**AUTOPSY FINDINGS OF ALUMINUM PHOSPHIDE INTOXICATED CORPSES in QALYUBIYAH GOVERNORATE, Egypt**

**Yasmina B. Abd El-magid\*, Nermeen Adly Hassan, Ibrahim Zamzam and Sahar M. Abo El-Wafa**

Forensic Medicine & Clinical Toxicology Department, Faculty of Medicine, Benha University, Egypt

**\*Corresponding author:** Yasmina B. Abd El-magid, Forensic Medicine & Clinical Toxicology Department, Faculty of Medicine, Benha University, Egypt,

[**yasmeena2014@yahoo.com**](mailto:yasmeena2014@yahoo.com)**,** +2 01006301728

**ABSTRACT**

**Objectives:** As a cost-efficient and powerful pesticide, rodenticide, and fumigant, aluminium phosphide (ALP) has been widely utilized. It can result in widespread suicide poisoning and high mortality due to its ease of availability. Phosphine is the primary component of ALP (PH3). The human body will suffer total damage as a result of exposure to PH3. **Aim of work:** The goal of the current study was to examine the autopsy results from various human organs that had been poisoned with ALP at the Forensic Medicine Authority in Benha, Qalyubiyah, Egypt. **Subjects & Methods:** In this work, 10 control cases and 10 chemically confirmed autopsy cases were included and thoroughly analyzed on a gross level. Additionally, tissue samples from various organs were stained with hematoxylin and eosin and examined under a microscope. **Results:** This study includes a number of gross autopsy observations that have been made. Upon concentrating on the pathological changes in the major organs, we discovered substantial PH3-induced damage in many systems, particularly the stomach, duodenum, lung, and kidney. We also noticed extensive myocardial, splenic, and hepatocellular deterioration in the heart, spleen, and liver tissues. **Conclusion:** We believed that these characteristic abnormalities were a possible indicator of PH3 poisoning and partially explained the substance's deadly nature (inhibition of mitochondrial oxidative phosphorylation). We expect that this study will contribute to a better understanding of PH3 toxicity in both forensic and clinical conditions.

**Keywords:** Aluminum phosphide (ALP), Autopsy findings, Pathology, Poisoning.

**INTRODUCTION**

Aluminum phosphide (ALP), known as grain tablets in Egypt, is a solid fumigant pesticide & easily available under brand name "celphos " tablet. It is widely used as a grain preservative around the world and is regarded as an ideal pesticide due to its affordability, effectiveness, and ease of market availability ***(Saleh and Makhlof, 2018).*** Patients who had taken more than three tablets each dose all died. 95% of patients die within 24 hours, with the average time from intake of AlP to death being 3 hours (1-48 hours) (***Kalawat et al., 2016)***.

If airborne moisture comes into contact with aluminum phosphide, it has the ability to release phosphine gas (PH3). Phosphine gas is odorless, very combustible, explosive, and highly toxic to insects. It can be used as a fumigant to keep pests away from stored goods, and it has a distinct garlic- or decaying-fish-like odor (***Yan et al., 2018).*** Also, phosphine toxicity through inhalation has been recorded ***(Gregorakos et al., 2002).***

All tissues can quickly absorb phosphine, which is then eliminated in the urine and exhaled through the lungs (***Hashemi-Domeneh, et al., 2016).*** Human toxicity to phosphonates is caused by a common mechanism known as respiratory inhibition. Phosphine is a noncompetitive inhibitor of mitochondrial cytochrome oxidase that blocks electron transfer, inhibiting oxidative phosphorylation, which in turn prevents cellular respiration and the generation of peroxide radicals. This significantly lowers the potential of the mitochondrial membrane and limits oxidative respiration by 70% (***Abdollahi and Mehrpour, 2014; Singh et al., 2014).*** Phosphine can inhibit catalase and deplete glutathione, which may also lead to malfunction of the cellular wall and canals. Multisystem toxicity is probably caused by interfering with cellular respiration (***Hsu, et al., 2002; Hashemi-Domeneh et al., 2016).***

Even in skilled and well-equipped facilities, the fatality rate in cases of aluminum phosphide poisoning ranges from 60% to 90% ***(Yan et al., 2017).*** The reported cases are based on hospital admission data; therefore, they surely only represent a small portion of the true occurrence. A cross sectional research by***Mwaheb &Hassan (2020)*** assessed 96 deaths from ALP poisoning in Fayoum Governorate between June 2012 and June 2019 were evaluated and collected from reports of the Forensic Medicine Authority reflectively. Additionally, ***Sheta et al. (2019)*** conducted a prospective study on all patients admitted to Alexandria Main University Hospital with acute AlP poisoning for 6 months, from 1 November 2017 to the end of April 2018, and found that 13 patients (43%) of the 30 patients admitted during the study period died. In the prospective research of AlP poisoning by ***Abdel-Hady et al. (2019),*** 44 patients with a mortality rate of 45.5% who were admitted to the emergency room of the Assiut university hospital were included.

So, the magnitude of AlP poisoning and its numerous related deaths in man prompted us to undertake this study. The purpose of the current study was to examine autopsy results in various human organs following ALP overdose that resulted in death. The study's conclusions might help to clarify how PH3 generally harms the body systems.

**SUBJECTS AND METHODS**

* **Number of cases in the present study,**
* Ten (10) medical-legal autopsy cases involving aluminium phosphide were investigated. On the basis of a chemical examination of the viscera at the analytical toxicology lab, all of the study cases were determined to be the result of aluminium phosphide poisoning.
* Ten (10) control cases were chosen.
* All the cases were collected from Forensic Medicine Authority, Benha, Qalyubiyah, Egypt after Egyptian Ministry of Justice approval.
* **Required data:**

- A complete and exact history of the time and amount of aluminum phosphide consumption as well as the death time was attempted to be obtained.

