

Bilateral Optic Disc Edema in a Patient with Lead Poisoning

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Dear Editor,

In their important contribution as a “Case Report”, Abri Aghdam et al^[1] described a man with bilateral optic disc edema due to lead (Pb) poisoning and complicated by opium addiction.^[1] This is an excellent description of papilledema induced by Pb overexposure,^[1] which is a rare but serious optic nerve damage attributed to systemic Pb toxicity.^[2, 3]

However, we would like to emphasize the role of whole-blood in diagnosing the Pb poisoning. The patient’s serum contained very high levels of Pb, which was 164 µg/dL.^[1] In humans, the normal blood Pb level is zero.^[4, 5]

We wonder whether whole-blood Pb concentrations were determined. In our view, the serum is not the primary and proper indicator medium as a biomarker of Pb exposure. In fact, serum alone (with no red blood cells) does not adequately reflect the 2% of the total body burden of the Pb, which is found in the circulating whole-blood.^[4, 5]

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In toxicology, the whole-blood Pb concentrations have been used in conjunction with urinary Pb levels as a primary measure of Pb exposure in humans.^[6]

Toxicological studies suggest that exposure to Pb during developmental periods may lead to long-term visual deficits both in *in vitro* and in animal models.^[7]

Toxic optic neuropathy may be the unique clinically significant alteration in patients with Pb poisoning.^[2, 3] Fortunately, Pb poisoning is a rare circumstance not commonly encountered by ophthalmologists.^[8–11] Consistent with this notion, Pb and other toxic metals (i.e., organic mercury, thallium) are to be considered “neurotoxicants”, primarily due to toxic effects on the optic nerve.^[12–14]

With regard to the issue of toxic optic neuropathy, over the past two decades, we have noticed only one case in which papilledema was associated with overexposure to nickel salts. Their interesting case report^[1] reminds us that Pb poisoning is a topic of growing interest among ophthalmologists^[15] and conveys the fact that the eyes can be injured due to Pb intoxication.

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Conflicts of Interest

There are no conflicts of interest.

REFERENCES

1. Abri Aghdam K, Zand A, Soltan Sanjari M. Bilateral optic disc edema in a patient with lead poisoning. *J Ophthalmic Vis Res* 2019;14:513–517.
2. Boudouresques J, Guillot P. [Papilledema, solitary manifestation of lead poisoning]. *Mars Med* 1957;94:693–695.
3. Viaud M, Greau H, Colas J, Baron A, Lhermitte R. [Lead poisoning encephalopathy with papilledema; value of treatment with chelating agents; report of a case]. *Rev Otoneuroophthalmol* 1958;30:191–198.
4. Guzzi G, Spadari F, Bombeccari GP, Pigatto PD. Maxillofacial gunshot wounds and diagnostic tests for lead in the blood. *Br J Oral Maxillofac Surg* 2017;55:105.
5. Pigatto PD, Ronchi A, Guzzi G. Iron overload, G6PD deficiency, and lead levels on blood smears. *Int J Hematol* 2016;103:724.
6. Casarett LJ, Doull J, Klaassen CD. Casarett and Doull's toxicology: the basic science of poisons. 6th ed. New York: McGraw-Hill Medical Pub. Division; 2001. p. xix, 1236.
7. Fox DA, Kala SV, Hamilton WR, Johnson JE, O'Callaghan JP. Low-level human equivalent gestational lead exposure produces supernormal scotopic electroretinograms, increased retinal neurogenesis, and decreased retinal dopamine utilization in rats. *Environ Health Perspect* 2008;116:618–625.
8. Citirik M, Acaroglu G, Mutluay AH, Zilelioglu O. Lead poisoning: report of a case. *Ann Ophthalmol* 2004;36:32–36.
9. Gilhotra JS, Von Lany H, Sharp DM. Retinal lead toxicity. *Indian J Ophthalmol* 2007;55:152–154.
10. Nagpal AG, Brodie SE. Supranormal electroretinogram in a 10-year-old girl with lead toxicity. *Doc Ophthalmol* 2009;118:163–166.
11. Sharma P, Sharma R. Toxic optic neuropathy. *Indian J Ophthalmol* 2011;59:137–141.
12. Grzybowski A, Zulsdorff M, Wilhelm H, Tonagel F. Toxic optic neuropathies: an updated review. *Acta Ophthalmol* 2015;93:402–410.
13. Saldana M, Collins CE, Gale R, Backhouse O. Diet-related mercury poisoning resulting in visual loss. *Br J Ophthalmol* 2006;90:1432–1434.
14. Pamphlett R, Kum Jew S, Cherepanoff S. Mercury in the retina and optic nerve following prenatal exposure to mercury vapor. *PLoS One* 2019;14:e0220859.
15. Phelps J. Headliners: lead exposure and vision. *Environ Health Perspect* 2005;113:A163.