

Case Report



Accommodative spasm as the main manifestation of topical eye contact with insecticide

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Abstract

The patient is a 23-year-old Caucasian male farmer who, after topical eye contact with an insecticide, developed accommodative spasm and blurred vision in one eye. He was treated with frequent doses of 2% homatropine drop and recovered within a week.

Introduction

Organophosphate is a common name for phosphoric acid esters. Organophosphate compounds are produced and used in two forms: insecticides and war chemical agents (e.g., sarin nerve agent). These toxins act on neurons and disrupt cholinesterase activity at synapses (lack of degradation of acetylcholine in parasympathetic terminals).¹

In exposure to high doses of organophosphate as in the gases used in the chemical warfare, victims suffer from shortness of breath, diarrhea, nausea, abdominal pain, increased salivation, tearing, and sweating.²

However, in contact with much lower doses of organophosphate derivatives, like diluted pesticides or insect repellents, the symptoms resemble asthma attacks in susceptible people and in local contact, that part of the body becomes inflamed, red and irritated. In long term exposure, organophosphates can cause anxiety, depression and peripheral neuropathy.³

Accommodation in the eye is an increase in the refractive power of the crystalline lens that makes it easier to see close objects. To do this, the ciliary muscle of the eye is stimulated by the short ciliary nerves (of parasympathetic origin of the oculomotor nerve), and its contraction causes the relaxation of zonular fibers on peripheral sides

of the crystalline lens and its subsequent deformation to become thicker and thus creating more refractive power.⁴

Accommodative spasm (also known as ciliary spasm) is a continuous contraction of the ciliary muscle and an increase in the refractive power of the lens crystal, resulting in a change in the refractive state of the eye towards myopia. This condition can be resulted due to intraocular inflammation, diseases of the central nervous system like MS, head trauma, and drugs with cholinergic or psychogenic effects. The condition is often accompanied by blurred vision, ocular refraction change towards myopia, and pain.⁵

Case Presentation

The patient is a 23-year-old Caucasian male farmer whose left eye was accidentally contacted with an insecticide while spraying trees. After two hours, he had developed pain and blurred vision and had been referred to an ophthalmological emergency clinic. The patient was generally well and had no symptoms of shortness of breath or systemic symptoms.

Irrigation of the ocular surface with sterile solution and cleaning was done. Vision of the right eye was 20/20 and examination with a slit lamp did not show any abnormal findings. Examination of the left eye revealed miosis and

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some redness and mucous secretions indicating mild irritation of the ocular surface, but examining both eyes no relative afferent pupillary defect (RAPD) was observed. The patient also had no defects in the epithelium of the ocular surface or other signs of eye burn (Figure 1). The visual acuity of the left eye was 20/100, which had a refraction of -5 in retinoscopy, and by applying refraction, the vision improved to 20/25.

The patient was treated with repeated doses of 2% homatropine drop, lubricant, artificial tears and a low dose of topical corticosteroid drops. At the first follow-up examination, three days later, redness and eye pain had been disappeared and refraction was -1, uncorrected visual acuity was 20/30 which was improved with applying refraction to 20/20. At the second follow-up examination, seven days later, all symptoms had been resolved and the patient had uncorrected visual acuity of 20/20.

Discussion

Ocular symptoms caused by insecticides generally include irritative symptoms and cholinergic symptoms including miosis, tearing, and accommodation problems.⁶ Affecting the nerves and the central nervous system, they can also cause abnormal eye movements, diplopia, nystagmus and balance disorders.⁷⁻⁹

In farmers with chronic exposure to pesticides retinopathy has been reported.¹⁰ Optic neuropathy, vertical pursuit deficit and myopia have also been observed as the adverse effects of chronic exposure to such pesticides.¹¹ Also, in Japan high exposure to organophosphates is linked to advanced visual disease syndrome described as Saku disease, an optico-autonomic peripheral neuropathy which affects nerves of ocular region resulting in pupillary dysfunction, ocular movement problems and optic nerve



Figure 1. Miosis, fine injection of the conjunctiva and increase in tear after topical exposure of the eye with insecticide.

malfunction.¹²

Ciliary spasm is a part of broad spectrum of complications of warfare agents like sarin which are organophosphates having significant cholinergic effect.^{6,13} However, the victims experience many other critical life-threatening problems including respiratory failure that accommodative spasm would not be one of their major problems at the early phase of the exposure. At the low doses of poisoning with organophosphates, irritating symptoms and eye discomfort are usually predominant. Ciliary spasm in these patients has two major differences from previous cases in the medical literature. First, it was caused by a diluted insecticide rather than strong chemical warfare agents; second, it was patient's chief complaint without systemic problems nor severe chemical conjunctivitis.

