LETTERS TO THE JOURNAL

Hyperbaric Oxygen Treatment for Chronic Cystoid Macular Edema After Branch Retinal Vein Occlusion

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Cystoid macular edema occurs in association with a variety of inflammatory, vascular, degenerative, and surgical conditions.[1] Antiprostaglandin medications have a prophylactic effect on postoperative cystoid macular edema, but no pharmacologic agents have been proven effective for established cases of edema.[2] Recently, Proff and Thom[3] demonstrated that hyperbaric oxygen is beneficial for pseudophakic cystoid macular edema. We treated two patients with hyperbaric oxygen for chronic cystoid macular edema secondary to branch retinal vein occlusion.

In October 1985, a 69-year-old woman had a branch retinal vein occlusion in the upper temporal vein of her left eye. Two months later, cystoid macular edema developed (Fig. 1). Her visual acuity was 20/70. Scattered argon laser photocoagulation was applied over the affected area of the retina, sparing the macula. The macular edema persisted, and her visual acuity remained decreased. In February 1987, the patient underwent total body hyperbaric oxygen treatment in a chamber set at 2 atmospheres for one hour. This was repeated two times a day for 14 days. Her visual acuity substantially improved, reaching 20/25 by the conclusion of the treatment. Fluorescein angiography showed a reduction in leakage of dye from the perifoveal capillaries after the hyperbaric oxygen treatment (Fig. 2). Two months later, her visual acuity stabilized at 20/30.

A 75-year-old woman suffered a branch vein occlusion in the macular region of her right eye in June 1985. Her visual acuity was initially 20/25, but decreased to 20/70 with the develop-

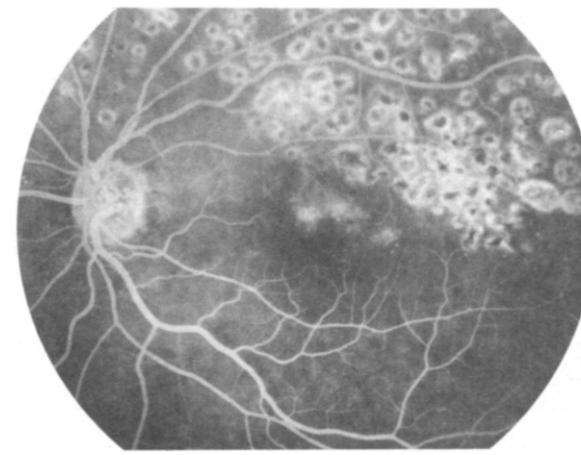

Fig. 1 (Ogura and associates). Arteriovenous phase of fluorescein angiogram before hyperbaric oxygen treatment. Note leakage of dye from the perifoveal capillaries and the presence of cystoid macular edema.

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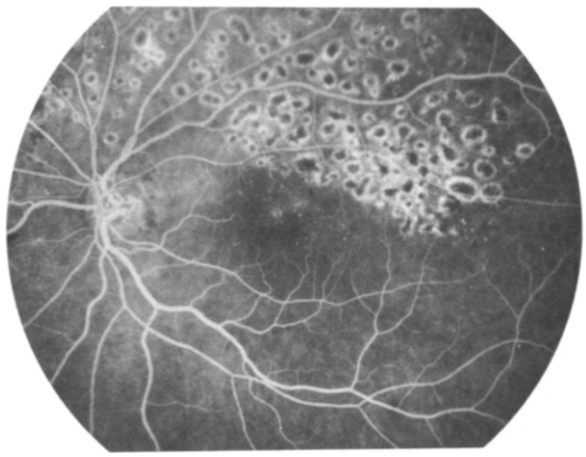

Fig. 2 (Ogura and associates). The same eye after hyperbaric oxygen treatment. The leakage of fluorescein is substantially reduced.

ment of macular edema. Laser photocoagulation failed to resolve the edema. In May 1987, the patient underwent treatment in a hyperbaric oxygen chamber set at 2 atmospheres for one hour twice a day for two weeks, and then once a day for a third week. Her visual acuity improved to 20/20 after four days of treatment. Results of static perimetry showed a reduced central scotoma. One month later her visual acuity was 20/20.

Macular edema commonly occurs after branch retinal vein occlusion. A previous study showed that 65% of the patients with branch vein occlusion have chronic macular edema and a poor visual prognosis.[4] The present study demonstrates that hyperbaric oxygen treatment may resolve macular edema and improve visual function, although the underlying mechanism is still unclear. Hyperbaric oxygen constricts blood vessels, hyperoxygenates tissue, and activates aerobic metabolism. It also reduces the formation of postischemic tissue edema in experimental animals.[5] Proff and Thom[3] speculated that capillaries constricted by hyperbaric oxygen may re-form injured junctional complexes between endothelial cells, which would prevent further leakage. Improvement in the metabolism of damaged cone cell membranes has also been suggested as a mechanism. Although further evaluation of the treatment is needed, it appears that hyperbaric oxygen treatment may be an effective modality for established cases of macular edema after branch retinal vein occlusion.

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Spontaneous Fungal Corneal Ulcer as an Ocular Manifestation of AIDS

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A 53-year-old man developed foreign body sensation, photophobia, and decreased visual acuity in the left eye to 20/100. Results of ocular examination showed two white-yellow anterior stromal corneal lesions that were positive for *Candida parapsilosis* (Figure). There was no history of ocular trauma, ocular surface abnormalities, blood transfusions, or intravenous drug usage.

In the preceding five years the patient had experienced numerous medical problems including leukopenia, pneumonia, and inversion of the T cell helper/suppressor ratio. Immunologic consultation had failed to establish a definitive diagnosis. After *C. parapsilosis* keratitis