Case Report

Clinical case report: Treatment of a central retinal vein occlusion with hyperbaric oxygen.

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Wright JK, Franklin B, Zant E. Clinical case report: Treatment of a central retinal vein occlusion with hyperbaric oxygen. Undersea Hyperb Med 2007; 34(5):315-319. A case of retinal central vein occlusion (CRVO) in a 43-year-old man is presented in which hyperbaric oxygen (HBO_2) was used as the only treatment method. CRVO is a relatively common cause of visual loss, with hypertension, diabetes, glaucoma and hypercoagulable conditions identified as risk factors. The patient in this report had none of these risk factors and declined treatments other than hyperbaric oxygen. HBO_2 was effective in sustaining the ischemic retina and controlling retinal edema until the retina revascularized and vision stabilized. The initial visual acuity in the left eye was 20/200 (corrected), and after two hyperbaric treatments it was 20/30 (corrected). Following three months of HBO_2 treatments the vision stabilized to 20/20 (corrected) in the affected eye. Treatment considerations in using HBO_2 as adjunctive therapy for CRVO are early institution of treatment, and continuation of HBO_2 until the retinal edema has resolved and vision has stabilized.

INTRODUCTION

Central retinal vein occlusion (CRVO) is a thrombosis of the central retinal vein with venous obstruction and venous stasis or hemorrhagic retinopathy (1). The clinical course is characterized by sudden diminished visual acuity which may be permanent or may slowly improve. Visual loss may be due to a variety of causes which include macular edema, dense intraretinal hemorrhages, retinal neovascularization, vitreous hemorrhage, retinal detachment, and retinal hypoxia (2). The hypoxic retina functions poorly, produces vascular endothelial growth factor and is prone to neovascularization with further loss of vision (3). The disease is classified as ischemic or non-ischemic, depending on the amount of retina which is poorly vascularized. Ischemic CRVO has a poor prognosis with fewer than ten percent of patients retaining vision better than 20/400 in spite of treatment (4). CRVO is a leading cause of blindness, second only to diabetic retinopathy as a cause for visual loss

(5). The disease is predominant in the elderly, and in those with coagulopathies and pre-existing hematologic and vascular disorders. It has been described in otherwise healthy individuals, particularly in athletes engaged in long distance running (6,7) and strenuous weight lifting (8).

CASE REPORT

A 43 year old Caucasian male awoke two days prior to presentation with blurred vision in the left eye, predominantly in the central visual field. Two days later he was examined by an optometrist and an ophthalmologist who diagnosed a left central retinal vein occlusion. There were no identifiable precipitating events or history of previous thrombotic events in the patient who was engaged in an athletic lifestyle consisting of running 12 to 15 miles per week, calisthenics for 30 minutes three times per week, and swimming for one and one half hours per week. Additionally he participated in parachuting, scuba diving, and occasional endurance running, though none of these activities occurred in the two weeks preceding the visual loss. He was on no medication, weighed 88.5 Kg and his height was 70 inches (178 cm.). Examination of the left eye revealed an edematous optic nerve with venous occlusion, macular edema and retinal hemorrhages **(**Fig 1A**).** Vision in the left eye was correctable to 20/200 where previously it has been correctable to 20/20. Visual fields were normal. The right eye was normal (Fig 1B).

Treatment with hyperbaric oxygen therapy was begun on the day the patient was initially examined, two days after the onset of symptoms. The hyperbaric oxygen treatment profile was 2.4 ATA (243.18 kilopascals) for 90 minutes with two five-minute air breaks at 30-minute intervals. Because an underlying hypercoagulable state was suspected the patient began aspirin, 325 mg daily, but this was discontinued by the patient one month after onset of the visual disturbance when no coagulopathy was found. There was a marked improvement in vision after the first two hyperbaric oxygen treatments with vision returning to 20/30 (corrected) in the left eye. Improvement in vision occurred after most hyperbaric oxygen treatments with a gradual diminution of visual acuity in the time period