Conclusion

This case suggests that accommodative spasm may be the main manifestation of topical eye contact with insecticides.

Authors' Contribution

SK performed a literature review, developed the study structure, and approved its final version for the intellectual content. Other authors helped in drafting the manuscript.

Conflict of Interest

There is no conflict of interest in any form.

Ethical Approval

Informed consent was obtained from the patient for publication of this report.

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References

1. Kwong TC. Organophosphate pesticides: biochemistry and clinical toxicology. *Ther Drug Monit.* 2002;24(1):144-9. doi: [10.1097/00007691-200202000-00022](https://doi.org/10.1097/00007691-200202000-00022).
2. Rusyniak DE, Nañagas KA. Organophosphate poisoning. *Semin Neurol.* 2004;24(2):197-204. doi: [10.1055/s-2004-830907](https://doi.org/10.1055/s-2004-830907).
3. Blain PG. Adverse health effects after low level exposure to organophosphates. *Occup Environ Med.* 2001;58(11):689-90. doi: [10.1136/oem.58.11.689](https://doi.org/10.1136/oem.58.11.689).
4. Glasser A. Accommodation: mechanism and measurement. *Ophthalmol Clin North Am.* 2006;19(1):1-12. doi: [10.1016/j.ohc.2005.09.004](https://doi.org/10.1016/j.ohc.2005.09.004).
5. Lindberg L. [Spasm of accommodation]. *Duodecim.* 2014;130(2):168-73.
6. Gore A. Broad spectrum treatment for ocular insult induced by organophosphate chemical warfare agents. *Toxicol Sci.* 2020;177(1):1-10. doi: [10.1093/toxsci/kfaa095](https://doi.org/10.1093/toxsci/kfaa095).
7. Hata S, Bernstein E, Davis LE. Atypical ocular bobbing in acute organophosphate poisoning. *Arch Neurol.* 1986;43(2):185-6. doi: [10.1001/archneur.1986.00520020071024](https://doi.org/10.1001/archneur.1986.00520020071024).
8. Pullicino P, Aquilina J. Opsoclonus in organophosphate poisoning. *Arch Neurol.* 1989;46(6):704-5. doi: [10.1001/archneur.1989.00520420126037](https://doi.org/10.1001/archneur.1989.00520420126037).
9. Jay WM, Marcus RW, Jay MS. Primary position upbeat

- nystagmus with organophosphate poisoning. *J Pediatr Ophthalmol Strabismus*. 1982;19(6):318-9. doi: [10.3928/0191-3913-19821101-09](https://doi.org/10.3928/0191-3913-19821101-09).
10. Jaga K, Dharmani C. Ocular toxicity from pesticide exposure: a recent review. *Environ Health Prev Med*. 2006;11(3):102-7. doi: [10.1265/ehpm.11.102](https://doi.org/10.1265/ehpm.11.102).
 11. Ishikawa S, Miyata M, Aoki S, Hanai Y. Chronic intoxication of organophosphorus pesticide and its treatment. *Folia Med Cracov*. 1993;34(1-4):139-51.
 12. Dementi B. Ocular effects of organophosphates: a historical perspective of Saku disease. *J Appl Toxicol*. 1994;14(2):119-29. doi: [10.1002/jat.2550140214](https://doi.org/10.1002/jat.2550140214).
 13. Egoz I, Nili U, Grauer E, Gore A. Optimization of the ocular treatment following organophosphate nerve agent insult. *Toxicol Sci*. 2017;159(1):50-63. doi: [10.1093/toxsci/kfx119](https://doi.org/10.1093/toxsci/kfx119).