CASE REPORT

Hyperbaric Oxygen Treatment in Purtscher's Retinopathy Induced by Chest Injury

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We present a patient who had Purtscher's retinopathy in both eyes after sustaining a chest contusion. After multiple sessions of hyperbaric oxygen (HBO) therapy were given, visual function and retinal appearance improved. This case suggests that HBO therapy may be a treatment option for Purtscher's retinopathy. [J Chin Med Assoc 2006;69(9):444–448]

Key Words: hyperbaric oxygen therapy, Purtscher's retinopathy, retinal ischemia

Introduction

Recent advances in understanding the effects of hyperbaric oxygen (HBO) on retinal anoxia has stimulated interest in the possibility of using it as therapeutic treatment for ischemic conditions of the retina and optic nerve. HBO has been used clinically to treat several retinal conditions, including retinal artery occlusion, retinal vein occlusion, ischemic optic neuropathy, acute retinal necrosis syndrome, cystoid macular edema as a complication of postoperative cataract surgery, and diabetic retinopathy/neuropathy.¹ The effect may be attributed to its ability to: (1) increase oxygen delivery to injured tissues; (2) reduce swelling and inflammation; (3) increase the formation of new blood vessels to injured tissue; (4) improve wound healing; (5) improve the body’s resistance to infections; and (6) renew damaged neurons. Unfortunately, in most cases, the treatment effect has only been transient. Purtscher’s retinopathy is a traumatic angiopathy, frequently following head or chest injuries. The visual prognosis is variable, depending on the severity of the initial injury. The disease is associated with retinal vascular occlusion. The efficacy of HBO in treating Purtscher’s retinopathy has not been established.² We present a case of Purtscher’s retinopathy with vision loss following chest trauma. HBO therapy was given, with resulting improvements in visual acuity and visual field.

Case Report

A 50-year-old man, who sustained a compressive chest contusion from a fallen steel pipe, immediately developed blurred vision in both eyes. No chest injury or other systemic disorders were found. After 2 weeks, he visited our clinic. His initial best-corrected visual acuity was counting fingers (CF)/100 cm in the right eye and CF/10 cm in the left eye. The anterior segment was normal. Fundus examination showed a macular yellowish-white patch in the right eye and confluent white patches with scattered retinal hemorrhage in the posterior pole of the left eye. Purtscher’s retinopathy in both eyes was diagnosed. Visual field examination showed a macular yellowish-white patch in the right eye and large visual field defect in the left eye. Fluorescein angiography showed bilateral macular arteriolar occlusion and paramacular capillary dropout (Figure 1), which were more extensive in the left eye (Figure 2). Multiple sessions of HBO therapy at 2 atm for 90 minutes were given twice a day, for 39 courses. No systemic or ocular complications were noticed during and after treatment. Visual function and retinal appearance gradually improved. One month after HBO therapy, fluorescein angiography of the right eye appeared normal (Figure 3), and the nonperfusion area of the left eye improved (Figure 4). Best-corrected visual acuity recovered to 0.8 in the right eye, and CF/50 cm in the left eye.

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Figure 1. (A) Initial fluorescein angiography of the right eye showed a nonperfusion area and late-phase vessel leakage; (B) color fundus showed a cotton wool patch at the macula.

Figure 2. (A) Initial fluorescein angiography of the left eye showed a large area of capillary dropout at the macula and late-phase vessel leakage; (B) color fundus revealed scattered retinal hemorrhage with multiple patches of retinal lesions.

Figure 3. (A) Follow-up fluorescein angiography of the right eye 1 month after hyperbaric oxygen therapy; (B) the right eye fundus appeared normal.
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eye at the end of 3 months of follow-up. The visual field test also showed improvement in both eyes (Figures 5 and 6).