- The viscera were chemically analyzed, and the cases where the viscera tested positive for alcohol or any other poison were excluded from the current study.

- The signs and symptoms seen in patients who were hospitalized and those who were not, as well as the care offered to those who were, have been studied.

- Detailed external and internal examination of all the poisoned and control cases were undertaken.

* **Histopathological examination:**

The Pathology Department of the Faculty of Medicine at Benha University conducted the analysis on each group's total number of cases, and the findings were reviewed there. After the autopsy, the lungs, heart, small intestine, liver, kidneys, spleen, and other organs that require accurate morphologic study were fixed in bouin's solution, which is made of picric acid, acetic acid, and formaldehyde in an aqueous solution.

Tissue specimens are fixed for 6 to 8 hours prior to getting transferred to 70% alcohol to "wash away" the yellow before being sent to histology for automated dehydration, paraffin embedding, sectioning, and staining. ***(Bancroft and Gamble, 2008).***

**RESULTS**

At the end of the study period all cases were subjected to the following studies:

1. **Observations during the study:**

In the present study, 10 medicolegal autopsy cases of Aluminum phosphide were studied. 5 were females and 5 were males, varying in age from 15 - 45 years with higher incidence in 2nd & 3rd decade and rare incidence in the extremes of age. No homicidal case was noted, 100% of the cases were recorded as suicidal incidence. The amount of aluminum phosphide tablets ingested may be estimated based on the history provided by the deceased's relatives, and it ranged from half a tablet to three tablets. The survival time in research cases ranged between 3-48 hours, whereas the post-mortem interval ranged between 7-48 hours.

With focusing on the distinctive observations of AlP poisoned corpses from the 5 control corpses on the external examination during autopsy, distinct bluish discoloration of the face was seen in 2 cases. Froth was present around the nostrils in one case which was blood tinged. In 4 cases, a characteristic garlicky odor associated with aluminum phosphide poisoning was spotted near to the body.

Internal examination revealed that the trachea was congested in all cases and that froth was present in 4 of them. In all cases, the lungs were found to be congested and edematous after being sliced. In all cases, little hemorrhages were found in the interlobular gaps and on the lungs' margins. Frothy, dark hemolyzed blood was seen flowing out of the cut. In 7 cases, the stomach had greyish brown fluid or pasty material. In 8 cases, the characteristic odor was detected. Sloughing of mucosa was also detected in all cases, with a greater prevalence in the fungal zone. In all cases, the liver, spleen, and kidneys were found to be congested.

1. **Histopathological results**

*The following histopathological findings were observed in the studied cases of the control group* ***(Fig 1,2):***

* + **Stomach:**

Histopathological examination of the stomach of most control cases showing normal architecture of the gastric mucosa with normal epithelial lining. The lamina propria is occupied with normal fundic glands.

* + **Small intestine:**

Histopathological examination of small intestine of all the control cases showing normal fundic mucosa with normal architecture of mucosal villi and submucosal layer containing normal burner’s glands with normal intraepithelial lymphocytes.

* + **Kidneys:**

Histopathological examination of the kidney of control cases showed normal architecture in all cases in the form of cortex contains normal renal corpuscles. Well-arranged renal glomeruli and tubules were observed.

* + **Lung:**

Histopathological examination of the lung of most control cases showed normal architecture in all cases with alveoli lined with flattened epithelium and normal bronchial vessels.

* + **Heart:**

Histopathological examination of the heart of all the control cases showing normal architecture of well-arranged myocardial cells.

* + **Liver:**

Histopathological examination of the liver of the control cases showed normal architecture in all cases in the form of hepatocytes arranged in hepatic plates radiating from central vein. Hepatocyte is polygonal in shape; cytoplasm is acidophilic with one or more round nucleus. Portal canal with normal vessels present at some angles of hepatic tubules.

*The following histopathological findings were observed in the study cases aluminium phosphide poisoning deaths* ***(Tab 1, Fig 1,2):***

* + **Stomach:**

On microscopic examination, the stomach wall was congested in all hospitalized cases, with patchy submucosal congestion in the remaining instances. Edema was seen in all cases, ranging from mild to severe. All of the cases had necrosis of the mucosa of the fundus region, and 5 had necrosis in other parts of the stomach in addition to the fundus region. In seven cases, inflammatory cell invasion reached the muscle layer.

* + **Small intestine:**

On microscopic examination, the intestinal wall of the duodenum was congested and edematous in each case. All of the cases involved duodenal mucosal necrosis, but in three of them, the muscular layer was also affected.

* **Kidneys:**

All of the cases involved congested kidneys. In each case, there was tubular dilating, edema, necrosis, and areas of tubular epithelial disintegration. There were inflammatory cell infiltration & intratubular hemorrhage in 4 cases. Also, there was glomerular necrosis in all the cases.

* + **Lung:**

On microscopic examination of lungs, all cases showed congestion and edema, which varied from mild to severe. In all cases, hemolyzed red cells and dilated capillaries thickened the alveoli, and 2 cases also showed cell infiltration around the bronchioles.

* + **Heart:**

All of the cases had congested and edematous myocardium, with focal myocardial necrosis identified in 9 cases and inflammatory cell infiltration seen in 3.

