Chapter 2.3.5

ACUTE ISCHEMIC OPHTHALMOLOGICAL DISORDERS

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Abstract: In the field of ophthalmology, a beneficial effect of hyperbaric oxygenation therapy has been described for retinal artery occlusions, non-arteritic optic neuropathy and macular edema secondary to retinal vein occlusion or uveitis. Here we describe the pathology of retinal vascular occlusions and discuss the use of HBO in these conditions.

Keywords: Hyperbaric oxygenation therapy; central retinal artery occlusion; CRAO; branch retinal vein occlusion; BRVO; central retinal vein occlusion; CRVO; branch retinal vein occlusion (BRVO); anterior optic neuropathy; AION; macular edema; retina; optic disc; optic nerve; ophthalmic artery; ciliary artery; carotid artery; emboli; arteritic AION; non-arteritic AION; arteritic CRAO; non-arteritic CRAO

1. VASCULAR SYSTEM OF THE EYE

The arterial vascular system of the eye derives from branches of the ophthalmic artery which is fed from the internal carotid artery. Before entering the eye, one branch is following the optic nerve into the eye to feed as the central retinal artery the inner retina. Other branches of the ophthalmic artery penetrate the sclera as short and long ciliary arteries to feed the choroid which is supplying the outer retina. Therefore, the retina has two vascular systems each supplying half of the retina. The venous system drains with its branch retinal veins converging to the central retinal vein the inner retina. The central retinal vein passes through the optic nerve together with the central retinal artery and later drains into the ophthalmic vein. The venous system of the choroid drains through four vortex veins through the

\[ \text{D. Mathieu (ed.), Handbook on Hyperbaric Medicine, 527–535.} \]
sclera into the orbital vein. The optic nerve disc receives its blood supply through the short posterior ciliary arteries.

![Diagram of vascular supply of the eye](image)

*Figure 2.3.5-1. Vascular supply of the eye*

2. **RETINAL ARTERY OCCLUSION**

2.1 **Pathomechanisms and clinical picture**

Retinal artery occlusion leads to a sudden severe and painless vision loss in the affected eye\(^1\). Patients with CRAO are rarely younger than 40 years, the mean age is 65 years. Occlusion of the ophthalmic artery is rare, usually the central retinal artery or one of its branches is occluded, leading to a central retinal artery occlusion (CRAO) or a branch retinal artery occlusion (BRAO). The retina is very sensitive to ischemia, experimental data have shown that a total occlusion of the central retinal artery causes irreversible damage to the inner retina if occlusion time exceeds 100 minutes\(^2\). Clinically, CRAO is seen as incomplete CRAO, subtotal CRAO or total CRAO. Most CRAO observed clinically are subtotal CRAO. Within 48 hours, about 75% of CRAO spontaneously recanalize\(^3\). However, at that time point irreversible damage at the inner retina has occurred, allowing only ambulatory vision. The leading cause of CRAO are emboli deriving from arteriosclerotic plaques in the aorta or from the carotids. Further sources of emboli are calcified heart valves, thrombi originating from areas of restricted heart movements or in patients with atrial arrhythmia. Rare emboli sources

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2.3.5.2

are less common.

Embolization of the ophthalmic artery is a high-risk procedure (AIG). The outcome of acute occlusion is poor, 10% of patients maintain

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Figures 2.3.5-2 and 2.3.5-3 are informative.

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are bacterial emboli in sepsis cases, air or bone marrow emboli in trauma patients.

Besides embolism, inflammation of the arterial wall in conditions like M. Horton or M. Wegener can cause a CRAO. Arterial hypertension is a major risk factor for CRAO. In this context the anterior ischemic optic neuropathy (AION) has to be mentioned producing a sudden and painless loss of visual acuity due to infarction of the optic nerve disc due to closure of the short posterior ciliary arteries. The risk factors are similar to CRAO, but arteritis is more frequently (50% of patients) found in AION.

