

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

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ABSTRACT

Introduction: 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. ALP's active ingredient is phosphine (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 in the Medico-legal Department, Benha, Qalyubiyah, Egypt. **Methodology:** In this work, 10 control cases and 10 chemically confirmed autopsy cases were included and examined in gross detail. Samples of tissue from various organs were stained with hematoxylin-eosin and examined microscopically. **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:** This study considered that these characteristic abnormalities were a possible indicator of PH₃ poisoning and partially explained the substance's deadly nature (inhibition of mitochondrial oxidative phosphorylation) and that may 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 the 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*).

Aluminum phosphide release phosphine (PH₃) gas if it comes in contact with moisture in the air. PH₃ gas is colorless, extremely

flammable and explosive; very toxic to insects; and useful as a fumigant for stored products to control pests; it has the characteristic smell of garlic or decaying fish (*Yan et al., 2018*).

All tissues can quickly absorb phosphine, which is then eliminated in the urine and exhaled through the lungs (*Hashemi-Domeneh et al., 2016*). 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*). It is likely that interference with cellular respiration may lead to multisystem toxicity (*Hsu et al., 2002; Hashemi-Domeneh et al., 2016*).

The incidence of ALP poisoning in Egypt start to increase sharply although government efforts to control their widespread use, and accounts for a reasonable proportion of the patients admitted to Egyptian poison centers to reach the present magnitude and require more extensive clinical and autopsy studies (*Abdelhamid, 2021*).

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 (2021)* 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 humans prompted us to undertake this study.

The purpose of this study was to analyze autopsy findings in ALP poisoning deaths that could provide information about the cause of PH₃-induced systemic damage.

MATERIALS AND METHODS

- In the present study, ten medico-legal autopsy cases of ALP poisoning were

investigated (five females and five males) upon chemical analysis of the viscera in the Forensic Science laboratory. The cases were aged 15–45 years, with a higher incidence in the 2nd and 3rd decades and a rare incidence at the extremes of age. A total of five controls were selected who died from thermal or mechanical injuries. All cases were collected from the Medico-legal Department, Benha, Qalyubiyah, Egypt.

- **Required data:**

- A detailed and accurate history concerning the time and quantity of aluminum phosphide ingestion and the cause of death was obtained
- This study excluded cases with viscera that were positive for alcohol or any other toxins. Hospitalized and non-hospitalized patients were observed for signs and symptoms as well as treatment. An in-depth investigation of both the poisoned and control cases was undertaken.

- **Autopsy (External & Internal examination):**

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

- **Histopathological examination:**

Histopathological examinations were done on all cases and examined at the Pathology Department, Faculty of Medicine, Benha University. After the autopsy, lungs, heart, stomach, small intestine, spleen, liver, and kidneys were immediately soaked in Bouin's solution (composed 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 experimental period, all observations were recorded and analyzed for all cases:

There was no homicidal case was recorded, 100% of the of them were suicides. Based on the history given by the deceased's relatives, the number of aluminum phosphide tablets varied from half a tablet to 3 tablets. The survival varied between 3 and 48 hours, and the post-mortem interval ranged between 7 and 48 hours.

I. Autopsy Findings:

On external examination during the autopsy, a distinct bluish discoloration of the face was seen in two cases. Froth was present around the nostrils in one case, which was blood-tinged. In four cases, a distinct garlicky odor associated with ALP poisoning was present within the body.

On internal examination, the trachea was congested in all cases, and froth was present in four cases. Lungs on the cut were congested and edematous in all cases. There were small-sized haemorrhages observed in all cases in the interlobular spaces and lung margins. On the cut, frothy, dark haemolyzed blood was observed coming out. The stomach contained greyish-brown fluid or pasty material in seven cases. The distinct odor was perceived in eight cases. Sloughing of the mucosa was also observed in all cases, especially in the fungal region. The liver, spleen, and kidneys were congested in all cases.

II. Histopathological results:

The following histopathological findings were observed in the studied cases of the control & ALP poisoned cases (**Figure 1 & Table 1**):

On microscopic examination, the stomach wall was congested in all hospitalized cases, and in the rest of the cases, there was patchy submucosal congestion (100%). Edema

was observed in all cases and varied from mild to severe (100%). Necrosis was observed in fundus region mucosa in all the cases and in just five cases, necrosis was also present in different areas of the stomach. In seven cases, round cell infiltration was seen up to the muscular layer (70%).

The intestinal wall of duodenum on microscopic examination was congested & edematous in all the cases. Necrosis of mucosa of duodenal region was observed in all the cases (100%), while three cases showed necrosis up to muscle layer (30%).