* + **Liver:**

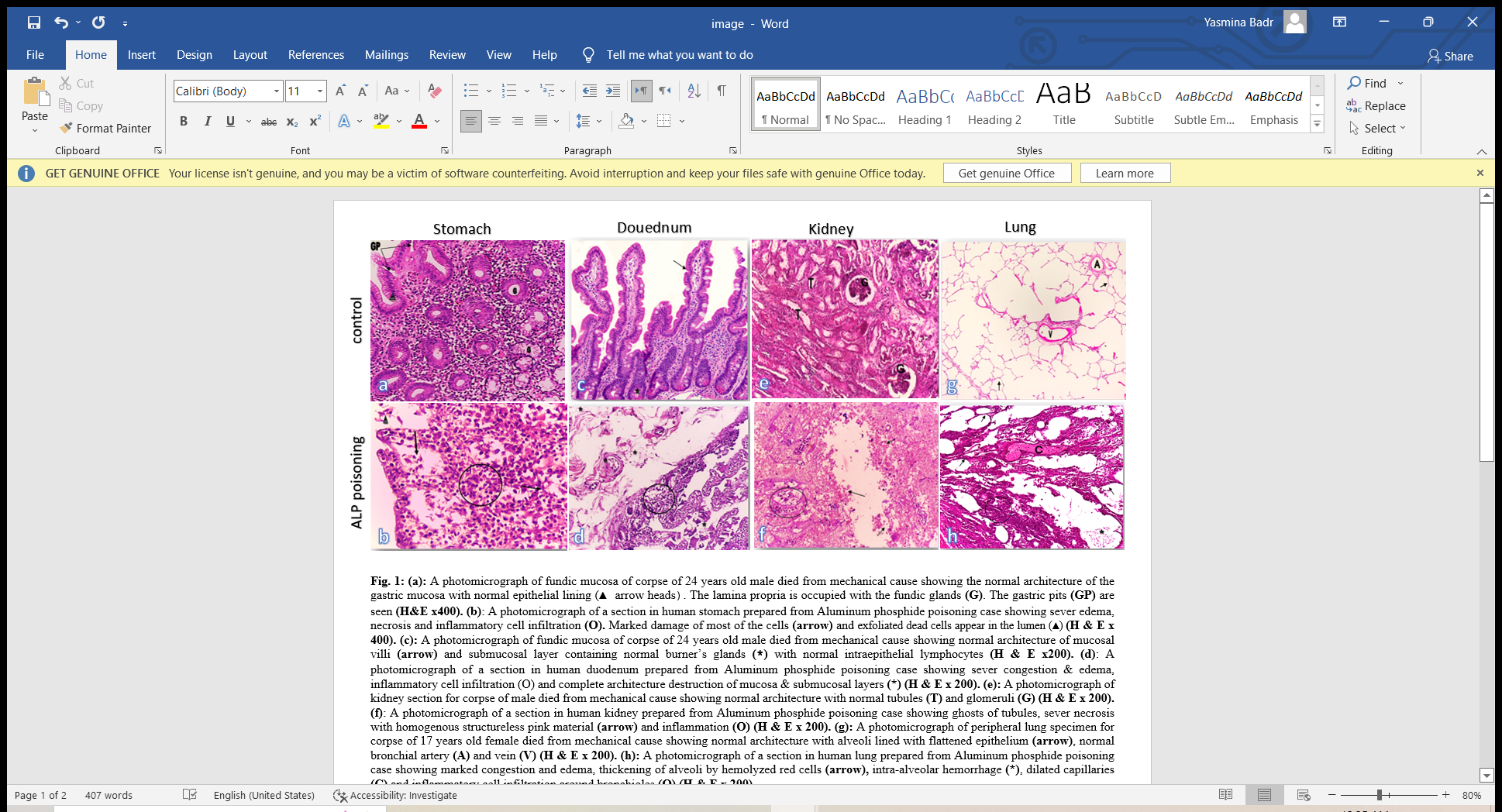
Liver was congested in all cases; mild fatty alteration was observed in 6 cases; and patches of Centrizonal hemorrhagic necrosis were observed in all cases.

