

Branch Arterial and Venous Occlusion in a Young Patient

Patrícia Sumarová^{1,2*} and Veronika Matušková^{1,2}

¹Ophthalmology Clinic of FN Brno, Czech Republic ²Ophthalmology Clinic, Faculty of Medicine, Masaryk University Brno, Czech Republic

*Corresponding Author: Patrícia Sumarová, Ophthalmology Clinic of FN Brno and Ophthalmology Clinic, Faculty of Medicine, Masaryk University Brno, Czech Republic.

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Abstract

Objective: The aim of the thesis is to point out the possibility of combined vascular occlusion in young people and a detailed description of the diagnosis and treatment of this relatively rare problem.

Results: Significant gain of central visual acuity and improvement of anatomical conditions due to the early therapeutic effect of anticoagulant treatment.

Conclusion: Combined branch retinal artery and vein occlusions are extremely rare, especially in young people, are associated with significant systemic comorbidity, and can have a good visual outcome if recognized early and treated appropriately, regardless of the completeness of all follow-up examinations.

Keywords: Retinal Artery and Vein Branch Occlusion; Anticoagulant Treatment; Antiplatelet Treatment; Cystoid Macular Edema

Introduction

Retinal vascular occlusions are generally associated with well-defined classic risk factors in the elderly, namely hypertension, diabetes, hyperlipoproteinemia and smoking. Autoimmune hypercoagulable disorders are often causal in younger individuals. The prevalence of the disease increases significantly with age. It mostly affects the temporal retinal vessels with symptoms of sudden and painless vision loss. If central vision is not affected, it can be asymptomatic. Combined branch retinal artery (BRAO) and vein (BRVO) occlusions are extremely rare and not well characterized. Treatment is based on interdisciplinary cooperation.

Case Description

A young man, 26 years old, was referred to us at the Ophthalmology Clinic via the drop eye ambulance for further examination and treatment of a combined occlusion of the branch of the retinal artery and vein in the left eye. Subjectively, for several days, he perceived blurred vision in his left eye, which gradually worsened. In general, he was not treated for anything, did not actively play sports, was of normal habit, abstinent, non-smoker. A few days ago he had a respiratory illness that was treated with antibiotics. Ophthalmic and family history were unremarkable. The vision of the right eye (VOD) was initially with a myopic correction of 4/4, the vision of the left eye (VOS) with a myopic correction of 4/25. Bilateral slit-lamp examination did not show pathology of the anterior segment, the fundus

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of the right eye was intact. In the left eye, in the area of the upper temporal arcade, there was a finding of splatter-like hemorrhages reaching up to half of the macula from above, to which a band of ischemia connected temporally, clinically on the vessels without visible microembolization. We documented the finding with a fundus camera and also performed optical coherence tomography (OCT), which clarified the extent and height of the macular edema (Figure 1 and 2). The central retinal thickness (CRT) of the left eye was initially 829 μm. Fluorescence angiography (FAG) was not indicated given that the extent of the intraretinal hemorrhage reached the central periphery at most. The wider periphery was unaffected. Ultra-wide field fluorescence angiography (UWFA) is not available at our institution. During hospitalization, we started vasodilator and hypotensive treatment (Cavinton i.v. inf., Diluran tbl, Mannitol i.v. inf., Azopt gtt.) to treat arterial occlusion. To ensure the treatment of venous occlusion, we therapeutically applied Fraxiparin s.c., which was, due to the patient s weight, 92 kg, in a dose of 0,6 ml-0-0,8 ml s.c. During hospitalization, the effectiveness of the treatment was monitored in the form of anti-Xa sampling. As part of the examination of the general condition, we performed basic and immunological blood sampling to rule out possible vasculitis. In the investigation of the primarily arterial cause, an electrocardiogram, chest X-ray, transthoracic (TTE) and transesophageal echocardiography (TEE) were performed to rule out a possible foramen ovale aperture, as well as duplex ultrasonography of the major arteries (UZV MMT) and internal examination. All results were negative. Due to the young age of the patient, a complete hematological examination was indicated, which ruled out a genetic mutation or a thrombophilic condition and clarified a possible venous cause of the occlusion. An ambulatory magnetic angiography of the brain and orbits was planned to rule out a vascular malformation. The patient was discharged with a therapeutic dose of Fraxiparin 0,6 ml-0-0,8 ml s.c. During treatment, the patient's liver tests worsened, based on these results, we completed an abdominal ultrasound, which demonstrated splenomegaly without dilatation of the portal tract. The set total treatment was reduced in the short term. During the outpatient check-up, which took place one month after discharge, we observed a significant improvement in VOS, which was 4/5 with myopic correction. Biomicroscopically hard exudates persisted in the macula, intraretinal hemorrhages in the papilla, otherwise no focal changes. On OCT, we demonstrated foveolar depression, without neuroretinal edema, but with its partial atrophy. The clinical findings were again verified by a color image taken with a fundus camera. Due to the improved clinical findings, the hematologist gradually reduced the therapeutic dose of heparin with a change of preparation to Clexane 0-0-1 ml s.c., which was partially reduced again to a dose of 0-0-0,6 ml s.c. during the next check-up every 4 weeks. During the last visit, which took place 2 and a half months after discharge, the VOS with myopic correction was 4/5. Biomicroscopic findings with the image of persistent hard exudates, but no longer hemorrhages in the papilla, optical coherence tomography without evidence of edema and with persistent partial atrophy of the neuroretina, CRT 189 µm (Figure 3 and 4). Clexane s.c. was in a dose of 0-0-0,6 ml s.c. applied for another 2 weeks, then Clexane 0-0-0,4 ml s.c. every 2 weeks, and after 3 months of the total therapeutic dose of heparinization, treatment was subsequently terminated. The patient continued to be monitored, he was subjectively satisfied, VOS and OCT remained stable. The next check-up is already planned with the results of the magnetic angiography of the brain and eyeballs - it has not yet taken place.



