Visual Recovery from Radiation-Induced Optic Neuropathy
The Role of Hyperbaric Oxygen Therapy

F.-X. Borruat, M.D., N. J. Schatz, M.D., J. S. Glaser, M.D.,
L. G. Feun, M.D., and L. Matos, M.D.

Optic neuropathy resulting in permanent visual loss is an infrequent delayed complication of radiation therapy. Hyperbaric oxygen therapy (HBO) has been used to treat such a complication, but its efficacy is controversial. We report a patient who presented with radiation-induced optic neuropathy 17 months after irradiation for a left maxillary antrum melanoma. HBO fully reversed visual loss in the more recently involved eye, and slightly improved vision in the earlier affected eye.
Key Words: Hyperbaric oxygen—Radiation necrosis—Optic neuropathy.

 Delayed necrosis of the intracranial optic nerves or chiasm is a recognized complication of ionizing radiation, characterized by abrupt and permanent visual loss affecting one or both eyes (1–3). Intervals from irradiation to symptom onset vary from less than 6 months to 3 years. Treatment of radiation optic neuropathy (RON) with hyperbaric oxygen (HBO) has been shown to stop or reverse the visual loss (4), but its efficacy has been challenged (5,6). We here report the benefits of HBO in a patient who presented with visual loss, occurring 17 months after radiotherapy.

CASE REPORT

A 45-year-old woman presented in May 1990, with recent onset of left nasal obstruction and bleeding. A melanoma of the left maxillary sinus invading the nasal cavity was found and extensive sinus surgery was performed. Despite postoperative 8-mm inferior displacement of the left orbital contents, visual acuity remained at 20/20 and visual fields were full in both eyes. Between July 5 and August 7 of 1990, 5,000 rads were delivered to the left maxillary sinus (20 sessions of 250 rads over 33 days), and 4,250 rads to the left neck (17 sessions of 250 rads over 27 days), without overlap of radiation fields. On October 15, 1990, chemotherapy was commenced with intravenous dacarbazine and oral piritrexim, an experimental folate antagonist (7). Dacarbazine had to be discontinued on August 1991, due to intolerable fatigueability following infusion and the patient remained on only oral piritrexim for the next 7 months. She remained in partial remission and visual function was unchanged in both eyes.

Early February 1992, 21 months after surgery and 17 months after completion of irradiation, the
patient noted left visual loss. On February 10, 1992, visual acuity was 20/20 OD and 20/70 OS with a left pupillary afferent defect. Goldmann visual field revealed an arcuate nasal inferior defect in the left eye (Fig. 1, top). The right optic disk was normal and the left showed temporal pallor. No retinal lesions were seen. Magnetic resonance imaging (MRI) was recommended but not carried out.

At 15 days later, the patient returned with further visual loss: left vision now "no light perception" (NLP), right acuity still 20/20, but with temporal hemianopia on Humphrey visual field (Fig. 1, middle). MRI performed the same day revealed an enlarged left intracranial optic nerve and left hemichiasm, which enhanced after gadolinium injection (Fig. 2). No recurrence of the tumor was detected either clinically or by MRI. The patient underwent HBO (three periods of 30 minutes of 100% oxygen at 2.4 atm, separated by 2 periods of 10 minutes of normal air breathing twice a day for 2 days, then 5 to 6 times/week, 35 sessions in to-

![Visual fields](image-url)

**FIG. 1.** Visual fields. **Top:** Goldmann perimetry: normal visual field in the right eye; the left eye shows an arcuate nasal inferior loss. **Middle:** Left vision is no light perception. Automated static threshold perimetry (Humphrey, 30-2) reveals a dense temporal hemianopia of the right eye. **Bottom:** Left vision is light perception. Automated static threshold perimetry (Humphrey, 30-2) shows a remarkable recovery of the temporal hemianopia.
The patient also received intravenous methylprednisolone 500 mg q6h for 5 days, followed by oral prednisone, 60 mg daily, tapering over 8 weeks.

At 13 days after therapy was initiated, marked resolution of the right temporal hemianopia was noted, further improving 2 weeks later (Fig. 1, bottom). Three weeks after completion of HBO therapy, vision was 20/20 OD and light perception OS; the right visual field (Humphrey, 30-2) was normal. MRI was repeated and showed decreased swelling of the left intracranial optic nerve and chiasm with only slight enhancement after gadolinium injection.

**DISCUSSION**

Delayed necrosis of the optic nerves and chiasm is a well-recognized complication of radiotherapy. Irradiation causes tissue ischemia secondary to a progressive obliterative endarteritis of the microvasculature. Pathology studies demonstrate myointimal and endothelial proliferation of small arteries and narrowing of the vessel lumen with fibrinoid necrosis (8). Skin biopsies from irradiated areas showed the presence of hypocellular and hypovascular tissue that is unable to regenerate supportive vessels and transcutaneous oxygen measurement in irradiated areas revealed tissue oxygen tension at 30% of nonirradiated areas (9). Despite the presence of an endarteritis, the role of high-dose steroids is uncertain as no cases of RON has been reported to improve with steroids only.

RON has been reported to occur as early as 2 months and as late as 7 years after completion of radiotherapy (Fig. 3). However, 90% of RON cases occur within 3 years of irradiation with a mean onset at 12 months (2,3,5,10). In the present case,
visual loss began suddenly 17 months after completion of irradiation. A left retrobulbar optic neuropathy evolved rapidly, progressing to no light perception. Two weeks later, evidence of chiasmal involvement occurred with a dense temporal hemianopia, but preserved visual acuity in the right eye. MRI revealed enlarged left intracranial optic nerve and swollen left chiasm, both enhanced with gadolinium (Fig. 2). These MRI characteristics have been previously demonstrated in RON (10). No other cerebral involvement was detected on MRI and no local recurrence of the sinus melanoma was evident.

HBO therapy produces a steep oxygen gradient between irradiated and non-irradiated tissues. Such an oxygen gradient directly enhances fibroblastic activity, collagen synthesis, and neovascularization in the irradiated tissues (9). HBO is presently the only available treatment capable of creating an environment that allows the reversal of radiation-induced tissue damage. From 20 to 30 sessions of 100% oxygen breathing at 2.4 atm for 90 min each day has been successfully used for problem wounds in oral and maxillofacial surgery following irradiation (9). Since angiogenesis is a progressive event with an initial lag phase, we suggest the use of twice-daily treatments during the first week if vision is deteriorating.

Guy and Schatz (4) first reported visual improvement in RON when treated early with HBO after onset of visual loss, within 2 days if possible. The effectiveness of HBO in RON was challenged by Roden and coworkers (5), but in that report all patients were treated 2 to 12 weeks following visual loss and breathing oxygen at only 2.0 atm.

Our patient was treated 15 days after the onset of left visual loss and 2 days after the loss of the right temporal visual field. There was full recovery of the right hemifield and the left vision slightly improved to light perception. The role of early therapy of RON is emphasized; the key element is the commencement of HBO therapy as soon as vision starts to deteriorate. The dramatic visual recovery in our case emphasized both the benefits of hyperbaric oxygen therapy and the need for starting therapy soon after visual loss, within 2 days, if possible.

REFERENCES