* + **Spleen:**

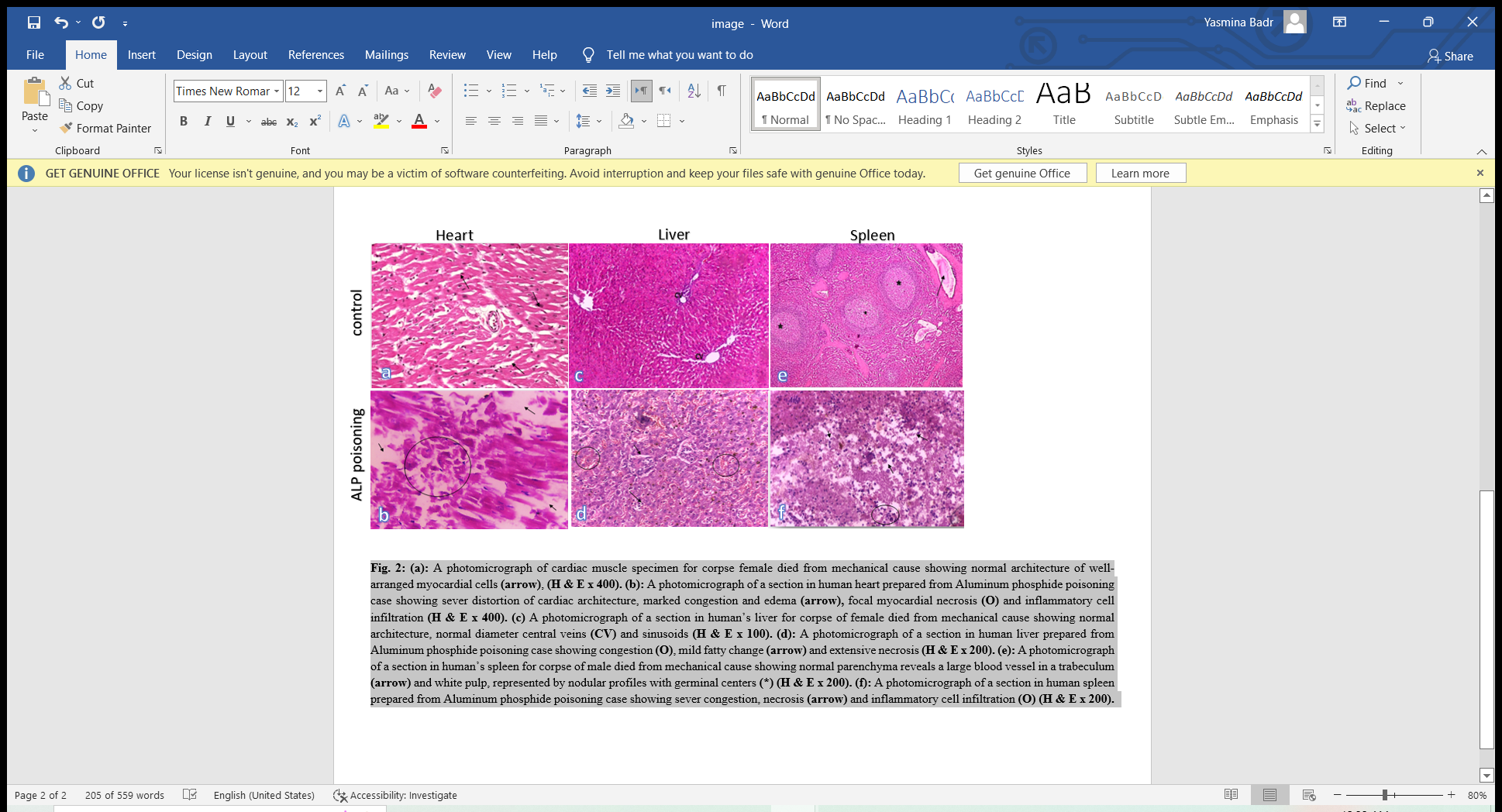
In all of the cases, there was splenic congestion. In 7 cases, splenic necrosis was seen.

**Table (1):** Histopathological findings of various organs in the present study on deaths with Aluminum phosphide poisoning:

|  |  |
| --- | --- |
| Histopathological findings | Percentage of each finding in poisoned cases (N= 10) |
| 1. Stomach: |
| 1. Congestion | 100% |
| 1. Edema | 100% |
| 1. Necrosis of mucosa | 100% |
| 1. Round cell infiltration up to mucosal layer | 70% |
| 1. Small intestine: | |
| 1. Congestion | 100% |
| 1. Edema | 100% |
| 1. Necrosis of mucosa | 100% |
| 1. Round cell infiltration up to mucosal layer | 30% |
| 1. Kidney: |
| 1. Congestion | 100% |
| 1. Necrosis, degeneration of tubular epithelium. | 100% |
| 1. glomerular necrosis | 100% |
| 1. Inflammatory cell infiltration. | 40% |
| 1. intratubular hemorrhage | 40% |
| 1. Lung: |
| 1. Congestion | 100% |
| 1. Oedema | 100% |
| 1. Thickening of alveoli by hemolyzed RBC and dilated capillaries | 100% |
| 1. Round cell infiltration | 20% |
| 1. Heart: |
| 1. Congestion | 100% |
| 1. Focal myocardial necrosis | 90% |
| 1. Round cell infiltration | 30% |
| 1. Liver: | |
| 1. Congestion | 100% |
| 1. Mild fatty infiltration | 60% |
| 1. Centrizonal necrosis | 100% |
| 1. Spleen: | |
| 1. Congestion | 100% |
| 1. Necrosis | 70% |



**Fig. 1: (a):** A photomicrograph of fundic mucosa in the stomach of control corpse of 24 years old male showing the normal architecture of the gastric mucosa with normal epithelial lining (▲ arrow heads). The lamina propria is occupied with the fundic glands **(G)**. The gastric pits **(GP)** are seen **(H&E x400).** **(b)**: A photomicrograph section in human stomach prepared from Aluminum phosphide poisoning case showing sever edema, necrosis and inflammatory cell infiltration **(O).** Marked damage of most of the cells **(arrow)** and exfoliated dead cells appear in the lumen (▲) **(H & E x 400).** **(c):** A photomicrograph of fundic mucosa in the duodenum of corpse of 24 years old male died from mechanical cause showing normal architecture of mucosal villi **(arrow)** and submucosal layer containing normal burner’s glands **(\*)** with normal intraepithelial lymphocytes **(H & E x200). (d)**: A photomicrograph section in human duodenum prepared from Aluminum phosphide poisoning case showing sever congestion & edema, inflammatory cell infiltration (O) and complete architecture destruction of mucosa & submucosal layers **(\*)** **(H & E x 200).** **(e):** A photomicrograph of kidney section for corpse of male died from mechanical cause showing normal architecture with normal tubules **(T)** and glomeruli **(G)** **(H & E x 200).** **(f)**: A photomicrograph section in human kidney prepared from Aluminum phosphide poisoning case showing ghosts of tubules, sever necrosis with homogenous structureless pink material **(arrow)** and inflammation **(O)** **(H & E x 200).** **(g):** A photomicrograph of peripheral lung specimen for corpse of 17 years old female died from mechanical cause showing normal architecture with alveoli lined with flattened epithelium **(arrow)**, normal bronchial artery **(A)** and vein **(V) (H & E x 200). (h):** A photomicrograph section in human lung prepared from Aluminum phosphide poisoning case showing marked congestion and edema, thickening of alveoli by hemolyzed red cells **(arrow),** intra-alveolar hemorrhage **(\*)**, dilated capillaries **(C)** and inflammatory cell infiltration around bronchioles **(O)** **(H & E x 200).**



