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Optic Neuropathy Related to Hydrogen Peroxide Inhalation

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Abstract

Optic neuropathy related to toxins is a complex, multifactorial disease potentially affecting individuals of all ages. We report a case of presumed toxic optic neuropathy secondary to H_2O_2 exposure. This has not been previously reported, and the temporal relationship of the exposure to the optic neuropathy is compelling, although not definite, evidence of a causal relationship.

Key Words: optic neuropathy, hydrogen peroxide

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Toxins, such as arsenic, and adverse reaction of some drugs such as ethambutol, phenytoin, cisplatin, and chloramphenicol may damage optic nerve.[1–5] Withdrawal of the toxic agent or early treatment may recover the vision or prevent the visual loss.[1,6] In this case report, we present a single case of optic neuropathy after hydrogen peroxide (H_2O_2) inhalation. Up to date, some studies of experimental optic neuritis have shown the toxic effects of H_2O_2 to the optic nerve, but to our knowledge, no human has developed optic neuropathy.

CASE

We report a 51-year-old jeweller man in present case. He had broken the bottle fully filled with H_2O_2 and inhaled it during 15 minutes. After complaints of burning of eyes and watery eyes, blurred vision have developed until he could see only shadows within 2 hours. Three days later, he was brought to our medical attention complaining of bilateral visual loss. He had no family history or biography in relation to disorders. At the time of our initial consultation, he said that he was only able to see shadows in both eyes. Neuro-ophthalmological examination revealed visual acuity of 2/10 in both eyes. The direct and consensual pupillary light reflexes were decreased, whereas the extrinsic ocular motility was normal. A campimetric study showed visual loss in all fields prominent in central areas of both eyes. Color perception was impaired. Fundus examination demonstrated swelling of the optic discs. Other neurological examination findings were normal. Routine biochemical and microbiological blood examinations, and serum levels of B12 were normal. The patient was initially given the diagnosis of bilateral optic neuropathy and, he has been submitted to the following tests in the ophthalmology unit: (1) visual campimetry, which showed marked decrease of retina sensitivity in both eyes; (2) cerebrospinal fluid examination revealed normal results; (3) visual evoked potential showed conduction disturbances, and it was unobtainable; and (4) retina angiofluorescein showed tortuous retinal vessels. When he was admitted to our unit, we decided to perform muscle biopsy (left brachial biceps muscle), with the intention of searching for abnormalities suggestive of mitochondrial cytopathy, such as the presence of ragged-red fibers. The results were normal. Our clinical suspicion of Leber hereditary optic neuropathy remained high, and a blood sample was therefore submitted to genetic analysis in relation to the principal mutations of this disorder. Superoxide dismutase test was normal. Results of cranial magnetic resonance imagings of the skull and both optic nerves were normal.

The patient underwent pulse therapy using methylprednisolone 1 g/d for 5 days and maintenance therapy of prednisone 80 mg/d. He showed visual improvement (visual acuity of 2/10 at right eye and seen fingers from 5 m at left eye) at the sixth day of therapy, and he showed similar visual acuity findings and bilateral optic atrophy at the 30th day examination in comparison with 6th day examination. Prednisolone therapy was stopped. During follow-up, he has been medicated using vitamin B complexes to improve cellular adenosine triphosphate usage.

DISCUSSION

It was evident that optic neuropathy was clearly due to the H_2O_2 inhalation, but we also performed muscle biopsy as well as genetic

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analysis to perform differential diagnosis from mitochondrial myopathy or Leber optic neuropathy which are common causes of bilateral vision loss in younger ages, respectively. Painless, bilateral, and insidious loss of central vision should alert the physician to the possibility of a nutritional, toxic, or iatrogenic optic neuropathy.[6] A diagnosis of Leber hereditary optic neuropathy should be suspected whenever young males develop bilateral visual loss, usually sequential, with a positive familial history.[7] DNA mutation analysis is available to detect genetic abnormalities.[8,9] We excluded the diagnosis of Leber optic atrophy and multiple sclerosis via the genetic analysis and cranial magnetic examinations.

Hydrogen peroxide is a strong oxidant that can diffuse in the perivascular space and induce peroxidation of lipids in axonal membranes and myelin in the interstitial optic nerve.[10,11] The H_2O_2 and/or hydroxide radical generated from perivascular H_2O_2 discharged by phagocytic mononuclear cells may play a role in the pathogenesis of demyelination.[11] The H_2O_2 and/or hydroxide radical accumulation in the optic nerve head and the optic nerve sheath appeared to provide a reservoir for diffusion of H_2O_2 into the retrobulbar optic nerve and adjacent perineural nerve contributing to the frequent predilection for optic nerve involvement in experimental allergic encephalomyelitis.[12] Using a modified cerium method, Guy et al[13] found that electron-dense cerium-derived H_2O_2 reaction product was localized in the perivascular space and in the optic nerve head in the presence of intravascular leukocytes. The perivascular and intravascular distribution of cerium-derived reaction product suggests that H_2O_2 may play role in the pathogenesis of altered vascular permeability in experimental optic neuritis and supports observations of suppression of blood-brain barrier permeability by detoxification of H_2O_2 with the exogenous administration of antioxidant enzymes.[13] To our knowledge, in the literature, there is not a human with optic neuropathy caused by H_2O_2. It was interesting also why the optic nerves were selectively involved in present case.

In the present case, there was generalized depression of visual fields—this itself is fairly nonspecific and can accompany visual loss from many causes. The defect was prominently central, patterns more typical for optic neuropathy in general and toxic optic neuropathy in particular. On the orbital magnetic resonance imaging, there was neither increased T2 signal in the optic nerves which was commonly seen in acute demyelination, whether from inflammatory or toxic causes nor presence of enhancement. Because of all these reasons, we think that our case is the first clinical report showing the same evidence of a causal relationship between optic neuropathy and H_2O_2 revealed in experimental studies, although not definite, and optic nerve demyelination was not seen in magnetic resonance imaging.

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