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Autopsy and histopathological study in the cerebellum in case of aluminium phosphide poisoning

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Abstract

The present study was conducted to study the effect of Aluminium Phosphide on brain. Autopsy sample were collected from the case brought to mortuary of AIIMS, Patna, Bihar. A study was made to observe the gross and Histopathological findings in brain in case of above poisoning. The study was undertaken as the mentioned poison has been the leading cause of suicidal deaths in Northern India for more than a decade and the commonest cause of poisoning in Central India.

The sample was chemically confirmed for the presence of Aluminium Phosphide poisoning. Detailed gross examination was made and the samples were sent for histopathological analysis to Department of Pathology, AIIMS, Patna.

Keywords: Cerebellum, aluminium phosphide, autopsy and histopathological

Introduction

The agricultural background of India has been in a need of grain preservatives for storage of grains to feed the alarmingly growing population.

Aluminum phosphide is a solid fumigant pesticide, commonly used as a food grain preservative, in India. Use of Aluminium Phosphide products has tremendously increased over last decade due to the increasing need of grain storage.

Aluminum Phosphide (AIP) poisoning has commonly been used as an agent of self-harm in north western India. It is one of the most common methods of committing suicide ^[1].

It is available in 3 gm tablets and pellets. The ingredients of the tablets are Aluminum Phosphide and ammonium carbonate (commonly marketed in India as Celphos and Quickphos tablets). These compounds are cheap, easily available, and effective grain fumigants with little residue. These properties make them ideal for poisoning ^[2].

It is available as dark brown or grayish tablets of 3 grams each and pellets of 0.6 grams, size-diameter 20 mm thickness 5 mm. It is kept in tens and twenties in sealed airtight Aluminium containers. Tablets of Aluminium Phosphide are also referred to as "rice tablets" or "wheat tablets". Trade names- Alphas, Bidphos, Celphos, Quickphos, Phosphotex, Phosphume and Phosphotoxin. Each Tablet weighing 3 gram and has the capacity to liberate 1 gram of Phosphine.

Fatal dose-1-3 grams, 1-3 tablets, 1-2 gm.

Inhalation of phosphine at a concentration of 400 to 600 ppm is fatal within 30 minutes (maximum upto 1 hour). Deaths are caused by even half unexposed tablets i.e. 1.5 gram tablet. Fatal period is 24 hours and ranges 1-4 days. Mortality is high up to (35-100%) ^[2, 3].

Although the cause of death in first 24 hrs is cardiogenic shock, the vital organs are affected due to hypoxic effect of the poison following its consumption ^[4, 5].

The changes in brain has been less reported, although the clinical manifestations need to be take into account when considering the clinical presentations of the poison.

Neurological manifestations following Aluminium Phosphide poisoning

CNS: headache, vertigo, tremors and unsteady gait. It gradually progresses to convulsion, coma and death.

As found by Wilson *et al.*, in cases of acute poisoning, CNS manifestations included headache accompanied with drowsiness, dizziness and paraesthesia.

On physical examination, tremors were reported along with ataxia, convulsion and coma in patients who died [6].

WHO had reported disturbances in taste, smell and dizziness in shipyard workers of Norway?

Further study by Misra *et al.* had reported drowsiness, stupor and delirium, whereas Misra U K *et al.* had reported gastritis, altered sensorium and peripheral vascular failure [7].

Peripheral nervous system

Studies have reported that some patients develop paraesthesia, fatigue and weakness. Peripheral neuropathy has been reported in some cases but no studies of the effects of Phosphine on Peripheral Nervous system have been reported [6, 7].

Autonomic nervous system

The direct toxic effect of Phosphine on Autonomic nervous system, indirectly it can induce tachycardia, hypotension, shock and gastrointestinal disorders.

Case History

A 40 year old male resident of Phulwarisharif, Patna, property dealer by profession, left his home after taking lunch at 2:00 p.m. He was found lying unconscious at 4:00 pm on 26 /06/2022, 13 km from his residence at empty land, sold by him. Nearby villagers informed police and relatives. He was admitted to the local hospital where he gave history of consuming 3 tablets of cephos. Further patient was referred to AIIMS Patna, higher centre for treatment where he died at around 7:00 pm. His body was kept overnight in cold chamber. Autopsy was performed the following day at AIIMS Mortuary, Department of FMT, AIIMS Patna.

The autopsy samples were collected from the above mentioned case brought for post-mortem and sent for histopathological analysis to Department of Histopathology, AIIMS, Patna. On the basis of above reports, case report was prepared.

Results

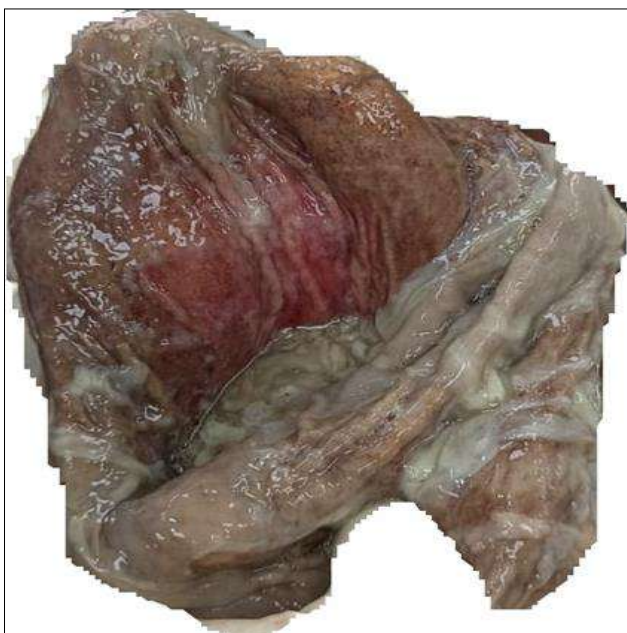


Fig 1: Stomach mucosa showing congestion along with areas of necrosis



Fig 2: Spleen showed sinusoidal dilation with red pulp exudation.