Discussion

Purtscher’s retinopathy was first described by Otmar Purtscher in 1910, with findings of multiple white retinal patches and superficial retinal hemorrhage in patients with vision loss following head trauma. A similar retinal appearance has been reported in a wide variety of conditions including compressive chest injury, pancreatitis, fat embolism from multiple fractures, lymphoproliferative disorder, and retrobulbar anesthesia. The pathogenesis of Purtscher’s retinopathy is unknown and may be secondary to more than 1 initiating factor. Either direct blunt trauma or a compressive effect in the thoracic region, as in this case, may lead to the development of Purtscher’s retinopathy. We believe that an increase in the thoracic pressure leads to a reflux in the venous system. Subsequent endothelial
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Damage can cause incompetence of the microvascular circulation, resulting in occlusion and ischemia. The diagnosis of Purtscher’s retinopathy is based on clinical presentation and patient history. Purtscher’s retinopathy must be differentiated from commotio retina, shaken baby syndrome, Terson’s syndrome, and Valsalva retinopathy (Table 1). Retinal lesions with or without macular involvement rapidly develop within a few hours to days following the injury. Unless the changes are very mild, visual acuity may decrease immediately, sometimes to 20/200 or worse. The retinal whitening and hemorrhage typically fade away in a few weeks, but blurred central vision and scotoma persist in about half of the cases. Eventually, the fundus may appear normal, or be left with residual pigmentary mottling and disc atrophy. The pathophysiology of this condition has not been completely elucidated. In the majority of patients, recovery of useful vision to 6/12 or better is expected; however, if macular arterioles are involved, the prognosis is generally poor. Visual acuity may remain decreased secondary to infarction of either the foveal photoreceptors or the optic nerve itself. There is no standard treatment for Purtscher’s retinopathy. Papaverine HCL, a peripheral vasodilator, has been reported to treat Purtscher’s retinopathy based on the rationale of dilating retinal arterioles to increase oxygen supply. Atabay et al described late visual recovery in a patient who received megadose steroid treatment for 3 weeks after the trauma. HBO has been applied to treat ischemic intraocular conditions. The increase in partial oxygen pressure in the lung during HBO therapy results in an increased uptake of oxygen in the arterial blood. This state of serum hyperoxia enhances oxygen delivery to oxygen-starved tissue and enables correction of limited hypoxia. Dollery et al reported that HBO contributed

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<thead>
<tr>
<th>Ocular disorder</th>
<th>Ocular sign</th>
<th>Patient history</th>
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<tr>
<td>Commotio retina</td>
<td>Retinal edema; choroidal rupture; exudates and hemorrhage</td>
<td>Blunt trauma to the globe</td>
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<tr>
<td>Shaken baby syndrome</td>
<td>Retinal hemorrhage; periorbital ecchymosis; retinoschisis; retinal folds; vitreous disorder</td>
<td>Whiplash-like child abuse</td>
</tr>
<tr>
<td>Terson’s syndrome</td>
<td>Vitreous hemorrhage; peripapillary hemorrhage</td>
<td>Subarachnoid hemorrhage</td>
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<tr>
<td>Valsalva retinopathy</td>
<td>Preterinal, vitreous, subconjunctival, and orbital hemorrhage; periorbital skin petechiae</td>
<td>Heavy weightlifting, vomiting, forceful coughing, straining at the toilet</td>
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Figure 6. (A) A large visual field defect was noted; (B) improvement after hyperbaric oxygen therapy was noted, with a small paracentral scotoma remaining.

Table 1. Differential diagnoses of Purtscher’s retinopathy
to an increase in oxygen supply from the choroid, thereby increasing the supply of oxygen to the inner retina, even in cases with decreased retinal circulation. The favorable effects of HBO on microcirculation indicated that it might be used to treat Purtscher’s retinopathy. In our present case, although the possibility of spontaneous recovery cannot be completely excluded, the dramatic improvements in visual function and retinal appearance in both eyes closely following HBO therapy suggest the usefulness of this treatment. However, the visual acuity in our patient’s left eye remained limited, and a persistent segmental visual field defect was noted. This may be due to the severe and extensive destruction of foveal avascular zone in the left eye. Our experience with this case suggests that HBO might be a new remedy for selected cases of Purtscher’s retinopathy and calls for a controlled clinical study to accurately assess its efficacy.

References