Case Report

Recurrent branch retinal vein occlusion in young age (case report)

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Abstract. We present a case of a 34 year old man with recurrent branch retinal vein occlusion (BRVO). He was examined with fluorescein angiography, digital fundus photography, serological and immunological blood tests. In contrast to the common belief that BRVO in young people usually have a beneficial development, we point out an atypical case with prolonged course and tendency of permanent damage of the visual acuity. The precise diagnostics of such cases and their adequate in-hospital treatment are the most important prerequisites for the satisfactory final recovery.

Key words: recurrent branch retinal vein occlusion, fluorescein angiography, macular edema.

Branch retinal vein occlusion (BRVO) is a relatively common pathology, which normally develops at 55 years of age [1-3]. If the condition is not diagnosed and properly treated on time, it can cause significant visual disability. All pathologic conditions affecting the structure of the blood vessel wall, as well as those connected with a decrease in the minute volume of the heart and increase in blood viscosity can enhance the development of BRVO [4-6]. Occlusions commonly occur in patients with longstanding arterial hypertension, in diabetics and in patients with arterio-sclerosis. Predisposing factors are also vasculitis, different kinds of hemopathies, changes in the lipid metabolism, dehydratations, congenital abnormalities, periflebits, etc [6,7]. Usually the clinical picture is presented by a sudden loss of central vision, corresponding to the severity of the thrombosis. Comparatively atypical are the cases of BRVO in young people known as papillophlebitis or optic disc vasculitis. They are found only in 10-20% of all cases of BRVO and affects people in the age group of 35-45 years. Although studied a lot [8-10], BRVO in young age still raises some unknown questions and is a challenge in regard to proper diagnostics and treatment.

The aim of our survey is to describe an atypical case of a young man with recurrent BRVO, admitted on July 14, 2004 to the clinical ward of the ophthalmology department of the Military Medical Academy in Sofia, Bulgaria. We want to point out the specific clinical picture, the prolonged course of the disease and the ways of treatment. Careful and precise diagnosis in such cases and their immediate hospitalization are important prerequisites for the final therapeutic success.

Case

We present a clinical case of a 34 year old man with BRVO, accepted and treated in the Military Medical Academy in Sofia. He underwent a complete ophthalmologic check up including – visual acuity, perimetry, direct and indirect funduscopy. He has also been examined with fluorescein angiography (FA), digital fundus photography, and serological, immunological assessment for HLA and haemostatic examination.

We report a case of papillophlebitis in a young male, who was accepted on July 14, 2004 to the Ophthalmology Department of the Military Medical Academy in Sofia with complaints of blurred vision and distortion of the objects in front of the right eye. The history revealed that the complaints started a couple of days ago. At the primary visit, visual acuity of the right eye was 20/70 and 20/20 on the left eye. On the funduscoppy, numerous feather-like hemorrhages along the inferior branch of the central vein were found. Single hard exudates and pathological tortuosity of the affected blood vessel has been depicted. Slight swelling at the borders of the papilla was also present and we considered that to be a sign of initial edema of the optic nerve. This picture totally corresponded to

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the typical findings of a BRVO of the inferior branch of the central retinal vein. The fluorescein angiographic pictures, which we performed, confirmed our diagnosis. In the arterial-venous phases slower and irregular filling with dye of the inferior temporal branch has been found. The typical symptom known as “parietal glow” - staining with dye firstly of the vessel walls and later on filling of the vessel lumen with the fluorescent was also present (Figure 1).

Figure 1. FA picture of the 34 year old patient at his first admission to the ward. The diffusion of fluorescein in the inferior temporal branch of the central retinal vein is slow and irregular. Lots of hemorrhages are found along the damaged vessel.

The penetration of the stain into the affected vessel was very difficult and extremely slow. Its filling even in the latest phases of the FA had been still irregular and incomplete. On the FA pictures a mild hemorrhagic syndrome was presented. We observed dark zones corresponding to retinal hemorrhages along side the affected vessel. The increased diffusion of the dye through the walls of the damaged vessel in the late stages of the fluorescein angiography has been considered as a sign of increased permeability of the vessel walls. The borders of the optic nerve were indistinct and there was a profound leakage of fluorescein around the papilla, while in the nasal part oval shaped, enlarged vessels were found, possibly representing newly forming optico-cilliar shunts.

The patient underwent thorough laboratory check up. We have been trying to find any possible inflammatory cause, which could have provoked the BRVO. The cardio-vascular system was also examined. Results from the laboratory tests however showed no signs of inflammation what so ever. The hematological tests were as follows: Hemoglobin-150g/l (Normal reference range (NRR) 135-180 g/l); Leukocyte-8.7 g/l (NRR 3.5-10.5. 10/9 g/l); Thrombocyte-338g/l (NRR 130-360 10/9 g/l); Glucose-5.6 (NRR 3.5-6.0 g/dl); Cholesterol-4.76 mmol/l (NRR 3.0-5.7 mmol/l); Creatinine-103 umol/l (NRR 30-140 umol/l); Bilirubin-12 mmol/l (NRR-3.4-21 mmol/l); Urea-5.5 mmol/l (NRR 1.7-8.3 mmol/l); Potassium-4.8 mmol/l (NRR 3.5-6.0 mmol/l); Sodium-145 mmol/l (NRR 135-150 mmol/l); Calcium-2.5mmol/l (NRR 2.1-2.8 mmol/l); AST-26 u/l (NRR 5-40 u/l); ALT-36 u/l (NRR 5-40 u/l). The hemostasis was in normal ranges. All tests for rare infections - AST, Wasserman, Right-Hundelson, Waale-Rose, tests for Adenoviruses, Herpes viruses, Hepatitis A and B, toxoplasmosis and Chlamydia were negative.