**Fig. 2: (a):** A photomicrograph of cardiac muscle specimen for corpse female died from mechanical cause showing normal architecture of well-arranged myocardial cells **(arrow)**, **(H & E x 400). (b):** A photomicrograph section in human heart prepared from Aluminum phosphide poisoning case showing sever distortion of cardiac architecture, marked congestion and edema **(arrow),** focal myocardial necrosis **(O)** and inflammatory cell infiltration **(H & E x 400). (c)** A photomicrograph section in human’s liver for corpse of female died from mechanical cause showing normal architecture, normal diameter central veins **(CV)** and sinusoids **(H & E x 100). (d):** A photomicrograph section in human liver prepared from Aluminum phosphide poisoning case showing congestion **(O)**, mild fatty change **(arrow)** and extensive necrosis **(H & E x 200). (e):** A photomicrograph section in human’s spleen for corpse of male died from mechanical cause showing normal parenchyma reveals a large blood vessel in a trabeculum **(arrow)** and white pulp, represented by nodular profiles with germinal centers **(\*)** **(H & E x 200).** **(f):** A photomicrograph section in human spleen prepared from Aluminum phosphide poisoning case showing sever congestion, necrosis **(arrow)** and inflammatory cell infiltration **(O) (H & E x 200).**

**DISCUSSION**

Aluminum phosphide poisoning is a serious and frequent scourge in developing countries, where it became an epidemic state and constituting a healthcare problem and a real challenge for the medical profession ***(Kenza Zniber et al., 2021).***

Poisoning from suicidal or unintentional intake of aluminum phosphide is a common medical problem encountered worldwide. Aluminum phosphide, when exposed to moisture, emits the highly toxic gas, phosphine. Toxic symptoms of phosphine overdose usually appear quickly, sometimes within 15 minutes. The vast majority of deaths occur within the first 12/24 hours, mainly as a result of cardiac arrest. Deaths that occur after 24 hours are frequently caused by liver failure. ***(Changal et al., 2017).***

The present work studied on medicolegal deaths of aluminum phosphide poisoning, aimed to assess autopsy findings of Aluminum phosphide poisoning.

Results of this study showed varying in age distribution of aluminum phosphide poisoning deaths from (15- 45) years with higher incidence in 2nd & 3rd decade and rare incidence in the extremes of age.

These findings are consistent with the findings of ***Mathai and Bhanu (2010)*** who observed that the majority of poisoned cases were young, ranging in age from 21 to 40 years.

Also,***Shaheen et al. (2018) study,*** whichwas in agreement with the current work, explained these observations by those younger persons are easily excited. On the other side, as individuals get older, they take more mature steps.

During the current study, neither homicidal nor accidental case was noted, 100% of the cases were recorded as suicidal incidence.

In agreement with these results, ***Mwaheb and Hassan (2020),*** found that all deaths in their studies were (100%) suicidal attempts in Egypt. They stated that due to its accessibility, its higher toxicity to humans and our social problems.

Furthermore, ***Meena et al. (2015)*** revealed that suicide was the most common cause of death from aluminum phosphide poisoning (94%), followed by accident (5%), and homicide (1%).

In contrast, ***Gunnell D et al. (2007)*** found that each year, 4% of pesticide self-poisoning deaths occur in the European region, 50% in the Western Pacific region, 33% in Africa, and 0.07% in North America.

This Contrast in the results may be due to the difference between different global regions, especially between developed and developing countries, in the culture, social life and problems.

The current study showed distinct bluish discoloration of the face of 2 cases (20%) during the external examination. Also, Froth was present around the nostrils in one case which was blood tinged. Furthermore, a characteristic garlicky odor related to Aluminum phosphide poisoning was present near to the body in four (40% of the cases).

***Shaheen et al. (2018) and*** ***Jain et al. (2005)*** coincided with the current observations and reported that Gross examination during postmortem revealed the following:

* Face was cyanosed and congested in 22% & 41.17% cases respectively.
* Garlic pungent smell was observed in 50% & 47.05% cases.
* Froth at and around mouth and nostrils was observed in 12% & 76.47% while blood-tinged froth was observed in 68% & 64.70% respectively.

In the current study, internal examination revealed that the trachea was congested in all cases (100%) and froth was detected in four cases (40%). Lungs were found to be congested and edematous in all cases (100%). Small haemorrhages were typically observed in all cases (100%) in the interlobular spaces and lung borders. Frothy, dark hemolyzed blood was seen flowing out of the cut. ***Hugar et al. (2014) and Jain et al. (2005)*** previously reported similar findings.

Furthermore, the current study found that on internal examination, the stomach had greyish brown fluid or pasty material in 7 (70% of the cases). The distinct odor was detected in 8 out of 10 cases (80%). Sloughing of mucosa was also observed in all the cases (100%), more in the fungal region.

These findings were in harmony with those of ***Jain et al. (2005),*** who observed that the stomach contained greyish brown fluid or pasty material in 29 cases (58%). The distinct odor was recognized in 33 (66% of the studied cases). Sloughing of mucosa was also observed in all the cases, more in the fungal region which explained by raising and accumulating of phosphine gas vapors in fundal region ***(******Shaheen et al., 2018)***. On the other hand, he explained that the odor indicator is likely to be observed during autopsy in non-hospitalized cases as well as in hospitalized cases where gastric lavage was not performed.

