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Autopsy, histopathology and nano technological study of the spleen in a case of aluminum phosphide poisoning in India

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Abstract
Introduction: Phosphine poisoning occurs infrequently. As of 1984, 94 cases had been reported in the world literature, with 28 deaths, but its frequency has increased recently. Cases of aluminum phosphide poisoning were first reported in India in 1980. Before then poisoning by
aluminum phosphide in India was unknown. These poisoning are mostly suicidal.
Material and Method: A 40 year old male resident of Phulwari Sharif, Patna, INDIA consumed 3 tablets of Aluminum phosphide. The patient was referred to AIIMS Patna higher center for treatment where he died at around 7:00 p.m. His body was kept overnight in AIIMS Mortuary cold chamber. An autopsy was performed at Mortuary of AIIMS Patna, INDIA. Chemical analysis report given by FSL, Patna came out positive for aluminum phosphide. All viscera sent for histopathology to Department of Pathology, AIIMS Patna, India. Spleen was sent for electron microscopic examination to CDRI, Lucknow, India.
Result: The spleen was congested and edematous weighing 140 g; on histopathology, the spleen was markedly congested and showed focal areas of exudation and small hemorrhages with necrosis. Electron microscopy of a cut section of the spleen showed that the sinusoids were dilated and congested, and the splenic mucosa showed hemorrhagic infarction followed by necrosis.
Conclusion: The histopathological report of the section of spleen showed hemorrhagic infarction, which is quite possible in Celphos poisoning, as it causes widespread organ damage with congestion and petechial hemorrhages, as well as edematous organs. Splenic necrosis is a sequela of infarction of the spleen. Sinusoidal dilatation of the spleen is due to its edema and the hypoxic damage002E.

Keywords: Electron microscopy, sinusoids hemorrhagic infarct, petechial hemorrhage, aluminum phosphide

Introduction
Phosphine poisoning occurs infrequently; as of 1984, 94 cases had been reported in the world literature, with 28 deaths, but its frequency has increased recently [1]. Cases of aluminum phosphide poisoning were first reported in India in 1980 [2, 3]. Before then, poisoning by aluminum phosphide in India was unknown. These poisonings are mostly suicidal. Aluminum phosphide is a lethal solid fumigant pesticide, insecticide, and rodenticide; it is a common outdoor and indoor pesticide in India and considered an ideal grain and tobacco preservative.
Aluminum phosphide is available as dark brown or grayish 3-g tablets or 0.6-g pellets with diameters of 20 mm and thicknesses of 5 mm. These are kept in groups of ten and twenty in sealed airtight aluminum containers. Tablets of aluminum phosphide are also referred to as “rice tablets” or “wheat tablets,” with many trade names: Alphos, Bidphos, Celphos, Quickphos, Phosphotex, Phosphume, and Phosphotoxin. Each 3-g tablet has the capacity to liberate 1 g of phosphine, which has a fatal dose of 1–3 g (1 to 3 tablets). Inhalation of phosphine at a concentration of 400 to 600 ppm is fatal within 30 min. Deaths are even caused by exposure to half tablets (i.e., a 1.5-g tablet). The time in which maximum people die when administered fatal dose is 24 h and spans 1–4 days, and mortality is high (reaching 35-100%) [3, 5].

Materials and Methods
A 40-year-old male resident of Phuwari Sharif, Patna who was a property dealer left his home after taking lunch at 2:00 p.m. He was found lying unconscious at 4:00 p.m. on June 26, 2022, 13 km from his residence on empty land that he had sold to his customer. Nearby villagers informed police and relatives.