Contrary to the initial expectations, no infectious agents were found. In the cardiologic examination we found out that our patient had been suffering from high blood pressure, which required serious treatment. He has been treated in the Department of Ophthalmology for two weeks with i.m. Etamsylate (Dicynone), i.v Pentoxifylline (Trental) and later on with oral Pentoxifylline (Agapurin Retard), Troxerutin (Troxevasin), Dipyridamole (Antistenocardin), Endotelon (Endotelon) and Neurobex (Neurobex Forte) pills. After 14 days, he was discharged with 20/40 vision and given an appropriate supportive treatment at home.

After 45 days on August 24, 2005 we diagnosed a second thrombosis of the same branch, which has followed the first one and has developed proximally from the site of the first occlusion. He was accepted immediately to the ward. The visual acuity of the right eye has dropped drastically to 20/100 in comparison to the first incident. In the fundus we observed a severe hemorrhagic syndrome with multiple, different in shape and size, feather - like hemorrhages, scattered in the whole posterior pole (Figure 2).

Figure 2. Digital fundus photography of the same patient after the second BRVO. In the posterior pole swelling of the papilla and lots of hemorrhages are clearly seen.
Some of them has even progressed to the vitreous cavity and has been found over the optic nerve going up into the vitreous cavity. The optic disc was still with blurred, indistinct boarders and with multiple hemorrhages around it. On the fluorescein angiography the edema of the optic nerve has been clearly diagnosed as well as the multiple hemorrhages some of which has been prominating into the vitreous cavity (Figure 3).

![Figure 3](image1.png)

Figure 3. On the FA the papillo-edema and the hemorrhagic syndrome are clearly distinguished. One of the hemorrhages goes into the vitreous cavity.

This time the leading symptom in the clinical findings was the hemorrhage. There was also a massive edema in the posterior pole. The severe fundus picture was in contrast to the relatively good visual acuity of the patient. In some of the FA pictures dark zones of probable retinal ischemia has been depicted. Apart from the ophthalmologic examination additional hematological testing has been performed. The patient was again consulted to a cardiologist and we added Clopidogrel (Plavix) and Enoxaparin Sodium (Clexane) to the previous therapy. We continued the treatment with i.m Etamsylate (Dicynone), i.v. Pentoxifylline (Trental), per os Endotelon (Endotelon) and Betamethason (Diprophos) to reduce the oedema syndrome in the posterior pole. Patient’s vision improved due to the immediate and adequate treatment and the final visual acuity at the time he was discharged has been 20/40. On the funduscopy the hemorrhages have been fully resorbed, the papillo-edema has been minimized and formation of initial effective shunts has been detected. The FA confirmed the total resorption of the hemorrhages. Free diffusion of the dye from the optic nerve has been still detected, but only in the late phases of the fluorescein angiography. Effective optico-ciliar shunts have been documented on the nasal side of the papilla (Figure 4).

![Figure 4](image2.png)

Figure 4. FA of the same patient after treatment. The hemorrhages have been resolved; the swelling of the papilla is diminished. We observe formation of effective optico-ciliar shunts.

Only in the macula region a mild edema has remained, keeping the danger of development of micro and macro cystoid degeneration of the macula. The patient is still followed up with no further changes of the ophthalmological status to the current moment.

**Discussion**

The reported clinical case shows that BRVO in young people is one peculiar form of thrombosis, characterized by a specific clinical picture and way of development [1,2]. Although recent studies [3] have shown a comparatively higher incidence of the disease in the Western European countries, as a rule the condition is a rare finding. According to some studies [6,11] it is due to a spasm of the vessel wall, which can further lead to the occlusion. This hypothesis is useful in explaining the possibility of recurrences of the thrombotic occlusion in the same vessel, as well as to justify the relatively quick recovery of the visual acuity after that.

However according to other investigators [7,8] thrombi formation in the retinal veins is due to congenital anomalies in the blood vessel walls, occurring at the level of lamina cribrosa. This explains the more frequent onset of BRVO among members of certain families. The generally accepted hypothesis for the etiology of BRVO in young, which we also support [3,4] is the inflammatory cause. In many cases the thrombotic occlusion precedes the clinical picture of vasculitis, which makes the task of finding the etiological cause much more difficult. Arterial hypertension in young people is also considered as a predisposing factor for vein occlusions as well as diabetes mellitus [5,12].

No matter what the etiological cause is, we can conclude that the clinical picture of BRVO in
young people is always filled with certain peculiarities. One of the most common finding in such cases is the dissociation between the ophthalmoscopic picture and the relatively good visual acuity. In our case the patient’s VA was 20/100 at his worst, while in the posterior pole we had a very serious hemorrhagic and edematous syndrome. Furthermore the visualized findings in the posterior pole very much resembled trunk vein occlusion in elderly patients. The edematous syndrome of the papilla and around was much more expressed than in typical cases. The hemorrhagic findings have been concentrated predominantly in the papillo-macular region, which is also different from the occlusion in the elderly patients. According to the majority of the authors [8,9] BRVO in young people usually have a beneficial prognosis and resolve without causing much damage to the visual acuity. In our report we would like to point out that there are atypical cases with different clinical picture and outcome. We have presented a case of occlusion with a progressive, recurrent course and a tendency toward chronic macula edema, or ischemic outcome, both leading to a significant impairment of vision. Detailed knowledge of such atypical cases, their appropriate hospital treatment and their regular fluorescein angiographic control are the most important prerequisites for successful therapy and good final recovery after this sight-threatening incident.

References