Fig 3: Heart had no remarkable histological presentation

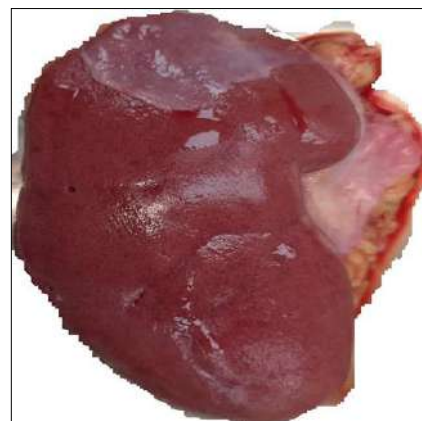


Fig 4: Kidney showed unremarkable presentation histologically along with few congested blood vessels.

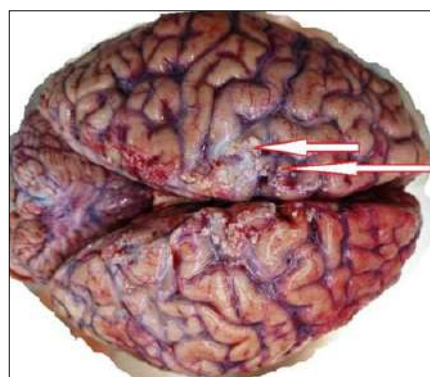


Fig 5: On removal of brain, marked congestion was noted along with edema and necrotic patches.

Discussion

According to the book, Essentials of Forensic Medicine by Reddy K S N, the findings in brain include congestion and edema [3]. Same has been reported by Aggarwal A in Textbook of Forensic Medicine and Toxicology along with disorganisation of different layers and degeneration of neurons. Paucity of glial cells and appearance of necrotic patches have also been reported in the above case of poisoning [2, 3].

Gross findings

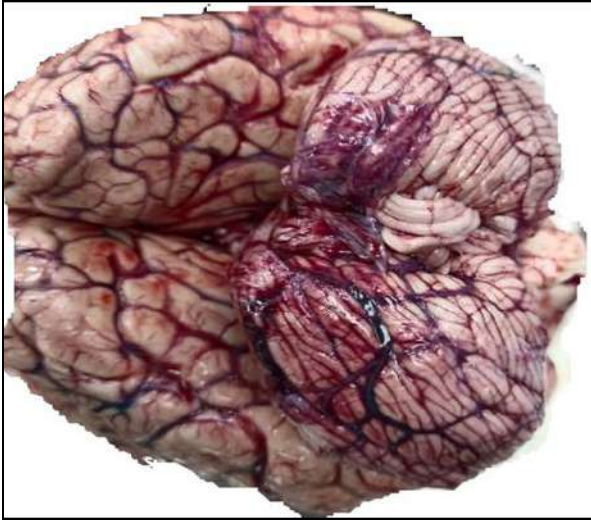


Fig 6: Macroscopically, the brain showed areas of necrotic patches on the surface of cerebellum, due to degeneration of neurons.

Histopathological findings

In case of Aluminium Phosphide poisoning, different grades of cerebral microvascular congestion has been seen. Typical inflammatory presentation in cells is normally absent [8].

As reported by Sinha U S *et al.* brain was found to be congested in most of the cases of AIP poisoning. The microscopic changes in brain included congestion and coagulative necrosis [9].

The histopathological findings included disorganization of different layers in cerebral cortex along with round shaped neurons having a convex border. The other major findings included degenerated Nissl granules in the cytoplasm and degenerated eccentric nucleus.

Further, the findings in the cerebellar cortex included degenerated neurons, the infiltration of round cells into the molecular layer and the disappearance of the processes of Purkinje cells.

In the study conducted by Tripathi SK *et al.* in 2007, microscopic examination showed major changes in the cerebral and cerebellar cortex following the consumption of Celphos.

The subcortical zone of the brain revealed a paucity of glial cells, degeneration of nerve fibres and the appearance of necrotic patches [10].

Omid Mehrpour has reported mild capillary dilation and congestion of cortex in histopathological examination of the brain tissue. Degeneration of Nissl granules in the cytoplasm along with intraparenchymal and subarachnoid hemorrhage (SAH).

Brain showed degeneration of white matter showing cystic vacuoles along with dark shrunken triangular nuclei with haloes around. It was suggestive of cytotoxic edema. [11]

Conclusion

Mortality is high, 35 to 100% in moderate and severe aluminum phosphide poisoning due to ingestion of aluminum phosphide. Human toxicity occurs either due to ingestion of aluminum phosphide (commonest mode), inhalation (uncommon), or even after absorption through skin (rare). In cases of early deaths of the victim after consumption of Aluminum Phosphide tablets which happens due to refractory cardiogenic shock as a result of haemorrhagic myocardial lesions mostly sub endocardial hemorrhages. 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 aluminum phosphide 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 (AIP) unexposed and further release of phosphine gas which is the active principle and responsible for widespread hypoxic organ damage.

The manner of death in this case is most likely due to suicide, celphos comes under an ideal suicidal poison group.

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Not available

Author's Contribution

Not available

Conflict of Interest

Not available

Financial Support

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