Figure 2.3.5-2 Branch retinal artery occlusion (BRAO). Note the whitish edema in the inferior retina and the white emboli located at the proximal inferior artery on the optic disc

Figure 2.3.5-3 Central retinal artery occlusion (CRAO). Note the whitish edema of the retina and the interrupted blood column with the retinal vessels
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**Figure 2.3.5-2.** Branch retinal artery occlusion (BRAO). Note the whitish edema in the inferior retina and the white embolus located at the proximal inferior artery on the optic disc

**Figure 2.3.5-3.** Central retinal artery occlusion (CRAO). Note the whitish edema of the retina and the interrupted blood column with the retinal vessels
2.2 Treatment options

Treatment options in CRAO are limited due to retinal sensitivity to ischemia and late presentation of patients. Massage of the eye ball is being attempted in order to move emboli further distal in the artery. A reduction of perfusion resistance into the eye is achieved by lowering the intraocular pressure either by paracentesis of the anterior chamber or by administration of anti-glaucoma medication. Hemodilution therapy is attempted to improve rheologic parameters, especially to lower the hematocrit. A systemic lysis therapy has so far been reported in single case reports and considered as to dangerous. Local intravascular fibrinolysis (LIF) using a catheter through the carotid artery to release urokinase (rTPA) into the ophthalmic artery has been described and reported to show better results compared to controls. In AION patients hemodilution therapy, and in some centers, surgical optic sheath fenestration to alleviate compression of swollen nerve axons, is being performed. In arteritic CRAO, BRAO or AION, immediate systemic steroid therapy is mandatory to protect the fellow eye from vision loss and to limit the damage in the affected eye.

2.3 HBO in retinal artery occlusion

Several reports exist describing a positive effect of HBO therapy on retinal artery occlusion. The aim is to maintain an oxygen supply to the retina through the choroidal vasculature under hyperbaric oxygenation therapy (HBO) until spontaneous recanalisation occurs. The bradytrophic vitreous could serve as an oxygen deposit after HBO therapy, and a local release of tissue plasminogen activator under HBO could further help to accelerate reperfusion. Takahashi described a combination of HBO-therapy with a stellate ganglion block in a small case series of CRAO patients. Beiran and coworkers combined HBO with nifedipine treatment in recent onset CRAO patients. In a study with 8 patients with CRAO and 10 patients with BRAO, a beneficial effect of HBO therapy compared to a control group was described by Alsentré and coworkers. In their study, patients were treated with 3x30 min at 240 kPa which was applied 3 times a day on admission, twice daily on day 2 and 3 and once daily for at least another 4 days. In a study at our clinic, 21 patients with CRAO were treated with HBO. We included only patients with an onset of symptoms of less than 12 hours and normal values for ESR and CRP, thus excluding arteritis. HBO therapy was performed 3 times within 24 hours after admission followed by two treatments daily for up to 2 days at 2.4 ata following the Marx scheme. In our study, about 10% of our patients regained a visual acuity which allows reading. Meanwhile, we have treated 40 patients with CRAO where
15% regained reading vision after HBO-treatment (figure 2.3.5-3-2.3.5-4). Meta-analysis with other interventional studies is difficult to perform as inclusion criteria as initial visual acuity and latency of symptoms vary, nevertheless our results have been very good.

In a case report, Bojic and coworkers described improvement in visual acuity in 2 patients with non arteritic AION\textsuperscript{10}. The improvement has been observed 3-5 months after the event. Such developments have been described also in the natural history of AION\textsuperscript{11}, but it is possibly worth to perform a pilot study with a larger patient number.

A sufficient oxygenation of the retina can be achieved by HBO in retinal artery occlusion. However, the glucose and nutrients transport provided by retinal artery circulation is vital for retinal ganglion cell survival. If retinal artery recanalization does not occur in time, the neural retinal cells die despite of sufficient oxygenation. Additionally, there is a risk of ischemia-reperfusion injury. It can be discussed if a neuroprotective medication like glutamate antagonists should be given along with HBO to reduce this risk.

Hyperbaric oxygenation leads to vasoconstriction in the autoregulated retinal vascular system\textsuperscript{12} which is not welcome in arterial occlusions. In order to minimize this effect some authors performed a stellate ganglion block\textsuperscript{6} or applied vasodilatative medication\textsuperscript{7}. It is also possible to add 5% of carbon monoxide to the inhalation gas to reduce vasoconstriction\textsuperscript{13}. However, the solubility of carbon dioxide compared to oxygen is higher under hyperbaric conditions, and therefore the effect is difficult to predict. In our opinion it is generally not necessary to block autoregulation as in ischemic tissues autoregulation fails. Also, the capability of retinal vessels to constrict declines with age, at the age of 50 the effect is only marginal\textsuperscript{14}.