***Hugar et al. (2014)*** supported our findings by reporting in his case report study that the stomach contained approximately 300 mL of dark brown colored fluid (altered blood) and showed hemorrhagic regions with ulcers on the mucosal surface.

The histological findings in the stomach specimens of aluminum phosphide poisoning cases included congestion in all hospitalized cases and patchy submucosal congestion in the remaining cases. Edema was seen in all cases, ranging from mild to severe. Necrosis of the mucosa of the fundus region was seen in all cases (100%), with 5 cases (50%) showing necrosis in regions other than the fundus region. Inflammatory cell infiltration was observed up to the muscle layer in 7 (70% of the cases).

These findings are in line with those of ***Jain et al. (2005),*** who reported that the stomach wall was congested on microscopic examination in 56% of the hospitalized cases, with patchy submucosal congestion in the other cases. Edema was seen in 68% of the cases, with mild edema in 52%, moderate edema in 14%, and severe edema in 2%. Almost all of the cases (98%) had necrosis of the mucosa of the fundus region, while 48% had necrosis in other parts of the stomach in addition to the fundus region. Inflammatory cell infiltration was found up to the muscle layer in 12% of the cases.

The current work showed the intestinal wall of duodenum on microscopic examination was congested & edematous in all the cases. All of the cases had necrosis of the duodenal mucosa, with three cases having necrosis up to the muscle layer. These findings are also not discussed in the previous studies.

As regard histopathological changes in the present study, microscopic examination of the kidney specimens revealed congestion, necrosis and areas of degeneration of tubular epithelium in all the cases (100%). Inflammatory cell infiltration & intratubular hemorrhage in 4 cases (40%). Also, there was glomerular necrosis in all the cases (100%).

These findings were consistent with those of ***Liang et al. (2020)*** who reported that epithelial necrosis was detected in both the proximal and distal renal tubules of all eight decedents in his study. Tubular dilatation, edema, and even balloon degeneration was occasionally reported. Interstitial bleeding with inflammatory cell infiltration was discovered in study deceased no. 1.

In partially agreement with the current findings,***Manoj and Rohini (2020)*** reported that the glomeruli generally appeared normal and showed mild congestion in few cases, but that not correlated with our findings. Another important finding in kidney was intratubular hemorrhage with RBC within tubules; that correlated with the present work.

Also, the microscopic examination of the lung specimens in the present study showed congestion and edema, which varied from mild to severe in all the cases (100%). Thickening of alveoli by hemolyzed red cells and dilated capillaries were seen in all the cases (100%) and inflammatory cell infiltration around bronchioles in 2 cases (20%).

These findings are in accordance with those of ***Liang et al. (2020) and Jain et al. (2005),*** who discovered severe edoema and mild haemorrhaging in numerous alveoli in aluminium phosphide poisoning cases. Many areas had collapsed alveoli and bronchioles, as well as alveolar dilatation. Necrosis and exfoliation of the bronchial mucosa epithelium were occasionally detected, together with peribronchiolar inflammatory infiltrate.

The current work showed histopathological changes of the heart specimens of aluminum phosphide poisoning cases. In all cases (100%), the myocardium was congested and edematous, with localized myocardial necrosis detected in 9 cases (90%) and inflammatory cell infiltration seen in 3 cases (30%).

These results were in a harmony with ***Memiş et al. (2007) and Jain et al. (2005)*** who reported that postmortem examinations of aluminum phosphide heart specimens have revealed focal myocardial infiltration and necrosis widespread small vessel injury.

In partially agreement with the current work findings, ***Liang et al. (2020)*** reported that we continually observed congestion, myocardial contraction band necrosis, coagulation necrosis, vacuolar degeneration, edema in all the study cases and this what is correlated with the current study. But he also reported that there was scattered inflammatory cells in the heart tissues of all the study cases (100%) which is not correlated with our findings.

The reason of myocardial cellular infiltration as stated in ***Liang et al. (2020***) study was minor in current work, which might be attributed to the fact that the full-blown infarct happened just a short time before the patient died ***(Shah et al., 2009).***

As regard histopathological changes in the present study, microscopic examination of the liver specimens of aluminum phosphide poisoning cases revealed congestion and areas of Centrizonal hemorrhagic necrosis in all cases (100%) in addition to mild fatty changes in 6 cases (60%).

The results of the present study coincided with those of ***Liang et al. (2020)*** who reported that liver specimens of aluminum phosphide poisoning cases showed focal hepatocyte necrosis with inflammatory cell infiltration, varying degree of edema and congestion in most of the study cases.

These findings were in agreement with the results of ***Jain et al. (2005),*** who reported that the liver was congested in 44 cases (88%), there was mild fatty change in 19 cases (38%), and regions of centrizonal hemorrhagic necrosis were identified in 10 cases (20%). Furthermore, ***Mehrpour et al. (2008)*** found that central venous congestion, microvacuolizition, hepatocyte degradation, and mononuclear infiltration were the most common histological findings in the liver.

The current work showed congestion in all the spleen specimens (100%) and Splenic necrosis in 7 cases (70%).

These results are in line with ***Shaheen et al. (2018) and Jain et al. (2005)*** who found that administration of NVP induced histopathological changes of spleen specimens including congestion, edema and areas of splenic necrosis in most of the cases.

