Case Report
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A case of acute aluminum phosphide poisoning leading to demyelinating optic neuropathy

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Abstract

Aluminum phosphide has been known, to be a major cause of non-accidental poisoning all over the world. The most common clinical manifestations being gastrointestinal and cardiovascular. Ocular manifestations have not been documented in the literature. We report a unique and rare presentation of aluminum phosphide poisoning. A 16 year old girl was brought with alleged ingestion of aluminum phosphide tablet and, developed redness and decreased vision in both the eyes. Visually Evoked Potential was suggestive of demyelinating optic neuropathy. She was treated with eye padding and eye drops to prevent dryness. Recovery and improvement in vision was seen after 5-6 days of ingestion. Aluminum phosphide poisoning may cause demyelinating optic neuropathy as a rare complication. In the presence of ocular symptoms, further ophthalmological and neurological evaluation is recommended. It is a diagnosis of exclusion and other possible etiologies must be ruled out before making the diagnosis.

Keywords: Aluminium phosphide; demyelinating optic neuropathy

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1. Introduction

Aluminum phosphide is a common household chemical used for grain preservation in developing countries. It is one of the most common causes of non-accidental poisoning in children and its estimated incidence in children in India is 9.14% which comprises a significant group. (1)The most common clinical manifestations following ingestion include nausea, restlessness, abdominal pain, palpitation, arrhythmias and shock.(2)In this article we discuss a rare manifestation of aluminum phosphide ingestion i.e. demyelinating optic neuropathy, not previously described in literature.

2. Case History

A 16 year old girl hailing from a rural household in Karnataka, India was brought with alleged consumption of 1 tablet of *(QUICKPHOS*) aluminum phosphide (56%). She was brought to medical attention within 1 hour of ingestion, managed by gastric lavage with normal saline, Vitamin k and dexamethasone, referred for complaints of progressive redness and decreased vision in both the eyes along with drooling of saliva and difficulty to swallow. It was accompanied by retrosternal burning pain. Medical, family, and psychosocial history was unremarkable. Seen at our tertiary care hospital 36 hours post ingestion, her vital parameters were normal.Systemic examination revealed altered sensorium, sluggishly reacting pupils, optic disc of left eye was hyperemic, fine crepitation were present all over lung fields, and epigastric tenderness. Redness of both the eyes associated with photophobia were present. Visual acuity at presentation was counting fingers at 1 meter.

Her blood counts, renal functions, liver functions, coagulation profile and Electrocardiography were normal. Chest x ray revealed normal lungs and heart. 2D ECHO done showed normal ejection fraction. Ophthalmic evaluation suggested a differential of demyelinating optic neuropathy or retro bulbar neuritis. Magnetic resonance

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imaging of the brain and spine were normal. Visual Evoked potential of both the eyes showed prolonged latencies of p100 in both the eyes as shown in figure -1 and figure -2, suggestive of demyelinating optic neuropathy.

Intravenous dexamethasone was continued at a dose of 18mg/m^2/day; magnesium sulphate for the first 72 hours, along with Amoxicillin, sodium bicarbonate, esomeprazole, and eye padding. Dexamethasone given initially for 5 days and then gradually tapered off in 2 weeks. Ocular symptoms resolved by 7th day with a visual acuity of counting fingers at 3 meter and no residual visual loss. Psychiatric counselling was given and at discharge child was normal with visual acuity of 6/6.