CRAO and BRAO could be a symptom of a subtotal carotid artery stenosis. Together with neurologists it should be discussed whether patients with this condition may receive HBO treatment or if generally a carotid artery duplex sonography should be mandatory before starting HBO therapy.

In our clinic we have reserved HBO treatment to non-arteritic CRAO and BRAO cases as the pathomechanism of ischemia in arteritic cases is different and requires systemic steroid medication. As we do not have experience with HBO treatment in arteritic retinal occlusion we recommend HBO to be used in early presenting cases of CRAO and BRAO which have not recanalized and have a visual acuity of less than 0.1.

It is also crucial to see patients with retinal artery occlusions as early as possible. We have therefore sensitized our personnel at the hospital admission to send patients complaining of an acute painless vision loss directly to the doctor, bypassing the waiting room.
3. RETINAL VEIN OCCLUSION

3.1 Pathomechanisms and clinical picture

Retinal vein occlusions are relatively common condition usually affecting people older than 40 years. In analogy to the retinal artery occlusions, the terminology in retinal vein occlusion is central retinal vein occlusion (CRVO) and branch retinal vein occlusion (BRVO), both of which can lead to a severe vision loss. The vision loss is less acute compared to retinal artery occlusions. In some cases the patient did not realize the condition as the visual acuity decreased slowly over weeks in the affected eye. Clinically, ischemic CRVO is discriminated from non-ischemic CRVO. Both forms differ in the extent of retinal hemorrhages and so called “cotton wool spots”, which represent localized ischemia of retinal nerve fibers. Ischemic vein occlusions usually have a visual acuity of 0.1 (10 %) or less on presentation. Both forms of CRVO can convert into the other form. While ischemic CRVO has a poor prognosis, non-ischemic CRVO in some cases can regain full visual acuity. Persistent ischemic CRVO or BRVO require panretinal laser coagulation to avoid complications due to retinal neovascularisation. The final visual acuity also depends on the extend of the cystoid macular edema, a common complication in CRVO and BRVO,
which can develop weeks after the disease’s onset. Visual acuity (VA) can improve spontaneously even after several months, however, in general an initial VA of 0.1 or less is an unfavourable prognostic factor. The main risk factor for CRVO and BRVO is arterial hypertension. Further risk factors are glaucoma, smoking, hyperopia, oral contraceptives, increased blood viscosity, a high hematocrit, and phlebitis. Often a retinal vein occlusion is situated at an anatomical narrowing, for example arteriovenous crossings or in the optic nerve head at the level of the lamina cribrosa.

3.2 Treatment options

Current treatments aim at the improvement of risk factors, e.g. reduction of arterial hypertension, isovolemic hemodilution to lower the blood viscosity and the hematocrit. In BRVO it has been attempted to surgically separate retinal veins from arteries in cases where the occlusion site was located at an arteriovenous crossing\textsuperscript{15}. In CRVO, incisions into the optic nerve sheath have been performed in order to decompress the swollen nerve fibers. The outcome after these experimental surgical interventions is not satisfactory so far\textsuperscript{16}.

3.3 HBO in retinal vein occlusions

Hyperbaric oxygenation therapy (HBO) has shown to produce an improvement in visual acuity by reducing macular edema secondary to conditions like uveitis\textsuperscript{17}. In a pilot study we treated patients with chronic macular edema secondary to ischemic CRVO with HBO therapy. We used the Marx scheme at 2.4 ata, with one session per day for 15 days followed by one session per week in the following 6 weeks. We observed in some cases dramatic improvement in visual acuity, but also observed a decline in visual acuity back to initial values weeks after HBO treatment was stopped\textsuperscript{18}. The effect of HBO on macular edema is not well understood, it can be speculated that vascular constriction within the retina reduces the edema, in these case some autoregulation must be functional. As the observed effect has been temporary, we currently do not perform HBO-treatment in retinal vein occlusion or macular edema secondary to other diseases.
REFERENCES


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