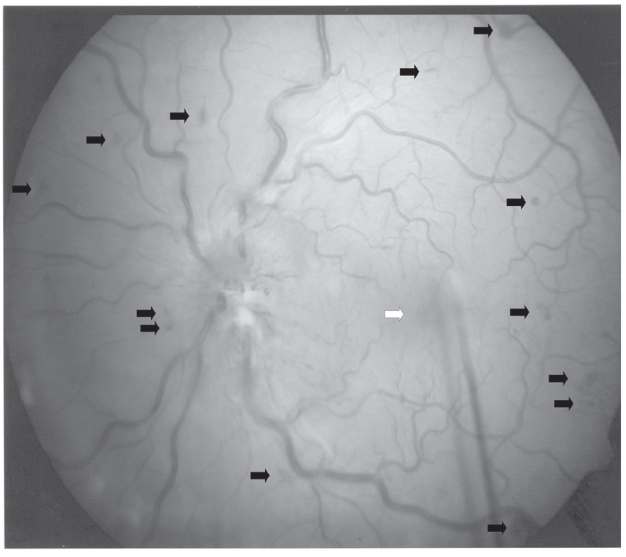

Fig. 1A. left retina showing papilledema, retinal edema, retinal flame hemorrhages (black arrows), and macular edema (white arrow).

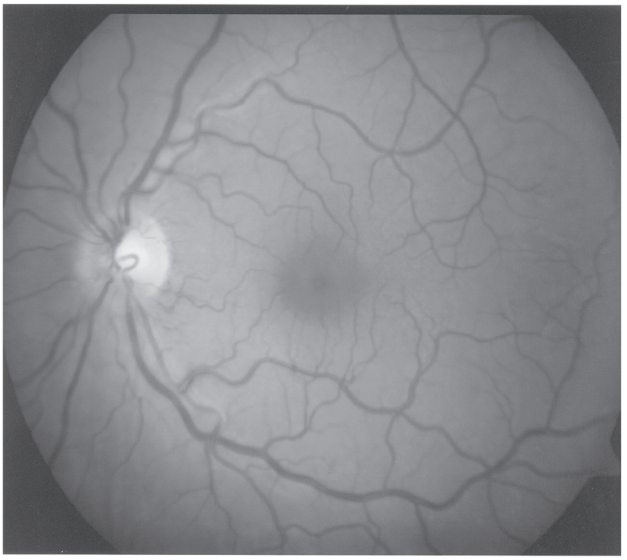

Fig. 1B. right retina (normal).

following hyperbaric oxygen treatment. The patient declined other therapy and opted for HBO_2 treatment each time the visual acuity diminished to a level causing difficulty in reading. Treatment with hyperbaric oxygen continued on a daily basis for 30 hyperbaric oxygen treatments and was then reduced in frequency to two to three hyperbaric oxygen treatments per week for a total of 60 treatments completed three months after the initial onset of the CRVO. The vision in the left eye remained variable for three months from 20/25 to 20/40 corrected. The optic nerve and fovea remained edematous until six months after the injury. On the days when vision had deteriorated hyperbaric treatment was followed by a rapid and consistent improvement in vision. The hyperbaric treatments were continued until the vision had stabilized. By seven months after the CRVO, vision was 20/20 (corrected) in the left eye and the retinal edema had resolved.

A search for a hypercoagulable state was instituted; cardiolipin antibody levels were drawn and found to be initially elevated. Cardiolipin immunoglobulin G (IgG) was 22 IgG phospholipid units (GPL) (normal = 0-15 GPL), and cardiolipin immunoglobulin M (IgM) was 31 IgM phospholipid units (MPL) (normal = 0-13 MPL). The erythrocyte sedimentation rate, glycosylated hemoglobin, cryoglobin, hemoglobin electrophoresis, venereal disease research laboratory test (VDRL), fluorescent treponemal antigen (FTA), antinuclear antibodies (ANA) titer, prothrombin time and partial thromboplastin time were normal. Hematology evaluation for hypercoagulable conditions was unproductive without evidence of sustained cardiolipin antibody elevation or coagulopathy. The cardiolipin antibody level returned to normal during the hematologic workup performed one month after the initial event. The initial elevation in cardiolipin antibody was thought to be the result of the thrombotic event causing CRVO.