Previous studies assumed that the primary mechanism of PH3 toxicity was ATP depletion caused by suppression of mitochondrial oxidative phosphorylation ***(Bumbrah et al., 2012; Moghadamnia AA, 2012).*** As our study found, this can cause widespread organ damage. Furthermore, the cardiac and hepatocellular fatty degeneration we found might be regarded typical pathological changes of PH3 poisoning and help to explain the harmful mechanism of PH3 (inhibition of mitochondrial oxidative phosphorylation), as indicated by ***Liang et al. (2020) study.***

**CONCLUSION**

We believed that these characteristics abnormalities were an indication of PH3 poisoning and that they helped to describe the harmful mechanism of PH3. We discovered serious damage caused by PH3 in several body systems, including the stomach, duodenum, lung, and kidney, after focusing on the pathological changes on the major organs. Furthermore, we found extensive myocardial, splenic, and hepatocellular deterioration in the heart, spleen, and liver tissues. When taken together, these findings may help to increase knowledge of PH3 toxicity and give indications for potential therapies in acute PH3 poisoning.

**RECOMMENDATIONS**

* Raising public awareness about its toxicity and fatality through the media is an urgent requirement.
* A thorough study is required to comprehend the predisposing aspects and mechanism of action in order to assess and establish an effective treatment in such cases.
* Treatment options should be widely available in hospitals.
* It should be illegal to store it or sell it, and only people with permission should be able to use, store, or sell it.

**CONFLICTS OF INTEREST**

There are no conflicts of interest declared by the authors.

**REFERENCE**

***Abdel-Hady R., Mohamed A., Khalifa M. & Rahman K. (2019):*** Supportive Measures in the Treatment of Aluminum Phosphide Poisoning as a Trial to Reduce Mortality at Assiut University Hospital, Egypt. Arab Journal of Forensic Sciences & Forensic Medicine. 1. 1210-1222. 10.26735/16586794.2019.008.

***Abdollahi, M. and Mehrpour, O. (2014):*** Aluminum Phosphide. Encyclopedia of Toxicology, 3rd Edition, Elsevier Inc., Academic Press, pp. 164–166.

***Bancroft JD and Gamble M {Eds.} (2008):*** Fixatives. In: Theory and practice of histology techniques. 6th ed., Churchill Livingstone, Elsevier, China, pp.72-75.

***Bumbrah G. S., Krishan K., Kanchan T., Sharma M., & Sodhi G. S. (2012):*** Phosphide poisoning: a review of literature. Forensic science international, 214(1-3), 1–6. <https://doi.org/10.1016/j.forsciint.2011.06.018>

***Changal K.H., Latief M., Parry M., Abbas F. (2017):*** Aluminium phosphide poisoning with severe cardiac dysfunction and the role of digoxin. BMJ Case Rep. 2017 Aug 11;2017: bcr2017220125. doi: 10.1136/bcr-2017-220125. PMID: 28801325; PMCID: PMC5614089.

***Gregorakos L., Sakayianni K. & Harizopoulou, V. (2002):*** Recovery from severe inhalational phosphine poisoning. Report of two cases. Clinical Intensive Care, 13, 177–179.

***Gunnell D., Eddleston M., Phillips M.R. & Konradsen F. (2007):*** The global distribution of fatal pesticide self-poisoning: systematic review. BMC Public Health. 7:357. doi: 10.1186/1471-2458-7-357. PMID: 18154668; PMCID: PMC2262093.

***Hashemi-Domeneh B., Zamani N., Hassanian-Moghaddam H., Rahimi M., Shadnia S., Erfantalab P., & Ostadi A. (2016):*** A review of aluminium phosphide poisoning and a flowchart to treat it. Arhiv za :higijenu rada i toksikologiju, 67(3), 183–193. <https://doi.org/10.1515/aiht-2016-67-2784>

***Hsu C. H., Chi B. C., Liu M. Y., Li J. H., Chen C. J., & Chen R. Y. (2002):*** Phosphine-induced oxidative damage in rats: role of glutathione. Toxicology, 179(1-2), 1–8. <https://doi.org/10.1016/s0300-483x(02)00246-9>.

***Hugar B., Praveen S., Sh Hosahally J., Kainoor S. & Shetty, A.R. (2014):*** Gastrointestinal Hemorrhage in Aluminum Phosphide Poisoning. Journal of Forensic Sciences. 60. 10.1111/1556-4029.12588.

***Jain A.K., Nigam M.K., Garg S., Dubey B.P., & Arora A. (2005):*** ALUMINIUM PHOSPHIDE POISONING AUTOPSY FINDINGS. Journal of Indian Academy of Forensic Medicine, 27, 35-39.

***Kalawat S., Thakur V., Thakur A., & Punjabi N. (2016):*** Cardiovascular profile of aluminium phosphide poisoning and its clinical significance. International Journal of Advances In Medicine, 859-864. doi: 10.18203/2349-3933.ijam20163505.

***Kenza Z., Abdoulaye D., Dania S., Yasmina B., Sofia K., Khalid A., Amine A. Z. et al. (2021):*** Reversible Myocardial Injury Associated With Aluminum Phosphide Poisoning: Case Report. Sch J Med Case Rep, 9(5): 476-479.

***Liang Y., Tong F., Huang F., Liu Y., Zhu L., Le Grange J.M., He G. & Zhou Y. (2020):*** Pathological changes induced by phosphine poisoning: a study on 8 children. Int J Legal Med.;134(1):217-228. doi: 10.1007/s00414-019-02169-z. Epub 2019 Nov 11. PMID: 31713064.