Figure 1: Input color fundus image of the right and left eye.

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Figure 2: Input OCT image of the left eye.



Figure 3: Color image of the fund at the last check.



Figure 4: OCT image of the last follow-up examination.

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Discussion

Combined branch retinal artery (BRAO) and vein occlusion (BRVO) appears to be uncommon and extremely rare, affecting both young and old patients with significant visual morbidity and systemic associations [1]. The pathogenetic mechanisms by which combined BRVO and BRAO occur are unclear. We hypothesize that BRVO may be the initial event that occurs due to compression of the vein by atherosclerotic arteries at arterio-venous crossing sites, leading to turbulent blood flow and dynamic obstruction or actual thrombus formation and mechanical blockage. If it is severe enough, a sudden rise in intravascular pressure in the venous tree to values above systolic pressure can lead to the transfer of "back pressure" to the arterial circulation and subsequent deterioration of arterial perfusion, which is manifested as BRAO. In the younger age group, the presumed hypercoagulable blood state predisposes to venous thrombosis and increased back pressure on the arterial circulation leads to occlusion of the artery. As part of the diagnosis, a careful personal anamnesis is necessary with the exclusion of cardiovascular risk factors, a complete blood count including immunological parameters must be examined, TTE or TEE of the heart must be performed to rule out foramen ovale aperture or other malignancies affecting the arterial bed. In the younger age group, known risk factors for combined occlusion are coagulation disorders, e.g. anticardiolipin syndrome, von Willebrand syndrome, protein S deficiency or factor V mutation. Especially in young patients, examination is appropriate brain magnetic resonance to rule out intracranial or intraorbital pathology. In their study, Lam and colleagues [2] investigated risk factors for BRVO. They reviewed the medical records of 60 consecutive patients younger than 49 years of age with BRVO. It recorded patient s age, sex, body mass index, history of smoking, diabetes, hypertension, hyperlipidemia, and hormone replacement therapy in woman. An increased risk of BRVO was found in patients with a history of systemic hypertension, hyperlipidemia, and elevated body mass index, but not in patients with diabetes, smoking, or hormone replacement therapy. Tain., et al. [3] presented a six-year review in which 17 eyes of 15 patients younger than 50 years (mean age 34 +/- 8 years, 71% male, 29% female) were identified with retinal artery occlusion. Systemic diseases in patients with branch retinal artery occlusion (BRAO) included Susac disease (n = 1), sickle cell disease (n = 2), foramen ovale aperture (n = 1), HIV positive (n = 1), the rest were undetected pathology. In some cases, combined branch retinal occlusion may be preceded by slightly elevated antiphospholipid IgM antibodies with a one-year interval. This case of a 30-years-old patient was published by Consigli, et al [4]. Treatment of retinal arterial occlusion consists of hypotensive and vasodilatory treatment, administration of antiplatelet therapy is recommended. Administering antiplatelet agents helps reduce the risk of secondary complications. In the treatment of retinal vein occlusion, retinal laser photocoagulation (LFK) can be used to treat peripheral ischemic parts of the retina, which was not necessary in our case. The anti-VEGF treatment used to treat macular edema in our case was not indicated for maximum regression of the edema upon established heparinization. However, in his retrospective study, Singh found no significant differences in visual acuity or prevalence of cystoid macular edema (CME) at the last visit in patients taking antiplatelets/anticoagulants compared with those not taking these agents [5].

Conclusion

Combined branch retinal artery and vein occlusions are extremely rare, are associated with significant systemic comorbidity, and can have a good visual outcome if recognized early and treated appropriately, regardless of the completeness of all investigations. In the case of a young man, in whom the systemic cause has not yet been found, we point out a significant gain in central visual acuity (CVO) due to the early therapeutic effect of anticoagulant treatment. During diagnosis and treatment, it is always necessary to think about arterial as well as venous issues.

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