

Hyperbaric oxygen therapy and optic neuritis: Case report and literature review

S. David Register M.D.¹, Maria E. Aaron M.D.², Helen B. Gelly M.D.³

¹ Hyperbaric Physicians of Georgia, Inc., Atlanta, Georgia; now at Baptist Health Medical Center, North Little Rock, Arkansas USA

² Department of Ophthalmology, Emory University School of Medicine, Atlanta, Georgia USA

³ HyperbaRxs, LLC, Atlanta, Georgia USA

CORRESPONDING AUTHOR: Dr. Dave Register – aslandev@aol.com

ABSTRACT

A history of optic neuritis has long been considered a relative contraindication to hyperbaric oxygen therapy [1,2]. However, the published medical literature regarding the use of hyperbaric oxygen therapy in patients with previous optic neuritis is very limited, and patients who might benefit from hyperbaric oxygen therapy may be denied its benefit without adequate consideration of the risk-benefit ratio. We present a case report of a patient with a history of optic neuritis who underwent comprehensive ophthalmologic evaluation before and after 40 treatments with hyperbaric oxygen therapy, with no detectable ophthalmologic deficit.

INTRODUCTION:

*First get your facts;
then you can distort them at your leisure.*

– Mark Twain

In 1969 Nichols *et al.* [3] reported a single patient with transient unilateral diminution of vision after exposure for six hours to 2.0 atmospheres (ATA) of inspired oxygen during an experimental protocol studying pulmonary tolerance of oxygen in human subjects. The patient, a 21-year-old male, had a history of unilateral retrobulbar optic neuritis. Following his hyperbaric oxygen therapy he was diagnosed with recrudescence of unilateral retrobulbar optic neuritis. This was an extreme exposure to hyperbaric oxygen (HBO₂) and one that would not be experienced in current HBO₂ treatment regimens. Additionally, optic neuritis is known to recur. However, this single case report has been repeatedly cited as evidence that patients with a history of optic neuritis are at increased risk of hyperbaric oxygen-induced optic neuritis with subsequent permanent visual impairment.

Often overlooked is the fact that many patients with multiple sclerosis (MS) have undergone hyperbaric oxygen therapy as treatment for their MS. Since optic neuritis is a common manifestation of MS, numerous patients with a history of optic neuritis (and patients with active optic neuritis) were treated with hyperbaric oxygen therapy without reports of new-onset visual impairment. Moreover, hyperbaric oxygen has been used as therapy

for radiation-induced optic neuropathy [4,5]. HBO₂ has also been found to be of proven or likely benefit in a number of ischemic disorders of the retina and optic nerve [10,11].

This case report is not intended to imply that a patient history of optic neuritis should not be sought before beginning hyperbaric therapy. Instead, it is intended to show that the medical literature may not support an automatic exclusion of a patient with a history of optic neuritis if there is an indication for hyperbaric oxygen therapy. Ophthalmologic evaluation of the patient, both before and after hyperbaric oxygen therapy, is recommended.

CASE REPORT

M.P. was a 35-year-old female recently diagnosed with malignant neoplasm of the right breast. She underwent bilateral mastectomies with simultaneous reconstruction via latissimus dorsi flaps and tissue expanders, followed by both radiation therapy and chemotherapy. She subsequently developed infection of the tissue expander of the right breast, which required surgical removal of the implant and administration of intravenous antibiotics. She was then referred for hyperbaric oxygen therapy in an effort to salvage ischemic, edematous and marginally viable breast tissue compromised by radiation, infection and surgery. Her past medical history included multiple sclerosis with a history of bilateral optic neuritis 10 years earlier, attention deficit disorder and chronic pain syndrome.

The neuro-ophthalmologist who had cared for the patient 10 years earlier did not wish to make any recommendations regarding the advisability of hyperbaric oxygen therapy. A second opinion was sought and resulted in a complete ophthalmologic evaluation before and after hyperbaric therapy. Ophthalmologic evaluation prior to the initiation of hyperbaric therapy revealed that the patient was severely myopic but had a best-corrected visual acuity of 20/40 in both eyes. She had a normal pupillary exam, with no evidence of pupillary defect, a normal anterior segment examination, normal color vision and a dilated fundus examination that revealed some mild temporal pallor in the right optic nerve and lattice degeneration inferiorly in the left eye. Automated visual field testing was performed and demonstrated a mildly enlarged blind spot in the right eye and some inferior nasal depression in the left eye.

During the course of hyperbaric oxygen therapy, which consisted of 40 treatments of oxygen at 2.4 ATA, the patient noted no subjective visual changes other than the progressive myopia that often occurs during HBO₂ therapy [6]. After the completion of the course of HBO₂, the ophthalmologic examination was repeated. Of note, best-corrected visual acuity had improved to 20/25, with a half-diopter more myopic correction. Her pupillary, anterior segment, color vision and dilated fundus exams were all unchanged. Her visual fields were repeated and were unchanged from the pretreatment visual fields.

