Electron microscopy of a spleen section showed dilated and congested sinusoids, with evidence of haemorrhagic infarction and subsequent necrosis in the splenic mucosa[22].

Medicolegal and histopathological findings: This inexpensive, extremely dangerous chemical was easily accessible, which contributed to the unexpected spike in AlP poisoning cases. As per the microscopic study of Sinha et al. findings show there was bleeding and congestion in the stomach and coagulative necrosis and congestion in the brain. Alveolar thickness, oedema, dilated capillaries, collapsing alveoli, and hemorrhage were all seen under microscopy of pulmonary tissues. Degeneration, infiltration, tubular dilatation, and obscured edema were the alterations seen in the kidney. The liver had fatty alteration, sinusoidal dilatation, bile stasis, centrilobular necrosis, Kupffer cell hyperplasia, central venous congestion, degeneration, and bleeding[25].Study performed by the Kapoor et al. with 1752 medicolegal autopsies were performed in the mortuary throughout the research period, 205 were suspected of being poisoned, and 83 of these cases had circumstantial evidence of AlP poisoning.The most susceptible age group, which accounted for 40.5% of poisoning deaths, was 21 to 30 years old. The gender ratio was 2:1. The rainy season had the highest rate of poisoning-related mortality (41.95%). The majority of victims—61.5%—came from rural areas. It has been noted that the poisoning with AlP was mostly utilized for suicide, a failed suicidal act with a non-fatal result is called an attempted suicide. It is possible for some individuals who attempt suicide to pass away unintentionally while doing so[26].

Deaths of the victim after consumption of AlP tablets which happens due to refractory cardiogenic shock as a result of haemorrhagic myocardial lesions mostly sub endocardial haemorrhages. Refractory Cardiogenic shock is the most common cause of death and occurs within 24 hours and causes fatality in 30 to 40% of the victims dying due to AlP poisoning. In small number of cases brain damage may contribute in cause of death as there are a number of neurogenic manifestations, which can lead to severe blood loss, shock and hypotension. These findings are in favour of death due to consumption of celphos tablets AlP unexposed and further release of phosphine gas which is the active principle and responsible for widespread hypoxic organ damage[3].Histopathological findings can demonstrate the impact of poison consumption at the tissue and cellular levels. Such studies can be instrumental in discovering an antidote, which is currently unavailable. This research can offer guidelines for clinical procedures, aiding in treatment decisions based on the prognosis in these cases[9].

Possible Antidotes and future perspectives in dealing with Aluminium Phosphide poisoning cases.

Possible Antidote: There is currently no known treatment for AlP poisoning; supportive treatment is the sole available strategy for action[27].The current treatment for phosphine exposure focuses on supportive care until the substance is excreted. For accidental inhalation, patients should be moved to fresh air, while ingestion requires gut decontamination. Gastric lavage with magnesium sulphate or potassium permanganate is common, and sodium bicarbonate and coconut oil are also used. These substances oxidize and neutralize gastric acid, preventing phosphine release and absorption, with coconut oil forming a protective layer over the stomach lining[28–30].Activated charcoal and antacids adsorb and reduce the absorption of phosphine from the gastrointestinal tract[31]. Melatonin has been suggested to manage cardiotoxicity by scavenging reactive oxygen species (ROS), accelerating ATP production, and preventing apoptosis by inhibiting cytochrome c release[32]. Researchers emphasize the following steps to follow to combat the situation with AlP poisoning, first is to inhibit phosphine absorption through the GIT and enhance its excretion through the lungs and kidneys. Secondly Prevent the release of phosphine from AlP.Third step is to reduce organ toxicity and implement supportive measures as soon as possible[29].

Future perspective: The comprehensive review of aluminum phosphide (AlP) poisoning provides crucial insights into its devastating effects on vital organs and highlights gaps in current treatment strategies. Moving forward, several areas warrant attention to improve outcomes and mitigate the impact of AlP poisoning:

- Antidote Development,one of the most pressing needs is the development of a specific antidote for AlP poisoning. Current management focuses on supportive care and symptomatic treatment, which are often inadequate. Research efforts should prioritize identifying compounds that can neutralize phosphine gas or mitigate its toxic effects.
- Toxicological Studies,further toxicological studies are essential to elucidate the exact mechanisms through which phosphine gas disrupts cellular function and leads to multi-organ failure. Understanding these mechanisms at a molecular level could inform targeted therapies and interventions.
- Clinical Protocols,standardized clinical protocols andguidelines should be developed based on the latest research findings to optimize patient care and improve survival rates.
- Public Health Initiatives: Public health campaigns are needed to raise awareness about the dangers and toxicity of AlP and promote safe handling practices among agricultural workers and consumers. Education on proper storage, handling, and disposal of AlP-containing products could reduce accidental exposures.

This study identifies key areas that warrant further investigation and presents opportunities for future research to develop innovative solutions to tackle the pressing issue of AlP poisoning, with the ultimate goal of preventing its harmful effects on a global scale.

CONCLUSION

This review comprehensively examines the toxicological impact of AlP poisoning on various human organs, highlighting bothmicroscopic and gross pathological changes. AlP, though beneficial as an effective grain fumigant and rodenticide, poses severe toxicity risks to humans, particularly in cases of intentional ingestion. The liver, lungs, heart, spleen, kidneys, and brain are significantly affected vital organs, leading to high mortality rates reason behind is the multi-organ failure and cardiogenic shock. The liver commonly shows congestion and necrosis, the lungs present with edema and alveolar thickening, the heart exhibits myocardial necrosis, the spleen shows haemorrhagic infarction, the kidneys reveal acute tubular necrosis, and the brain demonstrates cortical disorganization and neuronal degeneration.

These findings underline the critical need for enhanced clinical management strategies and the development of specific antidotes for AlP poisoning. The ultrastructural and gross pathological data provide valuable insights for medical professionals, helping to improve treatment protocols and patient outcomes. This review emphasizes the importance of further research to understand the mechanisms of AlP toxicity better and to develop effective therapeutic interventions. In conclusion, addressing the complex challenges associated with AlP poisoning requires a concerted effort from the scientific community, healthcare providers, policymakers, and public health advocates. By advancing research, improving clinical management, and enhancing preventive measures, we can strive towards reducing the significant burden of AlP-related morbidity and mortality worldwide.

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