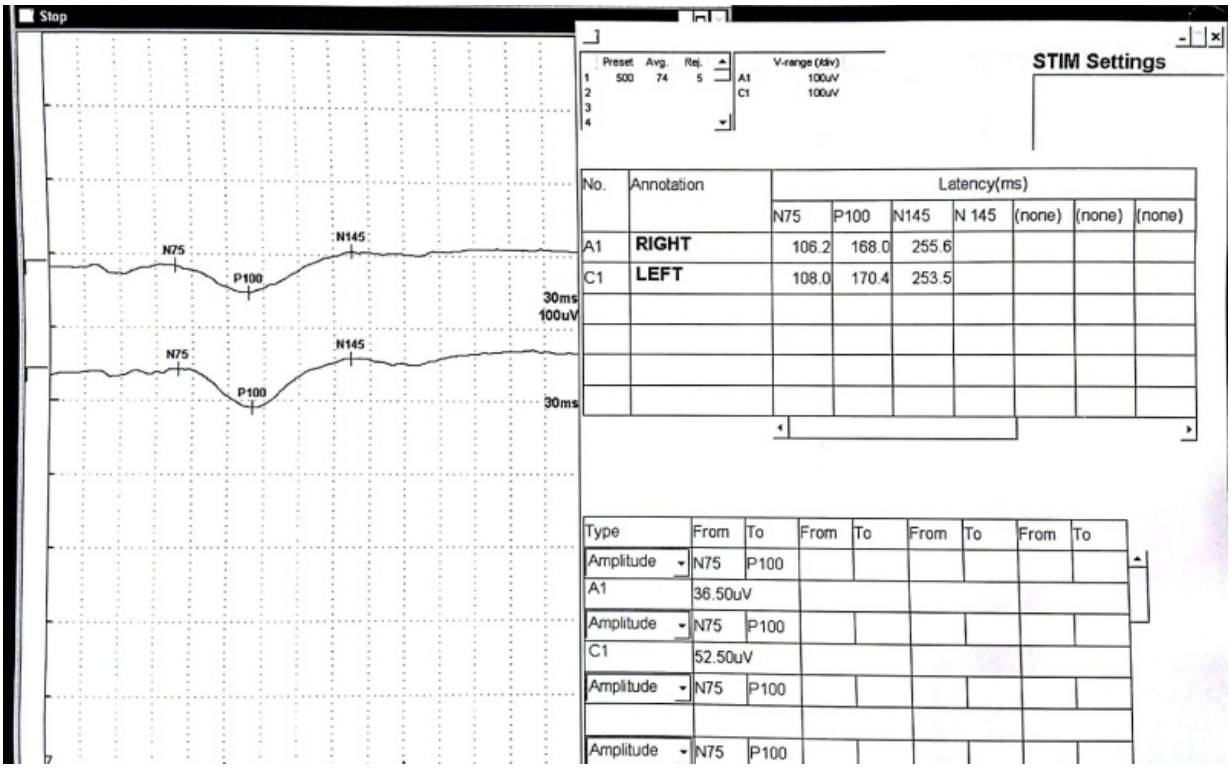


Figure 1. Visually evoked potential showing prolonged p100.

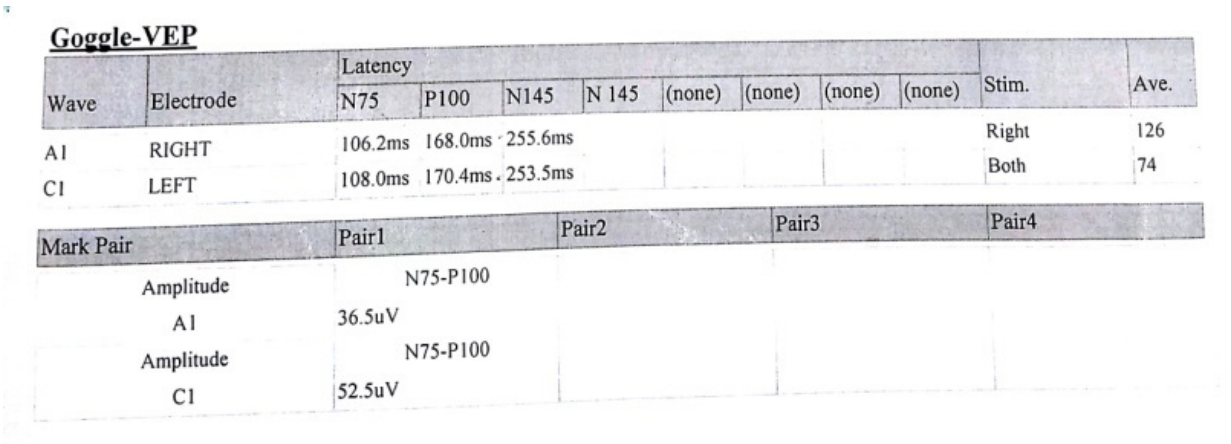


Figure 2. VEP showing prolonged p100.