DISCUSSION

Optic neuritis is a specific type of optic neuropathy resulting from inflammation of the optic nerve; if the inflammation occurs in the segment of the optic nerve posterior to the globe, the inflammation is termed retrobulbar optic neuritis. The most common etiology of optic neuritis is probably an autoimmune response to a viral infection. In some people, optic neuritis may be the initial presentation of multiple sclerosis; in fact, optic neuritis is the first symptom in 15-20% of patients who develop multiple sclerosis [10,11].

Oxygen administered in high inspired partial pressure is known to be toxic to multiple body tissues, especially the central nervous system, the lung and the eye. Visual symptoms during hyperbaric oxygen therapy may be a manifestation of CNS toxicity (twitching of the eyelids, blurred vision) [6] or a manifestation of retinal oxygen toxicity (reversible visual field constriction) [7] or lenticular oxygen toxicity (myopia and cataract) [6].

The patient reported by Nichols *et al.* was a 21-year-old male who had a history of recurrent bouts of pain in the right eye and intermittent diminished visual acuity of the right eye. Based on the finding of two paracentral scotomas on tangent screen examination by an ophthalmologist in his right visual field, a diagnosis of retrobulbar optic neuritis had been made. His pain recurred several times during the first year and lasted for several days.

Approximately one year after his initial diagnosis of retrobulbar neuritis, he was found to have recurrence of the pain and a small central scotoma of the right eye. These recurrent episodes of optic neuritis preceded any exposure to hyperbaric oxygen. In 1966 the man volunteered for inclusion in a study of pulmonary oxygen toxicity and underwent pre-HBO₂ ophthalmologic evaluation which revealed no scotoma but “slight pallor of the right optic disc.” After breathing 100% oxygen at 2.0 ATA for two hours, he noted a temporal visual field constriction and a “bluish haze” of the right eye. He was unable to read letters with his right eye but able to read normally with his left eye. He continued to breathe 100% oxygen at 2.0 ATA and, after four hours, described vision from his right eye as being “dark gray.”

Nevertheless, he continued breathing oxygen at 2.0 ATA for another two hours (six hours total), at which time his right eye visual acuity was “light perception only.” The patient noted improvement in his visual fields 15 minutes after terminating oxygen breathing and switching to compressed air. The volunteer was treated with corticotropin (ACTH) and methylprednisolone and monitored with daily eye exams.

Over the first two weeks, his visual acuity improved to 6/6, and the paracentral scotomas found on initial exam cleared. Exam at two months revealed normal visual acuity but a larger area of temporal pallor. Nichols *et al.* [3] postulated that two distinct processes were evident in this subject: a rapidly-reversible constriction of the visual fields as described by Behnke *et al.* [7] and a transient recrudescence of previously existent retrobulbar neuritis. Nichols *et al.* stated that the “previous history of two episodes of retrobulbar neuritis and the presence of temporal pallor” (prior to beginning hyperbaric oxygen therapy) “make the existence of some degree of vascular limitation likely” in this case.

Our patient had a neuro-ophthalmologist-documented history of optic neuritis 10 years prior to initiation of HBO₂ therapy. However, her ophthalmologic testing and examination prior to beginning hyperbaric oxygen therapy were normal; the pallor of the optic disc

described by Nichols *et al.* was not found. Moreover, although she was treated with oxygen at 2.4 ATA, treatments were for 30 minutes times three, with five-minute air breaks interspersed. The patient was questioned regularly about subjective visual changes and, other than the myopia, no deficits were reported. Post-HBO₂ testing and exam revealed no recrudescence of optic neuritis. Had the patient developed any subjective symptoms of visual difficulty, the HBO₂ therapy would have been terminated, limiting the oxygen exposure, and ophthalmologic evaluation immediately repeated.

This case report is not intended to cause clinicians to no longer seek a history of optic neuritis as part of the evaluation of the patient for possible hyperbaric oxygen therapy. It is intended, instead, to provide a counterpoint to the automatic exclusion of patients with a history of optic neuritis as candidates for HBO₂ therapy. No reports of recrudescence of optic neuritis have been reported in the large numbers of patients with multiple sclerosis who have undergone HBO₂ as therapy for their MS. Butler, Murphy-Lavoie and Jain have deleted a history of optic neuritis as a relative contraindication to hyperbaric oxygen therapy [8]. We suggest careful pre-HBO₂ ophthalmologic evaluation and close daily follow-up of the patient's visual situation, as well as immediate termination of therapy and re-evaluation should any symptoms be reported. Increased reporting of the ophthalmologic response of patients with a history of retrobulbar neuritis subsequently treated with hyperbaric therapy will provide additional evidence on which to base future recommendations. ■