He was admitted to the local hospital where he gave a history of consuming 3 tablets of Celphos. The patient was referred to AIIMS Patna higher center for treatment where he died at around 7:00 p.m. His body was kept overnight in the AIIMS Mortuary cold chamber. An autopsy was performed the following day. All viscera was sent to Forensic Science Laboratory, Patna for chemical analysis, and samples of the spleen, heart, liver, lungs, and kidneys were sent for histopathology at the Department of Pathology, AIIMS Patna. The spleen was sent to Central Drug Research Institute, Lucknow, India for electron microscopic examination

Results

The autopsy was conducted at 9:25 a.m. on June 27, 2022. No external injuries were noted. Rigor mortis was present all over the body, and post-mortem staining developed and fixed over the back except at pressure areas. Blood-tinged froth was oozing out of both nostrils, and the nails of fingers and toes were cyanosed, as were the tip of nose and the lips. On gross examination, all organs were edematous and congested. There was a garlicky/decaying-fish-like odor at the mouth and nostrils.

The mucosa of the stomach was congested with hemorrhagic spots and contained 10 mL of grayish/brownish pasty material with a slightly offensive garlicky/decaying-fish-like odor. The mucosa and membrane of the small intestines were congested.

The weight of the heart was 380 g, with all chambers containing liquid and blood clots dark brown in color.

The spleen was congested and edematous weighing 140 g; on histopathology, the spleen was markedly congested and showed focal areas of exudation and small hemorrhages with necrosis. Electron microscopy of a cut section of the spleen showed that the sinusoids were dilated and congested, and the splenic mucosa showed hemorrhagic infarction followed by necrosis.

Figure format

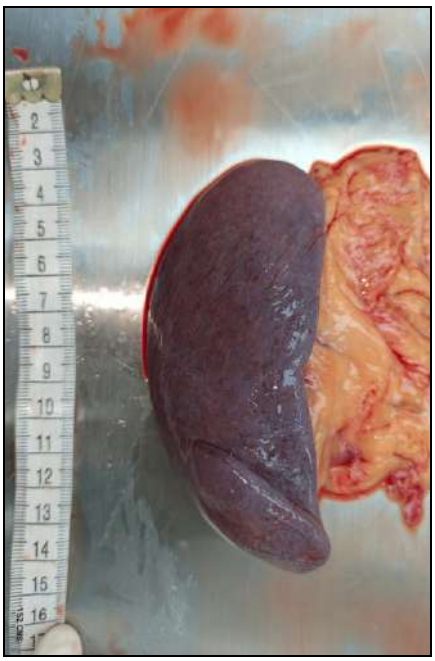

Fig 1: Spleen showing enlargement, edema, intense congestion, and petechial haemorrhage on the external surface.

Fig 2: Cut section of spleen showing intense congestion of spleenic pulp, with the presence of petechial hemorrhage and necrosis of spleen fibromuscular tissue due to aluminium phosphide poisoning.

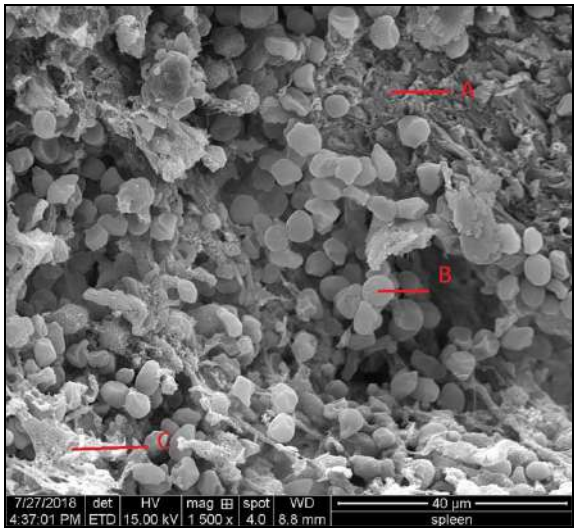

Fig 3: Electron microscope images of (A) necrosis of spleenic mucosa, (B) sinusoidal congeestion, and (C) haemorrhagic and infarction area of spleen.