***Manoj K. P. & Rohini M. (2020): Study of Renal Histopathology In Case of Celphos Poisoning by SEM. IAR J MED Sci; vol (1), iss-1. 10.47310/iarjms.2020.v01i05.003***

***Mathai A., &*** ***Bhanu M. S. (2010):*** Acute aluminium phosphide poisoning: Can we predict mortality?. Indian journal of anaesthesia, 54(4), 302–307. <https://doi.org/10.4103/0019-5049.68372>

***Meena M.C., Mittal S. & Rani Y. (2015):*** Fatal aluminium phosphide poisoning. Interdiscip Toxicol. 2015 Jun;8(2):65-7. doi: 10.1515/intox-2015-0010. PMID: 27486362; PMCID: PMC4961899.

***Mehrpour O., Dolati M., Soltaninejad K., Shadnia S., & Nazparvar B. (2008):*** Evaluation of Histopathological Changes in Fatal Aluminum Phosphide Poisoning. Journal of Forensic Medicine, 2, 34-36.

***Memiş D., Tokatlıoglu D., Koyuncu O., & Hekimoglu S. (2007):*** Fatal aluminium phosphide poisoning. European Journal of Anaesthesiology, 24(3), 292-293. doi:10.1017/S0265021506001451.

***Moghadamnia AA (2012):*** An update on toxicology of aluminum phosphide. Daru 20:25. <https://doi.org/10.1186/2008-2231-20-25>

***Mwaheb, M. & Hassan S. (2020):*** Fatal Aluminium Phosphide poisoning in Fayoum Governorate, Egypt (2012-2019). The Egyptian Journal of Forensic Sciences and Applied Toxicology. 21. 10.21608/ejfsat.2020.28015.1141.

***Saleh A. & Makhlof M. (2018):*** Outcome of Toxicity and Mortality Predictors Of Aluminum Phosphide Poisoning In Fayoum Governorate, Egypt. Zagazig Journal of Forensic Medicine, 16(2), 40-52. doi: 10.21608/zjfm.2018.6009.1019

***Shah V. Baxi S. & Vyas T. (2009):*** Severe myocardial depression in a patient with aluminium phosphide poisoning: a clinical, electrocardiographical and histopathological correlation. Indian Journal of Critical Care Medicine: Peer-reviewed, Official Publication of Indian Society of Critical Care Medicine. 13(1):41-43.

***Shaheen M.A., Murrad Z. & M. M. M. Z. B. (2018):*** Pattern of Aluminium Phosphide Poisoning and Autopsy Findingsat KEMU Lahore. Journal of Fatima Jinnah Medical University, 5(3). Retrieved from https://jfjmu.com/index.php/ojs/article/view/282.

***Sheta A.A., El-Banna A.S., Elmeguid R.A., Mohamed H.E. & Gad N.H. (2019):*** A study of the predictive factors of mortality in acute poisoning with aluminum phosphide with special reference to echocardiography and SOFA score.Environ Sci Pollut Res Int. 26(32):33135- 33145.

***Singh, Y., Joshi, S.C., Satyawali, V. et al. (2014):***Acute aluminium phosphide poisoning, what is new? Egypt J Intern Med 26, 99–103. <https://doi.org/10.4103/1110-7782.145298>

***Yan H., Chen H., Li Z., Shen M., Zhuo X., Wu H., & Xiang, P. (2018):*** Phosphine Analysis in Postmortem Specimens Following Inhalation of Phosphine: Fatal Aluminum Phosphide Poisoning in Children. Journal of Analytical Toxicology, 42(5), 330–336. https://doi.org/10.1093/jat/bky005.

**الملخص العربي**

**نتائج التشريح للجثث المسممة بفوسفيد الألومنيوم بمحافظة القليوبية، جمهورية مصر العربية**

يستخدم فوسفيد الألومنيوم المعروفة بأقراص الغلة على نطاق واسع كمبيد حشري اقتصادي وفعال، ومبيد للقوارض. حيث أن المكون النشط لـه هو غاز الفوسفين، فتوفر فوسفيد الألومنيوم بسهولة يؤدى إلى استخدامه كوسيلة انتحار بشكل متزايد مع ارتفاع معدل الوفيات. فغاز الفوسفين يؤدى إلى حدوث تلف كامل بجسم الإنسان. لذلك تهدف الدراسة الحالية إلى تحليل نتائج تشريح الجثة في أعضاء مختلفة من جسم الإنسان بسبب تسمم فوسفيد الألومنيوم في هيئة الطب الشرعي ببنها، القليوبية، جمهورية مصر العربية. حيث استعرضت هذه الدراسة 10 حالات تشريح مؤكد كيميائيًا بتسممها بفوسفيد الألومنيوم وتم فحصها بالتفصيل بشكل كبير وتم صبغ عينات من الأنسجة من مختلف الأعضاء بواسطة الهيماتوكسيلين إيوزين وتم ملاحظتها مجهريًا. بعد التركيز على التغيرات المرضية على الأعضاء الرئيسية وجدنا أضرارا شديدة يسببها غاز الفوسفين في العديد من الأجهزة وخاصة في المعدة والاثني عشر والرئة والكلى. علاوة على ذلك، لاحظنا التنكس الشديد في أنسجة عضلة القلب والطحال والكبد. الخلاصة. ولذلك اعتبرنا أن هذه التغييرات المميزة إجمالا هي علامة توحي بالتسمم غاز الفوسفين بسبب الآلية السامة لهذا الغاز وهي (تثبيط الفسفرة المؤكسدة للميتوكوندريا). فنأمل أن يتمكن هذا البحث من تحسين فهم سمية غاز الفوسفين في كل من الطب الشرعي والممارسة السريرية.