REFERENCES

1. Foster JH. Hyperbaric oxygen therapy: Contraindications and complications. *J Oral Maxillofac Surg*; 50: 1081-1086, 1992.
2. Kindwall EP, Whelan HT. *Hyperbaric Medicine Practice*. Flagstaff: Best Publishing Company, 1999: 83-98.
3. Nichols CW, Lambertsen CJ, Clark JM. Transient unilateral loss of vision associated with oxygen at high pressure. *Arch Ophthalmol* 1969; 81: 548-552.
4. Guy J, Schatz NJ. Hyperbaric oxygen in the treatment of radiation-induced optic neuropathy. *Ophthalmology* 1986; 93: 1083-1086.
5. Fontanesi J, Golden EB, Cianci PC, Heideman RL. Treatment of radiation-induced optic neuropathy in the pediatric population-A case report and review of the literature. *Journal of Hyperbaric Medicine* 1991; 6: 245-248.
6. Butler FK, Hagan CE: Ocular considerations in hyperbaric oxygen therapy. In: *The Physiology and Medicine of Hyperbaric Oxygen Therapy*: Neuman TE, Thom S, eds; Saunders/Elsevier; Philadelphia; 2008.
7. Behnke AR, Forbes HS, Motley EP. Circulatory and visual effects of oxygen at 3 atmospheres pressure. *Amer J Physiol* 1936; 114: 436-442.
8. Butler F, Murphy-Lavoie H, Jain KK. HBO therapy and ophthalmology. In: Jain KK ed. *Textbook of Hyperbaric Medicine*. Cambridge: Hogrefe Publishing, 2009: 399-420.
9. Butler FK, Hagan C, Murphy-Lavoie H: *Hyperbaric Oxygen Therapy and the Eye*. Undersea Hyperb Med 2008; 35:333-387.
10. Emedicine.medscape. Adult optic neuritis. Available at: <http://www.emedicine.medscape.com/article/1217083-overview>. Accessed May 1, 2011.
11. Uptodate.com. Optic neuritis: Pathophysiology, clinical features, and diagnosis. Available at <http://www.uptodate.com/optic-neuritis-pathophysiology>. Accessed May 1, 2011. □

Notes on the use of HBO₂ with optic neuritis

Aside from the Nichols case report mentioned in the preceding paper [1], there is only one other published case report of vision loss during a hyperoxic exposure in a patient with a history of optic neuritis that we are aware of [2]. Both of these cases reverted to pretreatment condition after the cessation of hyperbaric oxygen. Kindwall refers to an anecdotal (unpublished) case where the patient went “completely blind during the course of HBO treatment and remained so.” However, this is the only such case.

There are at least 14,000 cases of multiple sclerosis treated with hyperbaric oxygen in the published literature, and none of them report complications related to optic neuritis [3]. Up to 50% of patients with MS develop optic neuritis, and for 20-30% it is their presenting symptom [4].

Optic neuritis is an optic nerve inflammatory process; there is no pathophysiologic reason to suspect that hyperbaric oxygen would worsen this condition: Indeed, multiple cases of optic neuropathy from other causes have been treated successfully with hyperbaric oxygen therapy.

Relying on one anecdotal unpublished case to justify listing optic neuritis as a relative contraindication to HBO₂ appears unwarranted. The most conservative approach would be to follow the recommendations outlined in this article and monitor the patient very closely during treatment for any signs of visual decline, remembering that oxygen toxicity may affect the lens, the retina, and the CNS – all of which may produce vision loss unrelated to optic neuritis. Based on current evidence, withholding HBO₂ because of a history of optic neuritis may not be appropriate.

Heather Murphy-Lavoie M.D., UHM, FACEP, FAAEM
*Associate Professor of Medicine, Section of Emergency Medicine; Assistant Residency Director, Emergency Medicine Residency Associate Program Director;
Undersea and Hyperbaric Fellowship LSU School of Medicine/
MCLNO, New Orleans, Louisiana*

Frank K. Butler M.D. CAPT MC USN (Retired)
*Chairman, Committee on Tactical Combat Casualty Care
Adjunct Associate Professor, Military and Emergency Medicine; Uniformed Services University of the Health Sciences*

REFERENCES

1. Nichols CW, Lambertsen CJ, Clark JM. Transient unilateral loss of vision associated with oxygen at high pressure. *Arch Ophthalmol* 1969; 81: 548-552.
2. Lambrou GN, Kopferschmitt J, Jaeger A, Brini A. [Slowly reversible central scotoma: iatrogenic effect of hyperbaric oxygenation in the treatment of multiple sclerosis]. *J Fr Ophtalmol.* 1987;10(1):51-9.
3. Kindwall, EP, et al. Treatment of multiple sclerosis with hyperbaric oxygen: Results of a national registry. *Arch Neurol*, 1991, 195-199.
4. James PB. HBO therapy in multiple sclerosis. In: Jain KK ed. *Textbook of Hyperbaric Medicine.* Cambridge: Hogrefe Publishing, 2009: 292-298.

□