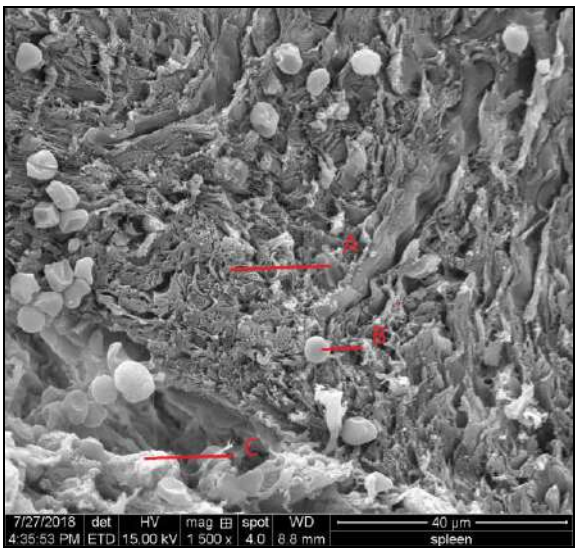

Fig 4: Electron microscope images of (A) Haemorrhagic and infarction area of spleen, (B) sinusoidal congestion and edema, and (C) Necrotic spleenic mucosa.

Discussion

Gray to grayish-brown fluid or pasty material may be seen in gastric cavity in 56% cases of phosphine poisoning. This occurs due to hemorrhage with mucosal shedding of gastric mucosa into the contents of the stomach [4]. Post mortem examination reveals severe congestion of the brain, lungs, liver and the heart. Heart cavity was filled with dark blood [6]. The histopathological report of the section of spleen showed hemorrhagic infarction, which is quite possible in Celphos poisoning, as it causes widespread organ damage with congestion and petechial hemorrhages, as well as edematous organs [7]. Hence, marked congestion of the spleen with focal areas of exudation and small hemorrhages [1] during post-mortem examination were noted. Congestion in the spleen is apparent in 82% of aluminum phosphide poisoning cases. Splenic necrosis is seen in 20% of cases [2]. Splenic necrosis is a sequela of infarction of the spleen. Sinusoidal dilatation of the spleen is due to its edema and the hypoxic damage caused by aluminum phosphide poisoning [8]. Therefore, hemorrhagic infarction of the spleen with sinusoidal dilatation and enlarged spleen due to edema is quite a possibility in aluminum phosphide poisoning. Bleeding diathesis or hemorrhagic infarction of the spleen may also be caused by widespread capillary damage [3]. No external injury was noted on the body of the deceased, which indicates that the manner of death in this case was suicidal in nature, as struggle marks are seen in homicidal poisoning. The manner of death was reported to be suicidal in 100% of the cases by a study done in 2005 [2]. As celphos is very cheap and easily available and it causes rapid death hence it is also called as an ideal suicidal poison [7]. The liver, lungs, and kidneys were congested, as there was widespread hypoxic organ damage with congestion and petechiae [5, 8].

Conclusion

Mortality is high (35-100%) in moderate and severe aluminum phosphide poisoning due to ingestion. Human toxicity occurs due to ingestion of aluminum phosphide (Most common mode), inhalation (Uncommon), or even after absorption through the skin (Rare). Rapid death of the victim after consumption of aluminum phosphide tablets happens due to refractory cardiogenic shock as a result of hemorrhagic myocardial lesions (Mostly subendocardial hemorrhages). Refractory cardiogenic shock is the most common cause of death and occurs within 24 hours, causing death in 30-40% of aluminum phosphide poisoning victims. In a small number of cases, splenic damage may contribute to cause of death, as there is mainly hemorrhagic infarction of the spleen, which can lead to severe blood loss, shock, and hypotension. These findings point to death due to consumption of Celphos tablets (Aluminum phosphide) and further release of phosphine gas, which is its (Aluminum phosphide) active principle and is responsible for widespread hypoxic organ damage.

The manner of death in this case was most likely due to suicide, as Celphos is an ideal suicidal poison.

Acknowledgments

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