DISCUSSION

CRVO involves thrombus in the central retinal vein, usually induced by a hypercoagulable state. The causes of CRVO are glaucoma, ocular hypertension, trauma, diabetes, hypertension, sarcoidosis, retinal vascular anomalies, coagulopathies, and hyperviscosity syndromes (9). Hypertension, diabetes, and glaucoma have been identified as risk factors (10). Subsequent to the initial retinal venous thrombosis intraretinal hemorrhages, venous tortuosity, and capillary nonperfusion occur with resultant retinal hypoxemia (11). CRVO can be manifested by non-ischemic areas of retina characterized by stasis or ischemic areas characterized by hemorrhagic retinopathy (12). In the patient described here the retinopathy was ischemic and multiple retinal hemorrhages were present (Fig 1A). Anterior segment neovascularization and glaucoma occur in about eight per cent of cases (13).

Cardiolipin antibodies are produced as an immune response to the lipids in platelets and cell membranes in response to thrombotic events and in some autoimmune diseases (14). They are indicators of inappropriate thrombosis and are elevated in hypercoagulable states and are associated with some autoimmune diseases. While the cardiolipin antibodies were elevated initially they returned to normal within three weeks and it was felt that they were indicative of a thrombotic event rather than anti-phospholipid antibody syndrome.

Other than controlling the underlying pathology there is no commonly agreed upon therapy for CRVO. Treatment recommendations have ranged from observation to intraocular steroid injections, photocoagulation (15), surgical chorioretinal anastamoses (16), injection of thrombolytic agents and tissue plasminogen activator into retinal veins (17,18),

and optic nerve decompression (19). Injection of steroids into the eye is not without risk and retinal necrosis has been described following this treatment (20). Surgical decompression of the optic nerve has been associated with elevated intraocular pressure and endopthalmitis (21).

HBO_2 treatment has produced dramatic improvement in some patients with central retinal artery occlusion (CRAO) where treatment is started within a few hours of the CRAO and there is adequate collateral circulation in the retina to support oxygenation (22). Considerations when instituting HBO_2 for CRAO are 1) early institution of treatment, within hours of the event if possible, and 2) continuation of treatment until clinical improvement has stabilized. This approach has been used successfully for treatment of CRVO in the past (23-28). A literature review revealed 109 documented cases of CRVO treatment with HBO_2; all but two were outside North America. Based on our previous experience treating retinal arterial occlusion with HBO_2, we felt immediate HBO_2 therapy was indicated in this case of CRVO with retinal ischemia and treatment was started on the day the patient presented. Even though the patient was offered intraocular steroid injections, he declined, as the HBO_2 was immediately effective. There was no evidence of anterior segment neovascularization in this patient.

While non-ischemic CRVO may eventually resolve without significant loss of vision, the ischemic version of CRVO described in this case report needed prompt intervention to salvage vision. The rapid improvement in vision once HBO_2 therapy was instituted bears this out. In spite of the improvement in vision the retinal edema persisted for several months as expected with CRVO. This recurrent edema was the cause of retinal hypoxia, cellular dysfunction, and the gradual visual deterioration following each HBO_2 treatment. The edema-reducing properties of HBO_2, along with down regulation of inflammatory cytokines may have contributed to the improvement in vision immediately following each treatment (29,30). An advantage of HBO_2 treatment for CRVO is that it can be utilized periodically during the healing process to assist in reduction of retinal edema and restoration of vision. It is possible that, if started early in the course of ischemia, HBO_2 alone may be effective treatment for CRVO, but this would require verification through controlled studies before abandoning other treatment modalities. This case supports the use of HBO_2 in the acute management of CRVO, and the clinical course suggests that visual acuity may respond to HBO_2 therapy until the retinal edema resolves.

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