Kidneys were congested in all the cases. Tubular dilation, edema, necrosis and areas of degeneration of tubular epithelium was seen in all the cases (100%). Inflammatory cell infiltration & intratubular hemorrhage in four cases (40%). Also, there was glomerular necrosis in all the cases.

On microscopic examination of lungs, all cases showed congestion and edema, which varied from mild to severe (100%). Thickening of the alveoli by hemolyzed red cells and dilated capillaries were seen in all cases and cell infiltration around bronchioles in two cases (20%).

Myocardium was congested & edematous in all the cases, focal myocardial necrosis seen in nine cases (90%) and inflammatory cell infiltration seen in three cases (30%).

Liver was congested in all the cases (100%); A mild fatty change was seen in six cases (60%) and areas of centrilobular hemorrhagic necrosis was seen in all the cases (100%).

Congestion in the spleen was apparent in all cases (100%). Splenic necrosis was seen in seven cases (70%).

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

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

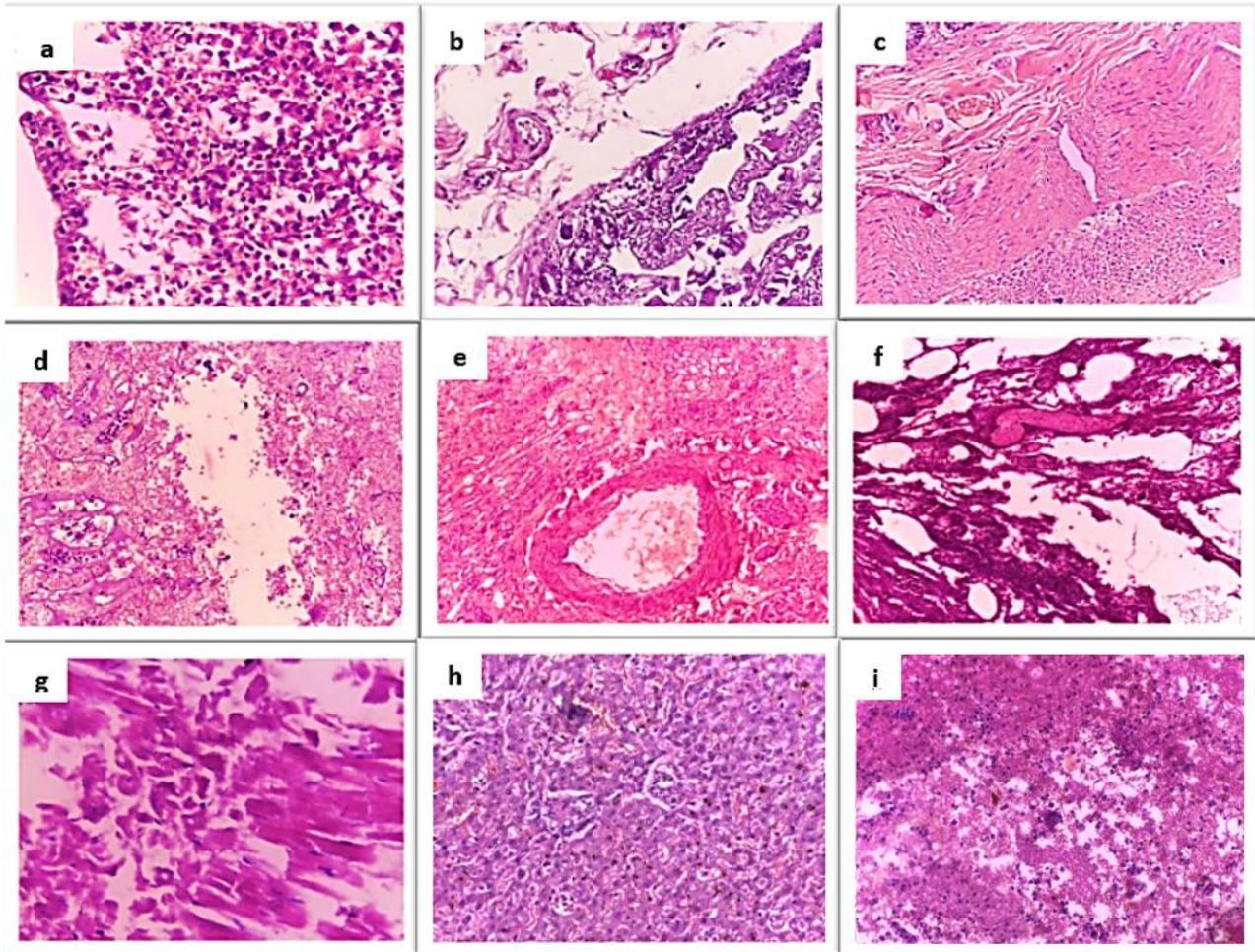


Fig. 1: A photomicrograph sections of different organs of the ALP poisoning cases. **(a)** stomach section showed sever edema, necrosis and inflammatory cell infiltration (H and E x 400). **(b)** human duodenum section showed sever congestion & edema, necrosis and cell infiltration (H and E x 200). **(c)** human duodenum section showed sever congestion & edema, necrosis and cell infiltration (H and E x 200). **(d)** human kidney section showed ghosts of tubules, sever necrosis with homogenous structureless pink material and inflammation (H and E x 200). **(e)** human kidney section showed marked tubular dilatation with intratubular hemorrhage, in addition to massive tubular and glomerular necrosis with marked congestion and inflammation (H and E x 200). **(f)** human lung section showed marked congestion and edema, thickening of alveoli by hemolyzed red cells, dilated capillaries and cell infiltration around bronchioles (H and E x 100). **(g)** human heart section showed marked congestion and edema, focal myocardial necrosis, and inflammatory cell infiltration (H and E x 400). **(h)** human liver section showed congestion, mild fatty change and centrilobular hemorrhagic necrosis (H and E x 200). **(i)** human spleen section showed sever congestion, necrosis, and inflammatory cell infiltration (H and E x 200).

DISCUSSION

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

Poisoning from suicide or unintentional intake of ALP 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 to 24 hours of ingestion, 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 medicolegal deaths of ALP poisoning, aimed to assess autopsy findings of these cases.

Our results showed the higher incidence of ALP poisoning was among cases aged 20 to 30, while older cases were rare.

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

Also, Shaheen et al. (2011) study, which agreed with the current work, explained these observations by younger persons who are easily excited. On the other side, as individuals get older, they take more mature steps.

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

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).