Figure 3. Poison ingested by the patient.

3. Discussion

Aluminum phosphide is a fumigant extensively used since 1940's for indoor fumigation of agricultural compounds and commonly known as "rice tablet".(3) It is available as tablets or pellets in trade names like *Celphos, Quikphos, synfume and phosfume.* The tablets on exposure to moisture release phosphine gas which has a fishy odor, is detectable and is easily absorbed. Phosphine inhibits mitochondrial cytochrome oxidase and oxidative phosphorylation.(4) In presence of aluminum phosphide, cellular superoxide and peroxide radicals are generated, with subsequent cellular damage by lipid peroxidation.

Aluminum phosphide poisoning is a common mode of suicide in the agricultural community in northern India with an incidence of 11.82% with an overall mortality of 53.2% in affected individuals.(5) Since it is easily available, cheap and its use being unregulated, it remains to be one of the most common household poisons.

Poisoning with metal phosphides cause nausea, restlessness, abdominal pain, palpitation, pulmonary edema, cyanosis, hypotension, shock and cardiac arrhythmias.(6) Other rare effects include hepatitis, acute tubular necrosis, disseminated intravascular coagulation and respiratory alkalosis. The inhalation of phosphine gas causes diarrhea, cold and clammy sweats, tremors, convulsions, delirium, coma and death from respiratory and cardiac arrest. The other complications in acute aluminum phosphide poisoning are hepatic failure, renal failure, cardiac failure, clotting abnormalities and pleural effusion. Ocular manifestations following aluminum phosphide has not been described so far. (7)

Treatment with oral activated charcoal, sorbitol suspension or sodium bicarbonate solution is recommended.(8) Intravenous administration of magnesium sulfate, sodium bicarbonate and calcium gluconate is also an alternative.(8) Specific therapy with intravenous magnesium sulfate is also recommended. Hydrocortisone 200–400 mg every 4–6 h is given intravenously to combat shock, reduce the dose of dopamine, check capillary leakage in lungs (ARDS) and to potentiate the responsiveness of the body to endogenous and exogenous catecholamines.(9) No known specific antidote is however, available for metal phosphide poisoning. Metabolic acidosis must be treated by administering sodium bicarbonate and shock should be treated with appropriate vasopressors.(6)

Demyelinating optic neuropathy has been previously described in the literature with methanol, tobacco or alcohol amblyopia, characterized by bilateral involvement. (10) Visual evoked potentials can be used to diagnose optic nerve involvement but do not distinguish between different acute optic neuropathies, although a delayed but well preserved P100 waveform is characteristic of demyelination. Visual evoked potentials with pattern electroretinograms can distinguish between optic nerve and macular pathology.

The patient in our case was managed as described in previous studies with high dose dexamethasone and magnesium sulphate and other supportive measures. Improvement was seen over 5 days gradually with no other significant complications.

4. Conclusion

Aluminum phosphide poisoning may cause demyelinating optic neuropathy as a rare complication and maybe anticipated. In the presence of ocular symptoms, further ophthalmological and neurological evaluation is recommended. It is a diagnosis of exclusion and other

possible etiologies must be ruled out before making the diagnosis.

5. Key Messages

- Aluminum phosphide poisoning is the most common cause of non-accidental poisoning in the developing world with a high case fatality.
- Demyelinating optic neuropathy can be a rare clinical manifestation following its ingestion.
- There is no antidote at present and treatment is mainly supportive.

6. Conflict of Interest

None Declared

7. References

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