Jain et al. (2005) and Shaheen et al. (2011) coincided with the current observations and reported that Gross examination during postmortem revealed

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 hemorrhages 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. Jain et al. (2005) and Hugar et al. (2014) previously reported similar findings.

In the current study, 70% of cases showed grayish-brown fluid in the stomach, with a distinct odor in 80% of cases. Sloughing of the mucosa was also observed in all cases, particularly in the fungal region. These observations were consistent with those of Ashok et al. (2005), who reported that the stomach was found to contain grayish-brown fluid or pasty material in 58% of cases. The distinct odor was detected in 66% of the studied cases. Sloughing of the mucosa was also observed in all cases, especially in the fungal region. This was explained by the raising and accumulating of phosphine gas vapors in the fundal 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., 2011). 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 five cases (50%) showing necrosis in regions other than the fundus region. Inflammatory cell infiltration was observed up to the muscle layer in seven (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 Jain et al. (2005) and Liang et al. (2020), who discovered severe edema and mild hemorrhaging 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 Jain et al. (2005) and Memiş et al. (2007) 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 they 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, microvacuolization, 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 Jain et al. (2005) and Shaheen et al. (2011) who found that administration of ALP 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, 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

This study believed that these characteristics abnormalities were an indication of PH3 poisoning and that they helped to describe the harmful mechanism of PH3. This work discovered serious damage caused by PH3 in several body systems, including the stomach, duodenum, lung, and kidney, after focusing on the pathological changes in 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 ALP 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 or sell ALP, 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.

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المخلص 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 له هو غاز الفوسفين، فتوفر فوسفيد الألومنيوم بسهولة يؤدي إلى استخدامه كوسيلة انتحار بشكل متزايد مع ارتفاع معدل الوفيات. فغاز الفوسفين يؤدي 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 مؤكدة كيميائياً بتسممها بفوسفيد الألومنيوم وتم فحصها بالتفصيل بشكل كبير وتم صبغ عينات من الأنسجة من مختلف الأعضاء بواسطة الهيماتوكسيلين إيوزين وتم ملاحظتها مجهرياً. بعد التركيز على التغيرات المرضية على الأعضاء الرئيسية وجدنا أضراراً شديدة يسببها غاز الفوسفين في العديد من الأجهزة وخاصة في المعدة والاثني عشر والرئة والكلى. علاوة على ذلك، لاحظنا التتسكس الشديد في أنسجة عضلة القلب والطحال والكبد. الخلاصة. ولذلك اعتبرنا أن هذه التغيرات المميزة إجمالاً هي علامة توحى بالتسمم غاز الفوسفين بسبب الآلية السامة لهذا الغاز وهي (تنشيط الفسفرة المؤكسدة للميتوكوندريا). فنأمل أن يتمكن هذا البحث من تحسين فهم سمية غاز الفوسفين في كل من الطب الشرعي والممارسة السريرية.